Respiratory Physiology

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Preface

During my 18 years of teaching medical physiology to almost 5,000 students, I have tried many different instructional approaches to each subject. Some of these proved ineffective and were dropped. The techniques that I found most effective have been retained and refined, and are used in this volume. The author, and any students who may find this text helpful, are in debt to these student "guinea pigs" of mine.

What sort of book is it? No text can be all things to all students, and this one has been especially tailored for those taking a course in medical physiology. Even within this limited context, however, different sorts of coverage remain possible. The material I have selected for inclusion and the order in which I present it represents my best attempt to expedite student understanding. Reasonable teachers can (and certainly will) disagree with my choices. I apologize to them in advance.

I have also chosen to include the problems and the problem-answers following each chapter. Along with most educators, I believe that memorization of normal values and equations, and learning to understand concepts are necessary steps in a medical student's education in physiology. I do not believe students have acquired the most relevant education, however, until they can solve non-trivial problems by using the knowledge. Thus, the problems at the end of each chapter require the application of previously acquired information to new situations, some of them clinical. The answers given after the problems not only identify the correct numerical result or the correct alternative choice, but also present some of the logical sequences that might have been used in arriving at those answers.

This volume will be of interest to medical students, graduate students in physiology, and advanced students, physicians, and researchers interested in reviewing the basic physiology of the respiratory system.

Allan H. Mines

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I am grateful to my wife, Susan, for proofreading the various stages of this text. She endured my wordiness and misspellings with equal patience.

Finally, I owe a debt to my students, on whom I have tried many of the approaches which appear in this text, as well as many others (which mercifully do not appear). I hope to repay that debt in some small way by making the study of respiratory physiology somewhat easier for students to come.

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1

Introduction to Respiration

Respiration is often divided into two parts: Internal respiration and external respiration. The former deals with the processes by which the mitochondria metabolize various foodstuffs, usually involving O_2 as the oxidizing agent, and resulting in the production of high energy bonds, CO_2 and H_2O. This subject is adequately covered in any good textbook of biochemistry, and will hardly be mentioned here. The latter, external respiration, which involves the exchange of gases between the environment and the mitochondria, is the subject of this book. The first chapter is designed to give an overview of respiration: It begins with a consideration of the structure of the system from several viewpoints, and then goes into a brief description of the muscles of respiration. The symbols for variables that are commonly used in respiratory physiology are then tabulated and defined in Table 1. Next, two simple models of the respiratory system are discussed, followed by a brief consideration of the control systems that regulate our breathing. The chapter ends with the definition of the various lung volumes, and with some examples of their measurement using common methods.

STRUCTURE

A brief consideration of the anatomy of the system is probably warranted, since an understanding of function so often depends on a knowledge of the underlying structure. The human lung is extremely complex. The main conducting tube, the trachea, divides into two bronchi, which in turn divide into two tubes each, and each of these tubes then divides into two more, and so forth. In all, there are 20–23 such divisions, resulting in 1–8 million terminal tubes. Each of these has at its end numerous blind sacs, called alveoli, where gas exchange occurs. There are about 300 million of these in man, each of which has a diameter of 75–300 μm. Breathing results in a flow of gas between the environment and the alveoli, while the right heart causes blood to flow through smaller and smaller tubes in the pulmonary circulation, and finally through a meshwork of fine capillaries encasing each alveolus, before returning to the left heart to be pumped through the body. At rest in a normal-sized person, breathing brings about 4 liters of environmental gas into the alveoli per minute

TABLE 1. *Symbols in respiratory physiology*

General variables		Modifying symbols	
C	Compliance	A	Alveolar gas
C	Concentration, content	B	Barometric
D	Diffusing capacity	D	Dead space gas
f	Respiratory frequency	E	Expired gas
F	Fractional concentration (dry gas)	I	Inspired gas
P	Gas pressure	T	Tidal gas
R	Resistance		
R	Respiratory exchange ratio	AW	Airway
Q	Volume of blood	CW	Chestwall
$\dot{Q}$	Volume of blood per unit time	ES	Esophageal
V	Gas volume	IP	Intrapleural
$\dot{V}$	Gas volume per unit time	L	Lung
		T	Total system
		TC	Transchestwall
		TP	Transpulmonary
		TT	Transtotal system
		a	Arterial blood
		b	Blood (general)
		c	Capillary blood
		p	Pulmonary
		t	Tissue
		$\bar{}$	Mean value
		$\dot{}$	Time derivative

Thus, $P_{A_{O_2}}$ = Partial pressure of O_2 in alveolar gas
$P_{a_{O_2}}$ = Partial pressure of O_2 in arterial blood
$\dot{V}_{O_2}$ = O_2 consumption per unit time
$F_{E_{CO_2}}$ = Fraction of CO in dried, expired gas
$\dot{V}_A$ = Ventilation of the alveoli per minute or per second
$\dot{V}_A/\dot{Q}$ = Ventilation/perfusion ratio

(and about that much alveolar gas out into the environment), and the right heart pumps about 4–5.5 liters/min of blood through the pulmonary circulation. During severe exercise, the gas flow must be capable of increasing by 30- –40-fold, and the blood flow by perhaps 5- –6-fold.

These flows of gas and blood must be apportioned reasonably equitably among the 300 million alveoli. Personally, I find that numbers like million and billion, let alone the vastly larger ones which students of biology must deal with, are hard for me to comprehend. This same difficulty was expressed by a student of mine after reading this description, and we found that the concept of "1 million" became clearer to both of us when we calculated that 1 million seconds is equal to 11 days, 13 hours, and 47 minutes. A billion seconds is 31.7 years! A system designed to be able to distribute *one* fluid reasonably equitably to 300 million separate sites would be noteworthy. Our drip irrigation systems, which are now extensively employed and which must cause similar low flows of water out of each of thousands of "spigots" (some of which are elevated

above the others because of nonflat terrain), are a hydraulic engineer's nightmare. The pulmonary distribution system, capable of distributing two fluids reasonably equitably among each of 300 million alveoli (some of which are above and some of which are below the pump), fair boggles the mind. In fact, although this distribution system works astonishingly well in health, the distortions of pulmonary architecture which occur pathologically can cause severe mismatching of ventilation and blood flow ($\dot{V}A/\dot{Q}$ mismatching). Under these conditions, arterialization of blood is impaired and severe disability can result.

The surface area at which these two fluids meet is very large (about 70 m^2) and the average distance between the gas and blood (thickness of the membranes separating the gas and blood) is a small fraction of a micron in healthy lungs. It should be intuitively obvious to the reader that both of these factors facilitate the diffusion of gases between blood and alveolar gas. Equally obvious is that a decrease of the surface area available for exchange, or a thickening of the membranes separating the blood and gas, can lead to inadequate gas exchange, poor arterialization of blood, and clinical disability.

Even in the normal, healthy individual, about 2% of the blood draining from the body tissues bypasses the gas exchange process of the lungs. This small flow of venous blood mixes with the blood that has been arterialized in the lung, lowering the O_2 content of the arterial blood slightly, and lowering its partial pressure of O_2 quite measurably. The two circulations involved in this normal, physiological "venous admixture" are the coronary and bronchial circulations. Some of the coronary venous blood returns to the heart, not through the coronary sinus, but directly into the cavity of the left heart through the thebesian veins; some of the bronchial venous blood returns to the left heart through the pulmonary veins. Various cardiovascular and pulmonary diseases can greatly increase the magnitude of the venous admixture [also called right-to-left (R-L) shunt] causing extreme clinical disability.

On a gross anatomical scale, the lungs and airways share the chest cavity with the heart and great vessels, and the esophagus. The lungs are encased in a thin membrane called the visceral pleura, and another similar membrane, called the parietal pleura, lines the chest cavity. Between the two membranes is a very thin film of fluid that allows the two surfaces to slide past one another easily; but normally, the membranes cannot separate from one another. Why they must stay closely apposed to one another was succinctly summarized by Jere Mead in April, 1961 when he wrote in *Physiological Reviews:*

> Regard the occupancy of the chest cavity as a competition between solids, liquids, and gases. The liquids are removed down to a vestige because the capillary pressure in the visceral pleura is considerably lower than its colloid osmotic pressure. The gases are removed because the total gas pressure in venous capillary blood is considerably less than atmospheric due to the relative capacity of blood for carbon dioxide and oxygen. The lungs, chest wall, and diaphragm are then pressed into service by atmospheric pressure and occupy the space, as it were, by default.

So, normally the lungs and chest wall must move as a unit; the lung volume only changes by a liter if the chestwall volume has also changed by a liter. I emphasize this here because the diagrams we tend to make of the respiratory system often show the lungs as a smallish balloon inside a large chest cavity. We do this so that there is plenty of room to write in pressures and volumes, but the student should remember that anatomical accuracy is sacrificed in this effort.

MUSCLES OF RESPIRATION

Normally, in quiet breathing, the inspiratory muscles pull the respiratory system above its "equilibrium volume" [Functional Residual Capacity (FRC)], then relax, allowing the elastic recoil of the system to effect expiration passively. The main inspiratory muscles are the diaphragm and the external intercostal muscles, and in quiet breathing, the diaphragm may be the only active inspiratory muscle. The diaphragm is attached all along the circumference of the lower thoracic cavity; its contraction pulls its central part down, enlarging the thoracic cavity. The motor nerves to the diaphragm leave the spinal cord in the ventral roots of C 3–5, and travel in the phrenic nerves. The external intercostal muscles are innervated by intercostal nerves, which leave the spinal cord in T 1–11. Their contraction raises the anterior end of each rib, pulling it upward and outward, and increasing the anterior–posterior diameter of the thoracic cavity. Other muscles, the "accessory" muscles of inspiration, become active only when breathing is greatly increased, as in severe muscular exercise. These include the scalenes, the sternocleidomastoids, the trapezii, and various muscles that reduce resistance to air flow. Maximal contraction of the inspiratory muscles with the glottis closed (the "Müller maneuver") can cause intrapleural pressure to become 60–100 mm Hg less than atmospheric.

Although expiration is passive during quiet breathing, it can become active when breathing is greatly increased or when significant airway obstruction exists. The abdominal muscles (including the rectus abdominus, external oblique, internal oblique, and transversus abdominus) are the principal expiratory muscles. They are innervated by nerves emerging from the spinal cord at the last six thoracic segments and at the first few lumbar ones. Their contraction increases intraabdominal pressure, thus forcing the diaphragm upward and increasing intrapleural and intrapulmonic pressures. They are absolutely essential for such functions as coughing, straining, and vomiting. The internal intercostals can also aid expiration and are innervated by intercostal nerves leaving the spinal cord at T 1–11. Forced maximal contraction of the expiratory muscles against a closed glottis (Valsalva's maneuver) can result in sustained intrapulmonary pressures above 100 mm Hg. Such pressures in the abdomen and chest will decrease venous return to the heart from the lower extremities and from the head and upper extremities. You already know that less cardiac input means less cardiac output; less cardiac output means less cerebral circulation; less

cerebral circulation can mean loss of consciousness in seconds. This sequence of events leads to CNS asphyxia and unconsciousness even more rapidly if a period of hyperventilation (which constricts cerebral vessels) precedes the Valsalva maneuver.

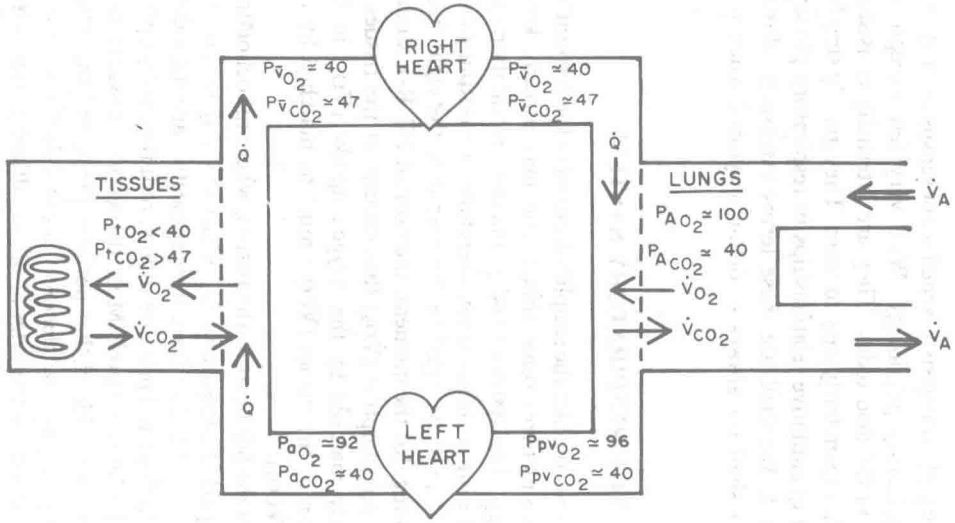
SYMBOLOGY

In 1951 a group of respiratory physiologists reached a consensus on a system of symbols to be used in respiratory physiology. With very few exceptions, these are used by all workers in the field today. They are internally consistent and well conceived, which makes them fairly easy to learn. They greatly simplify the consideration of the many quantitative relationships in respiratory physiology. They are listed in Table 1. We shall use these terms extensively during the respiration chapters, but I shall try always to define a symbol when it is first used.

A MODEL OF THE RESPIRATORY SYSTEM

Let us first use these symbols to consider the simplified model of the respiratory system shown in Fig. 1. It emphasizes some things you may already know from cardiovascular physiology. The systemic and pulmonary circulations are in series with one another. During a steady state, therefore, the output of the left heart (systemic circulation) must be equal to the output of the right heart (pulmonary circulation) (ignoring, for the moment, the normal 2% R-L shunt). In the same way, the oxygen consumption ($\dot{V}O_2$) that occurrs at the tissues in a steady state must be exactly matched by the oxygen uptake ($\dot{V}O_2$) in the lungs, and the CO_2 production at the tissues ($\dot{V}CO_2$) must be matched by the CO_2 excretion by the lungs ($\dot{V}CO_2$).

Arterial blood is pumped by the left heart to the tissues, where mitochondrial metabolism keeps the tissue partial pressure of O_2 (Pt_{O_2}) lower than, and the tissue partial pressure of CO_2 (Pt_{CO_2}) higher than, their respective arterial values. Thus, both gases diffuse from high partial pressure to low, resulting in O_2 uptake by the tissues and CO_2 uptake by the blood. Most tissues use nowhere near the total amount of O_2 present in the blood that perfuses them, and the mixed venous blood oxygen content ($C\bar{v}_{O_2}$; the bar denotes "mean value") is, at rest, normally about 75% of what it was in the arterial blood, although the partial pressure of O_2 in the mixed venous blood ($P\bar{v}_{O_2}$) is about 40 mm Hg, while the arterial P_{O_2} (Pa_{O_2}) is near 100 mm Hg. This discrepancy between the fall of content and fall of partial pressure reflects the unusual sigmoid relationship between those variables in the case of O_2. Almost as much CO_2 is exchanged at the tissues as is O_2 [Respiratory Quotient (R.Q.) = $\dot{V}CO_2/\dot{V}O_2$ = about 0.8 (the dot denotes "time derivative")], yet the rise of P_{CO_2} from the arterial blood to the mixed venous blood is normally only about 6–8 mm Hg. Thus, in the



IN STEADY STATE:

$$\dot{V}_{CO_2\,tissues} = \dot{V}_{CO_2\,lungs} = \dot{Q}[C\bar{v}_{CO_2} - Ca_{CO_2}] = \dot{V}_A[F_{A_{CO_2}} - F_{I_{CO_2}}]$$

$$\dot{V}_{O_2\,tissues} = \dot{V}_{O_2\,lungs} = \dot{Q}[Ca_{O_2} - C\bar{v}_{O_2}] = \dot{V}_A[F_{I_{O_2}} - F_{A_{O_2}}]$$

$$\dot{Q}\ right\ heart = \dot{Q}\ left\ heart$$

FIG. 1. A simplified model of the respiratory system. P_{CO_2} and P_{O_2} are given normal, resting sea-level values in the tissues (Pt_{O_2}), in the mixed venous blood ($P\bar{v}_{O_2}$), in the alveolar gas ($P_{A_{O_2}}$), in mixed pulmonary venous blood (Ppv_{O_2}), and in the arteries (Pa_{O_2}).

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normal range the curve relating content of CO_2 to its partial pressure has a very different shape from that of the O_2 curve.

The mixed venous blood is then pumped by the right heart to the lungs where it exchanges with alveolar gas, losing CO_2 to the gas and gaining O_2 from it. The partial pressures in the mixed pulmonary venous blood are normally very nearly the same as those in mixed alveolar gas, and the partial pressures and contents in arterial blood are again very nearly the same as those in mixed pulmonary venous blood. The alveolar ventilation ($\dot{V}A$) brings a flow of fresh environmental gas, which is rich in O_2 (20.93%) and almost free of CO_2 (0.04%), through the alveoli. This flow of gas tends to replace the O_2 taken up by the blood and to carry the CO_2 given off by the blood out into the environment. Again in the steady state, the amounts of O_2 and CO_2 exchanged at the alveolar membranes must be equal to the amounts exchanged with environment per minute.

PRESSURE PROFILE OF O_2 FROM ENVIRONMENT TO MITOCHONDRION

The respiratory system can also be viewed as a delivery system that passes O_2 down a series of steps between environment and mitochondrion, as seen in Fig. 2. There is a limited "pressure head" for that delivery, which is set by P_{O_2} in the environmental gas, which in turn is determined by the barometric pressure times the fraction of the dry gas molecules which are O_2 ($P_B \times F_{O_2}$). If the environmental gas is totally dry, which normally occurs only if a subject breathes from a tank of dried compressed air, then the environmental $P_{O_2} = 760 \times 0.2093 = 159$ mm Hg. It is easy to see that lowering either P_B (as in the ascent to high altitude) or F_{O_2} (as in breathing a mixture of air and N_2) will lower the available "pressure head" for O_2 delivery, and tend to lower the maximum possible $\dot{V}O_2$. As soon as the gas enters the warm, moist respiratory tract it is humidified, and by the time it reaches the trachea it has become saturated with water vapor. At a body temperature of 37°C, the water vapor pressure is 47 mm Hg, and the total pressure exerted by the other gases (O_2, N_2, CO_2, etc.) can then be only $760 - 47 = 713$ mm Hg. The P_{O_2} in the trachea, defined as the inspired P_{O_2} ($P_{I_{O_2}}$), is then $(P_B - P_{H_2O})(F_{I_{O_2}}) = 713$ mm Hg $(0.2093) = 149$ mm Hg. So, P_{O_2} decreased as the gas became humidified, simply because the water vapor diluted the O_2. The gas then passes into the alveoli where its P_{O_2} (the $P_{A_{O_2}}$) is determined by a balance of two processes. The alveolar ventilation ($\dot{V}A$) brings a flow of O_2-rich gas into the alveoli and tends to raise $P_{A_{O_2}}$, while the pulmonary blood flow ($\dot{Q}$) removes O_2 from the alveolar gas and tends to lower $P_{A_{O_2}}$. As $\dot{V}A$ increases, clearly $P_{A_{O_2}}$ will tend to be brought closer to $P_{I_{O_2}}$, and vice versa. In this case, $P_{A_{O_2}}$ is shown to be a nice, normal sea-level value of 100 mm Hg. The pulmonary venous blood, here shown to have a P_{O_2} of 96 mm Hg, normally has a slightly lower P_{O_2} than the mixed alveolar gas. The two possible contributors to this drop of

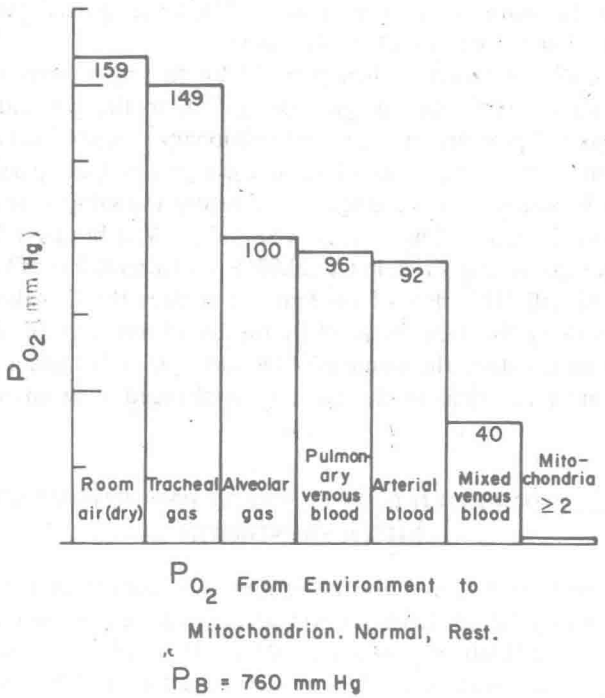

FIG. 2. Oxygen pressure profile from environment to mitochondrion in a normal resting male at sea level.

P_{O_2} are $\dot{V}_A/\dot{Q}$ mismatching and diffusion difficulties, but normally at sea level the latter contribution is too small to measure. As already mentioned in this chapter, however, pulmonary disease can result in either of these factors producing a drop in P_{O_2}, which can be severely debilitating. By the time the arterialized blood from the lungs has mixed with the 2% R-L shunt (coronary and bronchial circulations), and has been pumped into the systemic arteries, its P_{O_2} (now the arterial P_{O_2} or Pa_{o_2}) has decreased still further, and is here shown to be 92 mm Hg. This small difference of 8 mm Hg between PA_{o_2} and Pa_{o_2} can obviously be increased greatly if the R-L shunt is larger.

Since the arterial walls are far too thick to allow any significant gas exchange, neither Pa_{o_2} nor the O_2 content changes until the blood reaches the capillaries, where only a single endothelial cell separates blood from tissue. Tissue P_{O_2} (Pt_{o_2}) is kept quite low (< 40 mm Hg) so that O_2 diffuses from blood to tissue rapidly near the arterial end of the capillary, where the difference in P_{O_2} is large ($92 - Pt_{o_2}$), and then less and less rapidly as the P_{O_2} in the capillary decreases. The O_2, still following its partial pressure gradient, diffuses through the extracellular fluid, into the cells, finally to reach the mitochondria, the lowest level in this O_2 transport cascade, where P_{O_2} may get as low as 1–3

mm Hg. Here, O_2 is used to oxidize H_2 and C, which is the ultimate purpose of this O_2 delivery system. The blood, having passed through the tissue capillaries, now returns to the right heart through the systemic veins, its P_{O_2} having been decreased to about 40 mm Hg.

CONTROL OF RESPIRATORY SYSTEM

Unlike the heart, which has its own intrinsic rhythm, the respiratory muscles do not "beat" on their own, and therefore respiration needs to have neural systems capable of generating and maintaining a regular breathing cycle to carry out gas exchange between environment and alveoli. We know that the organism's environment is subject to change (ascent to high altitude, scuba diving, etc.) and that mitochondrial activity is subject to change (muscular exercise, body temperature change, altered thyroid function, etc.). Thus, there is clearly a need for systems that will modify this basic regular breathing cycle by achieving rates of gas exchange that will satisfy bodily needs under a wide variety of functional states. These systems must have receptors to sample the parameters of importance to the respiratory system (P_{O_2}, P_{CO_2}, pH, state of lung inflation, level of muscular activity, etc.). Using that information, and setting priorities when conflicting needs exist, the systems must drive the muscles of respiration smoothly, dependably, and automatically. They must be capable of voluntary override to allow for speech and the myriad other activities in which we engage using nose and mouth. These systems will be discussed at some length in the chapter entitled "Regulation of Breathing."

LUNG VOLUMES

Clinically, it is useful to be able to measure the volume of gas in the lungs under a number of different circumstances. The various divisions and subdivisions of the lung volume are shown in Fig. 3. Total Lung Capacity (TLC) is the maximum amount of gas the lung can contain *in vivo*, when the subject inspires as much as he can. When the subject starts from that position of maximum inflation, and expires as much gas as he can, he is exhaling his Vital Capacity (VC). At this point, there will be a significant amount of gas still left in the lung, the Residual Volume (RV), which cannot be exhaled *in vivo*. Normally, however, people do not exhale down to RV with each breath. During quiet breathing, they exhale down to FRC, at which point the elastic recoils of the two components (lungs and chest wall) just balance one another. FRC, then, is the volume to which the respiratory system returns at the end of a normal quiet breath, and is about 40% of the TLC. If a subject inhales as much as he can, beginning at FRC, he inhales his Inspiratory Capacity (IC). Normally, however, people inhale much smaller volumes with each quiet breath, called Tidal Volumes (V_T). After having inhaled a tidal volume, a person still has a considerable Inspiratory Reserve Volume (IRV) between end-inspiration and

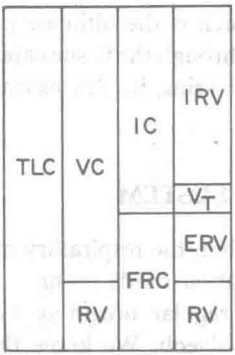

FIG. 3. Lung volumes in a healthy young male.

TLC. After having exhaled normally down to FRC, a person has a smaller, but still considerable, Expiratory Reserve Volume (ERV) between FRC and RV.

MEASUREMENT OF LUNG VOLUMES

It is easy to measure those lung volumes not involving the RV with a simple spirometer, shown in Fig. 4. It is simply an upside-down water-sealed can (called a bell) whose movements are recorded on moving paper. When a subject is made to breathe into and out of the spirometer, his breathing movements are traced on the paper. If, for instance, beginning at TLC, he exhales down to

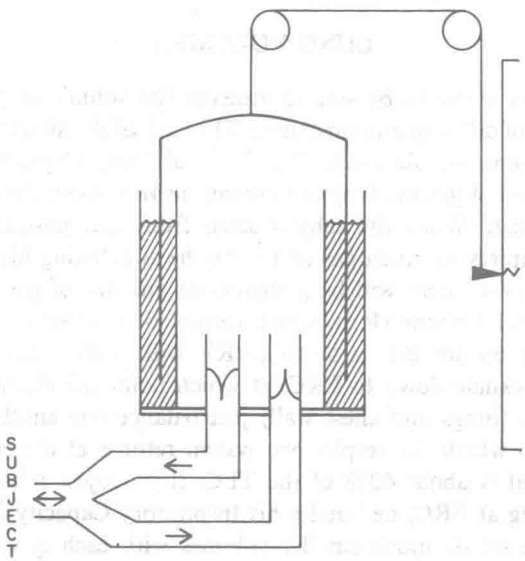

FIG. 4. A cross section through a spirometer.

RV, the pen will have moved a vertical distance proportional to his VC. All one needs to know is the constant of proportionality (which turns out to be the cross-sectional area of the bell) to calculate the volume that has been put into the bell (the volume of the right-circular cylinder which has been filled) as the subject exhaled his VC. Of course, the gas in the lungs was at 37°C and saturated with H_2O at that temperature (Body Temperature and Pressure, Saturated, or BTPS), while the gas will have decreased in volume as it entered the spirometer, which is at Ambient Temperature and Pressure, Saturated, or ATPS, so an ideal gas correction is necessary. Assuming that P_B = 760 mm Hg, body temperature = 37°C (at which temperature the vapor pressure of H_2O is 47 mm Hg), and spirometer temperature = 25°C (at which temperature P_{H_2O} = 24 mm Hg), the calculation proceeds as follows:

$$\frac{P_1V_1}{T_1} = \frac{P_2V_2}{T_2}$$

$$V_2 = \frac{P_1V_1T_2}{T_1P_2} = \frac{(736 \text{ mm Hg})(V_1)(310°K)}{(298°K)(713 \text{ mm Hg})}$$

Thus, $V_2 = 1.074(V_1)$, or, the actual volume moved out of the lungs is 7.4% greater than the volume moved into the spirometer. In a similar fashion, one could measure IC, IRV, ERV, and V_T, but wouldn't know the value of TLC, RV, or FRC without more information.

The additional information can be gotten through the use of either a volume of dilution method or a Boyle's law method. You may already have become familiar with the principle behind the volumes of dilution method. Figure 5 shows one way that it can be used to measure FRC, called the helium closed circuit method. At FRC, a subject begins rebreathing from a bag that initially contains 2 liters of 10% He. He continues to rebreathe until mixing is complete and the concentration of He is the same in the bag and the lungs. Since negligible amounts of He are taken up by the body, the initial amount of He present only in the bag must equal the final amount of He "present in bag and lungs." The calculation proceeds as follows:

$$\begin{aligned}
\text{Initial amount of He} &= \text{Final amount of He} \\
V_L(\%He)_i + Vbag(\%He)_i &= (V_L + Vbag)\ (\%He)_f \\
0 + 2 \text{ liters } (0.1) &= (V_L + 2 \text{ liters})\ (0.05) \\
0.2 \text{ liters} &= 0.05V_L + 0.1 \text{ liter} \\
2 \text{ liters} &= V_L
\end{aligned}$$

where V_L and Vbag are the volumes of lung and bag, respectively, and i and f refer to the initial and final (complete mixing) situations. This type of method will measure the volume of all gas able to exchange with the bag He, but will not measure that which is trapped beyond occluded airways.

The Boyle's law method measures the total Thoracic Gas Volume (TGV), whether in free communication with the airways or not. Boyle's law simply

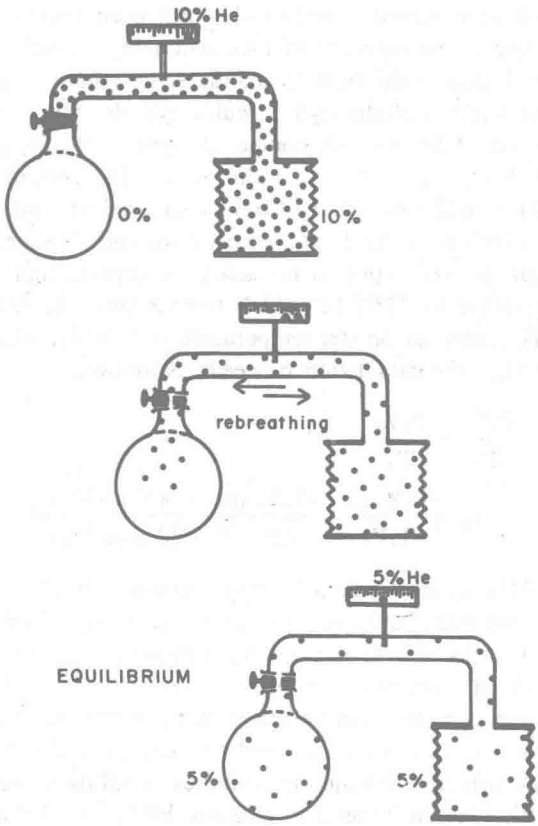

FIG. 5. Measurement of FRC: Helium closed-circuit technique. (Reproduced with permission from Comroe, J. H., Jr., et al.: *The Lung: Clinical Physiology and Pulmonary Function Tests,* 2nd Ed. Copyright © 1962 by Year Book Medical Publishers, Chicago.)

states that the pressure times the volume of a gas is constant, provided that both the number of moles and the temperature stay constant, or $P_1V_1 = P_2V_2$. This method requires the use of a body plethysmograph, one type of which is shown in Fig. 6. The subject is seated within an airtight box, breathing the outside air through a tube. A pressure gauge samples the pressure in the tube near his mouth, which will be equal to alveolar pressure only if there is no flow of gas through the tube. The volume changes of his lung are measured accurately by the Krogh spirometer. As he breathes in, expanding his chest by 1 liter, 1 liter of air around him is displaced up into the Krogh spirometer, and vice versa. To measure the FRC, one simply occludes the airway when the subject has just completed a normal expiration (subject at FRC) by turning the stopcock 90° counterclockwise. The subject makes inspiratory and expiratory efforts against the closed airway, thus decompressing and compressing the gas in his lungs, and these volume changes continue to be recorded by the Krogh

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