RELATING BASIC CONCEPTS OF PULMONARY MECHANICS TO CLINICAL SITUATIONS

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How the pressure-volume relationships of the lungs and chest wall interact to determine the compliance characteristics of the respiratory system is perhaps one of the more difficult conceptual areas in pulmonary physiology both to teach and for first-year medical school physiology students to master. An understanding of this material is nevertheless important, because it allows the student to integrate a large number of concepts in pulmonary mechanics that may not seem at first glance to be related. For example, in spirometry, the lung divisions, total lung capacity (TLC), functional residual capacity (FRC), and residual volume (RV) are frequently learned only in descriptive terms; e.g., FRC is the volume of air remaining in the lungs at the end of a normal expiration. An understanding of the pressure-volume characteristics of the respiratory system, however, allows these descriptive definitions to be replaced by more useful functional definitions; e.g., FRC is the lung volume at which the elastic recoils of the lungs and chest wall are equal in magnitude but opposite in direction. The latter has the distinct advantage in that it helps the student to understand why FRC is altered in such diseases as emphysema and pulmonary fibrosis, the clinical utility of determining FRC, and the calamitous consequences of a pneumothorax. An understanding of the compliance characteristics of the lungs is also critical for helping the student to understand why a vertical distribution of ventilation exists in the lungs and how, for example, in unilateral lung disease, arterial Po2 can have substantially different values depending on whether the patient is lying on the left or right side. According to the objective of this presentation is to provide a framework for teaching this material in a way that allows the student to understand the relationship of basic concepts to the disordered physiology that results from lung disease.

PLACING A DISCUSSION OF COMPLIANCE IN THE OVERALL CONTEXT OF FUNCTION: WORK OF BREATHING

The work required to ventilate the lungs can be divided into two major components, elastic and nonelastic work. Both of these components can be further divided. In this regard, the elastic work component is composed of work that must be done against lung elastic recoil, chest wall recoil, and surface tension. The nonelastic work component is primarily the effort required to overcome airway resistance but also has a small tissue resistance contribution. An understanding of how energy is expended in ventilating the lungs is important, because patients with lung disease may have a larger energy requirement to breathe than individuals with healthy lungs. Importantly, the precise mechanism responsible for the increased work of breathing may differ depending on which subcomponent is affected. For example, the work of breathing is increased in a patient with pulmonary fibrosis because of an abnormal reduction in lung compliance. In a patient with ankylosing spondylitis (a disease in which the vertebral joints and ribs become immobile), the work of breathing may be increased due to a reduction in chest wall compliance. Babies with infant respiratory distress syndrome have to work harder to breathe because of an increase in alveolar surface tension due to a deficiency of pulmonary surfactant. Finally, patients with asthma or bronchitis expend more energy breathing because of an increased airway resistance. These examples thus indicate that an understanding of the various components that contribute to the work of breathing allows the student to appreciate that patients with lung disease may be required to expend more respiratory energy but for very different reasons. In the remainder of this discussion, we will concentrate on developing an understand-
ing of the lung and chest wall components of elastic work.

**A “Time-Out” For Some Basic Definitions**

At this time, it is important to define three of the lung capacities and volumes that are generally considered during a discussion of spirometry. These are TLC, FRC, and RV, and they are defined in Table 1. When introducing these concepts, I find it useful to have the students inhale maximally to TLC, exhale passively to FRC, exhale maximally to RV, and then finally inhale passively to FRC. This allows me to point out that they had to do considerable work in both reaching and maintaining TLC and RV. This sets the stage for a discussion of the factors that require this energy expenditure. I also point out that when they reached FRC (after either exhaling from TLC or inhaling from RV), their lungs automatically stopped at this position and that it takes no conscious work to keep the lungs at this volume. Note that the definitions given in Table 1 are purely descriptive. They give little insight into the functional significance of these capacities and volumes. As the discussion develops, these descriptive definitions will be replaced with functional definitions that have the advantage of allowing the student to understand the significance of measuring TLC, FRC, and RV in health and disease.

**DISCUSSION OF COMPLIANCE: FROM SPRINGS AND BELLOWS TO ISOLATED LUNGS TO INTACT LUNGS**

Figure 1 (top) shows a simple spring that is progressively being stretched in each succeeding frame by the addition of incremental amounts of weight. This behavior is plotted as a simple graph at the bottom, which shows that as the force (f) on the spring increases, the distance (d) the spring stretches also increases. For a while this relationship is linear, and the slope of this curve (Δd/Δf) is defined as the compliance of the spring. Note that after a critical amount of weight is added, the spring can no longer distend, and the curve becomes flat. At this point, we say that the spring has reached its elastic limit. The addition of further weight may cause the spring to break. This behavior has an analogy in the lung in that, in breath hold divers who do not exhale as they ascend from a dive, the air contained in the lungs expands. If this process continues, alveolar pressure may increase to values that exceed the elastic limits of the alveoli, and barotrauma may result. Finally, Fig. 1, middle, shows the effect of stretching a bellows, a structure that behaves in an analogous fashion to the spring. The compliance characteristics of the bellows are also shown on the graph plotted in Fig. 1. In this case, however, the data are plotted on volume (v)-pressure (p) axes (instead of the distance-force axes used for the spring), and the compliance is given as Δv/Δp. This representation is useful because this structure is more like a lung, i.e., air flows into the bellows as its volume is expanded.

Up to this point, we have discussed the compliance characteristics of a single spring or bellows with a set compliance. It is also possible to examine the pressure-volume characteristics of less compliant (stiffer) and more compliant (looser) springs and bellows (Fig. 2). As shown in Fig. 2, the compliance (Δd/Δf or Δv/Δp) is reduced for the stiff spring or bellows and is increased for the loose spring or bellows. In other words, it is harder to both stretch a stiff spring and to inflate a stiff bellows than it is to cause the same changes in normal springs or bellows.

It is also possible to measure the compliance of isolated lungs. An experimental setup for doing so is shown in Fig. 3, which depicts an isolated lung in a bell jar. The pressure surrounding the lung (pleural pressure) can be reduced in a step fashion by pumping air out of the jar. This causes the lung to inflate. In the figure, the volume of air entering the lungs is measured by a spirometer. The results of this maneuver are plotted on the graph at right and show the pressure-volume relationships of the lungs. Note that the inflation and deflation curves do not follow the

<table>
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<tr>
<td>TLC</td>
<td>Volume of air contained in the lungs after a maximal inspiration</td>
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<tr>
<td>FRC</td>
<td>Volume of air remaining in the lungs after a normal passive expiration</td>
</tr>
<tr>
<td>RV</td>
<td>Volume of air remaining in the lungs after a maximal expiration</td>
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TLC, total lung capacity; FRC, functional residual capacity; RV, residual volume.
same path. This is called “hysteresis” and is related to the presence of surface tension on the alveolar epithelial surfaces, because the degree of hysteresis is markedly reduced when the contribution of surface tension is eliminated by inflating the lungs with saline instead of air. For simplicity, a deflation compliance curve is shown in Fig. 4. This curve indicates that the lung exhibits nonlinear compliance characteristics. At low lung volumes, the compliance is high (i.e., a given change in pressure results in a relatively large change in volume), and at higher lung volumes, the compliance is reduced (i.e., the same change in pressure results in a smaller change in volume).

Another “Time-Out” For Some Basic Definitions

An often confusing aspect of understanding compliance is the term that is used to describe the pressure axis of the pressure-volume curve. In various text figures, the reader may see this axis labeled as “pressure surrounding the lung,” “pleural pressure,” “transpulmonary pressure,” or “elastic recoil pressure.” The first two of these terms are synonymous, i.e., pleural pressure is the pressure surrounding the lung. Likewise, under static conditions, transpulmonary pressure is synonymous with the term elastic recoil pressure. Pleural pressure and transpulmonary pres-
sure are two very different pressures, however. Pleural pressure is the pressure on the outside of the lung and during normal breathing is a negative (subatmospheric) value. The transpulmonary pressure, however, is a “transmural pressure,” i.e., the difference in pressure between the inside and outside of the lungs, and by convention is always positive. While we are breathing (when air is either flowing in or out of the lung), the two pressures are not the same, as shown in Fig. 5. If this is true, how can these terms be used interchangeably on the x-axis of the pressure-volume curve? The answer to this question is that they cannot, except under one unique set of conditions: when there is no airflow in or out of the lungs. Under these conditions, the pleural and transpulmonary pressures are equal in magnitude but opposite in sign (Fig. 5). Thus, when compliance is measured under conditions of zero airflow (i.e., “static conditions”), either measure of pressure may be used.

BACK TO COMPLIANCE OF THE LUNGS: MEASUREMENT OF COMPLIANCE IN THE INTACT LUNG WITHIN THE CHEST

It is also possible to measure the compliance of the lungs in people. As with isolated lungs, this measurement requires knowledge of the lung volume and some measure of the distending pressure of the lungs. The former can be easily measured using a spirometer, but the latter is complicated because of the difficulty of measuring pleural pressure when the lungs are...

**FIG. 2.**
Effect of changing compliance on distance-force and pressure-volume relationships in springs and bellows. Vertical arrows indicate distance or volume change that occurs for a comparable change in force or pressure.

**FIG. 3.**
Measurement of pressure-volume relationships in isolated lungs. [Borrowed with permission from J. B. West. Respiratory Physiology—The Essentials (5th ed.). Baltimore, MD: Williams & Wilkins, 1995 (11).]
enclosed within the thoracic cavity. Although it is possible to insert a needle to measure the pressure between the visceral and parietal pleural membranes, this procedure carries the risk of either puncturing the lungs or creating a pneumothorax. Few people would be willing to have this measurement made on themselves, and it is not likely that an institutional human review board would readily approve such a procedure. A less onerous procedure for obtaining a measurement of pleural pressure is to measure changes in the pressure within the esophagus during the respiratory cycle. This measurement is obtained by having the subject swallow a balloon-tipped catheter so that the tip comes to rest within the esophagus. What does the measurement of esophageal pressure tell about pleural pressure? The answer is that the esophagus is essentially a flaccid tube (but only when there is no esophageal muscular activity such as when the subject swallows), it expands as its outside pressure (pleural pressure) falls during inspiration, thus lowering its internal pressure. Thus the pressure within the esophagus changes as the pleural pressure changes during the respiratory cycle and can thus be used to track changes in pleural pressure.

**ALTERED LUNG COMPLIANCE IN INDIVIDUALS WITH LUNG DISEASE**

It is possible to use the above technique to determine the pressure-volume relationships of lungs of individuals with different lung diseases. Figure 6 shows representative compliance curves for an individual with normal lungs, a patient with pulmonary fibrosis, and another with emphysema. The figure shows that compliance is reduced in the patient with fibrosis and increased in the patient with emphysema. The work
The effort required to overcome the elastic recoil of the lungs is thus increased in the patient with fibrosis. This occurs because relatively noncompliant fibrotic tissue has been laid down in the lung, thus increasing its stiffness. On the other hand, the effort required to overcome the elastic recoil of the lungs is actually decreased in the patient with emphysema, because the lungs have lost elasticity due to tissue destruction. The figure also shows that TLC is reduced in the patient with fibrosis but may be greater than normal in the patient with emphysema. At this point, it is important to ensure that the student does not come away from this discussion with the idea that emphysema is a desirous condition to have because it is easier to inflate the lungs and TLC is greater. These effects are far outweighed by an increased work of breathing due to an increase in airway resistance caused by the loss of radial tension around noncartilaginous airways and disruptions in gas exchange due to loss of surface area for gas exchange and ventilation/perfusion inequality. Later in the discussion, we will discuss factors that may play a role in altering TLC in patients with these diseases.

COMPLIANCE CHARACTERISTICS OF THE CHEST WALL

Up to this point, we have discussed only the compliance characteristics of the lungs. When we breathe, however, we also have to expand the chest wall, which has its own unique set of compliance characteristics. (The term “chest wall” used in this context is a simplification and actually represents the rib cage, diaphragm, and abdomen.) The pressure-volume relationship for the chest wall is shown in Fig. 7. The curve shows that at ~55–60% of vital capacity, the transmural pressure of the chest wall is zero. This means that the pressures on the outside and inside of the chest wall are equal and that it requires no effort to keep the chest wall at this position. This is called the “resting position” of the chest wall. Work is required to either inflate or deflate the chest wall from this position, however. To deflate the chest wall, the transmural pressure must be made negative (outside greater than inside pressure), whereas to inflate the chest wall, the transmural pressure must be made positive.

COMPLIANCE OF THE RESPIRATORY SYSTEM

As indicated earlier, when we breathe, we must do work against the elasticity of both the lungs and chest wall. Accordingly, it is useful to show both curves on the same pressure-volume graph (Fig. 8). To determine the pressure-volume relationship of the entire respiratory system, the lung and chest wall curves are added to create a third curve that defines the work required to inflate the combined lungs and chest wall.1 This graph also contains another piece of very important information. Note that all three structures at some point have a particular volume where the recoil (transmural) pressure is zero. This means that the pressure on the inside of each structure is equal to that on the outside and that there is thus no force to make the structure inflate or deflate. These points are “equilibrium points” and represent the volumes that occur in the absence of external forces. In this regard, they represent the “resting positions” of the chest wall, lungs, and respiratory system. As indicated above, the resting position of the chest wall is at ~55–60% of vital capacity, and the resting position of the lungs is actually at some value below the residual volume. (This volume is termed the “minimal volume” of the lungs and is analogous to the small

1 Mines et al. (7) have developed a laboratory exercise in which the pressure-volume relationships of the lung, chest wall, and respiratory system at lung volumes above FRC can be determined. The original exercise was written using anesthetized dogs, but should be readily adaptable to smaller species.

2 This curve is called the “relaxation-pressure curve” because it can be obtained by measuring the airway pressure when the respiratory muscles are relaxed against an obstructed airway at various lung volumes (3). A positive pressure develops when the inflated chest is relaxed, and a negative pressure occurs when the deflated chest is relaxed.
volume of air contained in an uninflated balloon.) Thus, under normal conditions, the lungs never reach their minimal volume. The resting position of the respiratory system is somewhere in between those of the lungs and chest wall. Figure 8 shows that, at this point, the elastic recoils of the lungs and chest wall are equal but opposite. In other words, at this volume, the tendency of the lungs to recoil inward toward their minimal volume is exactly balanced by the tendency of the chest wall to recoil outward to its resting position. A standoff is achieved, which requires no effort, and this creates the resting position of the respiratory system. This volume is FRC. Previously, we gave this volume a descriptive definition (Table 1). It can now be defined as the resting position of the respiratory system, i.e., the lung volume where the tendency of the lungs to recoil inward is exactly balanced by the tendency of the chest wall to recoil outward (Table 2).

**UNCOUPLING THE LUNGS FROM THE CHEST WALL: CREATION OF A PNEUMOTHORAX**

The pressure-volume curves of the lung, chest wall, and respiratory system can also be used to understand what happens when a pneumothorax is created. Normally, the pleural pressure is subatmospheric (negative). If the chest wall is injured in such a way as to create a passage between the atmosphere and pleural space, the higher atmospheric pressure causes air to rush into the pleural space. If the passage is

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3 This may not be strictly correct. Submaximal neuromuscular blockade in seated humans has been found to produce a small (15%) reduction in FRC that results from a decrease in the outward elastic recoil of the chest wall (2). These data suggest that involuntary respiratory muscle activity contributes to the elastic recoil of the chest wall and thus somewhat influences the value of FRC.
sufficiently large, this process results in the lung becoming uncoupled from the chest wall. When the normal coupling is severed, both the lungs and chest wall are free to move toward their respective resting positions. As a result, the lung collapses to its minimal volume and the chest wall recoils outward to its resting position. To reestablish the coupling between the lung and chest wall, it is necessary to reestablish the subatmospheric pleural pressure. This is what the surgeon does by inserting a chest tube before closing the chest after thoracic surgery. The chest tube is used to evacuate air from the thoracic cavity and thus reestablish a subatmospheric pleural pressure.

**FUNCTIONAL DEFINITIONS OF TLC, FRC, AND RV**

Based on the previous discussion, it is now possible to replace the descriptive definitions of TLC, FRC, and RV with functional definitions that result from considering these volumes as lung volumes at which static balances have been achieved between opposing respiratory forces (Table 2). Thus, as indicated above, FRC represents the volume at which the opposing elastic recoil pressures of the lungs and chest wall are of equal magnitude. At TLC, muscular effort is required to maintain the lungs (which have a relatively low compliance at this volume; see Fig. 8) and chest wall at this volume. The value for TLC is thus determined by how successful the inspiratory effort is in balancing the combined tendencies of the lungs and chest wall to recoil inward. Finally, at RV, the lungs still have a small but finite recoil pressure (tendency to collapse), but this is overwhelmed by the much greater tendency for the chest wall to recoil outward. RV in young individuals without pulmonary disease is thus determined by the ability of the respiratory muscles to

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**TABLE 2**

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<tr>
<td>TLC</td>
<td>Lung volume at which a static balance has been achieved between the maximal inspiratory force that can be generated by the respiratory muscles and the expiratory force generated by the inward-directed elastic recoils of the lungs and chest wall.</td>
</tr>
<tr>
<td>FRC</td>
<td>Lung volume at which the elastic recoils of the lung and chest wall are equal but opposite.</td>
</tr>
<tr>
<td>RV</td>
<td>Lung volume at which a static balance has been achieved between the maximal expiratory force that can be generated by the respiratory muscles (and the elastic recoil of the lung) and the force generated by the outward-directed elastic recoil of the chest wall.</td>
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compress the chest wall. In older individuals and those with lung disease, however, RV may have additional determinants, which will be discussed in the next section.

These functional definitions have the advantage in that they provide the student with the means to begin to analyze how lung volumes may become altered in disease. For example, reductions in TLC could occur if the compliance of the lung or chest wall is reduced or if the inspiratory muscles become weakened.

INTERACTION OF LUNG AND CHEST WALL ELASTIC RECOILS IN HEALTH AND DISEASE

At this point, it is now possible to consider how the determination of FRC and TLC helps the physician to understand how the mechanical properties of the lungs can change with disease. In Fig. 9, FRC is depicted as a standoff between a tug-of-war between two rival individuals, lung and chest wall recoil. In emphysema, the elastic recoil of the lungs is reduced, because lung compliance is increased. The chest wall elastic recoil is thus proportionally greater, and the equilibrium point now occurs at a higher lung volume. Thus, in patients with emphysema, FRC is increased. Just the opposite occurs in pulmonary fibrosis, a disease where the lung becomes stiff and its elastic recoil is increased. The equilibrium standoff position has now been altered, because the increased elastic recoil of the lung results in the equilibrium position being at a smaller volume. Thus a hallmark of pulmonary fibrosis is a reduced FRC.

A similar approach can be used to describe the differences in TLC observed in these patients. If TLC represents the static balance between the combined tendency of the lungs and chest wall to recoil inward from TLC and the resisting maximal inspiratory effort required to keep the lungs at this volume, then changes in lung compliance and elastic recoil should result in changes in TLC. Thus TLC is reduced in the patient with pulmonary fibrosis because of a reduced lung compliance and may be increased in the patient with emphysema because of an increased lung compliance. Thus an abnormally high TLC and FRC lead the clinician to consider the possibility of emphysema, whereas reductions in these values suggest the presence of pulmonary fibrosis.

Thus far I have indicated that TLC, FRC, and RV can be considered to represent lung volumes at which static balances have been attained between opposing respiratory mechanical forces. It is important to note,

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4 Contraction of antagonist respiratory muscles may also play a role in limiting RV (1).

5 It is possible that the elevated FRC in some patients with emphysema may also have a dynamic component (6). In this regard, expiratory flow rates may be reduced by airway narrowing to such an extent that expiration could be prematurely terminated by the next inspiration, thus preventing the respiratory system from reaching its true resting position.

6 It is likely that alterations in the pressure-volume curve of the chest wall may also play a role in allowing TLC to increase to high volumes in some patients with emphysema. In this regard, Sharp et al. (10) observed that the thorax remained very compliant at the very large lung volumes observed in patients with emphysema that they studied.
however, that under some conditions, dynamic factors may prevent these static balances from being achieved. For example, in older individuals with no lung pathology, expiration at low lung volumes is slower than normal due to an increased airway resistance, and the individual terminates expiration and begins to inspire before the static balance that determines RV in younger subjects is reached (5). Additionally, as indicated in footnote 5, FRC may be determined dynamically in some patients with emphysema. Finally, in some diseases, such as asthma, airway closure occurs and air trapping contributes to an increased RV (6). It is important to emphasize, however, that although these lung volumes and capacities may not always be determined by the static balances discussed above, an understanding of these basic interactions provides the initial critical first step in helping the student to understand more complicated changes that may occur during aging or the development of disease.

ROLE THAT NONLINEAR LUNG COMPLIANCE PLAYS IN DETERMINING THE VERTICAL DISTRIBUTION OF VENTILATION IN THE LUNG

Due to the weight of the lung, the hydrostatic pressure is greater at the base of the lung compared with the apex. As a result, pleural pressure is more positive (less negative) at the base of the lung. There is no vertical gradient of alveolar pressure, however. Because transpulmonary pressure is the difference between alveolar and pleural pressure, the vertical gradient of pleural pressure makes the transpulmonary pressure of alveoli at the base of the lung less than that of those located at the apex. Because the pressure-volume curve defines the relationship between transpulmonary pressure and volume, this must mean that alveoli at the base of the lung are less inflated than those at the apex. For example, Fig. 10, depicting the pressure-volume curve for the lung, shows the location on the curve of alveoli located at the apex (Fig. 10A) and those located at the base of the lung (Fig. 10B). Thus, at FRC, alveoli at the apex of the lung may be ~70% inflated, whereas those at the base may be only ~30% inflated.

These initial volumes would be of no consequence if the pressure-volume curve of the lung was linear. If so, the increase in volume in all regions of the lung would be identical, because a given change in pleural pressure caused by the inspiratory effort would result in the same incremental increase in volume in all alveoli. This would occur, because the relationship between pressure and volume would be identical for all lung regions. In reality, the pressure-volume curve is not linear. For alveoli located on the steep section of the curve (Fig. 10B), a given change in pressure will result in a relatively large increase in volume. For alveoli located at the apex, where the slope of the curve is flatter (Fig. 10A), the same change in pressure will result in a proportionally smaller increase in volume. Thus, when we inhale from FRC, more of the air goes to alveoli located at the base of the lungs.

A POSSIBLE CONSEQUENCE OF THE NONLINEAR PRESSURE-VOLUME CURVE FOR ARTERIAL OXYGENATION IN UNILATERAL LUNG DISEASE

Remolina et al. (9) measured arterial P O2 in nine hospitalized patients with unilateral lung disease. These included cases of bacterial and aspiration pneumonia and bronchogenic and metastatic carcinoma. Large differences in arterial P O2 were observed in these patients when they were lying on their right or left sides. When the sick lung was dependent, severe reductions in arterial P O2 were observed that could be reversed if the patient rolled over to place the healthy lung in the dependent position (Fig. 11). These positional differences disappeared after recovery. The most likely explanation for this behavior is the development of regions of ventilation-perfusion heterogeneity when the sick lung was dependent. In this regard,
both blood flow and ventilation are normally higher at the bottom of the lung. When the sick lung was dependent, its blood flow was probably increased, but ventilation may not have been able to increase to maintain a normal ventilation-perfusion ratio. Placing the good lung down thus probably allowed ventilation to increase to more precisely match the larger blood flow.

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