

# The Effects of Anesthesia and 100 Per Cent Oxygen on the Functional Residual Capacity of the Lungs

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Functional residual capacities were measured with the helium-dilution technique in conscious subjects breathing air and then breathing 100 per cent oxygen. In seated subjects there was no change in FRC. In the supine position there was a small (3.3 per cent) decrease in FRC on breathing oxygen. FRC values of 11 patients were measured while they were awake and breathing room air and then during anesthesia while they breathed halothane in either 30 per cent or 100 per cent oxygen. There were consistent decreases in FRC following induction, not progressive with time and not related to the inspired mixture. The decreases in FRC correlated with the ratio of weight (kg) to height (cm), such that FRC after induction, expressed as percentage of control FRC, =  $137.7 - 164.4 \frac{\text{weight (kg)}}{\text{height (cm)}}$ . There was no evidence that the decreases in FRC were due to increased expiratory muscle tone. Spontaneous big breaths during anesthesia restored FRC only partly. A possible mechanism for the decrease in FRC during anesthesia in the supine position is airway closure at low lung volumes due to relative obesity and inadequacy of the sigh mechanism. (Key words: Functional residual capacity; Atelectasis; Oxygen; Sigh; Body weight; Airway closure.)

THE VOLUME of gas in the lung at the end of normal expiration (the functional residual capacity, FRC) is of considerable significance. Flow resistance increases with reduction of lung volume below FRC.<sup>1</sup> Studies of the distribution of ventilation in the lung suggest that at low lung volumes airways close and gas is trapped.<sup>2</sup> The proximity of the normal FRC to this closing volume, and the degree of change in FRC, determine the extent of interference with gas exchange. Continued perfu-

sion of those areas where gas is trapped will increase the physiologic shunt; atelectasis may or may not occur, perhaps depending on the type of gas trapped.<sup>3</sup> Increases in physiologic shunt following induction of anesthesia have been suggested.

It was our purpose to measure the changes in FRC following induction of anesthesia, during spontaneous ventilation with halothane in either 30 per cent oxygen in nitrogen or 100 per cent oxygen, and to follow any progression with time.

As a preliminary, we measured FRC in conscious volunteers breathing air and then oxygen, both sitting and supine.

## Method

### MEASUREMENT OF FUNCTIONAL RESIDUAL CAPACITY

FRC was measured with the closed-circuit helium technique,<sup>10</sup> using a catharometer (Cambridge Instrument Co., Ltd.) for measurement of helium. For subjects breathing air, the helium meter was calibrated for helium in air, the initial helium concentration being 13.6 per cent. For subjects breathing 100 per cent oxygen,<sup>11</sup> the spirometer and circuit were flushed with 100 per cent oxygen. Oxygen and helium were added to produce a helium concentration of 9.9 per cent. Known volumes of oxygen were then added and the reductions in helium concentration noted. In this way a graph showing the relationship of volume added to change in helium deflection was constructed. The same procedure was carried out in making measurements in the subjects; in this case the added volume was the FRC.

When subjects breathed 30 per cent oxygen in nitrogen, a similar calibration was carried out. The deadspace of the spirometer was filled with air; 1,000 ml air, 790 ml oxygen and 500 ml helium were then added. In this

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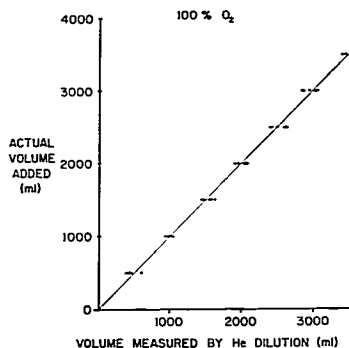


FIG. 1. A volume of oxygen is added to the circuit and the volume measured by the change in helium concentration plotted.

way a mixture of 8.76 per cent helium in approximately 30 per cent oxygen was made. Known volumes of 30 per cent oxygen in nitrogen were then added and the reductions in helium concentration noted. A graph relating helium concentration to added volume was constructed.

The reproducibility of this method is illustrated in figure 1 for 100 per cent oxygen and in figure 2 for 30 per cent oxygen, where the measured volumes fall within 10 per cent of actual volumes.

In addition, the helium meter was calibrated for halothane (Fluothane, Ayerst Laboratories). The spirometer was filled with oxygen. Different concentrations of halothane in oxygen were then added, and the decreases in helium deflection noted. Halothane and oxygen affect the helium meter independently.<sup>11</sup> The decreases in helium deflection were related to halothane concentrations in the spirometer. Halothane concentrations were measured with a Hook and Tucker analyzer, calibrated at zero and a single known concentration of halothane (Ohio Calibration standard 2.98 per cent). An appropriate correction could then be made in the anesthetized subjects by sampling the gas in the spirometer at the end of a measurement. The reproducibility of this method is illustrated in figure 3, where the measured volumes fall within 12 per cent of actual volumes.

In those patients breathing 30 per cent oxygen in nitrogen, a 50-ml sample of gas was withdrawn at the end of a measurement and the oxygen partial pressure measured by means of an Instrumentation Laboratory gas analysis system 113.

In all the measurements, oxygen was added to keep the end-expiratory line on the spirometer as near to horizontal as possible.

#### PROCEDURE

*Five conscious volunteers, seated.* FRC was measured at least twice while the subjects were breathing air. Subjects then breathed 100 per cent oxygen by means of a nonbreathing circuit for 30 to 70 minutes. FRC was measured at intervals, but not earlier than 15 minutes after the beginning of oxygen breathing.

*Five conscious volunteers, supine.* The procedure used for seated subjects was repeated with supine subjects.

*Anesthetized patients.* All patients were free of cardiac and respiratory disease, as shown by history and physical examination. Premedication consisted of atropine, 0.4 mg, plus meperidine (Demerol), 50 to 75 mg. Five patients also received pentobarbital (Nembutal), 100 mg. All patients were scheduled for surgical operations on the limbs. Approximately an hour after premedication,

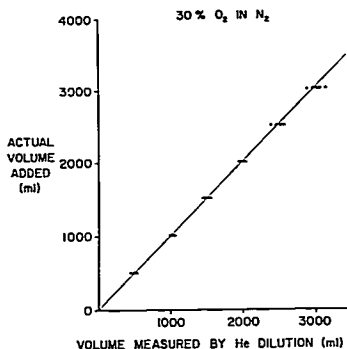


FIG. 2. A volume of 30 per cent oxygen in nitrogen is added to the circuit and the volume measured by the change in helium concentration plotted.

two or three measurements of FRC were made with the patient supine, breathing air.

Anesthesia was induced with 2.5 per cent sodium thiopental (sodium pentothal), 5 mg/kg, immediately followed by succinylcholine (Anectine), 1 mg/kg. The lungs were then ventilated with either 100 per cent oxygen or 30 per cent oxygen in nitrogen. Following topical application of 4 per cent lidocaine (Xylocaine), 3 ml, to the larynx, the trachea was intubated with a Magill rubber tube and the cuff inflated. Spontaneous respiration was established, and a nonbreathing system used. Ventilation was subsequently spontaneous during the period of study except for a period of hyperinflation prior to the last measurement in one subject. Depth of anesthesia was monitored only by the usual clinical guides of blood pressure, pulse, respiration and response to operation. An attempt was made to keep depth of anesthesia as light as possible. There were no episodes of significant hypotension or cardiac arrhythmias.

The patients were divided into two groups: six patients breathed halothane in oxygen, and five patients breathed halothane in 30 per cent oxygen in nitrogen. Four patients of the latter group were switched to halothane in oxygen after approximately 40 minutes of the 30 per cent oxygen mixture.

Measurements of FRC were made at intervals throughout the surgical procedures and are referred to in the text as postinduction measurements. No measurement of FRC was made within 15 minutes of starting a particular gas mixture.

#### POSITION OF APNEA

During the study it became apparent that it would be of interest to assess the position of the lungs and chest wall during apnea produced by induction of anesthesia with sodium thiopental. We therefore recorded, on a carefully balanced spirometer filled with oxygen, the respiratory patterns of four patients during quiet breathing. Sodium thiopental (approximately 5 mg/kg) was injected intravenously, and the position of apnea noted. It was also of interest to record the position of the respiratory system in the anesthetized patient during apnea produced by the intravenous injection

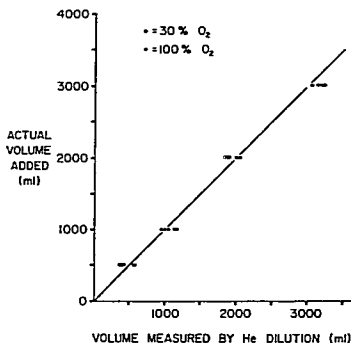


FIG. 3. A volume of halothane in either 30 or 100 per cent oxygen is added to the circuit calibrated appropriately. The volume is then measured by the change in helium concentration corrected for the halothane percentage in the circuit. The halothane percentages varied from 0.08 to 0.62, which was the approximate range found in anesthetized patients.

of succinylcholine. Following the protocol described under "Procedure," spontaneous respiration with oxygen and halothane was established. The endotracheal tube of the patient was then connected to a balanced spirometer, and ten to 15 breaths were recorded to establish a clear end-expiratory level. Succinylcholine (30–40 mg) was injected intravenously, and the position of apnea relative to the end-expiratory level noted.

#### Results

Results are shown in tables 1 to 5 and figure 4.

*Seated conscious volunteers* (table 1) showed no significant alterations in FRC when they changed from breathing air to breathing 100 per cent oxygen. In the supine position (table 2), there were decreases (mean 3.3 per cent) in FRC during breathing of oxygen compared with breathing of air, significant at the 5 per cent level.

*Anesthetized patients.* There were significant decreases in FRC after induction of anesthesia (tables 3 and 4). Two factors must be considered. First, inspection of the data shows that there was no progressive reduction in FRC during anesthesia. If the best-fit line

TABLE 1. Values of FRC in Conscious Seated Subjects

Subject	Age (Years)	Sex	Height (cm)	Weight (kg)	Breathing Room Air				Breathing 100 Per Cent Oxygen		
					FRC Time*	1910 -30	1925 -15	2005 -5	1875 +25	1935 +44	
1	28	F	170	71	FRC Time*	3204 -43	3229 -31	3119 -18	3204 -7	3229 +20	3194 +34
2	35	M	175	75	FRC Time*	2993 -87	2878 -75	2968 -47	2783 -12	2663 +22	2863 +34
3	29	M	170	68	FRC Time*	1790 -26	1800 -17	1780 -6		1735 +17	1710 +35
4	26	F	152	52	FRC Time*	2135 -40	2025 -20	2070 -5		2105 +14	2205 +30
5	25	F	162	54	FRC Time*					2050 +43	

\* Minutes before (-) or after (+) starting to breathe 100 per cent oxygen.

was made for the relationship of change in FRC after induction against time, for each patient (except patient 3 for whom only two postinduction measurements were made), the slopes of the lines were approximately equally positive or negative, and none were significant. This allowed us to compare mean postinduction values from any patient with mean pre-induction FRC. Second, it is evident that there were differences in the percentage decreases in FRC in different patients. Reductions in FRC were larger in patients with

greater body weights. When percentage decreases in FRC were plotted against the ratios of weight (kg) to height (cm) a linear relationship was found (fig. 4). The regression line is represented by the formula:

$$\frac{\text{FRC before anesthesia}}{\text{FRC during anesthesia}} \times 100 = 137.7 - 164.4 \frac{\text{weight (kg)}}{\text{height (cm)}}$$

The correlation coefficient was 0.9. Comparison of other data with this regression line is a

TABLE 2. Values of FRC in Conscious Supine Subjects

Subject	Age (Years)	Sex	Height (cm)	Weight (kg)	Breathing Room Air				Breathing 100 Per Cent Oxygen			
					FRC Time*	1530 -20	1515 -13	1595 -4	1485 +18	1430 +40		
1	28	F	170	71	FRC Time*	3495 -20	3490 -10		3390 +15	3160 +32		
2	35	M	175	75	FRC Time*	2051 -40	1961 -3		1885 +20	1996 +33		
3	29	M	170	68	FRC Time*	2800 -20	2785 -5		2560 +20	2580 +45	2600 +55	
4	28	M	185	88	FRC Time*	2850 -40	2900 -23	2770 -5	2840 +18	3010 +37	2860 +54	2940 +70
5	34	M	178	63	FRC Time*	1255 -45	1255 -15	1200 -5	1130 +29	1240 +40	1180 +50	1290 +60
6	33	M	175	79	FRC Time*							

\* Minutes before (-) or after (+) starting to breathe 100 per cent oxygen.

TABLE 3. Values of FRC in Patients Breathing Room Air While Awake, and Then Breathing Halothane in 100 Per Cent Oxygen during Anesthesia

Subject	Sex	Age (Years)	Height (cm)	Weight (kg)	Preinduction, Breathing Room Air (Mean)	Postinduction, Breathing 100 Per Cent Oxygen (Mean)
1	F	19	152	47	FRC 1340 1173 (1256) Per cent Time* -50 -35 (100)	990 1220 1110 1210 1120 (1130) (90) +20 +40 +60 +85 +120
2	M	43	180	81	FRC 2125 2225 (2175) Per cent Time* -30 -15 (100)	1488 1510 1430 1300 1400 (1426) (66) +15 +30 +45 +60 +90
3	F	55	167	61	FRC 2191 2231 (2211) Per cent Time* -35 -10 (100)	1790 1840 (1815) (86) +20 +45
4	M	50	180	86	FRC 2335 2216 (2276) Per cent Time* -25 -10 (100)	1380 1430 1150 1270 1210 (1288) (57) +20 +50 +65 +90 +120
5	M	56	177	106	FRC 2236 2416 (2326) Per cent Time* -25 -5 (100)	965 1220 1130 1010 770 (1019) (44) +25 +40 +65 +80 +85
6	M	47	170	81	FRC 3380 3380 (3380) Per cent Time* -20 -5 (100)	1470 1655 1780 1670 1620 (1639) (48) +25 +45 +57 +70 +85

TABLE 4. Values of FRC in Patients Breathing Room Air and then Breathing Halothane in 30 Per Cent Oxygen in Nitrogen

Subject	Sex	Age (Years)	Height (cm)	Weight (kg)	Preinduction, Breathing Room Air (Mean)	Postinduction, Breathing 30 Per Cent Oxygen (Mean)
7	F	54	164	70	FRC 2148 2024 (2086) Per Cent Time* -15 -5 (100)	1400 1260 1450 1530 1170 +29 +45 +65 +85 +105 1010 (1303) +120 (62)
8	F	41	162	61	FRC 1670 1630 (1650) Per Cent Time* -58 -40 (100)	1180 1050 1260 1190 1320† +17 +37 +42 +52 +94 1200† 1340† 1250† 1250† 1170† (1220) +112 +122 +132 +147 +163 (74)
9	F	65	152	73	FRC 1647 1537 1435 (1540) Per Cent Time* -25 -15 -8 (100)	880 900 1020 950† 980† (946) +20 +45 +55 +85 +100 (61)
10	F	18	172	63	FRC 1700 1725 (1712) Per Cent Time* -22 -12 (100)	1460 1440 1430 1360† 1410† +23 +38 +58 +78 +88 1390† (1415) +98 (83)
11	F	18	154	45	FRC 1994 2001 (1997) Per Cent Time* -28 -20 (100)	1720 1640 1650 1800† 1710† +15 +35 +52 +70 +90 1510† 1690† 1680† (1675) +110 +132 +150 (84)

\* Minutes before (-) or after (+) induction of anesthesia.

† Patient also breathed halothane in 100 per cent oxygen; these measurements are marked with daggers.

satisfactory method of evaluation, because two major variables, the height and weight of the patient, are controlled. If, therefore, the decreases in FRC in patients breathing halothane in 100 per cent oxygen differed from those in patients breathing halothane in 30 per cent oxygen, they should form a separate population on the regression line. Inspection of figure 4 shows that the results from these patients are scattered about the regression line, and therefore the concentration of oxygen does not affect the decrease in FRC. This was confirmed in those patients who breathed halothane in 30 per cent oxygen, then in 100 per cent oxygen, in whom no significant differences in FRC were found.

The decreases in FRC did not correlate well with the ages of the patients, although the three youngest patients (patients 1, 10, and 11) were among the lowest in ratio of weight to height. The presence of pentobarbital as additional premedication did not correlate with reduction in FRC. The presence or absence of surgical stimulation did not alter reductions in FRC in two patients in whom the first post-induction FRC values were measured before the surgical incision.

The positions of the lungs and chest wall during apnea produced by sodium thiopental in the four subjects showed an insignificant mean difference of 12 ml above the end-

expiratory positions (table 5). In four of the seven subjects given succinylcholine during surgical operation, the positions of rest of the respiratory system coincided with the end-expiratory levels (table 5); the mean for the seven patients was +14 ml.

## Discussion

Our results indicate that in the stated anesthetic circumstances there is a fall in the functional residual capacity of the lung. This finding is in agreement with the report by Laws,<sup>12</sup> who found a mean fall of 9.0 per cent during anesthesia with controlled ventilation on air. Colgan and Whang<sup>13</sup> did not find decreases in FRC in eight patients breathing halothane and oxygen. Their anesthetic protocol differed from ours in that they did not use barbiturates for induction, and they showed in dogs that the use of these drugs may be associated with decreases in FRC. In addition, the weights and heights of the patients in their study were not given.

If the concept that gas trapping will occur as FRC decreases, it would be expected that impairment of gas exchange will parallel the reduction in FRC. Panday and Nunn<sup>8</sup> suggested that the anesthetic state is associated with an increase in the AaDO<sub>2</sub> and that this increase does not vary with time. This would

TABLE 5. Effects of Intravenous Sodium Thiopental and Intravenous Succinylcholine on the End-expiratory Position of the Respiratory System during Surgical Operation

Subject	Sex	Age (Years)	Height (cm)	Weight (kg)	Thiopental		Succinylcholine	
					Dose (mg)	Apnea Point* (ml.)	Dose (mg)	Apnea Point* (ml.)
12	M	41	183	59	300	-50	30	-60
13	M	63	178	79	350	+180	40	0
7†	F	54	164	70	350	0		
14	F	35	170	61	300	-80	40	0
15	M	59	175	73			30	0
16	M	43	178	76			30	+80
11‡	F	18	154	45			30	+80
17	F	60	152	64			40	0
Mean					325	+12	34	+14

\* Volume at which the lungs are held above (+) or below (-) the end-expiratory level.

† Patient 7 in table 4.

‡ Patient 11 in table 4.

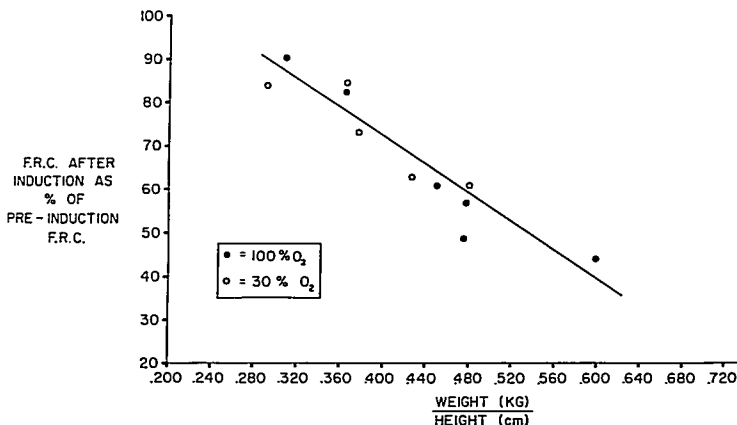


FIG. 4. Relationship of reduction in FRC during anesthesia to the ratio of height (kg) and weight (cm). The regression line is represented by the formula:

$$\frac{\text{FRC during anesthesia}}{\text{FRC before anesthesia}} \times 100 = 137.7 - 164.4 \frac{\text{weight (kg)}}{\text{height (cm)}}$$

fit with our finding of a reduction in FRC which was not progressive. However, it should be noted that their control comparisons were not in the same group of patients, and from our results it is not valid to compare different groups of patients unless account is taken of their physical characteristics. The variable reported effects of anesthesia on gas exchange<sup>5, 6, 7, 8, 13, 14</sup> might be explainable on the basis of the heights and weights of the patients.

It would be predicted from our results that obese patients will have marked reductions of FRC during anesthesia and will be subject to mismatching of ventilation to perfusion in the lung. Equally, a tall thin patient should have little upset of gas exchange. For any patient thought preoperatively to have an FRC close to the closing volume<sup>2</sup> the amount of decrease in FRC to be expected during anesthesia can be predicted from our formula. The presence of 100 per cent oxygen or 30 per cent oxygen in nitrogen as the inspired gas did not affect the decrease in FRC. This finding is not in agreement with the report by

Déry and co-workers,<sup>15</sup> who used a slightly different method and found an immediate decrease in FRC during breathing of 100 per cent oxygen. In other studies of oxygen toxicity<sup>16, 17</sup> consistent alterations in residual volume were not found. Our results show that the lung volume is reduced equally with the two concentrations of oxygen, but they do not exclude a greater incidence of atelectasis if 100 per cent oxygen is breathed when gas is trapped.

The reason for the decrease in FRC is difficult to define. The volume of the lung at end-expiration depends on the balance between the recoil of the lung and the tendency of the chest wall to expand. The electromyogram of the expiratory muscles in anesthetized patients is said to show activity throughout expiration, unlike that of conscious subjects in whom there is silence at end-expiration.<sup>18</sup> In the seven patients in whom we produced apnea during anesthesia by paralysis with succinylcholine, there were no significant changes in end-expiratory position. This procedure was carried out in one subject (patient 11)

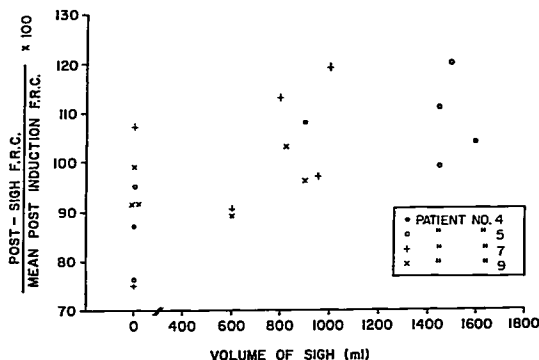


FIG. 5. Effects of spontaneous sighs on measured FRC. Where no sigh occurred, seven of eight measurements were below 100 per cent; with sigh volumes greater than 600 ml seven of ten measurements were above 100 per cent.

who had a measured deficit in FRC of 300 ml during anesthesia. Succinylcholine (30 mg iv) was given after the last measurement of FRC while the patient was still anesthetized; the position of apnea was only 80 ml above the end-expiratory level prior to injection. This suggests that increased expiratory muscle tone is not the cause of the reduction of FRC during anesthesia. It remains possible that a reduction of inspiratory muscle tone under anesthesia might decrease the volume of the lung at end-expiration.

The report by Laws<sup>17</sup> indicated that FRC was reduced immediately after sodium thiopental and succinylcholine were given to patients. From our results (table 5) we conclude that intravenous sodium thiopental does not affect the positions which the lungs and thorax adopt during apnea, compared with the previous end-expiratory positions.

In conscious subjects in the supine position the degree of airway closure and gas trapping is greater in obese subjects,<sup>19</sup> presumably because the weight of the thoracic cage reduces lung volume. It is possible that under anesthesia this effect may be accentuated, causing the reduction of FRC and explaining the relationship to relative obesity. With large inspiratory excursions from FRC, more airways will tend to be above their closing volumes, and small tidal volumes will have the reverse effect. We found that, on the whole, the patients had smaller tidal volumes under anes-

thesia than when conscious. However, in some patients the mean tidal volume during the measurement of FRC approached or, in three patients, exceeded control tidal volumes. We could not establish a relationship between tidal volume and degree of change in FRC. If during the measurement of FRC the patient takes a big inspiration and opens up airways behind which lie trapped gas, the concentration of helium will fall and a larger FRC will be measured. In four of the eleven patients a spontaneous big breath occurred during the measurement of FRC, and the measured FRC was slightly higher than when this sigh did not occur (fig. 5). In one of our subjects (patient 11) who was hyperinflated to 30 cm H<sub>2</sub>O pressure for ten seconds three times and a measurement of FRC immediately made, FRC was 85 ml greater than mean FRC after induction. There was still a deficit of 150 ml compared with control measurements. Our results indicate that the reduction in FRC during anesthesia is due to trapped gas at least in part, and that the one or two sighs during the measurement were insufficient for complete equilibration. It should be noted that atelectasis would also show as a decrease in the helium concentration if a big breath re-expanded the lung, because we added oxygen to maintain a horizontal baseline. However, another effect of atelectasis would be that the end-expiratory level to which the patient returns immediately



after the sigh might give some indication of the volume of gas lost to the lungs from the spirometer. In the patients who did sigh, the mean gain of volume by the lungs after the sigh was small, representing approximately 5 per cent of the decrease in FRC; in no case did it represent more than 12 per cent.

It seems possible, therefore, that the reduction in FRC represents a volume of gas trapped in the lung behind closed airways. This volume is greater in obese subjects, particularly when they are supine. Anesthesia may affect FRC by altering inspiratory muscle tone. An alternative explanation is that normal intermittent big breaths allow communication of the trapped gas with the airway, and that in the anesthetized patient this mechanism is deficient.

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