

# Thoracic Gas Volume Measured by Body Plethysmography during Anesthesia and Muscle Paralysis:

## Description and Validation of a Method

Göran Hedenstierna, M.D.,\* Per-Olof Järnberg, M.D.,† Ingeborg Gottlieb, M.Sc.‡

A method based on body plethysmography for the assessment of thoracic gas volume (TGV) in the anesthetized, paralyzed subject is presented. The compression of thoracic gas following inflation is detected by measuring the difference between the inflation volume and the "box volume" change caused by the expansion of the chest. Model experiments showed good agreement between true and measured volumes with a residual standard deviation of 2 per cent. In studies on human subjects with healthy lungs during halothane anesthesia, the coefficient of variation of repeated measurements was 5 per cent. Comparative measurements with resting lung volume (FRC) determined by multiple breath nitrogen washout disclosed a larger volume by the box technique, a difference which was reduced but not eliminated by deep breathing during the nitrogen washout. This difference, amounting to 0.2 liters on the average, may be explained by abdominal gas and by the detection of trapped gas by the box technique but not by the nitrogen washout. (Key words: Anesthesia. Lung: functional residual capacity; thoracic volume. Measurement techniques: plethysmography.)

MUCH INTEREST has been devoted to the determination of resting lung volume during anesthesia. Its exact determination may guide in the understanding of impaired gas exchange in the anesthetized subject.<sup>1-3</sup> The techniques used have been based almost exclusively on gas dilution principles, mainly on nitrogen washout. This technique has the drawback of not measuring any trapped gas behind occluded airways. Lung regions with very long time constants may also be excluded or inaccurately determined by gas dilution measurements. Body plethysmography, by the implication of Boyle's law, would be a superior technique under such circumstances. However, the presently used technique requires the patient's cooperation; he/she is expected to do panting maneuvers while the mouthpiece is occluded.<sup>4</sup> Accordingly, in a study on thoracic gas volume (TGV) by body plethysmography in anesthetized subjects, no direct TGV measurement was made in the paralyzed state.<sup>5</sup>

\* Assistant Professor of Clinical Physiology, Karolinska Institute.

† Assistant Professor of Anesthesiology, Karolinska Institute.

‡ Laboratory Engineer.

Received from the Departments of Clinical Physiology, Karolinska Institute at Huddinge Hospital, Huddinge, and Anesthesiology, Karolinska Hospital, Stockholm, Sweden. Accepted for publication April 21, 1981. Supported by grants from the Swedish Medical Research Council, no 4X-5315, Petrus and Augusta Hedlund Foundation, and the Karolinska Institute, Stockholm, Sweden.

Address reprint requests to Göran Hedenstierna: Department of Clinical Physiology, Huddinge Hospital, S-141 86 Huddinge, Sweden.

An elegant technique for assessing lung volume in apneic dogs by body plethysmography was described by Laver *et al.*<sup>6</sup> It was based on the administration of a small insufflation volume, and the subsequent pressurizing of the plethysmograph in order to restore trans-thoracic pressure to zero. The pressure measured in the occluded trachea and the initially administered volume were used for the calculation of lung volume according to Boyle's law. High pressures were required which put demands on the plethysmograph and made the technique less suitable for human studies.

This paper presents a modification of the technique of measuring TGV by body plethysmography so that it becomes suitable for measurements in the noncooperative, paralyzed human subject. The method is based on compression of thoracic gas following inflation, and the subsequent smaller expansion of the chest than would be expected from the insufflated volume. By assuming interdependence between lung units,<sup>7</sup> this method may also measure trapped gas and units with long time constants.

## Materials and Methods

### THEORY

When the lungs are inflated with a certain volume, the expansion of the chest will not correspond exactly to the insufflated volume because of gas compression within the chest. The volume deficit depends on the pressure increase and the volume of gas within the lung. The following equation gives the relationship:

$$\frac{(V_0 + \Delta V)P_0}{P_0 + \Delta P} = V_0 + \Delta V - V_{df} \quad (\text{equation 1})$$

where  $V_0$  is initial lung volume;  $\Delta V$  is insufflated volume;  $P_0$  is initial alveolar pressure, *i.e.*, barometric minus water vapor pressure;  $\Delta P$  is increase in alveolar pressure when  $\Delta V$  is insufflated volume; and  $V_{df}$  is volume deficit, *i.e.*, the difference between chest expansion and insufflated volume. Solving for  $V_0$

$$V_0 = \frac{\Delta V - V_{df} - \frac{P_0 \times \Delta P}{P_0 + \Delta P}}{\frac{P_0}{(P_0 + \Delta P)} - 1} \quad (\text{equation 2})$$

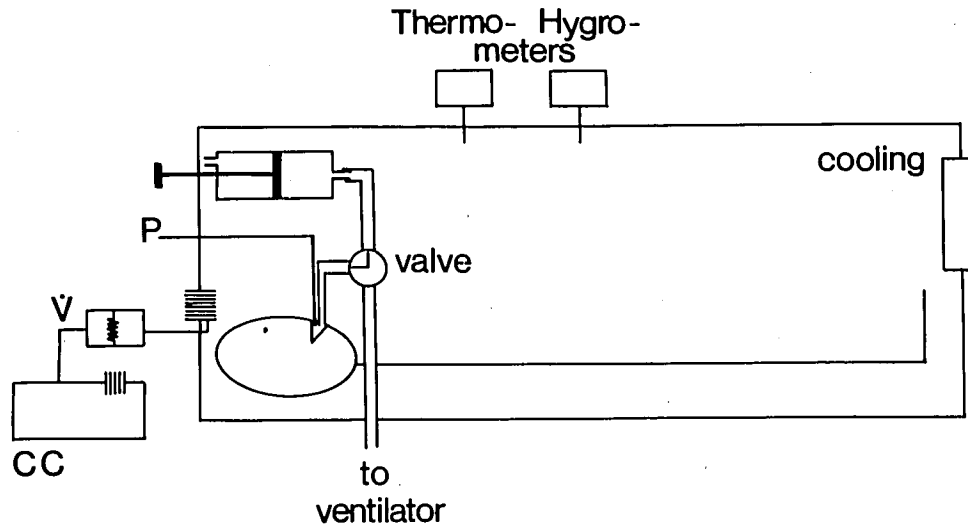


FIG. 1. The technical set-up. Note the location of the syringe within the box. The valve is electric but requires no energy (and causes no heat) except for switching. P: alveolar pressure;  $\dot{V}$ : gas flow into or out of the body plethysmograph; CC: compensation chamber. See text for further explanations.

The volume deficit can be sensed by means of body plethysmography. By substituting  $\Delta V - V_{df}$  by  $\Delta V_{box}$ , *i.e.*, the change in box volume (or box pressure),  $(P_0/P_0 + \Delta P)$  by P, and  $V_0$  by  $(TGV + V_{app})$  ( $V_{app}$  equals volume in tubings and syringe) equation 2 becomes:

$$TGV = \frac{V_{box} - P \cdot V}{P - 1} - V_{app} \quad (\text{equation 3})$$

The measurement requires the greatest precision. Thus, it can be shown that an error of 25 ml of the box volume reading may cause 25–30 per cent error in the TGV determination. An erroneous reading of the inflation volume ( $\Delta V$ ) has less influence, since it also causes an error in the box volume reading and these two errors almost cancel each other.

To improve the accuracy of the box volume reading, a volume equal to that used for inflating the lung can be taken from the body box. The meter which senses changes in box volume (or pressure) will then record the volume deficit ( $V_{df}$ ) and can be suitably calibrated for this small volume while any influence on box volume recording by the much larger  $\Delta V$  has been eliminated. In practice, this can be arranged according to figure 1 where a super syringe is used with the front end connected to the subject within the box and the back end connected to the body box. Modifying equation 3 accordingly:

$$TGV = \frac{V(1 - P) - V_{df}}{P - 1} - V_{app} \quad (\text{equation 4})$$

The determination of TGV according to the present technique is critically dependent on the humidity and temperature of the gas to be insufflated ( $\Delta V$ ). If this gas is of body temperature and saturated with water, then equation 4 can be used without any corrections and TGV will be expressed in BTPS. This can be ascertained by preheating and humidifying the gas in the syringe. How-

ever, by knowing the temperature and humidity of the gas within the syringe, a BTPS correction of  $\Delta V$  can be performed, the increase ( $\Delta V^1$ ) being:

$$\Delta V^1 = \Delta V \left( \frac{P_B - P_{H_2O_{BT}}}{P_B - P_{H_2O_{AT}}} \times \frac{273 + AT}{273 + BT} \right) - 1$$

where  $P_{H_2O_{AT}}$  = water vapor pressure at ambient temperature, AT = ambient temperature, and BT = body temperature.

$\Delta V^1$  causes an expulsion of gas out from the box (or a pressure increase) and thus opposite to the flow (or pressure) caused by the gas compression in the chest ( $V_{df}$ ). The net effect sensed by the meter of the box thus becomes

$$V_{net} = \Delta V^1 - V_{df} \quad (\text{fig. 2})$$

Rearranging  $V_{df} = \Delta V^1 - V_{net}$ , and inserting into equation 4:

$$TGV = \frac{\Delta V(1 - P) - (\Delta V^1 - V_{net})}{P - 1} - V_{app} \quad (\text{equation 5})$$

The withdrawal of gas from the box to the rear of the super syringe is a potential cause of changing the temperature of the gas. It is easily counteracted by placing the syringe within the box (see fig. 1).

#### EQUIPMENT

A specially designed integrated flow plethysmograph was constructed allowing measurements in supine subjects. The box was made of Lucite® and had the shape of a half cylinder. Its volume was 485 l. The whole body of the subject lay within the box. A stainless steel screen (mesh 450, diameter 76 mm) was mounted in an opening of the box wall and created a pressure drop of 1 mm H<sub>2</sub>O for a gas flow rate of 0.95 l/s. The pressure drop

was sensed by a differential pressure transducer (EMT 32®, Siemens-Elema) and was integrated to provide a volume signal. The atmospheric port of the transducer was connected to a compensation chamber with the same acoustical time constant as the body plethysmograph. The frequency response of the box was flat up to 12 Hz as tested with a 10-inch loud speaker. Airway opening (mouth) pressure was recorded with a pressure transducer (EMT 34®, Siemens-Elema). A remotely controlled shutter was connected to the mouthpiece. Pressure and volume were displayed on an X-Y recorder (Bryans 26000®). The box was cooled by thermoelectric (Peltier) heat pumps (LK-03®, Supercool). The cooling capacity corresponded to 80 watts.

A three-liter gas tight syringe, mounted within the box, was connected to the subject. The volume of the tubing between subject and syringe was 110 ml. The rear space of the syringe behind the plunger was open to the interior of the body box. The shaft, attached to the plunger and passing through the box wall to allow inflation of the lungs when the box is closed, caused small volume variations of the box during the inflation-deflation. This was corrected by subtracting 5 ml from  $\Delta V_{\text{box}}$  per liter  $\Delta V$ .

## MEASUREMENTS

### Lung Model Tests

The accuracy of the method was first tested in lung model experiments. The lung model consisted of one or two anesthesia rubber bags with a capacity of three liters each. The bags were pre-filled with different, known volumes of gas, but always so that the pressure within the bag was one or a few cm H<sub>2</sub>O above atmospheric. They were inflated with different gas volumes by the syringe. The inflated volume, the change in box volume and the increase in "alveolar" pressure were noted, and "TGV" was calculated according to equation 4.

### Human Tests

The method was also tested and compared to multiple breath nitrogen washout in anesthetized subjects who were to undergo elective surgery.

**Material.** The subjects were five men and three women, with a mean age of 42 years. Height and weight were normal for age. Subject data are shown in table 1. Clinical and radiologic examinations revealed no circulatory or pulmonary disease, although 3 of the subjects were moderate smokers (10–15 cigarettes/day). The scope of the investigation was explained to the subjects, and their informed consent obtained. The Ethical Committee of the Karolinska Institute had approved the investigation. There were no complications.

**Anesthesia.** After premedication with diazepam (0.15–

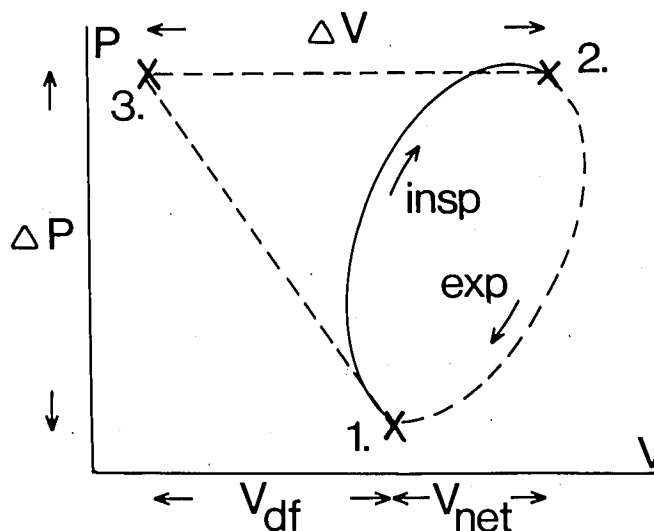


FIG. 2. Recording of airway pressure (P) and "box volume" (V) during manual inflation-deflation of the lungs. During inflation (from 1 to 2) P increases continuously, except for a small overshoot by the end of inflation, (caused by resistance in tubings and airways?).  $\Delta P$  indicates the change in alveolar pressure (static conditions). Simultaneously, V is first reduced, presumably indicating a short period of isothermal inflation, and then it is increased as a consequence of the heating and humidification of the inflated gas. The net volume change from 1 to 2 is  $V_{\text{net}}$ , while the calculated increase in volume of the inflated volume ( $\Delta V^1$ ) corresponds to the distance along the volume axis between 2 and 3.  $\Delta V^1$  is calculated from the inflated volume ( $\Delta V$ ), and its change in temperature and humidity. The volume deficit ( $V_{\text{df}}$ ) caused by gas compression equals ( $\Delta V^1 - V_{\text{net}}$ ). The dotted line from 1 to 3 shows the hypothetical change in alveolar pressure and  $V_{\text{df}}$  during isothermal inflation.

0.20 mg/kg) administered rectally and atropine (0.5 mg), administered intramuscularly approximately one hour previously, anesthesia was induced with thiopental (4 mg/kg). Pancuronium bromide (0.1 mg/kg) was then administered. Endotracheal intubation, using a widebore Portex® endotracheal tube, was performed after ventilating the lungs with oxygen. The subject was mechanically ventilated with an air-oxygen mixture. Anesthesia was maintained with halothane (1.0–1.5 per cent) and further doses of pancuronium bromide were given intravenously as required.

**Body plethysmography.** The box measurement was performed as described above. The inflation volume ( $\Delta V$ ) was two liters in all experiments. The gas in the super syringe had the same composition of air, O<sub>2</sub> and halothane as during the preceding mechanical ventilation so as to maintain an acceptable steady state of anesthetic gas exchange. TGV was determined 5 times within a 10 min-period to test the reproducibility of the method. Equation 5 was used for the calculation of TGV.

**Multiple breath nitrogen washout.** During oxygen breathing, the expirate was collected in a Douglas® bag, and its volume and nitrogen concentration was analyzed. The nitrogen concentration was measured by an ioniza-

TABLE 1. TGV by Body Plethysmography and by Multiple Breath Nitrogen Washout in Anesthetized Paralyzed Subjects\*

Subject Number	Sex	Age (yr)	Height (cm)	Weight (kg)	Body Plethysmography		Nitrogen Washout		TGV - FRC (l)	TGV - (FRC + Trapped Gas) (l)
					TGV (l)	Coefficient of Variation (Per cent)	FRC (l)	Trapped Gas (l)		
1	M	44	176	71	2.23	2	2.01	—	0.22	—
2	M	62	171	68	3.14	6	2.59	0.24	0.55	0.31
3	F	61	166	69	2.27	7	2.05	0.15	0.22	0.07
4	M	59	174	80	2.80	3	2.29	0.36	0.51	0.15
5	M	37	182	73	2.65	5	2.53	—	0.12	—
6	F	48	158	52	2.34	4	1.98	0.16	0.36	0.20
7	F	27	160	48	1.68	6	1.76	—	-0.08	—
8	F	35	171	57	2.08	5	2.10	—	-0.02	—
MEAN					2.40	5	2.16	(0.22)	0.24†	(0.18)†

\* Subject data are shown to the left. Body plethysmographic data are shown in the middle rows (TGV and coefficient of variation on five determinations of TGV). Nitrogen washout data are shown to the right: FRC and trapped gas, the latter obtained by collecting and an-

alyzing the expirate of three deep inflations after previous multiple breath nitrogen washout.

† Significant difference between TGV and FRC,  $P < 0.05$ .

tion meter (Nitralyzer 505®, Med. Science). By the end of the test, when nitrogen concentration was 2 per cent or less, the collection of expired gas was switched from the first bag to a second one. Three deep breaths (2-3 liters) were finally collected in the latter bag and the test was finished. Resting lung volume (FRC) was calculated according to:

$$\text{FRC} = \frac{V_E \times (\bar{F}_{E_{N_2}} - F_{e_{N_2}}) - V_D \times F_{i_{N_2}}}{F_{i_{N_2}} - F_{f_{N_2}}} \times 1.1$$

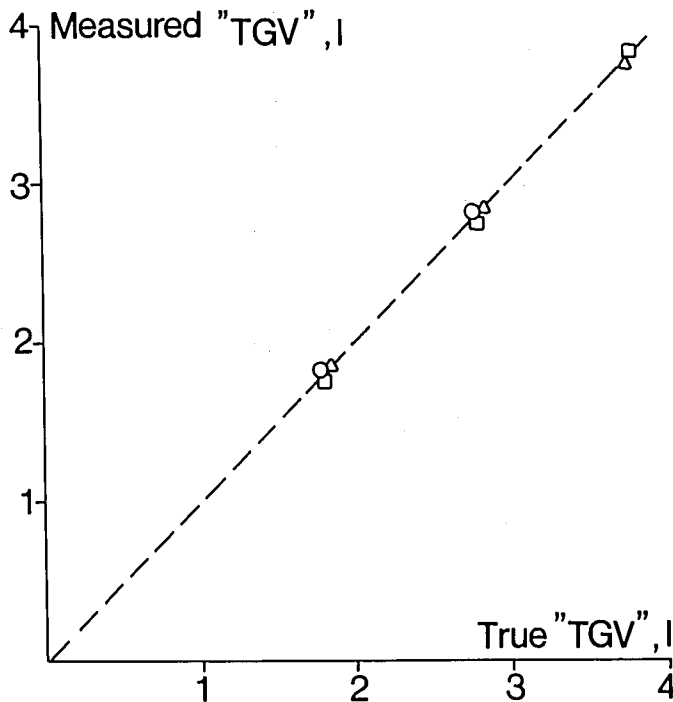


FIG. 3. "TGV" by body plethysmography in model tests. The lung model consisted of one or two anesthesia bags. Each point represents a mean of five determinations with an inflation volume of 0.1 (O), 0.25 (□) or 0.51 (Δ). The coefficient of variation for five consecutive determinations was on average 2 per cent.  $r: 0.99$  (linear regression analysis).

where  $V_E$  = the volume collected in the Douglas® bag;  $\bar{F}_{E_{N_2}}$  = mean expired  $N_2$  concentration;  $F_{e_{N_2}}$  =  $N_2$  impurities of inspired gas during oxygen breathing, set to 0.2 per cent, and  $N_2$  dissolved in body fluids and expired, set to 0.7 per cent, thus totalling 0.9 per cent;  $V_D$  = apparatus dead space, 190 ml;  $F_{i_{N_2}}$  = initial  $N_2$  concentration, before oxygen breathing; and  $F_{f_{N_2}}$  = final end-tidal  $N_2$  concentration, assumed to equal that in the alveoli. By adding the expired amount of nitrogen collected in the second bag, after subtraction for nitrogen impurities (0.2 per cent), and dissolution (set to 0.3 per cent) to the calculation of FRC, a larger than the initial FRC was obtained. This was considered to be due to the addition of trapped gas. Measurements were made in duplicate with a 10-min interval of air ventilation in between.

## Results

### LUNG MODEL TESTS

The results are shown in figure 3. The calculated "TGV" showed no systematic deviation from the true "TGV" for any inflation volume or true "TGV." The scatter was small and tended to be reduced further when inflation volume was increased and/or true "TGV" was small. With an inflation volume of 0.5 l and a true "TGV" of 1.8 l, the standard deviation was less than 0.05 l. The increased accuracy with a large inflation volume and small "TGV" follows from the greater change in "alveolar" pressure ( $\Delta P$ ) which has the effect of increasing the absolute value of the denominator in equation 4.

### HUMAN TESTS

Results from body plethysmography and multiple nitrogen washout are shown in table 1. It can be seen that the biological variability in TGV determined by plethysmography was no more than 5 per cent. FRC by ni-

trogen washout was smaller than TGV by body plethysmography ( $P < 0.05$ ). The difference was reduced but not eliminated ( $P < 0.05$ ) when trapped gas was taken into account by the additional three deep breaths of oxygen.

### Discussion

The body plethysmographic technique presented here for the determination of TGV has proved accurate in lung model tests and has also shown good reproducibility in measurements on anesthetized paralyzed humans with normal lungs. The higher TGV with body plethysmography than FRC with nitrogen washout, a difference that was reduced by large tidal volumes (equal to or larger than  $\Delta V$ ) during the washout, may be a sign of trapped gas when breathing at the resting level during anesthesia.<sup>8</sup> However, a difference of on average 0.18 l persisted even after deep breathing during the nitrogen washout. This difference may in part (but probably not in whole, as will be seen) be explained by the incorporation of abdominal gas into the measurement of TGV. The size of this contribution depends on the pressure abdominal gas is subjected to during the inflation with  $\Delta V$ , and the volume of the gas. From data by Grimby *et al.*<sup>9</sup> it can be calculated that gastric pressure increases by an average of 7 cm H<sub>2</sub>O when tracheal pressure is increased by 30 cm H<sub>2</sub>O in an anesthetized subject (the latter pressure being the mean increase when a  $\Delta V$  of 2 l was administered). While there appears to be no report on abdominal gas volume in the anesthetized subject, two studies in awake subjects indicate this volume to be on average 115 and 360 ml, respectively.<sup>10,11</sup> This should result in a contribution to the measured TGV by no more than 27 and 84 ml, respectively. These volumes are too small to affect the clinical implication of a measured TGV. It is also doubtful if abdominal gas is the single cause of the persisting difference between the box-TGV and the nitrogen washout-FRC; an abdominal gas volume of 780 ml would be required to completely account for the different results with the two techniques.

It may also be that lung units which still are closed by the end of a deep breath (if they exist), and lung units with long time constants, are included in the measurement of TGV by body plethysmography but not in the recording of FRC by nitrogen washout. The reason would be lung interdependence which creates forces to expand occluded or "slow" lung units.<sup>7</sup> Such units will be subjected to a lower pressure than surrounding units and so tend to reduce the net pressure increase in the lung ( $\Delta P$ ) for a given inflation volume ( $\Delta V$ ). The calculated TGV would thus be larger and closer to true lung volume than what would be the case in the absence of lung interdependence. Although reasonable to accept, it must be stressed that the influence of lung interdependence as described here remains a conjecture.

The exchange of anesthetic and other gases may complicate the plethysmographic determination of TGV. The anesthetic gas uptake can vary considerably not only between but also within subjects.<sup>12,13</sup> While a large uptake can be shown to have minor effects on the pressure-volume relationship during the administration of  $\Delta V$  (as shown in fig. 2; this is because  $\Delta V$  and  $\Delta V'$  both are subjected to errors which almost cancel each other) an unstable signal is created by the end of the inflation, and this will obscure the calculation of TGV. The use of the plethysmographic technique should therefore be restricted to anesthetic procedures which cause a moderate anesthetic gas exchange. The presently used inhalational anesthesia (which did not include nitrous oxide) caused a change in "box volume" after the inflation with  $\Delta V$  by on average 1 ml/s and imposed no problems in recording the pressure-volume signal on the X-Y recorder. The possibility remains to inflate the lungs with a gas that contains not only the anesthetic gas in expected alveolar concentration (as in present study) but also carbon dioxide, a procedure that further prevents a drifting signal.

### References

1. Bendixen HH, Hedley-Whyte J, Laver MB: Impaired oxygenation in surgical patients during general anesthesia with controlled ventilation. *New Engl J Med* 269:991-996, 1963
2. Nunn JF: Factors influencing the arterial oxygen tension during halothane anesthesia with spontaneous ventilation. *Br J Anaesth* 36:327-341, 1964
3. Marshall BE, Cohen PJ, Klingermaier CH, et al: Pulmonary venous admixture before, during and after halothane: oxygen anesthesia in man. *J Appl Physiol* 27:653-667, 1973
4. DuBois AB, Botelho SY, Bedell GN, et al: A rapid plethysmographic method for measuring thoracic gas volume: A comparison with a nitrogen washout method for measuring functional residual capacity in normal subjects. *J Clin Invest* 35:322-326, 1956
5. Westbrook PR, Stubbs SE, Sessler AD, et al: Effects of anesthesia and muscle paralysis on respiratory mechanics in normal man. *J Appl Physiol* 34:81-86, 1973
6. Laver MB, Morgan J, Bendixen HH, et al: Lung volume, compliance, and arterial oxygen tensions during controlled ventilation. *J Appl Physiol* 19:725-733, 1964
7. Mead J, Takishima T, Leith D: Stress distribution in lungs: a model of pulmonary elasticity. *J Appl Physiol* 28:596-608, 1970
8. Don HF, Wahba WM, Craig DB: Airway closure, gas trapping, and the functional residual capacity during anesthesia. *ANESTHESIOLOGY* 36:533-539, 1972
9. Grimby G, Hedenstierna G, Löfström B: Chest wall mechanics during artificial ventilation. *J Appl Physiol* 38:576-580, 1975
10. Bedell GN, Marshall R, DuBois AB, et al: Measurement of the volume of gas in the gastrointestinal tract. Values in normal subjects and ambulatory patients. *J Clin Invest* 35:336-345, 1956
11. Brown R, Hoppin FG Jr, Ingram RH Jr, et al: Influence of abdominal gas on the Boyle's law determination of thoracic gas volume. *J Appl Physiol* 44:469-473, 1978
12. Nunn JF, Bergman NA, Cole AJ: Factors affecting the arterial oxygen tension during anesthesia with artificial ventilation. *Br J Anaesth* 37:898-914, 1965
13. Ducek R, Young I, Calusen J, et al: Altered distribution of pulmonary ventilation and blood flow following induction of inhalational anesthesia. *ANESTHESIOLOGY* 52:113-125, 1980