Pressure-Volume Curves of the Respiratory System

R Scott Harris MD

The quasi-static pressure-volume (P-V) curve of the respiratory system describes the mechanical behavior of the lungs and chest wall during inflation and deflation. To eliminate resistive and convective acceleration effects, the measurement of volume and pressure must be performed during short periods of apnea or during very slow flow. There are 3 main techniques for acquiring quasi-static P-V curves: the supersyringe method, the constant flow method, and the multiple-occlusion (or ventilator) method. For the information to be interpreted correctly, one must understand the interaction between the lungs and the chest wall, the effects of the supine position, and the meaning of hysteresis. The P-V curve has been studied in many disease states, but it has been applied most extensively to patients with acute respiratory distress syndrome, in hopes that it might allow clinicians to customize ventilator settings according to a patient's individual respiratory mechanics and thus protect the patient from ventilator-induced lung injury. However, lack of standardization of the procedure used to acquire P-V curves, difficulties in measuring absolute lung volume, lack of knowledge regarding how to use the information, and a paucity of data showing a benefit in morbidity and mortality with the use of P-V curves have tempered early enthusiasm regarding the clinical usefulness of the quasi-static P-V curve. *Key words: lung mechanics, compliance, lung recruitment, pressure-volume curve, mechanical ventilation; acute respiratory distress syndrome; waveforms.* [Respir Care 2005;50(1):78–98.

Introduction

In spontaneously breathing subjects the diaphragm and chest wall together form a mechanical pump that moves air in and out of the lungs to exchange oxygen and carbon dioxide to and from the blood. When the respiratory system fails and mechanical ventilation is necessary, the respiratory system's mechanical behavior can change in characteristic ways that can be assessed by the mechanical ventilator or with simple equipment available in respiratory care departments. The quasi-static pressure-volume (P-V) relationship is one aspect of mechanical behavior that has been used to gain information about the way the lungs deform during breathing in health and disease.

It has long been hoped that with mechanically ventilated patients the P-V curve would allow the clinician to diagnose lung disease, customize ventilator settings, follow the course of disease, and make prognoses. Surprisingly, despite over a half-century of research on P-V curves, we still have a limited understanding of the meaning of the P-V relationship. So, though some of those goals have been realized, much research still needs to be done before P-V curves can be widely used clinically.

Equation of Motion

The equation of motion describes the pressure change at the airway opening during breathing. The equation assumes one degree of freedom, meaning that the lung expands equally in all directions (isotropic expansion). It is written as:

$$P_{AO} = \frac{V}{C} + \dot{V}R + \ddot{V}I - P_{mus} \quad (1)$$

in which P_{AO} is the pressure at the airway opening (mouth or endotracheal tube), P_{mus} is the pressure generated by the respiratory muscles, V is lung volume, C is respiratory-system compliance, $\dot{V}$ is gas flow, R is airway resistance, $\ddot{V}$ is convective gas acceleration, and I is impedance. What is evident from that equation is that a static P-V curve eliminates the resistive and impedance effects on pressure, such that only the compliance is assessed. Thus, static P-V curves are also called compliance curves. Also evident is the respiratory muscle contribution to pressure. If a subject is not sufficiently sedated (or paralyzed), the measured P-V curve may not be representative of the lung compliance properties alone and may include effects of the respiratory muscles.

Static Versus Dynamic Pressure-Volume Curves

Whenever one measures pressure when airflow has stopped, there is always a continual change in pressure—lowering in an exponential fashion. Because we must allow the subject to breathe, the measurement must be interrupted after a few seconds. Therefore, the system never reaches truly static conditions, so we obtain what has been called a "quasi-static" P-V curve. This means that the resultant curve depends somewhat on how long the clinician waits for static conditions. It is possible to use a slow flow rate to approximate static conditions, but the flow rate must be < 9 L/min to largely eliminate the pressure change from resistive elements of the respiratory system.[1,2]

The confusing term "dynamic compliance" has been used to mean various things, but it originally referred to compliance calculated during a tidal breath at the points of zero flow on the dynamic P-V loop. Some investigators have made arguments that perhaps P-V measurements should be made during breathing rather than in quasi-static conditions, since that might be more representative of the mechanical behavior that the lung experiences during breathing. Ranieri et al[3] used the shape of the inspiratory pressure-time curve during constant flow to calculate a "stress index," which, they argue, can detect overdistention or recruitment. Lichtwarck-Aschoff et al[4] advocate using the "slice method" to estimate static compliance from a dynamic P-V loop, using a linear resistance-compliance model. Karason et al[5] developed a method to calculate the alveolar pressure during dynamic/therapeutic conditions, which they call the "dynostatic pressure." They argue that this method has the advantage that it provides a breath-by-breath analysis of the P-V relationship, without requiring ventilator disconnects.

Adams et al[6] studied the effect of increasing constant flows on the shape of the inspiratory P-V loop. They measured a quasi-static P-V curve, using the supersyringe technique, and compared it with inspiratory P-V curves measured with 10, 30, and 50 L/min constant inspiratory flows in dogs with oleic-acid-induced lung injury. They found that the curve consistently shifted to the right at all inspiratory flows tested (Fig. 1). They also found that during the measurement of the dynamic P-V curves there was increased volume at the beginning of the dynamic inspirations while on positive end-expiratory pressure (PEEP), which, they concluded, was recruitment from tidal venti-

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R Scott Harris MD presented a version of this article at the 34th RESPIRATORY CARE Journal Conference, Applied Respiratory Physiology: Use of Ventilator Waveforms and Mechanics in the Management of Critically Ill Patients, held April 16–19, 2004, in Cancún, Mexico.

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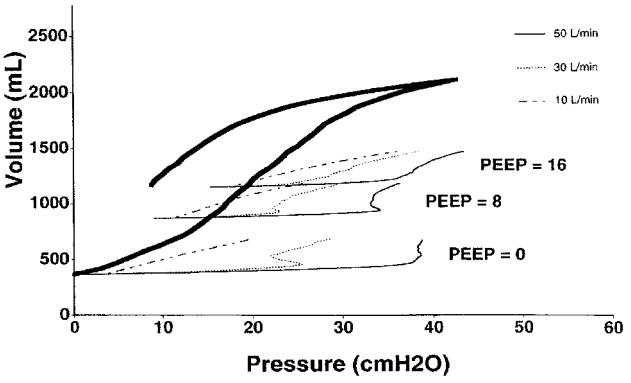

Fig. 1. Comparison of static and dynamic pressure-volume (P-V) loops. The thick solid line is the quasi-static P-V loop acquired with the supersyringe method. The thin solid, dotted, and dashed lines are dynamic P-V loops acquired with inspiratory flow rates of 50, 30, and 10 L/min, respectively. P-V loops were collected at 3 different PEEP levels. Note the rightward shift of the P-V curve with increasing flow rate, which represents increased pressure generated from airway resistance. Note also the increased volume at each PEEP level at the beginning of the curve, representing tidal recruitment. (From Reference 6, with permission.)

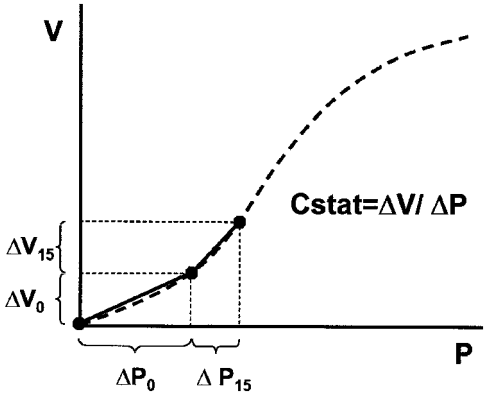

Fig. 2. Effect of PEEP on static compliance (C_{stat}). In this example, if C_{stat} is measured with a PEEP of zero ($\Delta V0/\Delta P0$) on one day and then a PEEP of 15 cm H_2O ($\Delta V15/\Delta P15$) the next day, one could erroneously interpret that as an improvement in the patient's lung disease (increased compliance). However, this change in compliance is simply a change in the position of the volume excursion on the same pressure-volume curve.

lation independent of PEEP. From that study it is clear that measuring P-V curves with tidal ventilation at high flow ($\geq$ 10 L/min) still leaves a large pressure change from resistive components, and, furthermore, that there is a PEEP-independent volume-recruitment effect. This last point will be discussed below, in the section that describes the multiple-occlusion method for measuring P-V curves.

Static Compliance, Chord Compliance, Specific Compliance, and the Pressure-Volume Curve

It is important when discussing P-V curves to understand the difference between the terms *static compliance*, *chord compliance*, and *specific compliance*. Static compliance usually implies compliance calculated from 2 volume points during quasi-static conditions. This is usually done by performing an end-inspiratory hold on the ventilator and measuring the pressure after a few seconds. The tidal volume (V_T) is divided by the pressure change. This compliance measurement assumes a linear relationship between volume and pressure, but the P-V relationship is not linear, so care must be taken in interpreting changes in static compliance measured that way. For example, an increase in static compliance after a PEEP increase on a ventilator could mean simply a shift in location on the P-V curve, rather than a change in intrinsic properties of the lung (Fig. 2). In plane geometry, a "chord" is a line segment that connects 2 points on a curve. Therefore, static compliance calculated this way is actually chord compliance. It would be more precise to say "static chord compliance." Specific compliance is compliance that is normalized by a lung volume, usually total lung capacity (TLC) or functional residual capacity (FRC). Thus, given the same volume excursion, a child will have lower chord compliance than an adult, but they will have the same specific compliance. Specific compliance is often used to better assess the intrinsic elastic properties of the lung, by eliminating the effects of variations in lung size.

Measurement

There are 3 main techniques for constructing a quasi-static P-V curve: the supersyringe method, the constant-flow method, and the multiple-occlusion method.

Supersyringe Method

The supersyringe method consists of connecting a supersyringe to the end of the endotracheal tube after allowing the respiratory system to reach relaxation lung volume, and then measuring airway pressure and insufflated volume. The syringe's plunger is moved in regular steps (usually about 100 mL each), with 2–3-second pauses to allow for quasi-static conditions (Fig. 3), and then a pressure-volume plot is drawn (Fig. 4). Usually, when airway pressure reaches 40 cm H_2O, inflation is stopped and deflation is performed in the same way. This method is simple and allows construction of both inflation and deflation curves. It also has disadvantages: it requires additional equipment, disconnecting the patient from the ventilator, and the patient has to be sedated and paralyzed. Changes associated with continuing gas exchange, changes in gas temperature

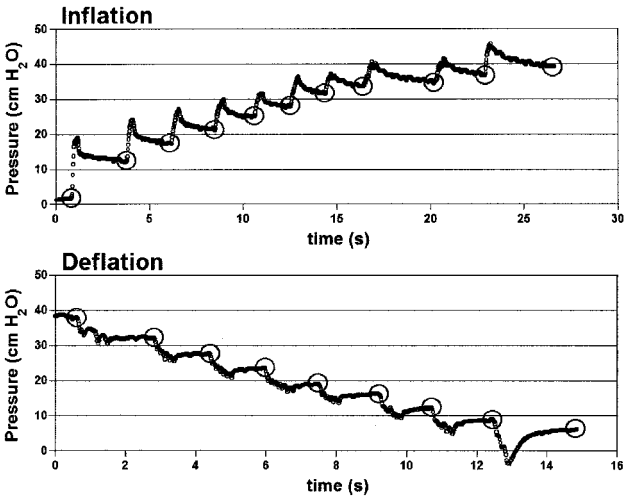

Fig. 3. Pressure-time plot acquired with the supersyringe method. The top plot is inflation and the bottom plot is deflation. Note the exponential decay of pressure that continues even before the next increment of volume is insufflated with the syringe. The time constants for the decay in pressure become longer the higher the pressure during inflation, but the opposite is true during deflation. The large open circles represent the pressure values that would be chosen to construct a quasi-static pressure-volume curve.

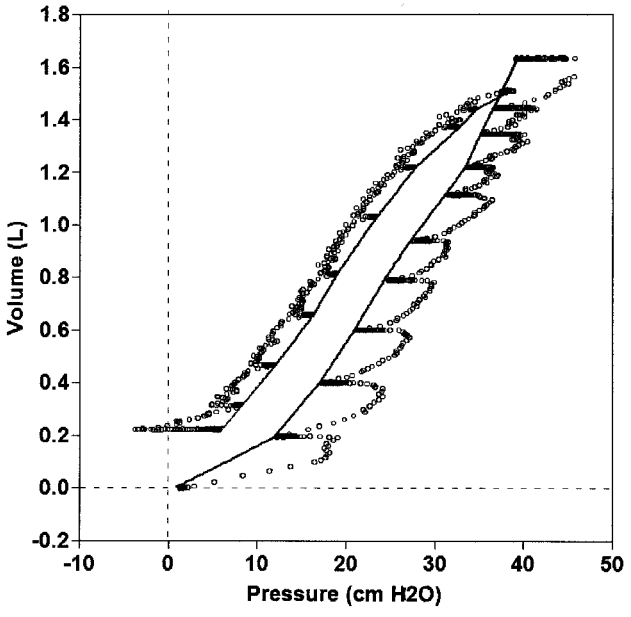

Fig. 4. Pressure-volume plot acquired with the supersyringe method. The open circles are the data points plotted continuously during the maneuver. The solid lines show the quasi-static points connected to form a smooth P-V curve. The inflation and deflation points are not connected, because they were performed separately in this example. Note the large changes in pressure with each volume step, which are due to airway resistance, and then the slow decrease in pressure, probably due to stress relaxation.

and humidity, and compression and decompression of the gas are not taken into account.

Constant-Flow Method

The constant-flow method is simple to perform and it can be done with some ventilators, so it can preserve some changes in volume history that are lost with the supersyringe method (because the patient is disconnected from the ventilator). However, the constant-flow method suffers from the effects of oxygen consumption, which increases hysteresis. One cannot easily perform a deflation curve unless a special system is employed to limit expiratory flow to a constant value. Lu et al[1] found that either 3 L/min or 9 L/min of constant flow gave similar slopes and lower inflection points, but the curve was shifted slightly to the right. Servillo et al[2] showed that slightly higher flows (15 L/min) substantially right-shifted the curve and its derived parameters. It recently became clear that higher flow causes a more substantial shift in the curve at higher volumes, presumably secondary to viscoelastic properties of the lung. Even at a constant flow as slow as 1.7 L/min,[7] there can be slight differences between P-V curves measured with the constant-flow method versus with the supersyringe method. There is a slight shift to the right on inflation and a shift to the left on deflation (Fig. 5).

Multiple-Occlusion Method

The multiple-occlusion method (or "ventilator method") has unique advantages. It is obtained by periodically interrupting tidal breathing at different lung volumes to obtain each P-V point. Normal tidal breathing then resumes for about 4 breaths and another, different point is obtained. Both inflation and deflation curves can be acquired, no ventilator-disconnection is needed, and there is no correction for oxygen consumption, because the measurements are interspersed with normal ventilation. The multiple-occlusion method still requires sedation and/or paralysis to prevent spontaneous breaths during the measurements. This measurement assumes a constant "relaxation lung volume," since absolute volume is not measured and the measurements are done intermittently, over many breaths. This method includes what some investigators call "recruited lung volume," because it takes into account time-dependent recruitment that occurs with normal ventilation during PEEP. It differs from the supersyringe method in having less hysteresis, less curvature at low volumes, and in showing increased pressure at high volumes. Those changes are probably due to reduced effects of oxygen consumption and recruitment, and to more effects of viscoelastic behavior at high lung volumes during the measurement.[8]

Physiologic Meaning

The shape of the quasi-static respiratory-system P-V curve in an upright, awake, and relaxed subject is sigmoi-

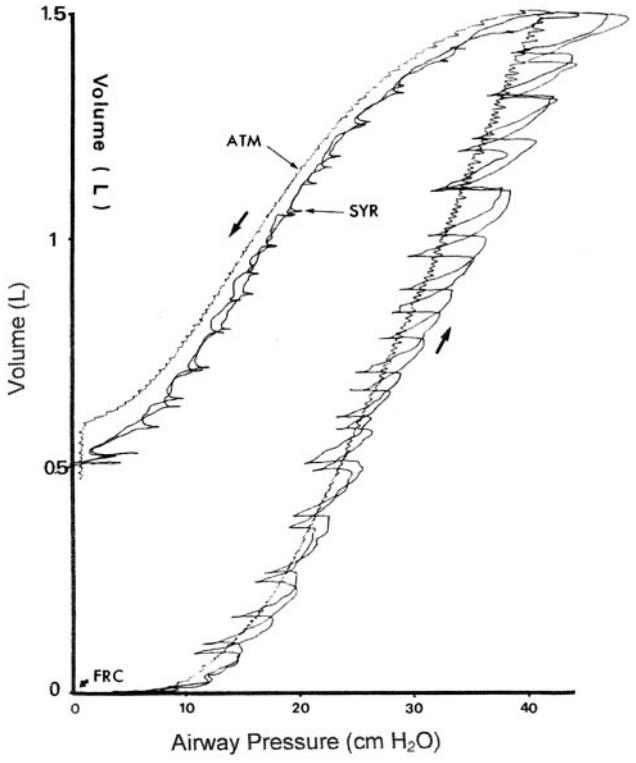

Fig. 5. Pressure-volume curves acquired using the slow constant-flow method (ATM) and the supersyringe method (SYR). The slow constant-flow curve was generated using a custom-built device designed to deliver a constant flow of 1.7 L/min for both inflation and deflation. Note that although the curves appear similar, the slow constant-flow curve is shifted slightly to the right on inflation and slightly to the left on deflation. (From Reference 7, with permission.)

dal, with upward concavity at low inflation pressure and downward concavity at higher inflation pressure (Fig. 6).[9] That sigmoidal shape reflects the balance of forces between the chest wall (diaphragm and rib cage) and the lung parenchyma. The lung volume where the outward expansive force of the chest wall balances the inward retractile force of the lung parenchyma is the FRC, which is also where alveolar pressure equals atmospheric pressure. The chest wall contributes most to the curvature below FRC, and the lung contributes most to the curvature above FRC.

Lung Versus Chest Wall

The measurement of the total-respiratory-system P-V curve includes both the chest wall (abdomen and rib cage) and the lungs. When compliance is altered in disease states, it is important to know the relative contributions of both to the total-respiratory-system P-V curve. For 2 elements in series the reciprocal of the total compliance is the sum of the reciprocals of the individual compliances:

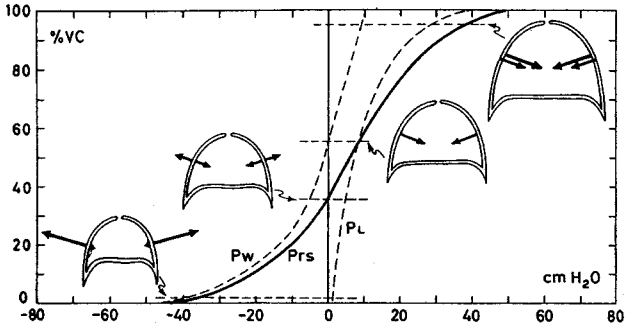

Fig. 6. The normal sigmoid pressure-volume curve in an upright, awake, and relaxed subject. The pressure-volume curve of the respiratory system (P_{RS}) is the sum of the pressures generated by the chest wall (P_W) and lungs (P_L) (dotted lines). Functional residual capacity (FRC) occurs where the sum of the 2 pressures are equal and opposite. Note that the chest wall contributes to much of the curvature below FRC, whereas the lung contributes most to the curvature above FRC. %VC = percent of vital capacity. (From Reference 9, with permission.)

$$\frac{1}{C_{RS}} = \frac{1}{C_L} + \frac{1}{C_{CW}} \quad (2)$$

in which C_{RS} is the compliance of the total respiratory system, C_L is the compliance of the lung, and C_{CW} is the compliance of the chest wall. For normal subjects during tidal breathing, it is assumed that the compliance of the total respiratory system, lung, and chest wall are linear, and we calculate a single compliance for each (chord compliance). Normal values are 100 mL/cm H_2O for C_{RS}, 200 mL/cm H_2O for C_L, and 200 mL/cm H_2O for C_{CW}.

The abdomen is less compliant than the rib cage for large volume changes, but in the range of normal, quiet breathing it is nearly as compliant as the rib cage.[10] It is slightly more compliant in the supine position. During spontaneous breathing in the conscious state, the rib-cage volume-displacement corresponds to 40% of the V_T, but this rises to 72% during anesthesia and artificial ventilation.[11] The relative contributions of the rib cage and abdomen displacements are not influenced by a change in V_T. Chord compliance of the chest wall becomes higher with a larger V_T, indicating a P-V relationship that is concave upwards.[11] In mechanically ventilated patients with respiratory failure secondary to chronic obstructive pulmonary disease (COPD) and pulmonary edema, it was found that abnormalities in total-respiratory-system mechanics essentially reflected alterations in lung mechanics rather than in chest wall mechanics.[12] This has been an argument for using total-respiratory-system mechanics as a surrogate for lung mechanics, obviating the use of an esophageal balloon and thus simplifying the acquisition of data. However, in certain disease states the chest wall can become an important contributor to changes in the total-respiratory-system P-V curve (discussed below).

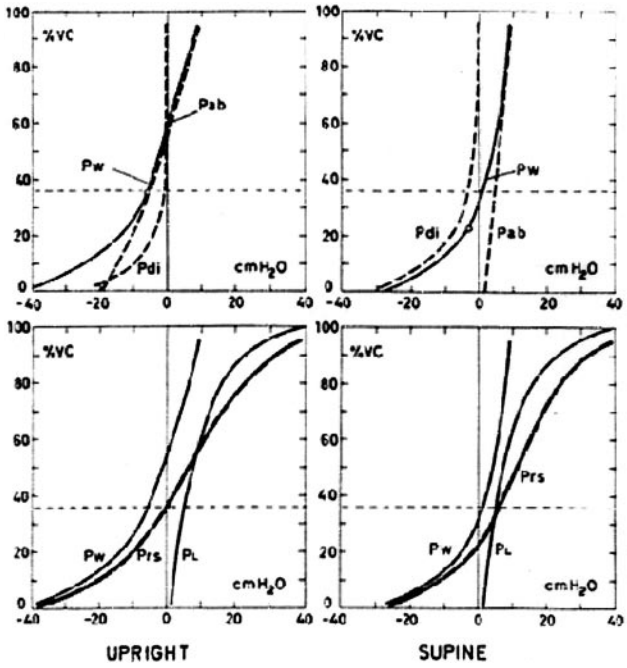

Fig. 7. Changes in the chest wall and abdomen pressure-volume curves from upright to supine posture. Note that the major effect of the supine position is a rightward shift of the abdomen compliance curve. This shifts the respiratory system curve to the right, lowering functional residual capacity, and slightly rotating the curve counterclockwise, increasing compliance. The abdomen generally has a high compliance, getting somewhat stiffer at higher lung volumes, as it is compressed slightly in both positions. However, the zero point of the abdominal compliance curve, which is below the diaphragm in the upright posture, moves to the level of the abdominal surface in the supine position. %VC = percent of vital capacity. P_{di} = diaphragmatic pressure. P_{ab} = abdominal pressure. P_w = chest wall pressure. P_L = lung pressure. (From Reference 9, with permission.)

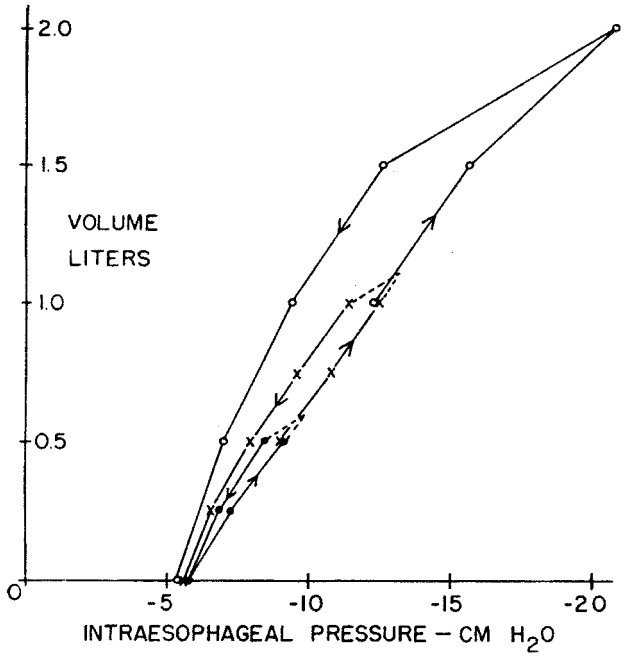

Fig. 8. The effect of tidal volume on hysteresis. Hysteresis is minimal at small tidal volumes, but becomes larger as the tidal volume increases. (From Reference 13, with permission.)

Supine Posture

In the supine posture the chest-wall curve moves to the right and rotates counterclockwise, increasing the compliance of the total respiratory system in the range above FRC, but dropping FRC to about half of the upright-posture value (Fig. 7).[9] This is the result of 2 effects: an expiratory effect of the abdomen and a slight inspiratory effect of the chest wall. The abdominal compliance curve shifts to the right, resulting in a similar shift of the respiratory-system curve to the right, lowering FRC, and slightly rotating the curve counterclockwise, increasing compliance. These effects are amplified by anesthesia and obesity (discussed below). The normal abdomen has a high compliance, getting somewhat stiffer at higher lung volumes, because the abdomen is compressed slightly. What changes is the zero point, which is below the diaphragm in the upright posture, but at the level of the abdominal surface in the supine position. In certain conditions, such as ascites or abdominal hemorrhage, intra-abdominal hypertension can markedly decrease abdominal compliance.

Hysteresis

An important phenomenon that is readily seen in P-V curves is hysteresis. Hysteresis refers to unrecoverable energy, or delayed recovery of energy, that is applied to a system. That is, the lung does not act as a perfect elastic system (in which any energy put in is immediately returned). Mead et al[13] demonstrated that hysteresis is minimal with small volume changes, but that it increases as the volume excursion increases (Fig. 8). In 1929, Von Neergaard[14] discovered that the main determinant of hysteresis is air-liquid surface forces in alveoli. Radford expanded on Von Neergaard's observations by performing both inflation and deflation P-V loops on excised cat lungs, either submerged in saline solution during saline injection or suspended in air during air inflation.[15] There was a marked difference in the pressure change at the same volume, both for inflation and deflation (Fig. 9). Although surface forces predominate in causing hysteresis with large tidal excursions, with small volume excursions the hysteresis does not seem to be related to surface forces and may be an intrinsic property of the tissues (Fig. 10).[16] The difference in pressure with large volume excursions was due to surface forces and could explain all of the hysteresis in lung inflation with gas. Those results were all with excised lungs.

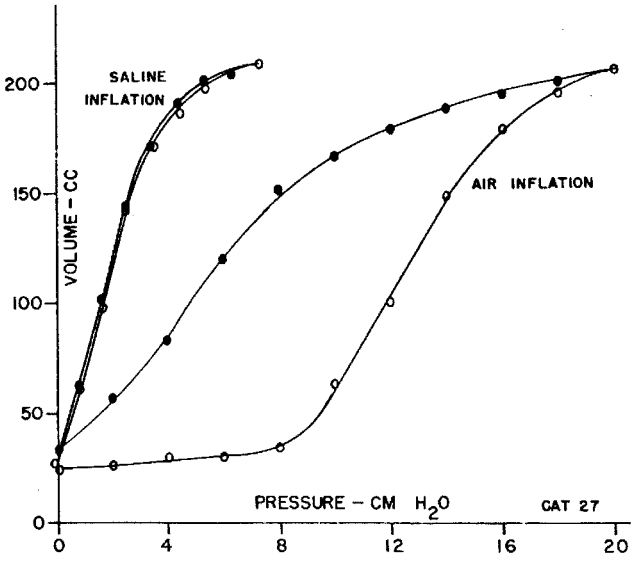

Fig. 9. Pressure-volume loops obtained from excised cat lungs submerged in saline. Note the marked pressure increase at all lung volumes when the lungs were filled with air rather than saline, both on inflation and deflation. Also, the saline inflation and deflation limbs are nearly identical, whereas with air inflation and deflation there is a large amount of hysteresis, due to surface forces at the air-liquid interface. (From Reference 15, with permission.)

What is the effect of the chest wall on hysteresis? The chest wall limits air-space collapse. Excised lungs, when inflated and deflated at lung volumes similar to those in an intact animal, show similar hysteresis. However, if the lungs are allowed to collapse by removing them from the chest well, hysteresis markedly increases (Fig. 11).[15] Therefore, important for hysteresis are the time taken for volume change, the initial lung volume, volume excursion, and whether the lungs have been previously collapsed or fully inflated in the minutes before the measurement. The resultant P-V curves are envelopes that define the limits of the lungs' elastic behavior for the defined volume history. Hysteresis, then, results from 4 processes: recruitment/derecruitment; surfactant; stress relaxation; and gas absorption during the measurement of P-V curves.

Alterations in Disease States

Obesity

Pelosi et al[17] measured FRC with helium dilution in 24 subjects undergoing general anesthesia. They studied 8 subjects who had body mass index (BMI) $\leq$ 25 kg/m^2, 8 whose BMIs were between 25 and 40 kg/m^2, and 8 morbidly obese patients whose BMIs were $\geq$ 40 kg/m^2. The most obvious and reproducible change with obesity was a drop in FRC that worsened with increasing BMI (Fig. 12), which is due to the marked expiratory action of the enlarged abdomen on the lungs, despite the slight inspiratory action of the chest wall going to the supine position. Interestingly, the chord compliance of the total respiratory system decreases exponentially as BMI increases, and it is mostly due to a drop in lung compliance, not the chest wall (Fig. 13). The loss of lung compliance is probably due to loss of air spaces secondary to atelectasis. Pelosi et al[18] also found that the chest wall compliance was reduced to a similar degree as the lung compliance, which agrees with previous research with awake obese subjects.[19] The cause of the reduced chest wall compliance is thought to be structural changes in the chest wall and rib cage (increased adiposity, kyphosis, lumbar lordosis) and a decreased thoracic volume bringing the chest wall P-V curve down to its less compliant region. The decrease in lung compliance can be improved by increasing PEEP,[20] reverse Trendelenburg position,[21] or laparotomy.[22] The total-respiratory-system P-V curve in sedated, paralyzed, obese subjects becomes curvilinear above FRC (Fig. 14), with upward concavity.[23]

Acute Respiratory Distress Syndrome

Early Use of the Pressure-Volume Curve. The P-V curve has been studied and most extensively applied with patients with acute respiratory distress syndrome (ARDS). From the first description of ARDS in 1967,[24] investigators noticed that the static compliance was reduced. Measuring chord compliance was suggested as a way of diagnosing different forms of respiratory distress.[25] A few years later the supersyringe technique was introduced and striking differences from normal were seen in ARDS. Matamis et al[26] found a nearly reproducible pattern of changes in the P-V curve according to the ARDS stage and chest radiograph findings (Fig. 15). These early findings led to much research on the P-V curve in ARDS, and attempts to define certain features of the curve and correlate them with other physiologic measurements, such as dead space, shunt, and oxygen delivery.

In ARDS the P-V curve appears sigmoidal in the volume range in which it is acquired (between end-expiratory lung volume and TLC). This is the same shape as a P-V curve from a healthy subject, except that in healthy lungs in the volume range between FRC and TLC the curve is concave down and relatively linear until high pressure is reached. In addition, the P-V curve in ARDS has a lower volume excursion to TLC and the entire curve is shifted down on the volume axis. That shift, however, is not apparent when performing a P-V curve, since the volume scale is usually referenced to the end-expiratory lung volume, not to absolute lung volume. Fig. 16 illustrates these concepts and some of the most common terms used to describe the P-V curve.

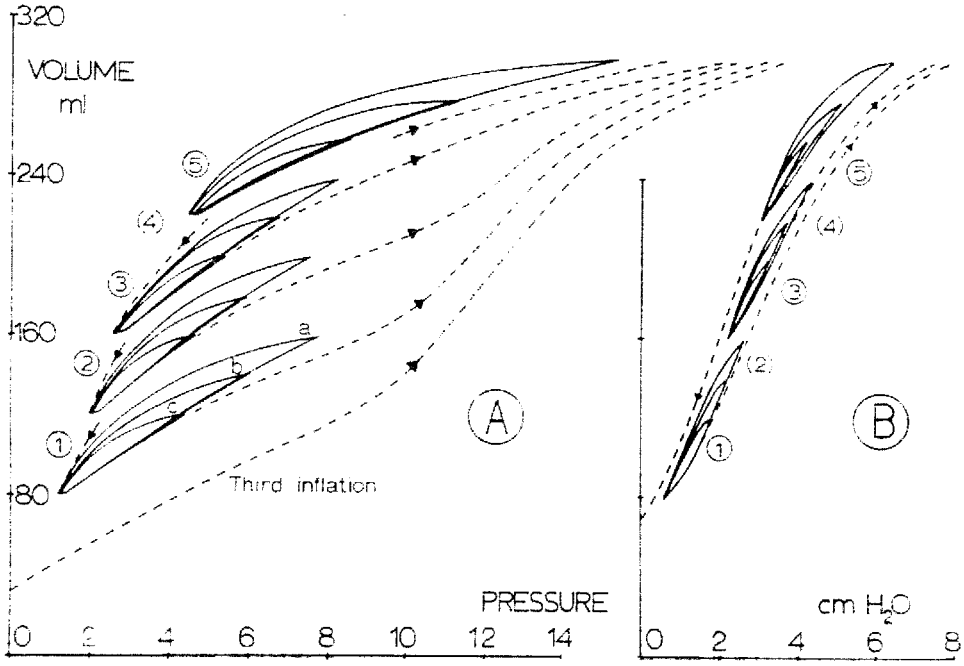

Fig. 10. The effect of volume excursion and surface forces on hysteresis in a cat lung. In panel A each set of pressure-volume (P-V) loops was done after inflation to total lung capacity. P-V loops were obtained at 80, 60, and 40-mL tidal volumes. Hysteresis increases with increasing tidal volume and also with increasing pressure and volume amplitude. In panel B the P-V loops were obtained on an excised lung filled with, and submerged in, saline. The behavior is similar, but with increased compliance. Note that the large excursions with air show the largest hysteresis, whereas hysteresis is absent with the saline-filled lungs. Therefore, that component of hysteresis is due to surface forces. (From Reference 16, with permission.)

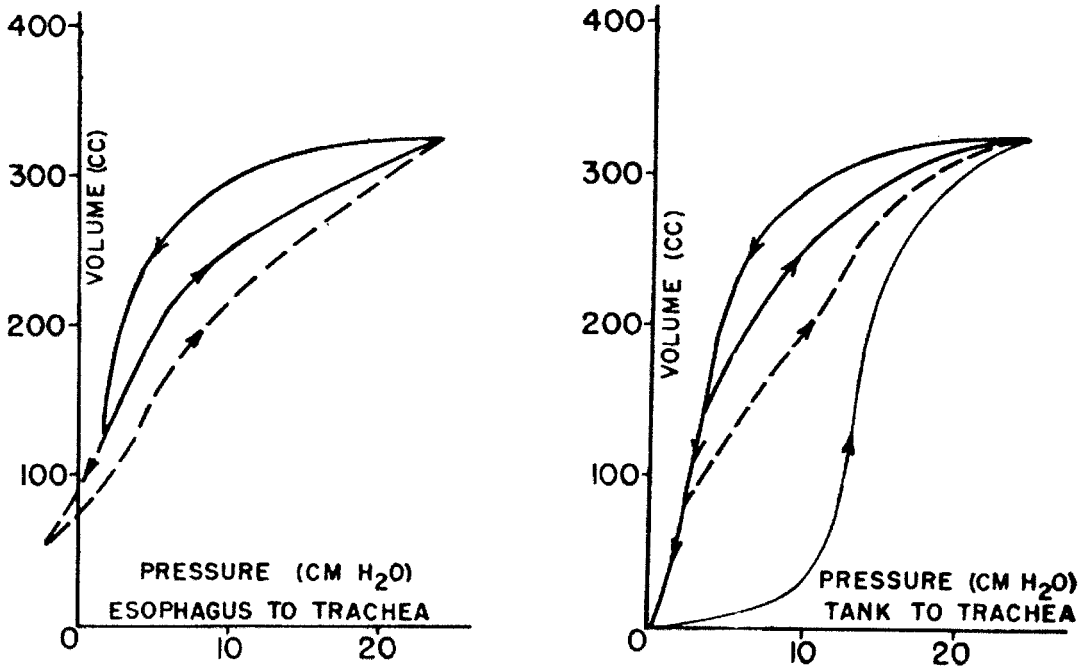

Fig. 11. The effect of the chest wall on hysteresis. The left panel shows the P-V curve of an anesthetized cat in an "iron-lung" (negative-pressure) ventilator. Note the increase in hysteresis when the lung is made to inflate from below functional residual capacity (dotted line). When the lungs are removed from the animal, the curves can essentially be reproduced if the starting volume is near the intact animal's functional residual capacity. However, if the lungs are allowed to collapse, there is a marked increase in hysteresis. (From Reference 15, with permission.)

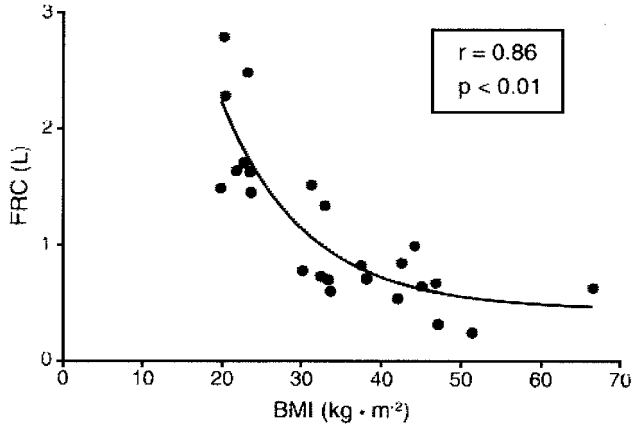

Fig. 12. Functional residual capacity (FRC) versus body mass index (BMI). In the supine position during general anesthesia, FRC decreases exponentially as BMI increases. (From Reference 17, with permission.)

The Lower Inflection Point. Suter et al[27] in 1975 showed that the end-expiratory pressure that resulted in the maximum oxygen transport and the lowest dead-space fraction resulted in the greatest total static compliance in 15 normovolemic patients in acute respiratory failure. Suter et al wrote, "The present data suggest that an optimal situation is achieved in acute pulmonary failure when tidal ventilation takes place on the steepest part of the patient's pressure-volume curve—that is, when the highest compliance is achieved."[27] This is where investigators and clinicians first started to get the idea that the majority of alveoli are closed below the "knee" of the P-V curve.

Gattinoni et al[28] coined the term "P_{flex}," which they defined as the pressure at the intersection of 2 lines: a low-compliance region at low lung volume and a higher-compliance region at higher lung volume (Fig. 17). Using computed tomography (CT), they found that (1) compliance correlated only with normally aerated tissue and (2) specific compliance was in the normal range, which led to the "baby lung" concept in ARDS. The amount of recruitment observed on CT was directly related to the ratio of the high compliance region to low compliance region (C_{inf}/C_{start}) on the P-V curve. Another way of saying this is, the more the curve was concave up, the more recruitable lung there was. Furthermore, Dall'ava-Santucci et al[29] found that increasing PEEP could eliminate curvature on the inflation limb (Fig. 18). Based on that and on the model presented by Suter et al, some intensivists began using P_{flex} to set PEEP, in order to optimize recruitment. In fact, by setting PEEP = P_{flex} + 2 cm H_2O in a pressure-limited, "open lung" ventilation strategy in ARDS, Amato et al[30] had less barotrauma, a higher weaning rate, and better survival at 28 days than a conventional ventilator strategy that did not use P-V-curve guidance.

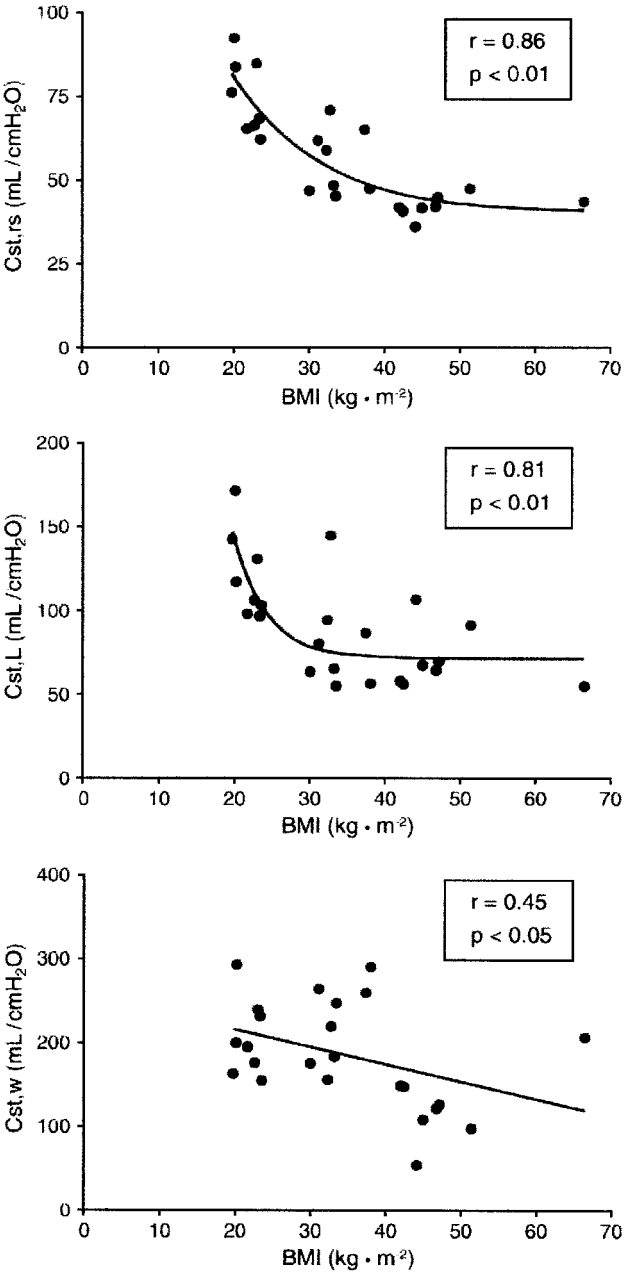

Fig. 13. Compliance of the total respiratory system (top), lung (middle), and chest wall (bottom) versus body mass index (BMI). Note the exponential decrease in total-respiratory-system compliance, which is due mainly to a similar decrease in lung compliance, not chest wall compliance (Cst). (From Reference 17, with permission.)

However, there are few theoretical or experimental reasons to justify the clinical use of P_{flex} to optimize alveolar recruitment. First, conceptually, PEEP is used to prevent collapse of lung units on deflation, not inflation. It would seem then that the deflation limb would be best for identifying the end-expiratory pressure to prevent collapse.[31–34] Second, there is probably a range of pressure in which

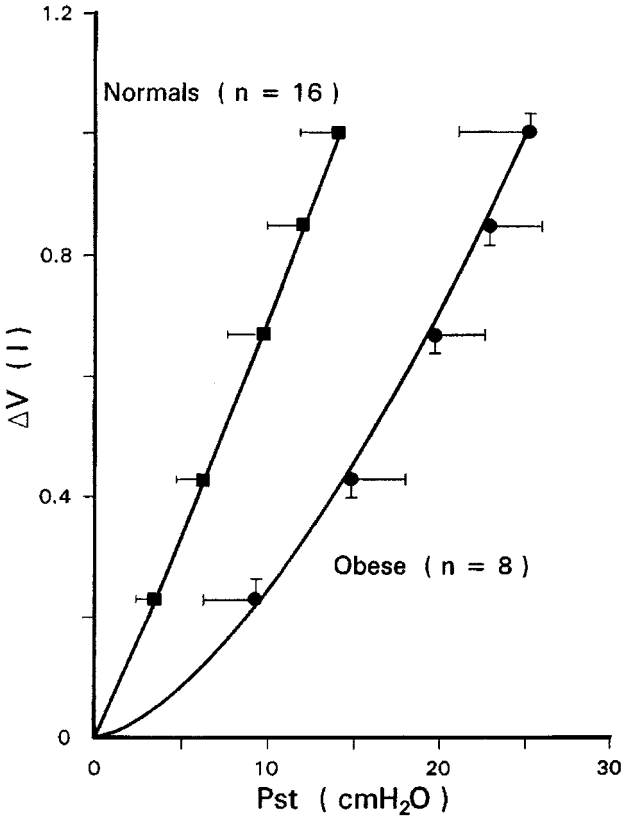

Fig. 14. The effect of obesity on the shape of the pressure-volume curve. The left curve is the average of 16 subjects of normal weight and the right curve is the average of 8 obese subjects. Note the rightward shift and appearance of a lower curvature in the obese subjects. Pst = static pressure of the total respiratory system. ΔV = change in volume. (From Reference 23, with permission.)

derecruitment takes place, rather than a single point. Holzapfel et al[32] compared the reduction in shunt fraction with features of the inflation and deflation P-V curves as PEEP was progressively increased in patients with ARDS. They found that the maximum reduction in shunt correlated best with the true inflection point (the point where concavity changes direction) of the deflation P-V curve. In addition, nitrogen washout studies with anesthetized patients showed that the deflation limb of the P-V curve can be used to estimate the pressure required to raise FRC above its closing volume.[35] Others have found, with mathematical models, that alveolar collapse may occur before the true inflection point has been reached.[36] These studies call into question the use of P_{flex} of the inflation limb to set PEEP.

Another problem with the use of P_{flex} is that there are at least 4 different definitions in the literature.[28,37–40] Many of the techniques used to obtain P_{flex} rely on graphical methods done by eye, which are subject to interobserver differences and intraobserver variability. Three studies have found substantial disagreement in obtaining P_{flex} by eye.[41–43] One study found the interobserver differences quite high, such that P_{flex} for the same set of P-V data differed by as much as 11 cm H_2O.[41] They found that the use of a curve-fitting equation, $V = a + b/(1 + e^{-(P-c)/d})$ (Fig. 19), described the P-V relationship well and could help reduce the variability in calculated parameters. Other investigators found good interobserver agreement,[44,45] perhaps related to training the observers on an exact P-V-curve analysis method.

The Upper Inflection Point. Roupie et al,[46] using the multiple-occlusion method, found that if the upper inflection point is a point that represents increased strain on alveoli, then many patients with ARDS were being subjected to increased parenchymal strain (Fig. 20). That study highlighted the possibility that V_T and pressure levels once thought to be safe might not be. The ARDS Network trial[47] of high-V_T versus low-V_T ventilation strategies, though it did not use P-V curves to set ventilator parameters, nevertheless supported this idea, because it found significantly lower mortality with a V_T of 6 mL/kg of ideal body weight than with 12 mg/kg. Interestingly, assuming the ARDS Network trial had patients similar to those in the Roupie et al study,[46] almost all patients would have had plateau pressure below their upper inflection point (see Fig. 20), so if the upper inflection point is representative of the average strain on alveoli, this measurement might be useful for minimizing lung parenchymal strain.

Pressure-Volume Curve or Recruitment Curve? Many investigators believed that the P-V curve consisted of 3 zones: one zone of low compliance, which represented the derecruited state; a zone of high compliance, which represented the isotropic expansion of open alveoli; and another zone of low compliance, which represented overdistention. Roupie et al[46] found that many patients are ventilated above their upper inflection points (upper P_{flex}). From that came the concept of ventilating between the upper and lower P_{flex} values. However, several investigators, using different techniques such as mathematical modeling,[48–51] in vivo microscopy,[52,53] and recruited volume,[54] have found that there is no precise point, such as P_{flex}, above which the lung is fully recruited and isotropically expands. All of these techniques suggest that there is continual recruitment up to the maximum pressure used to inflate the lung. Venegas et al[50] used the equation $V = a + b/(1 + e^{-(P-c)/d})$ to curve-fit a variety of P-V curves from both humans and animals with lung disease, including lung injury. Interestingly, the derivative of this equation resembles a Gaussian distribution, suggesting that the P-V curve is in fact largely the integral of a Gaussian distribution of opening and closing pressures of alveoli. That was in fact found to be the case experimentally, in both a canine oleic-acid-injury model[55] and in

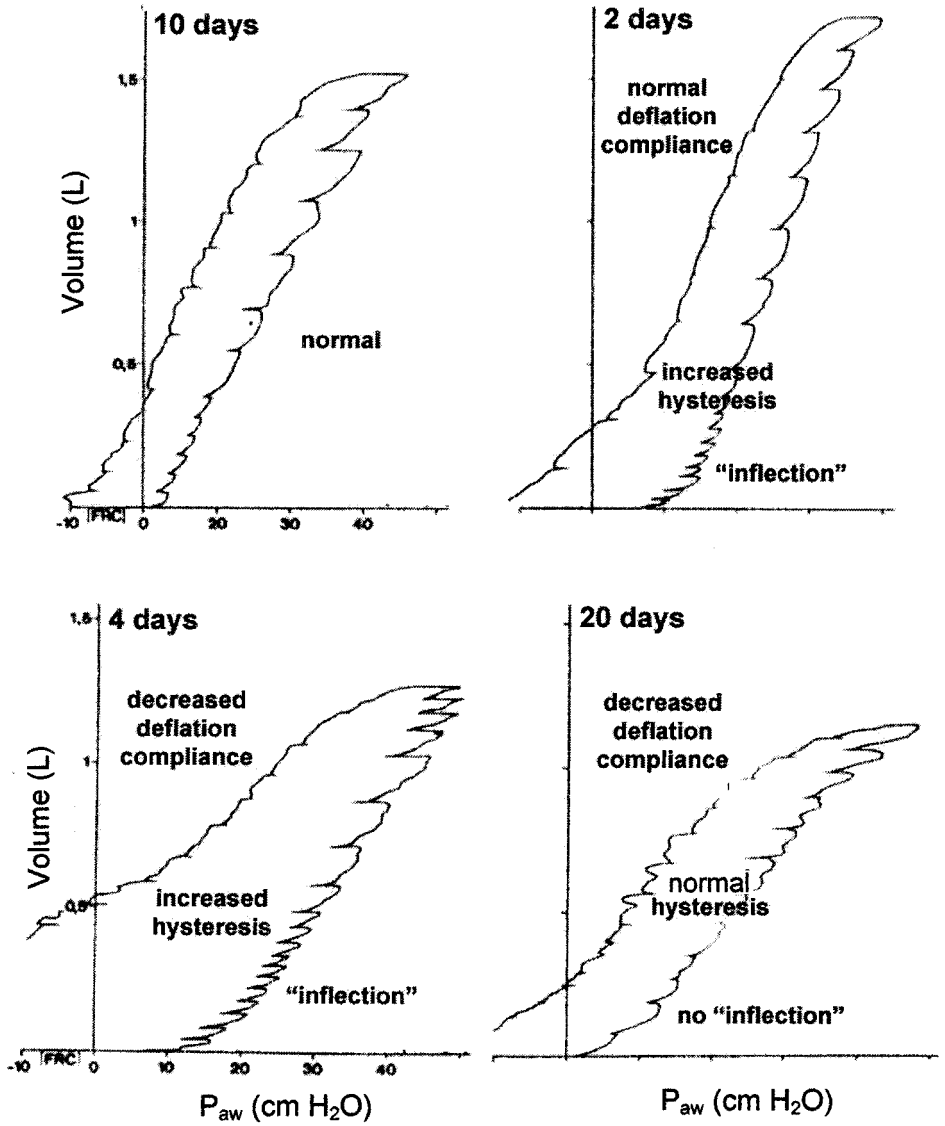

Fig. 15. Change in the pressure-volume curve over time in patients with acute respiratory distress syndrome (ARDS). The time at which each curve was measured is at the top of the volume (vertical) axis. In ARDS patients who recover (top left), the P-V curve looks normal. Early in ARDS (top right), there is normal deflation compliance, but hysteresis is increased and there is the appearance of an upward concavity ("inflection") in the inflation compliance. Later (bottom left) there is a decrease in deflation compliance, a marked increase in hysteresis, and an inflection point on the inflation curve. Finally, in late fibroproliferative ARDS (bottom right) there is a return towards a normal appearance of the curve, except for a continued low deflation compliance. P_{aw} = airway pressure. FRC = functional residual capacity. (From Reference 26, with permission.)

patients with ARDS.[56] In both those studies the mode of the Gaussian distribution of opening pressures was about 20 cm H_2O and of closing pressures about 5 cm H_2O.

The concept of the P-V curve representing recruitment and derecruitment has been illustrated by research from Bond and Froese,[57] Froese et al,[58] and Rimensberger et al.[33,34] From those data it appears that, at least in animal lung-lavage-injury models, it is better to ventilate the lungs on the deflation limb of the P-V curve to protect against lung injury. This seems to be especially true the smaller the V_T becomes, such as with high-frequency oscillation. Those data also illustrated the important concept of volume history in relation to the P-V curve. Where one is ventilating within the quasi-static P-V envelope depends on where one starts (eg, from a low lung volume or from a sustained inflation at high pressure). Although the end-expiratory pressure may be the same in both cases, the lung volume can be quite different (Fig. 21).

Although it seems reasonable to conclude from these data that the P-V curve largely represents opening and closing of airways or alveoli, some caution must be advised. There are still conflicting data on how alveoli de-

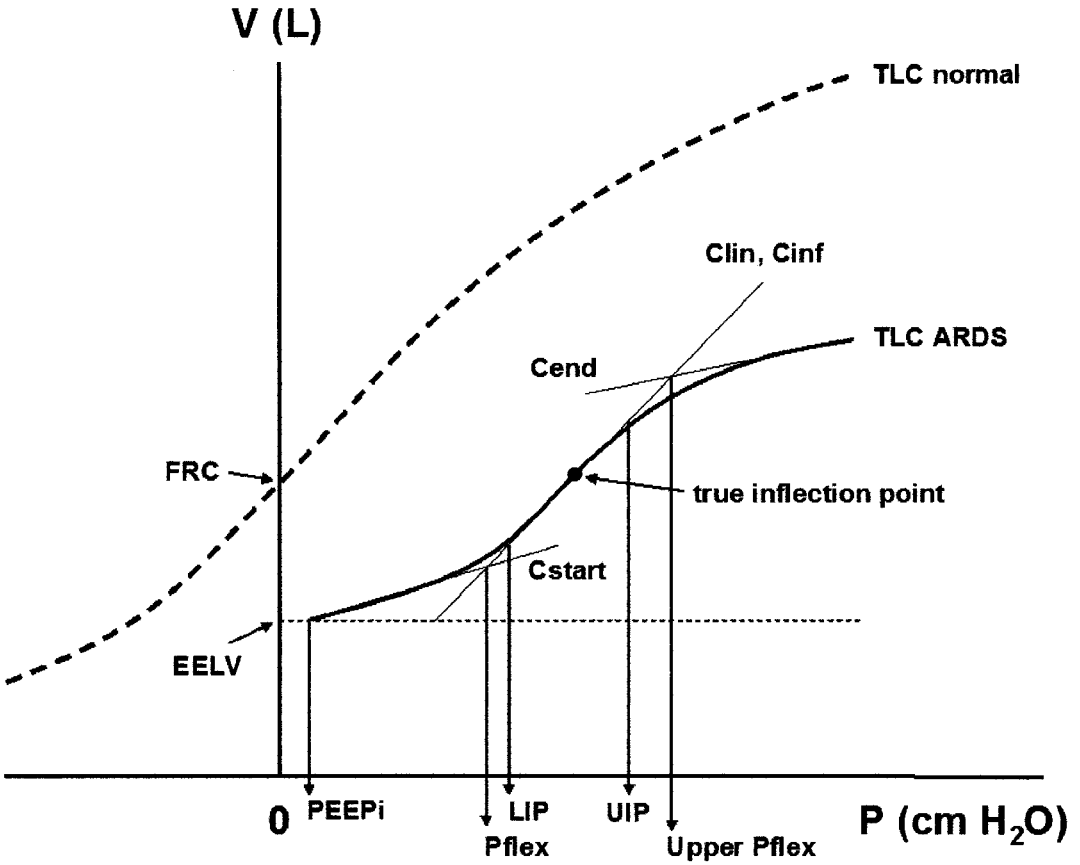

Fig. 16. The pressure-volume curve of a normal subject (dashed curve) and a patient with acute respiratory distress syndrome (ARDS) (solid curve). The pressure-volume curve is shifted downwards on the volume axis and has a reduced total lung capacity (TLC). The sigmoid shape of the curve is much more evident in ARDS. Note the small amount of pressure at the start of the ARDS pressure-volume curve, indicating a small amount of intrinsic positive end-expiratory pressure (PEEPi) at end-expiratory lung volume (EELV). Some investigators divide the curve into linear segments: C_{start}, C_{inf} or C_{lin}, and C_{end} (thin lines, explained below). Using these segments, the upper and lower P_{flex} (the pressure at the intersection of 2 lines: a low-compliance region at low lung volumes [C_{start}] and a higher-compliance region at higher lung volumes [C_{inf}]) were defined by the intersection of these lines. The lower inflection point (LIP) and upper inflection point (UIP) are defined by where the curve first begins to deviate from the line C_{lin}. Mathematically, these are not inflection points; the true inflection point (where concavity changes direction) is marked by the solid dot. V = volume. P = pressure.

form during a P-V maneuver. Schiller et al,[53] using in vivo microscopy in lung-injury models, demonstrated 3 behaviors of alveoli during mechanical ventilation: (1) those that do not change size, (2) those that change size throughout the inflation, and (3) those that "pop" open at a certain pressure and rapidly change size (Fig. 22). These data would seem to support the concept of recruitment. However, Hubmayr[59] showed that the features of an ARDS P-V curve can be obtained without having the alveoli open and close, but rather by forcing air into open, but liquid-filled, alveoli (Fig. 23). That discrepancy may be due to the different models. Carney et al[52] used a surfactant-deficiency model (lung lavage), whereas Hubmayr et al used an alveolar flooding model (oleic acid). To what extent those models represent what truly happens in human ARDS is unknown.

The Chest Wall in Acute Respiratory Distress Syndrome. Two published studies have addressed the effect of chest wall compliance in ARDS. Mergoni et al,[60] with a group of medical and surgical patients, found that the chest wall contributed to the lower inflection point and that the response to PEEP depended on whether the patient had a lower inflection point. If a lower inflection point was present, the patient tended to respond to an increase in PEEP. They also found that the total-respiratory-system P-V curve seemed accurate for estimating the lung upper inflection point. Ranieri et al[40] found that in normal lungs there was no lower or upper inflection point. In medical ARDS there was a lower inflection point from the lungs, which was on average 28% less than the lower inflection point of the total respiratory system. None of the patients had an upper inflection point. In surgical ARDS they found

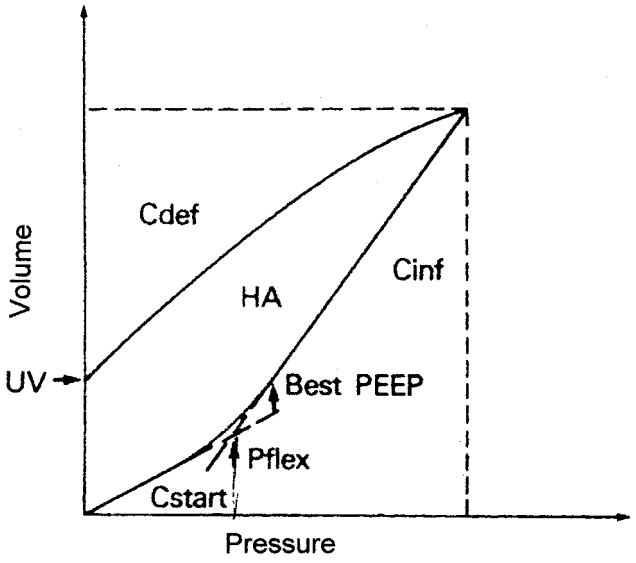

Fig. 17. Gattinoni et al[28] defined P_{flex} as the pressure at the intersection of 2 compliance lines: C_{start} (the low compliance at the beginning of inflation) and C_{inf} (a zone of higher compliance at higher lung volume). UV = unrecovered volume. C_{def} = deflation compliance. HA = hysteresis area. PEEP = positive end-expiratory pressure. (From Reference 28, with permission.)

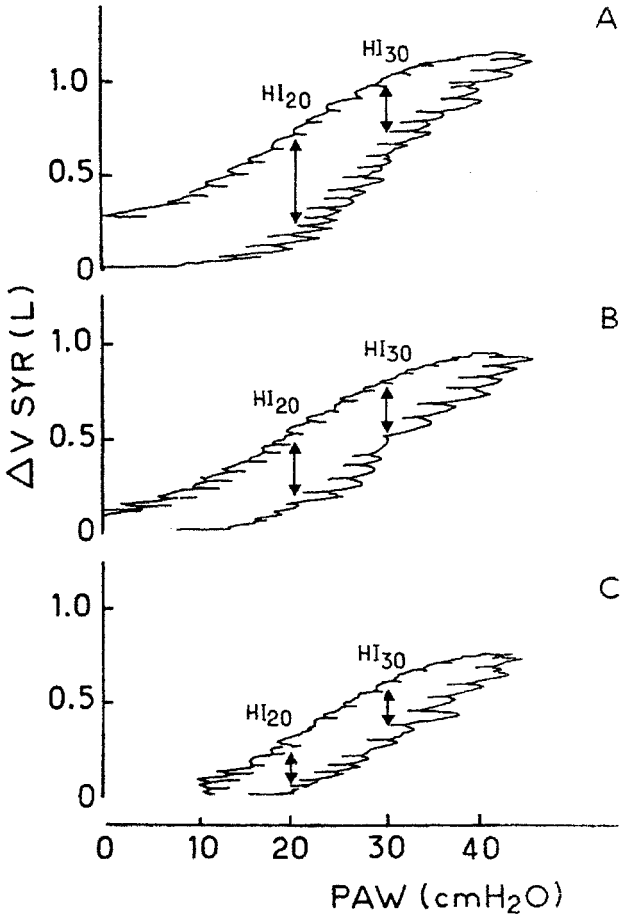

Fig. 18. Effect of positive end-expiratory pressure (PEEP) on the pressure-volume curve. Using a valve with the supersyringe, Dall'ava-Santucci et al[29] obtained pressure-volume curves during PEEP of zero (top), 5 (middle), and 10 (bottom) cm H_2O. Note the gradual loss of the upward concavity (or "knee") in the inspiratory limb of the pressure-volume curve. ΔV SYR = change in volume from the supersyringe. P_{AW} = airway pressure. (From Reference 29, with permission.)

an upper inflection point from the chest wall, which was higher than the total-respiratory-system upper inflection point by a mean of 28%, and no lower inflection point. A more recent study,[61] which used the sigmoid equation to fit total-respiratory-system, lung, and chest wall P-V curves from patients with ARDS, or pneumonia, or cardiogenic pulmonary edema found that the point of maximum compliance-increase ranged from zero to 8.3 cm H_2O and significantly influenced the total-respiratory-system inflection point in only 8 of 32 patients. That may be because there were only 5 extrapulmonary-ARDS patients among 26 total ARDS patients, and those would be patients comparable to the surgical patients in the Ranieri et al[40] study.

The Chest Wall in Pulmonary or Extrapulmonary ARDS. Gattinoni et al[62] proposed dividing ARDS into 2 types: pulmonary and extrapulmonary, based on the response to PEEP. Extrapulmonary ARDS generally responds better (by increasing P_{aO_2}) to a PEEP increase than does pulmonary ARDS. They also found that pulmonary ARDS had higher lung chord elastance, whereas extrapulmonary ARDS had higher chest-wall chord elastance. Those results were extended to the whole P-V curve by Albaiceta et al,[63] who found a significant impact of the chest wall P-V curve on total-respiratory-system mechanics in both pulmonary and extrapulmonary ARDS. There was a greater shift to the right because of decreased chest wall compliance in extrapulmonary ARDS. They also found that the total-respiratory P-V curve was shifted to a higher lung volume in extrapulmonary ARDS than in pulmonary ARDS. This is not surprising, given that pulmonary ARDS is associated with a dramatic reduction in air spaces because of consolidation.

Problems With the Pressure-Volume Curve in ARDS. Despite the apparent safety and reproducibility of P-V curves when performed using the same technique on the same day,[44,64] there are many problems with the routine use of P-V curves in ARDS. P-V curves are very dependent on the volume history of the lungs, so the clinician must be careful when comparing curves from different days, different patients, or different studies. There is no standard method for acquiring P-V curves, and different methods can yield very different P-V curves. The supersyringe method generates artifacts because of ongoing oxygen consumption during the maneuver, which causes a

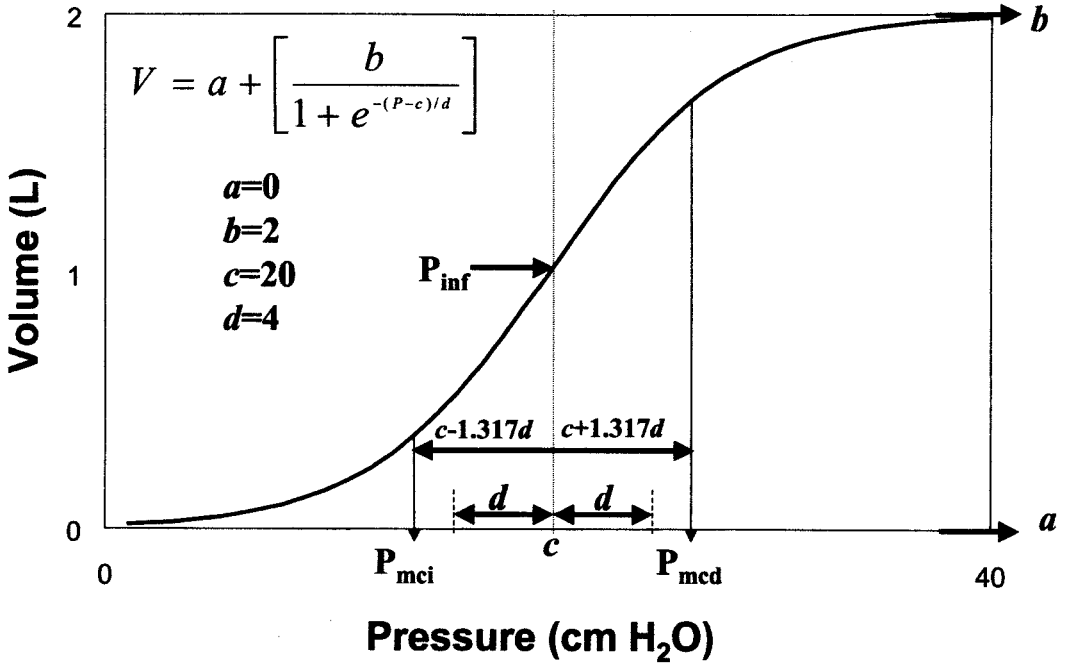

Fig. 19. A sigmoidal equation (upper left in the box) for curve-fitting pressure-volume data. The equation, with the coefficients a = 0 L, b = 2 L, c = 20 cm H_2O, and d = 4 cm H_2O, is shown plotted as the solid line. The inflection point (P_{inf}) is equal to c, and the points of maximum compliance increase (P_{mci}) and decrease (P_{mcd}) can be calculated as shown. (From Reference 41, with permission.)

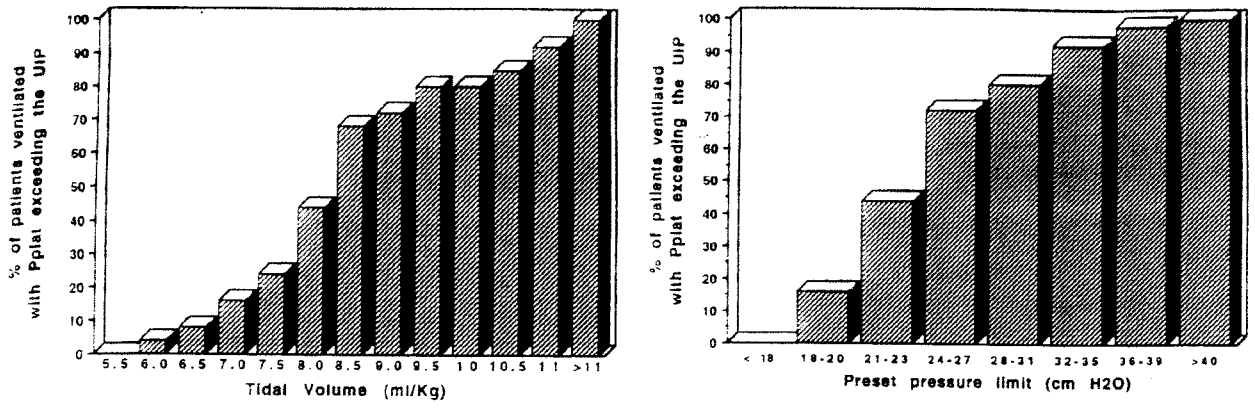

Fig. 20. Percentage of acute respiratory distress syndrome (ARDS) patients ventilated with a plateau pressure (P_{plat}) exceeding the upper infection point (UIP), grouped by tidal volume (left) or plateau pressure (right). Note the large percentage of patients ventilated above the UIP with tidal volume of 8–10 mL/kg of measured body weight (left). For comparison, the ARDS Network's low-tidal-volume strategy corresponds to about 7.2 mL/kg of measured body weight. Note also the large percentage of patients ventilated above the UIP with $P_{plat} < 32$ cm H_2O—a pressure zone once thought to be protective. (From Reference 46, with permission.)

loss of thoracic gas volume, which is not usually measured, as well as gas-volume changes due to changes in humidity and temperature. Fortunately, during the inflation maneuver the loss in thoracic gas volume is generally equally counterbalanced by an increase in thoracic gas volume (12%) from the added humidity and expansion caused by body temperature.[65,66] These effects, however, are in the same direction during deflation, and they increase hysteresis. The multiple-occlusion method may circumvent that problem.

There is no standard method for acquiring P-V curves, and the peak pressure before (ie, the volume history) and during the P-V maneuver affect the shape of the P-V curve.[45,67] Another problem with P-V curves is that they represent the aggregate behavior of millions of alveoli. In a lung with very heterogeneous mechanical properties, the

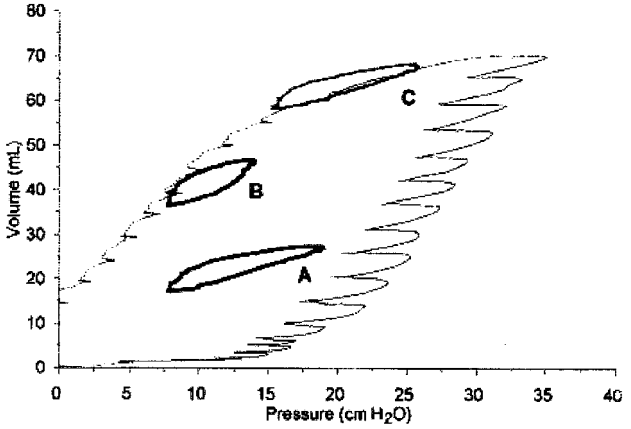

Fig. 21. Dynamic pressure-volume loops within the static pressure-volume curve, during 3 different ventilation modes. A: Ventilation with positive end-expiratory pressure (PEEP) less than P_{flex} (the pressure at the intersection of the low-compliance region at low lung volumes and the higher-compliance region at higher lung volumes) without a sustained inflation. B: Ventilation with PEEP less than P_{flex} after a sustained inflation. C: Ventilation with PEEP greater than P_{flex} after a sustained inflation. Note the marked lung-volume difference among the 3 groups, depending on the PEEP and the volume history of the lung. (From Reference 33, with permission.)

P-V curve may represent mainly the healthy alveoli.[28] In a CT-scan study of patients with ARDS, Vieira et al[68] found that in heterogeneously injured lungs the P-V curve may not demonstrate recruitment or overdistention that can be clearly seen on CT (Fig. 24). A study that used electrical impedance tomography had similar findings.[69]

Another problem is that lung recruitment is a time-dependent phenomenon that cannot be described by a single P-V curve.[70] Only one study has indicated that the use of P-V curves, as part of a volume-limited, open-lung approach, decreases mortality.[30] That study has been criticized, however, because of the high mortality (71%) in the control group. Also, the experimental group received recruitment maneuvers as part of their care, so it is unclear what had more impact in the mortality difference. Some would argue that we may not need to obtain P-V curves at all, given that the largest and most efficacious ARDS trial (the ARDS Network low-V_T versus conventional-V_T study[47]) combined with the ALVEOLI trial of high-PEEP versus low-PEEP,[71] suggest that as long as one limits V_T, only a modest amount of PEEP is necessary, and the ventilator settings may not need to be "customized" based on pulmonary mechanics. However, some animal data suggest that titrating PEEP based on the P-V curve is more effective at minimizing lung injury than is titrating PEEP to oxygenation, as was done in the ARDS Network trials.[72] So the hope still remains that the P-V curve will someday allow for individual titration of PEEP and perhaps further reduce mortality.

Congestive Heart Failure

With congestive heart failure, alveoli progressively fill with fluid, which impairs surfactant function and reduces gas volume. That situation is very similar to what occurs in early ARDS (noncardiogenic pulmonary edema). In animal experiments the reduction in compliance was greater than what could be accounted for by the loss of gas volume.[73] The discrepancy is due to a marked hysteresis related to the development of liquid bridges (foam) in small airways.[73] There is a paucity of data regarding the shape of the P-V curve in human subjects with congestive heart failure, but given the similar physiology to early exudative ARDS, the appearance would be expected to be similar. In fact, Hubmayr contends that fluid-filled alveoli (rather than derecruitment) can explain all of the features of the P-V curve in ARDS.[59]

Emphysema

Most of the early studies describing the P-V relationship in COPD were done with the hope of using the P-V curve to diagnose and establish the severity of emphysema. Since then, CT has supplanted the P-V curve as a means of diagnosing emphysema. Most of the early studies were done on spontaneously breathing COPD subjects rather than on mechanically ventilated subjects. This is probably because in mechanically ventilated subjects with COPD the major concern is for intrinsic PEEP and airway resistance, rather than lung elastic recoil or recruitment. Using the Salazar and Knowles equation[74] ($V = V_{max} - Ae^{-kP}$, in which V_{max} is the extrapolated volume at infinite pressure, A is a constant related to the intercept on the volume axis, e is the natural exponent [2.718], and P is static airway pressure), Gibson et al[75] found that the k factor was increased in COPD subjects. The k factor describes the concavity of the exponential fit and is independent of lung volume. Therefore, an increase in k means the curve has more concavity toward the pressure axis, without regard to its position. This relationship in COPD was also corroborated by Greaves and Colebatch,[76] who studied excised normal and emphysematous lungs. They found that when emphysema was present, k was increased by more than 2 standard deviations above the mean predicted value for age. They also found a direct correlation between k and mean alveolar size. Osborne et al[77] also studied the relationship between k and mean alveolar size in emphysema patients undergoing lung resection. They measured the P-V relationship prior to lung resection and then compared k to the morphologic changes on histology. They found that k reflects air-space size, except when airway closure subtracts the contribution of lung units from the deflation P-V curve. They also found that k generally correlates with severity of COPD until the contribution of large air

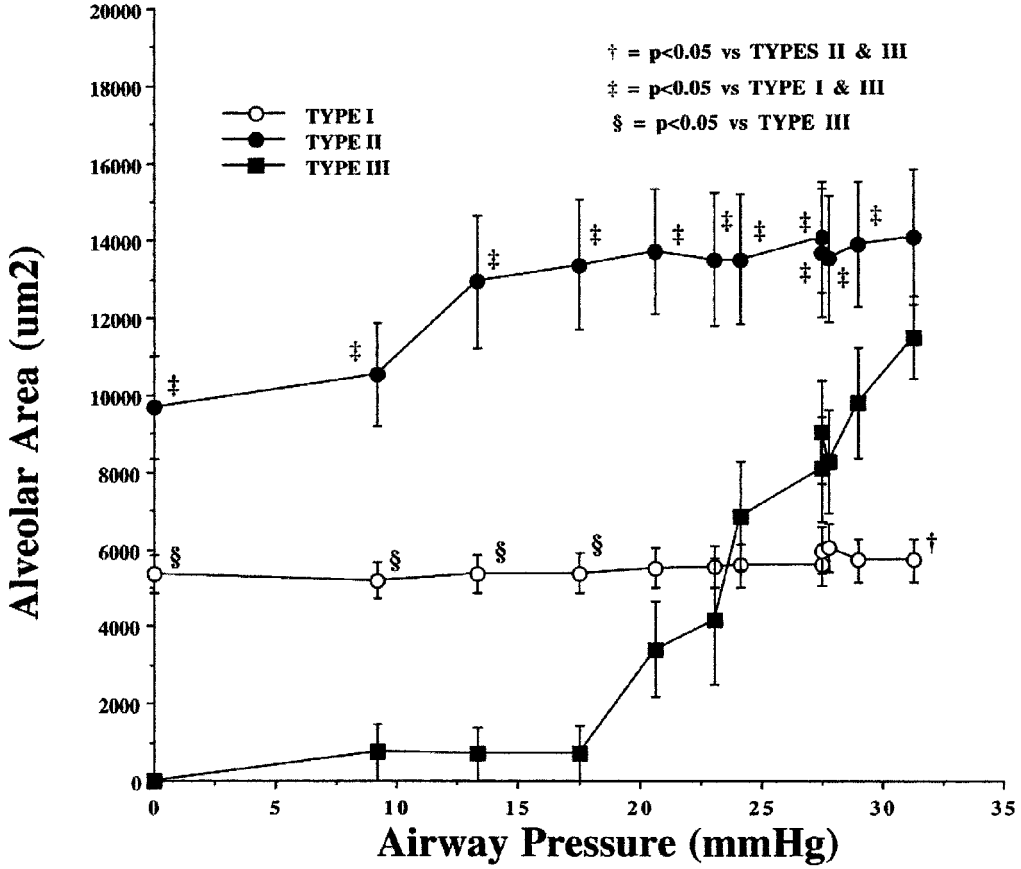

Fig. 22. Alveolar "pressure-volume curves" obtained using in vivo microscopy in a canine lung-lavage-injury model. Three types of behavior are seen: alveoli that don't change area during the inflation limb (type I); alveoli that expand during the inflation (type II); and alveoli that "pop" open at a threshold pressure (type III). (From Reference 53, with permission.)

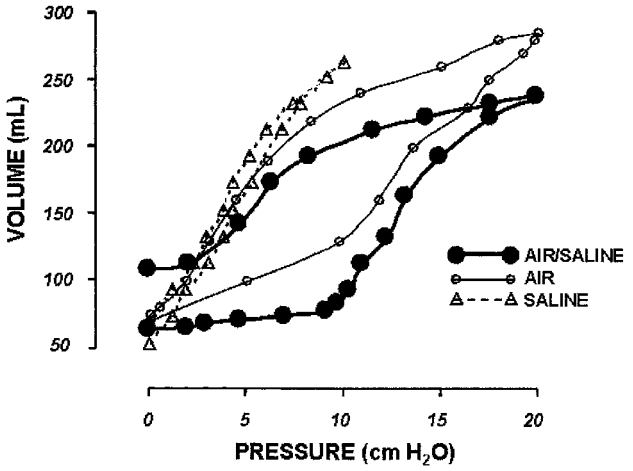

Fig. 23. Pressure-volume curves constructed using the parenchymal marker technique in a canine oleic-acid-injury model. The features of the typical lung-injury pressure-volume curve can be reproduced by delivering an air-saline mixture to the lung, rather than by opening collapsed alveoli. (From Reference 59, with permission.)

spaces to the shape of the curve was lost due to airway closure, such that k would decrease, sometimes back to the normal value. Three other research groups[78–80] had difficulty correlating k with severity of histologic changes of emphysema. So, although the shape of the P-V curve provides information about elastic recoil of the lung and perhaps even about air-space size, because it is an aggregate behavior of all alveoli, contributions of certain airways to the overall P-V relationship may become obscured because of airway closure or simply heterogeneous elastic behavior.

Asthma

Initially it was believed that TLC was increased in asthma, and one study that obtained (via plethysmography) P-V curves from patients having spontaneous asthma exacerbations showed a reduction in lung volume and an increase in elasticity when albuterol was administered.[81] Later, it was determined that plethysmography can give erroneously high TLC values, because mouth pressure fails to approximate alveolar pressure during panting, when there

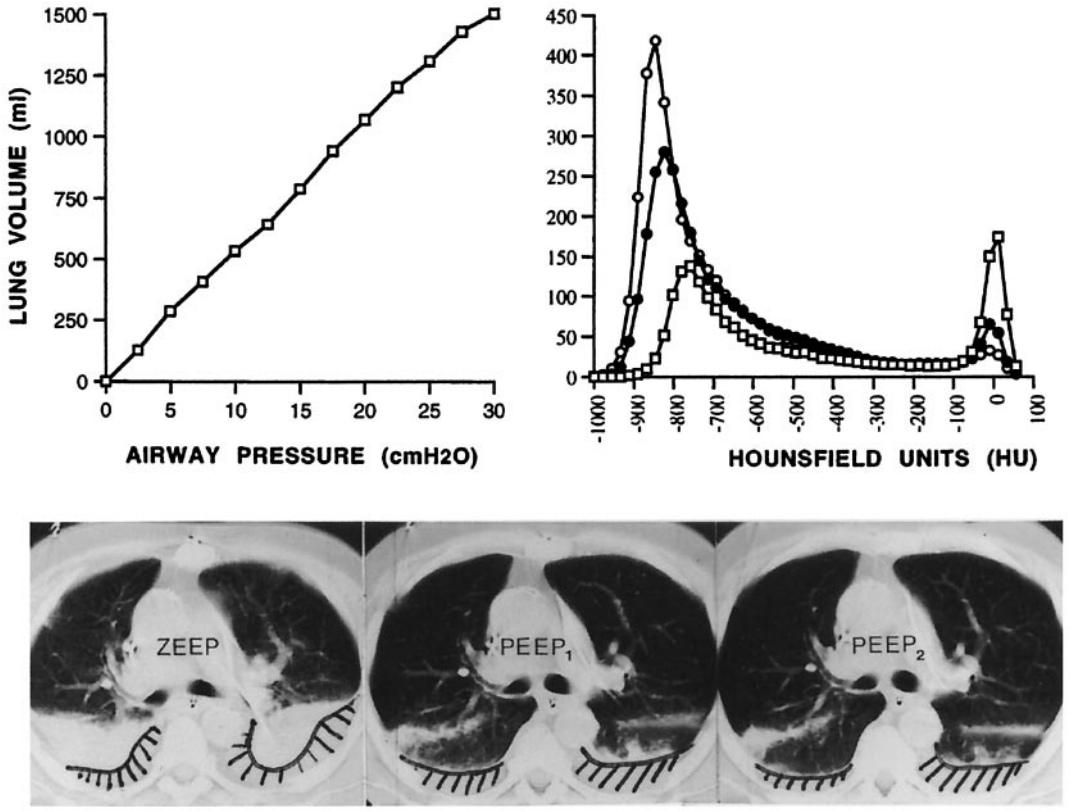

Fig. 24. Pressure-volume curve (top left), lung density histogram (top right), and computed tomograms (bottom) under positive end-expiratory pressure (PEEP) of zero (ZEEP) (open squares), 10 cm H_2O (closed circles), and 15 cm H_2O (open circles). Note that there is no concavity in the pressure-volume curve, yet there is both dramatic recruitment (indicated by the reduction in areas of zero attenuation) and overdistension (indicated by an increase in areas with attenuation values < –900 Hounsfield units). (From Reference 68, with permission.)

are heterogeneous time constants in the lung.[82] Therefore, later studies showed little change in the P-V curves of patients having asthma exacerbations.[78] However, there are no data describing changes in the P-V curve during status asthmaticus, in which airway closure may affect the shape of both the inspiratory and expiratory limbs of the P-V curve.

Interstitial Lung Disease

In interstitial lung disease, alveoli become fibrotic, reducing lung gas volume, which shifts the P-V curve downward on the volume axis (Fig. 25).[83] Though that shift is due to loss of gas volume, this does not necessarily mean a loss of total thoracic volume. Studies have shown an inconsistent change in tissue and blood volume, such that total thoracic volume may be decreased, unchanged, or even increased. Therefore, measuring chord compliance at fixed lung volumes can be misleading, and attempts have been made to distinguish between changes due to volume loss alone versus changes due to alveolar fibrosis. When fitted to the Salazar and Knowles equation,[74] the k factor was reduced in only 2 of 14 patients,[75] so it is appears that

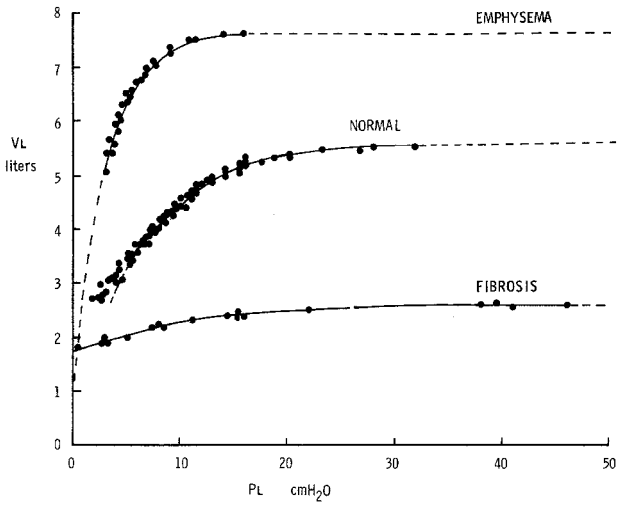

Fig. 25. Pressure-volume curves in emphysema, interstitial lung disease (fibrosis), and normal lungs. The solid line is the data fitted with the equation $V = V_{max} - Ae^{-kP}$ (see text). Note that emphysema causes a displacement of the pressure-volume curve to higher lung volume and an increase in concavity (increase in k), whereas fibrotic lung disease causes a displacement to lower lung volume and a decrease in concavity (decrease in k), compared with normal lungs. (From Reference 83, with permission.)

the shape of the P-V curve may not be a sensitive means of assessing alveolar fibrosis.

Intra-Abdominal Hypertension

Because the abdomen is an important part of the chest wall, factors that increase the stiffness of the abdomen may influence the P-V curve, such as pregnancy, ascites, and intra-abdominal infection or surgery. In a classic study by Mutoh et al,[84] the effects of abdominal distention on the lung and chest wall were studied in pigs by inflating a liquid-filled balloon placed in the abdominal cavity. Respiratory system, chest wall, and lung P-V curves were measured on deflation from TLC to residual volume, as well as in the tidal breathing range, before and 15 min after abdominal pressure was raised. Increasing abdominal pressure from 3 to 15 cm H_2O decreased TLC and FRC by approximately 40% and shifted the respiratory-system and chest-wall P-V curves downward and to the right. Much smaller downward shifts in lung deflation curves were seen, with no change in the transdiaphragmatic P-V relationship. All regional pleural pressures increased (became less negative) and, in the dependent region, approached zero at FRC. Tidal compliances of the respiratory system, chest wall, and lung were decreased 43%, 42%, and 48%, respectively. Abdominal distention appears to alter respiratory-system mechanics primarily by "stiffening" the diaphragm/abdomen part of the chest wall and secondarily by restricting lung expansion, thus shifting the lung P-V curve. In a study of ARDS patients with abdominal distention,[40] a flattening of the P-V curve at high pressures was seen, due to an increase in chest wall elastance. Abdominal decompression caused an upward and leftward shift of the P-V curves of the respiratory system, chest wall, lung, and abdomen.

Summary

If the P-V curve is to become a useful clinical tool, there are still a number of problems to solve. Currently, there is no standard method to obtain the curve or volume history. Some would argue that the static P-V curve does not represent the true stresses imposed on the breathing lung parenchyma and should not be used at all for setting the ventilator. In ARDS, it is still not known which limb of the P-V curve to use, although it would seem logical that the deflation limb should be used for setting PEEP. It also seems clear that features of the curve, such as P_{flex}, are more complicated than once thought. P_{flex} is a poorly defined and often subjective point, and does not indicate the end of recruitment. In fact, if there is no P_{flex}, there still might be the possibility of recruitment. To make matters worse, we still do not understand how alveoli deform during inflation and deflation in human ARDS, so that inferring from a P-V curve what is protecting alveoli or damaging them is exceedingly difficult. The effect of the chest wall on the shape of the total-respiratory system P-V curve is still not completely known. A bigger issue, perhaps, is that regional mechanical differences are obscured by the P-V curve of the whole lung, and this may be the most important piece of information clinicians need to know. Finally, it is difficult to interpret P-V curves without an absolute measure of lung volume. Being able to easily measure absolute lung volume versus pressure would be a big advance in our understanding and interpretation of the curves, but it remains a difficult task to do routinely. For now it appears that the P-V curve must remain a research tool, or in the clinical situation, reserved for selected patients when it is necessary to try to understand alterations in lung mechanics, provided that an understanding of its many limitations is taken into account.

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Discussion

Bigatello: You have been working on P-V curves now for a few years. What we struggle with every day is, how are we going to set the ventilator? Are you still using P-V curves? Do you have any abbreviated ways, or are there other ways that you use to come up with ventilator settings?

Harris: No. And no. I really don't use them much any more in the intensive care unit, except for research purposes. I think there's still a lot to learn about them, so I think the research environment is the best place to do P-V curves. I think that right now there's no good evidence that using the quasi-static P-V curve is a good way to set a ventilator; I don't think there's good evidence that you're going to improve your patient's outcome with it. Furthermore, where are you going to set it? The point keeps changing; it used to be P_{flex} plus 2 cm H_2O, now it's all the way up the curve, so it's a moving target, which reflects our lack of understanding of how to use the P-V curve.

Hess: I think you hit the nail right on the head when you asked, do we set the ventilator based on some physiologic measure or do we set the ventilator according to outcome data that we know are producing better patient outcomes?

Pierson:* Just to follow up on that, Dean, I want to remind people that the ALVEOLI study (the second ARDS Network study, which compared higher PEEP/lower F_{IO_2} to lower PEEP/higher F_{IO_2} in ALI [acute lung injury] and ARDS[1]) was stopped because it was determined that there was no difference between the groups, indicating that even though you may get more of the lung recruited at higher PEEP—and that would certainly be the assumption from what you've said—it didn't seem to make any difference in what happened to the patient.

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Harris: And that may go along with the idea that you bring up, Neil [MacIntyre], about maybe that lung that's closed, as Gattinoni would say, let's keep it closed, rest it, and maybe try to reduce the strain on the rest of the lung as much as possible. Perhaps it could be something as simple as just turning down the ventilator to the lowest settings possible that are still able to manage, ventilate, and oxygenate the patient. Maybe that's all.

MacIntyre: Just to put some details on what David [Pierson] said, it is true that the ALVEOLI trial was stopped after a little over 500 patients, for futility, despite rather impressive improvements in both compliance and P_{aO_2}/F_{IO_2} ratio [the ratio of arterial partial pressure of oxygen to fraction of inspired oxygen], so those beautiful numbers, in terms of mechanical function and gas exchange function, did not translate into a substantial improvement in outcome. I guess the good news is that it didn't hurt.

Campbell: One of the issues I have with the ALVEOLI trial is—and I think it speaks right to the point of the P-V curve—that it looked at gas exchange as the end point for determining the PEEP level. They were looking at a P_{aO_2}/F_{IO_2} ratio with PEEP tables, and I think that may be a problem with some patients. The P-V curve is a measurement of lung mechanics that may give us some useful information and help us manage the ventilator. Has your experience doing P-V curves changed your practice?

Harris: I'm not sure I understand the question.

Campbell: The first part was just a comment about the ALVEOLI trial. In the Amato et al study,[1] setting PEEP based on information obtained from the P-V curve was an important component of the lung-protective strategy that was associated with better outcome. The assumption in the ALVEOLI trial was that there is a linear relationship between lung injury and the P_{aO_2}/F_{IO_2} ratio, and certainly that can't be true in every patient.

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MacIntyre: What are you talking about? A linear relationship between what?

Campbell: I mean the assumption is that if you turn up the PEEP and the P_{aO_2} gets better, then you have adequately treated the problem. Or if your

P_{aO_2}/F_{IO_2} ratio is low, then you always need more PEEP.

MacIntyre: The ALVEOLI trial was driven by P_{O_2}, not P_{aO_2}/F_{IO_2} ratio.

Campbell: Well, P_{O_2} then; I mean gas exchange.

MacIntyre: Right. You're correct there; it was driven on gas exchange as a rational, simple way of trying to set the PEEP *and* the F_{IO_2}.

Campbell: But I believe there's a class of patients in whom, if you turn the PEEP up to your heart's content, you're not going to get any improvement in gas exchange.

Harris: I guess that's built into the PEEP/ F_{IO_2} tables; as you increase PEEP, if you're not getting better oxygenation, you'll increase oxygenation, and as well you'll increase F_{IO_2}, so I think it's just a way of minimizing pressure and F_{IO_2} to the minimum amount needed to maintain P_{aO_2} above 55 mm Hg; that's the only thing it's really doing, and they just chose from a consensus about what would be reasonable settings.

Campbell: My point is, can we identify that patient from the P-V curve?

Harris: I see. I don't think so, at least not easily. Rouby et al[1] showed some nice data about patients who have pulmonary versus extrapulmonary ARDS. You wouldn't think that they would necessarily respond to PEEP, but he showed that they do have a response; it's just kind of hidden. The P-V curve looks linear, so you wouldn't think that there's recruitment going on. But if you think about the study by Gattinoni et al[2] and what it said about the C_{inf} versus C_{stat}, if you have, in that situation, a ratio of 1, there shouldn't be a lot of recruitable lung. But Rouby et al[1] showed, by way of a CT scan, that there *is* recruitment going on, but it's also mixed in with isotropic expansion of *open* alveoli, and so you can't discern it in the P-V curve. So it's hard with the P-V curve to actually predict which patients are going to respond.

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Blanch: I agree with almost everything you said on P-V curves. In the same patient, P-V curves taken at different PEEPs always present with the same upper inflection point, and that upper inflection point may vary with patients, being in most cases lower than 32 cm H_2O. We may find different lower inflection points, different inflation slopes, but the upper inflection point is the same. It is particularly important to set a tidal volume that does not reach that limit, where the lung is open or quasi-open, because at higher tidal volume, overdistension can easily occur. Attempts to recruit the lung beyond that point are questionable.

Harris: When we first were doing these we used the supersyringe method, and an interesting thing was that we never saw an upper inflection point. We used to have these discussions about where are the upper inflection points that people are talking about. I think it's a difference in method, and I think the multiple-occlusion method may be better at detecting that overdistension, because with the supersyringe method you're allowing the lung to slowly distend and the surface forces and viscoelastic forces are changing, and they're allowing the lung to relax, so I don't think you see that upper inflection point. Maggiore et al[1] mentioned this in a recent review,[1] saying that—he calls it a *viscoelastic pressure*, which maybe the lungs experience during tidal breathing, and that the upper inflection point is reflective of that.

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Blanch: We have done some research comparing 2 techniques to create P-V curves: multiple-occlusion and low flow. Tidal recruitment might be present with the multiple-occlusion technique, whereas that was not observed with the low-flow technique. Tidal recruitment was not observed when P-V curves were taken at high PEEP. Those factors need to be taken into account when clinicians interpret P-V curves created using different techniques.