

Relationship of Preoperative Closing Volume to Functional Residual Capacity and Alveolar-Arterial Oxygen Difference during Anesthesia with Controlled Ventilation

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Patients undergoing peripheral surgical procedures were studied before and after induction of halothane anesthesia to determine the interrelationship of functional residual capacity (FRC), closing volume (CV) and alveolar-arterial oxygen difference (A-aD_o). Measurements of FRC, CV, and A-aD_o (F₁O₂ = 1.0) were made preoperatively. During anesthesia all patients were ventilated at normal P_aCO₂, with tidal volumes (V_T) of 5 and 10 ml/kg, the order determined randomly. FRC and A-aD_o were measured 30 and 60 minutes after institution of each tidal volume. FRC during anesthesia was unchanged from preoperative values at either tidal volume. Examining the relationship between preoperative CV and inspiratory lung volume (FRC + V_T) at the two tidal volumes administered revealed that when FRC exceeded CV, A-aD_o did not change with anesthesia and was not affected by tidal volume. When CV exceeded inspiratory lung volume, A-aD_o increased significantly with anesthesia. During anesthesia, changing from a small to a large tidal volume decreased A-aD_o when this increased inspiratory lung volume from below to above CV. (Key words: Lung, functional residual capacity; Lung, closing volume; Ventilation, mechanical; Anesthetics, volatile: halothane.)

AN INCREASE in pulmonary venous admixture relative to preoperative values has been demonstrated in anesthetized man.^{1,2} A proposed mechanism for this increased pul-

monary shunting is that anesthesia is associated with a reduction in functional residual capacity which in certain patients results in closure of dependent airways. This results in either equilibration of alveolar gas with venous blood or absorption of gas behind these closed airways, atelectasis, and consequently an increased pulmonary venous admixture. In support of this proposed mechanism, Don *et al.*³ have demonstrated that anesthesia with spontaneous ventilation increases the volume of trapped gas in patients in whom intraoperative functional residual capacity (FRC) is reduced to a volume less than the lung closing volume measured preoperatively. This interrelationship has not been examined previously during controlled ventilation.

The objectives of this study were to examine in anesthetized man during controlled ventilation: 1) The relationship of preoperative closing volume (CV), FRC, and alveolar-arterial oxygen tension difference (A-aD_o) to intraoperative FRC and A-aD_o. 2) The effect of ventilation with small (5 ml/kg) and large (10 ml/kg) tidal volumes (V_T) on these relationships. 3) The relationship of body stature to FRC and the relationship of age to A-aD_o.

Methods

Thirty-three patients scheduled for peripheral or lower abdominal surgical procedures were studied before and during surgery in the supine position. The day before operation, FRC, A-aD_o, and CV (24 of the patients) were measured. Patients were premedicated with atropine (0.4–0.6 mg) and morphine sulfate (5–10 mg). Anesthesia was induced with thiopental (2–3 mg/kg), suc-

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Received from the Department of Anesthesia, University of California, San Francisco, and the San Francisco Veterans Administration, San Francisco, California 94121. Accepted for publication December 18, 1973. Supported in part by SCOR Grant HL-14201.

TABLE 1. Ages and Body Statures in Groups I-III
(Means \pm SE)

	Age (Years)	Body Stature Height/Weight (cm/kg)
Group I (n = 8)	36.8 ± 6.6	2.49 ± 0.12
Group II (n = 8)	47.9 ± 5.2	2.41 ± 0.15
Group III (n = 8)	56.8* ± 1.8	2.37 ± 0.13

* Significantly different from Group I, $P < 0.05$.

cincholine was administered (60–100 mg) and endotracheal intubation was accomplished. Following intubation, the lungs were inflated manually for two consecutive breaths, and an end-inspiratory airway pressure of 30–40 cm H₂O was maintained for 30 seconds. Ventilation was then controlled and patients were not allowed to breathe spontaneously. Anesthesia was maintained with oxygen (flow rate 5 l/min) and halothane (0.75–1.0 vol per cent) administered by a standard circle system. *d*-Tubocurarine was administered in a dosage sufficient to abolish spontaneous ventilatory effort.

By random selection, half of the patients were first ventilated at a tidal volume of 5 ml/kg for one hour and then at 10 ml/kg for one hour. This sequence was reversed for the other patients. Respiratory rate was adjusted to maintain P_{aCO_2} at 40 torr (mean P_{aCO_2} 40 ± 1 SD). FRC and A-a D_{O_2} were measured in duplicate 30 and 60 minutes after institution of the two tidal volumes. FRC was measured using a closed-system helium-dilution method employing a catharometer and spirometer. Preoperative duplicate measurements were made with the patients breathing room air with oxygen added to the closed system to keep the volume constant. The mean difference between the high and low duplicate values of FRC was 161 ml ± 19 (SE). Intraoperatively the same system was used, filled with oxygen-helium and connected by a three-way valve so that the patient's airway could be connected either to the anesthetic circle system or to the spirometer. The spirometer was modified for measurement intraoperatively by the insertion of a self-inflating reservoir bag and unidirectional valving system.⁴ During the period of helium dilution, tidal volume and

respiratory frequency were kept identical to those of the ventilation pattern being studied. Halothane concentrations were measured by an infrared analyzer, and a correction factor was applied for the effect of halothane on the catharometer.⁵ The mean difference between the high and low duplicate values intraoperatively was 224 ml ± 28 (SE).

Closing volume was measured using the single-breath nitrogen washout curve described by Anthonisen *et al.*⁶ Each patient had at least two measurements of closing volume. The mean difference between the high and low duplicate values was 75 ml ± 21 (SE).

All volumes measured were corrected to body temperature and pressure, saturated.

Preoperatively, arterial blood was drawn after the patient had breathed pure oxygen through a mouthpiece connected to a one-way valve until end-expired nitrogen was less than 1 per cent. Intraoperatively, arterial blood samples were collected immediately before each FRC measurement. Samples were drawn in heparinized glass syringes, placed in ice, and analyzed for P_{O_2} , P_{CO_2} , and pH within three hours of sampling. Samples were corrected for changes produced by time and temperature.⁷ A blood-gas factor for the oxygen electrode was obtained daily, using a tonometer and a 30 per cent mixture of glycerol in water.⁸ PA_{O_2} was calculated from the following relationship: $PA_{O_2} = PI_{O_2} - Pa_{CO_2} - P_{\text{halothane}}$. Statistical analysis of the data was done by analysis of variance and linear regression.

Results

Patients were divided into groups according to the relationship of preoperative CV to intraoperative FRC plus V_T . $FRC + V_T$ is equal to inspiratory lung volume (ILV). Three groups with eight patients in each were apparent: Group I, $CV < ILV$ at either V_T ; Group II, $CV > ILV$ at V_T , but $< ILV$ at $V_{T_{10}}$; Group III, $CV > ILV$ at V_T or $V_{T_{10}}$. Table 1 lists mean ages and body statures of the patients in the three groups.

A-a D_{O_2} was smallest preoperatively and did not significantly increase with anesthesia at either tidal volume in Group I. A-a D_{O_2} in Group II increased significantly ($P < .001$) with anesthesia. In Group II, when the larger V_T was administered ($CV < FRC + V_{T_{10}}$), A-a D_{O_2} was significantly less ($P < .05$) than

TABLE 2. Relationship of FRC, CV, Tidal Volume, and A-aD_{O₂} (Means ± SE)

	Awake				Anesthetized					
					V _T 5 ml/kg			V _T 10 ml/kg		
	FRC (ml)	CV (ml)	ILV*-CV (ml)	A-aD _{O₂}	FRC (ml)	ILV*-CV (ml)	A-aD _{O₂}	FRC (ml)	ILV*-CV (ml)	A-aD _{O₂}
Group I	2,884 ±169	2,608 ±394	703 ±209	107 ±15	3,224 ±459	975 ±208	146 ±30	3,099 ±378	1,209 ±68	142 ±26
Group II	2,400 ±138	2,842 ±108	122 ±167	124 ±10	2,216 ±171	-251 ±117	239† ±25	2,427 ±171	334 ±125	188† ±20
Group III	2,775 ±261	4,528† ±537	-1,165† ±457	176† ±20	2,679 ±323	-1,475 ±358	241† ±31	2,661 ±340	-1,117 ±341	276† ±42

* Inspiratory lung volume, equal to FRC + V_T.

† Significant difference from awake values in Groups I and II.

‡ Significant difference from awake values in own group.

§ Significant difference from value for 5 ml/kg in own group.

A-aD_{O₂} at the smaller V_T (CV > FRC + V_T). In Group III, CV exceeded FRC + V_T at either tidal volume. A-aD_{O₂} was largest in this group preoperatively, increased with anesthesia, but was not significantly different at either tidal volume. Table 2 shows the data obtained for each group and also presents the difference between ILV (FRC + V_T) and CV to illustrate the correlation of these variables with A-aD_{O₂}.

Comparison of FRC's measured preoperatively and during anesthesia showed no significant difference at either tidal volume. In the 33 patients studied awake, FRC was 2,599 ml ± 139 (SE). FRC's measured intraoperatively were 2,566 ml ± 165 (SE) at V_T, 5 ml/kg and 2,536 ml ± 159(SE) at V_T, 10 ml/kg.

FRC's measured in awake patients and standardized to body weight correlated inversely with an index of obesity (ht/wt), $r = 0.49$; $P < 0.005$. The regression equation for this correlation is $y = 6.92x - 1.81$, where $y = \text{FRC(ml)}/\text{height(cm)}$ and $x = \text{height (cm)}/\text{weight (kg)}$. Age did not correlate with A-aD_{O₂} measured preoperatively ($F_{10} = 1.0$). Intraoperatively, A-aD_{O₂} correlated directly with age when patients were ventilated at tidal volumes of both 5 and 10 ml/kg. Correlation coefficients are V_T, 5 ml/kg, $r = 0.42$; $P < 0.025$; V_T, 10 ml/kg, $r = 0.48$; $P < 0.005$.

Discussion

In a previous report of the effect of tidal volume on A-aD_{O₂} during controlled ventilation, large tidal volumes were shown to

provide the optimum pulmonary gas exchange.⁹ In this study we found that the magnitude of A-aD_{O₂} can be related to the relationship between preoperative CV and inspiratory lung volume (FRC + V_T). In patients in whom inspiratory volumes always exceeded CV, A-aD_{O₂} remained unchanged when measured before and for two hours following induction of anesthesia (table 2, Group I). Of greater interest were the findings in patients classified in Group II (table 2). These were patients whose inspiratory lung volumes exceeded CV at tidal volumes of 10 ml/kg but not at 5 ml/kg. In these patients the higher tidal volume decreased A-aD_{O₂}. Thus, this study supports the postulate of Don *et al.* that the FRC-CV difference is an important determinant of pulmonary oxygen exchange during anesthesia.³ By studying anesthetized patients breathing spontaneously, these authors supported their postulate by measuring volume of trapped gas (VTG) and relating VTG to the relative magnitude of FRC and CV. This present study supports this thesis by measurement of a more direct index of pulmonary oxygen exchange, A-aD_{O₂}. However, if the relationship of CV to FRC is a major factor in oxygen exchange, why should anesthesia and controlled ventilation cause an increase in A-aD_{O₂} unless there is a change in FRC or closing volume? (Group III, Table 2.) One explanation is that a reduction in mixed venous oxygen concentration initiated by anesthesia and controlled ventilation would cause an increase in A-aD_{O₂}, even though the pulmonary shunt fraction remained constant.¹⁰

TABLE 3. Effects of Anesthesia with Controlled Ventilation on FRC

Author	Number of Subjects	Anesthesia		Surgical Procedure	When Measured	Effect on FRC
		Induction	Maintenance			
Laws ⁴	12	Thiopental Succinylcholine	Thiopental Succinylcholine	None	Immediately following intubation	10 per cent decrease from preoperative
Rehder <i>et al.</i> ¹⁵	12	Thiopental Succinylcholine	Thiopental Succinylcholine Meperidine	None	One hour post-induction	14 per cent decrease from preoperative
Westbrook <i>et al.</i> ¹⁴	5	Thiopental Succinylcholine	Thiopental Meperidine Hydrochloride	None	Throughout study	22 per cent decrease from preoperative
Wyche <i>et al.</i> ¹⁶	24	Thiopental Succinylcholine	N ₂ O/O ₂ Narcotic d-tubocurarine	Lower and upper abdominal	Within one hour of opening of abdomen	34 per cent decrease from tables of predicted normal
Weenig	33	Thiopental Succinylcholine	Halothane/O ₂ d-tubocurarine	Peripheral	30–60–90 and 120 minutes following induction	No change from preoperative

A decrease in mixed venous oxygen content could explain the increase in A-aD_{O₂} in this study, but fails to explain the decrease in A-aD_{O₂} found in Group II (table 2) when V_T was increased so that inspiratory lung volume exceeded the closing volume measured preoperatively.

A second explanation for these results is provided by West *et al.*¹⁴ These investigators have described different zones of perfusion of the lung which are dependent upon the relationship between pulmonary arterial, pulmonary venous, and alveolar pressure. When alveolar pressure is greater than pulmonary arterial pressure, pulmonary blood flow ceases. This relationship exists in the uppermost region of the lung. Patients in Group III (CV > FRC prior to anesthesia) had increased A-aD_{O₂}'s. Anesthesia with positive-pressure ventilation would raise alveolar pressure, thus increasing the relative size of West's Zone 1, and would direct a greater proportion of pulmonary blood flow to nonventilated alveoli in the dependent lung.

We have made comparisons between intraoperative FRC's and preoperative CV's. Present methods used for the measurement of closing volume preclude its estimation during anesthesia. The assumption that CV does not change with anesthesia and muscle paralysis may be in error, and investigation of the effect of anesthesia and muscle paralysis on CV is indicated.

It is not clear why FRC should change during anesthesia with paralysis and controlled ventilation. Controlled ventilation would be expected to reduce pulmonary blood volume, and paralysis would reduce inspiratory muscle tone if such tone is present at FRC. These effects, if they occur, would oppose each other. However, the relative magnitude and the occurrence of these effects have not been documented. FRC did not decrease in our study. Other studies of patients who were anesthetized and paralyzed, with ventilation controlled, have shown that FRC decreases (table 3). A difference in techniques of anesthesia or in the patient population may explain these findings. Laws *et al.* found a 12 per cent reduction in FRC after administration of thiopental, succinylcholine, and intubation.⁴ For this reason the design of our study included two large lung inflations (40 cm H₂O for 30–40 seconds) following intubation. Because of the well-documented reduction in FRC with anesthesia and spontaneous ventilation,^{12–14} patients were not allowed to breathe spontaneously following induction. Other investigators listed in table 3 did not administer large inflations and did allow spontaneous breathing before beginning controlled ventilation. If atelectasis resulted, subsequent end-inspiratory pressures may not have exceeded opening pressure. As pointed out by Wyche *et al.*,¹⁶ upper abdominal operations, with attendant retractors and packs, may significantly reduce FRC.

Of further interest in this study was the effect on FRC when a patient coughed or strained during anesthesia. This occurred in four patients. Anesthesia depth was increased and relaxants were administered to abolish expiratory straining.

No attempt was made to re-expand the lungs. Subsequent measurement of FRC revealed a mean reduction to 69.5 per cent of the preoperative value (range 63–76 per cent). Data from these patients were not included in our study.

Previous work indicates that during anesthesia with spontaneous ventilation, the reduction in FRC can be correlated with an index of body stature and is greater in relatively obese patients.¹² In this report an index of body stature (ht/wt) did correlate directly with FRC measured in supine, awake patients. This significant but relatively poor correlation ($r = 0.49$) compares favorably with other larger series which report that normal values for FRC have correlated best with body height. Correlation coefficients have ranged from 0.32 to 0.49 in these reports.¹⁷

Finally, although FRC was not reduced with anesthesia and controlled ventilation in this study, our work examined only a short duration of time under carefully controlled conditions. FRC might be reduced in other patients by many events that occur with anesthesia and surgery, such as apnea, endotracheal intubation, hypoventilation, rapid absorption of soluble anesthetic gases, expiratory straining, abdominal retractors, and in the postoperative patient, abdominal pain and use of opiates to relieve this pain. If this is the case, then placing the patient in a more upright posture, as Don *et al.* have suggested, might improve pulmonary gas exchange.³ If so, the change in FRC that results from moving from the supine to the upright posture, and the effects of body stature and surgical incision on this change, are important considerations.

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