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STATIC MECHANICS OF EXCISED WHOLE LUNG: THEORETICAL FRAMEWORK AND EXPERIMENTAL STUDIES

J. R. Ligas*
F. P. Primiano, Jr.*†
G. M. Saidel*‡

*Department of Biomedical Engineering
Case Western Reserve University
Cleveland, Ohio

†Department of Pediatrics
Case Western Reserve University and Rainbow Babies and Childrens Hospital
Cleveland, Ohio

‡Veterans Administration Medical Center
Cleveland, Ohio

A theoretical framework is presented in which to view models of static pulmonary mechanics. To test common simplifying assumptions of these models, we performed a set of experiments using normal lungs excised from dogs. Transpulmonary pressure (P_{tp}) and lung volume (V_L) were measured for air-filled lungs in air and saline-filled lungs in saline during stepwise-static deflations at different vascular volumes and temperatures. Simultaneously, we measured displacements between points on the lung surface. Changes in vascular volume shift the location but not the shape of the P_{tp}-V_L relationship. As long as the vascular pressure is in the normal range, changes in the volume (and weight) of the perfusate do not significantly stiffen the parenchyma. Furthermore, P_{tp}-V_L data obtained between 16°C and 40°C were superimposable, indicating that parenchymal mechanical properties evaluated at room temperature are valid at body temperature. Finally, the common assumptions of uniform deflation, homogeneity, and isotropy of bulk lung tissue appear consistent with the relationship between surface displacement and volume changes.

Keywords *– Parenchymal mechanics, Pressure-volume curves, Minimum lung volume.*

INTRODUCTION

Sophisticated analyses of static pulmonary behavior now rest upon a foundation of continuum mechanics (6,8,9,13,20). Such works are important for

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Address correspondence to Dr. Gerald M. Saidel, Biomedical Engineering Department, Case Western Reserve University, Cleveland, Ohio 44106.

several reasons. First, they allow detailed investigation of the physical mechanisms by which forces are transmitted throughout the lung parenchyma. Only with these models can "local compliance" be adequately understood and the interaction of alveolar membranes be addressed. Second, with modern computational techniques such as finite element analysis, more detailed models can be used to study a wide range of conditions for which simpler analyses would be inadequate. Third, estimated values of the parameters of realistic models can obviate the need for raw data storage, retrieval, and allow pattern recognition. And finally, when combined with appropriate experimental techniques, more detailed and realistic models may provide distinctions between normal and abnormal lungs.

These new analyses, however, are not without drawbacks. First, older theories and explanations of experimental results need to be rephrased in the lexicon of mechanics to provide a sense of continuity to the field. Second, to be acceptable, such models must describe deformations arising from nonuniform loading (9,13) in addition to those seen when the lung is expanded more uniformly by a distending pressure difference. Third, the parameter values should be easily and well-determined and should allow discrimination between normal and diseased lungs. As Wilson (22) pointed out, none of the proposed relationships between lung stresses and strains have been adequately tested by these criteria using rigorous quantitative methods in any of the work done to date.

Review of the proposed stress-strain relationships in the literature indicates that for each model, most parameters can be determined from pressure-volume data of excised, fluid-filled lungs. Although sophisticated experiments involving nonuniform deformations are necessary to validate a given model and to describe local nonuniform phenomena in the lung, clinically it is difficult to go beyond pressure-volume data. It is these data that will be used to discriminate between normal and diseased subjects (4). If parameter values determined from such experiments are not reliably determined or do not sufficiently discriminate normal from abnormal lungs, such models will not be used.

Accordingly, to show how a theoretical framework can be useful for possible application to pulmonary physiology and clinical pathology studies, we investigated the ability of simple experiments on excised lungs to provide the data required for the estimation of parameters embodied in more complicated models. In this paper we present a general theoretical framework for pulmonary mechanics, followed by simplification of this framework for normal lungs based on various hypotheses. We describe tests of several of these hypotheses.

THEORY

Analyses of the mechanics of a deformable body require specification of (a) an initial, undeformed geometry, (b) an equation of motion describing the movement of points of that body in response to applied loads, (c) appropriate boundary conditions relating the surface stresses to the applied

loads, and (d) constitutive relationships for the body materials, i.e., relationships between the relative displacements of the body material and the applied stresses. For a deformable body, each point within it moves at a rate and in a direction determined by the local forces. When the body is motionless, the equation of motion reduces to a force balance: the net force must be zero if the body is not to accelerate. In mathematical terms, the Eulerian force balance applies:

$$\nabla \times \Sigma + \rho g = 0 \tag{1}$$

where ∇ = the gradient operator
Σ = the local stress tensor for the material
ρ = the density
g = the acceleration of gravity.

The boundary condition is that at the body's surface the stresses must be equal to the applied force per unit area:

$$\Sigma_s = L_s/A_s \tag{2}$$

where Σ_s is the surface stress, L_s the resultant surface load, and A_s the surface area.

The third required equation, the constitutive relationship, relates stresses to relative displacements within the body:

$$\Sigma = f(E) \tag{3}$$

where f is a functional relationship, and E is some measure of the relative displacements, or strains. If an appropriate constitutive equation is supplied, one may predict the deformations caused by a given loading, or, conversely, predict what loading is necessary to produce a given deformation. This is the usual problem faced in solid mechanics. In the physiology laboratory, however, the applied loads *and* the resulting deformations can be measured. The problem then is either to formulate an accurate constitutive equation, or to estimate the material properties of the body in question using a given constitutive equation.

Application to Pulmonary Mechanics

Historically, investigations of pulmonary behavior have relied on simple models in which the parenchyma is depicted as a compliant sponge and the airways as flow-resistant ducts. With this lumped-parameter concept, the static transpulmonary pressure-volume curve for intact or excised lungs assumed importance as a measure of the elasticity of the parenchymal tissue/surfactant combination. In the clinical and pathology laboratories, this curve can be obtained very easily; the factors affecting it can be understood within the

framework of continuum mechanics discussed above. For simplicity the following discussion is restricted to excised lungs.

First assume that the lung's geometry can be adequately described. Because the lung is at rest when the pressure-volume data points are recorded, Eq. 1 applies at every point within the lung. In particular, to remain in static equilibrium each alveolar wall requires a force equal to the difference between the force of gravity acting on the wall and the intracapillary fluid and buoyancy force supplied to its surface by the intra-alveolar fluid, which is normally air but can be any experimental fluid. The required net force on an alveolar wall is thus

$$F_w = W_p + W_v + \rho_L V_L - \rho_s V_{TL} = \rho g \tag{4}$$

where W_p = weight of the parenchymal tissues
W_v = weight of the intravascular fluid
ρ_L, V_L = density and volume of the fluid within the airspaces, respectively
ρ_s = density of the fluid in which the lung is immersed
V_{TL} = total lung volume (airspace plus tissue plus intravascular volumes).

Equation 4 thus specifies the body force ρg for Eq. 1.

Next, consider the boundary conditions. An excised lung requires support before experiments can be undertaken: it must be stood upon its base, hung by the bronchus, or secured in some other manner. This generates local surface stresses, denoted S_s. Furthermore, at the surface of the lung the stress must equal the applied pressure difference, plus the local surface stress, minus the contribution from the local pleural tension, τ_p:

$$\Sigma = \Delta P + S_s - \tau_p(1/r_1 + 1/r_2) \ , \tag{5}$$

where r_1 and r_2 are the local principal radii of curvature.

Definition of the problem is complete upon specification of a constitutive equation. Although the lung is not perfectly elastic (19), the viscoelastic contribution is small at slow rates of deformation. Consequently, traditional models have treated the parenchymal material as elastic and attributed all time-dependent behavior to surfactant. The newer models essentially do the same (8,13).

For an elastic body the first requirement is a reference state from which deformations can be defined. It should be the state at which all applied forces are zero, and it should be reproducible, i.e., unloading the body should return it to this state. In the case of the lung, we assume that each alveolus has an unstressed size characterized by a volume V_0. Deformations from this baseline give rise to three stresses. First, the deformation of the alveolar walls produces a stress dependent upon the local alveolar wall mechanical properties, denoted by the vector θ_p. Second, pressures within the vessels and capillaries coursing through the alveolar membranes may produce a prestress S_v,

which may increase the inherent stiffness or resistance to deformation. And finally, surfactant lining the airspaces of the lung provides a surface tension, denoted S_L, dependent upon the area of the film. Because the lung and its surfactant lining are biologic materials, their properties may depend upon the temperature T.

Thus, from a consideration of the equation of motion and the boundary condition equation, one concludes that the static pressure-volume curve of an excised lung depends upon a group of factors independent of the parenchymal mechanical properties, namely $\{F_w, S_s, \tau_p, r_1, r_2, \Delta P\}$. Furthermore, consideration of the constitutive relationships for surfactant and lung parenchyma and of the preloading that could be caused by the vasculature leads one to conclude that the transpulmonary pressure-volume curve can also be influenced by the factors $\{S_L, S_v, V_0, \theta_p, T\}$. The combination of these factors can be expressed in functional form as:

$$V_L = f(\Delta P, \tau_p, F_w, S_s, r_1, r_2, S_L, S_v, V_0, \theta_p, T) \quad . \tag{6}$$

Note that we assume some standard volume or loading history to eliminate any time-dependent effects from consideration and that all quantities except ΔP and T may vary from point to point throughout the lung.

This seemingly abstract but theoretically straightforward framework encompasses the entire literature dealing with static lung mechanics. Every theory or experiment can be viewed in this light. Most older works are attempts to define conditions under which Eq. 6 can be further simplified. Simplification is important; if it can be carried out, more complex experiments and analyses can be avoided and use of the resulting model more easily undertaken.

The first simplifications may result from the structure and properties of the parenchyma itself. First, a body is said to be *homogeneous* if its properties are not a function of position within the body. Second, a body is *isotropic* if the properties at a point do not depend upon the directions chosen for the axes. If these attributes apply to the parenchyma, then the parameter vector θ_p is a global characteristic of the lung. If, in addition, the unstressed alveolar size distribution were constant, a single volume V_0 could characterize the unstressed state of the entire lung. Experimental manipulations may be employed to further simplify the analysis. If the lung is excised, degassed, and filled with and surrounded by saline, then the applied pressure difference is constant at every point on the surface and the effects of surfactant are eliminated. Furthermore, Eq. 4 reveals that since the weight and buoyancy forces are nearly equal under these conditions, the required support forces become minimal. In addition, if saline readily fills the vasculature and there is no transvascular pressure difference, then the vascular stress is also eliminated. Finally, if the pleural tension is negligible or if the local surface radii of curvature are large, Eq. 6 reduces to

$$V_L = f(\Delta P, V_0, \theta_p, T) \tag{7}$$

and Eqs. 1 and 5 become

$$\nabla \cdot \Sigma = 0 \tag{8}$$

$$\Sigma_s = \Delta P \tag{9}$$

These two equations are well known; their solution is a constant stress throughout the body:

$$\Sigma = \Sigma_s = \Delta P \tag{10}$$

This result implies that uniform deformation would occur throughout the lung, with important implications for the theoretical formulation. For uniform deformation, complicated stress analyses are not required and simple relationships between applied pressure difference and volume result. If the parameter values for the constitutive equation can be estimated from only pressure-volume data, the experiments required to evaluate the mechanical properties would be relatively simple, and the calculated values could be used in predictions of more complex lung behavior. Thus, whether the lung is homogeneous, isotropic, and deforms uniformly is of considerable interest, as are the properties of the pleura. Our studies dealing with whole-lung behavior are reported below; the investigation of pleural mechanical properties is reported in an accompanying paper (15).

METHODS

We performed experiments on lungs from 14 mongrel dogs. For removal of as much blood as possible from the lungs, the anesthetized animal was exsanguinated by severing the abdominal aorta. The trachea was unoccluded as the heart, lungs, esophagus, and trachea were dissected *en bloc* from the thorax. The major bronchi were then severed from the trachea and the lungs dissected free from the remaining tissues. We chose one lung for experimentation, weighed it, and ligated all vessels except the pulmonary artery. The pulmonary artery and bronchus were then cannulated and the lung submerged in water while partially inflated to detect air leaks. If encountered, these were repaired by ligating the holes in the pleura. If we could not stop the leaks, the lung was discarded. Three stepwise-static pressure-volume curves were obtained in air on deflation from 25 cm water distending pressure using the apparatus depicted schematically in Fig. 1. The lung was then degassed. In our early experiments this was accomplished by placing it in a closed container attached to wall suction of −40 cm water for 45–60 minutes. Saline in the bottom of the chamber prevented dehydration of the specimen. In later experiments we used Von Neergaard's method (21), the apparatus for which is shown in Fig. 2. An inflating pressure difference of 8–10 cm water was maintained as the absolute pressure was lowered with a vacuum pump. After 45 minutes the saline reservoir valve was opened and the lung permitted to

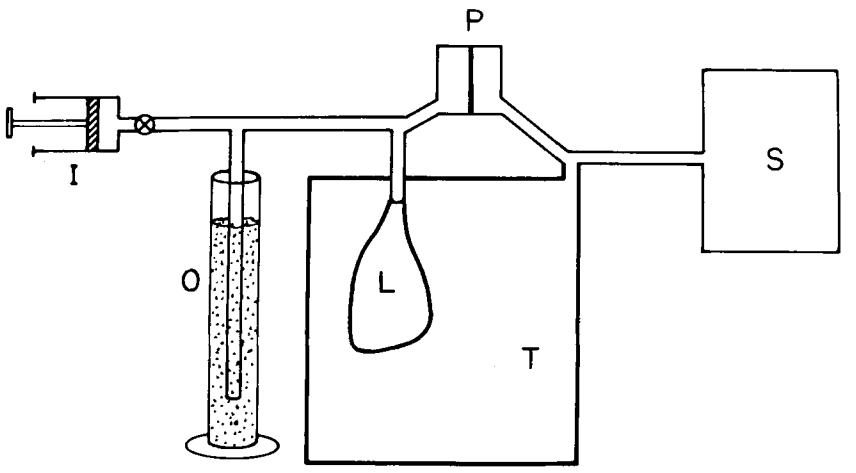

FIGURE 1. Apparatus for pressure-volume curves in air. L, lung; T, tank; P, pressure transducer; S, spirometer; O, over pressure relief device; I, injection syringe.

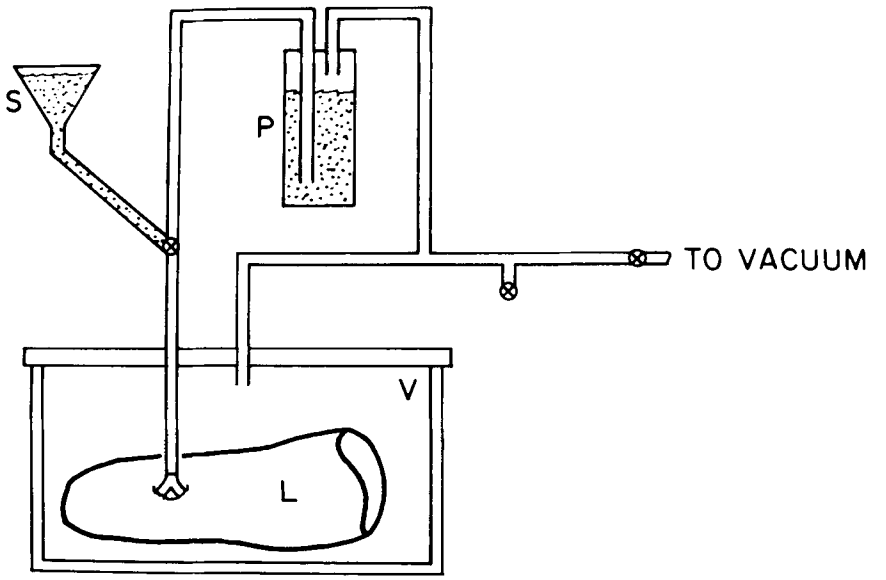

FIGURE 2. Von Neergaard apparatus (20) for degassing the lung. L, lung; V, vacuum chamber; P, device for maintaining constant distending pressure difference during degassing; S, saline reservoir.

fill with saline to roughly 30% total lung capacity. The vacuum chamber was then allowed to slowly fill with air to atmospheric pressure.

After removing the lung from this degassing apparatus, we placed it in the saline-filled plethysmograph depicted in Fig. 3. Here the lung was inflated by raising the reservoir attached to its bronchus and allowing both reservoirs

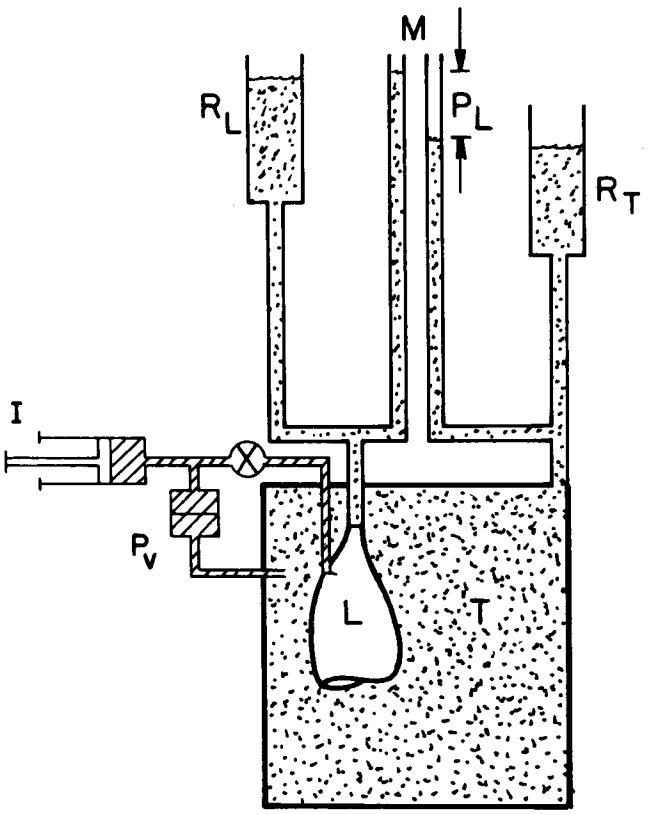

FIGURE 3. Plethysmograph for saline pressure-volume curves. L, lung; T, tank; R_L, lung reservoir; R_T, tank reservoir; M, manometer; P_L, lung distending pressure difference; I, injector syringe; P_v, vascular pressure gauge.

to come to equilibrium. The reservoir attached to the plethysmograph was calibrated for volume changes. The manometer measured the pressure difference applied across the lung. The lung had a slight tendency to float; thus, some forces were applied by the cannula at the hilum to keep the lung submerged in the tank. After equilibrium was achieved at a maximum pressure of 10–14 cm saline, stepwise-static deflation was begun by incrementally lowering the lung reservoir and allowing the fluid levels to reach equilibrium. As lung volume decreased, longer periods of time, often up to 30 minutes, were required to establish equilibrium. In four experiments the lung would not empty at low volumes, presumably because of kinking of the bronchus when the lung was suspended from it. In these cases the closed system was abandoned; the lid was removed from the tank to readjust the cannula and tubing. Tank saline height was kept constant and the lung forced to sit just under the surface. In this case the lung reservoir was used to both adjust the inflation pressure difference and to calculate volume changes. There was no observable difference between data obtained using this method and data obtained with the closed system with the exception that emptying continued to lower volumes at the very low pressures with the open system.

For three lungs the experiments were repeated at various temperatures over a range of 16–40°C by using cooled or heated air or saline. In experiments on six of the other lungs, the vasculature was perfused through the pulmonary artery with silicone oil (Harwick SF 200) to permit filling of the vessels without loss of intravascular fluid into the alveoli. That the oil did indeed traverse the capillaries is supported by the observation that oil ran out when the pulmonary veins were opened at the conclusion of the experiments.

To investigate the uniformity of expansion, for all lungs a grid of 6–10 cyanoacrylate glue dots approximately 3 mm in diameter (1) were randomly applied 1–2 cm apart to the lung surface, usually on one randomly chosen lobe. During both air and saline pressure-volume experiments, length changes were recorded photographically, except in a few instances when a flexible ruler was used. Errors in measurement were estimated to be 2% even when the plethysmograph was filled with saline. This estimate was derived by photographing bars of known length submerged in saline at various distances from the front surface of the plethysmograph.

EXPERIMENTAL RESULTS

Both degassing techniques gave similar results. Assuming a lung tissue density of 1.06 g/cc (9), differences between volume calculated from lung weight and volume measured by water displacement yielded residual air volumes of 30–90 cc, depending on lung size. Total lung capacity in air, calculated by adding the minimum volume to the volume required for full expansion, ranged from 0.5–1.8 liters.

At the beginning of the saline experiments, there seemed to be a maximum sustainable pressure difference of 10–16 cm water regardless of the quality of the lung preparation. Above this maximum pressure a red fluid, presumably hemoglobin in saline, seeped from the lung margins and the reservoir heights would not stabilize. In addition, at the conclusion of the experiments when the ties were removed from the pulmonary vessels, saline was present, indicating that saline can cross from the alveolar spaces to the pulmonary vasculature.

Figure 4 presents typical air and saline pressure-volume relationships for deflation. At higher pressure differences, volume tends to reach an asymptote. Total lung capacity for a given lung was the same in air as in saline. Static pressure-volume relationships for air-filled lungs in air and for saline-filled lungs in saline obtained at various temperatures are shown in Fig. 5. Temperature effects appear to be negligible in the range of 16–40°C.

The injection of oil into the vasculature appeared to increase the minimum lung volume V_0 and the total lung capacity by a small amount (Fig. 6). That is, at different vascular volumes the pressure-volume curves are slightly displaced along the volume scale with negligible change in curve shape.

To analyze the surface displacement data, volumes were scaled by total lung capacity (TLC) and lengths by length at TLC. If lung surface displacement were uniform over the entire range of lung volume change, then plots of the cube root of scaled volume versus scaled lengths should be linear with a slope

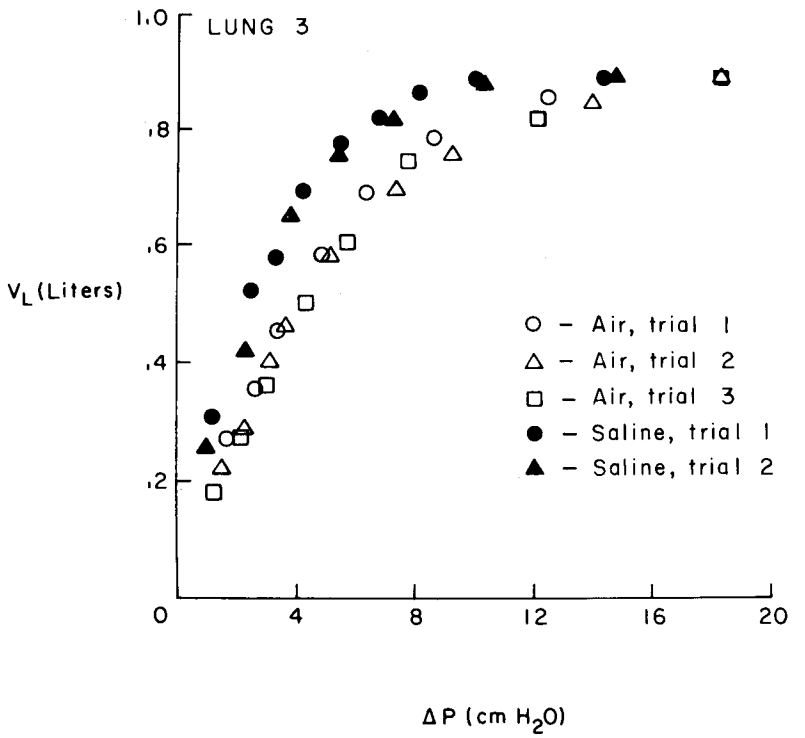

FIGURE 4. Saline and air pressure-volume deflation curves.

of 1.0. Figure 7 shows the relationship between these two variables for air-filled and saline-filled lungs. We found that the hypothesis that the mean slope differs from 1.0 is rejected by 2-tailed t test ($P < 0.05$).

DISCUSSION

Experimental Aspects

Both degassing methods we employed demonstrated that approximately 95% of the air volume can be removed by a single degassing. This is supported by others (16) and indicates that the use of 100% oxygen to flush the animal's lungs before sacrifice to ensure adequate degassing is unnecessary. During the deflation to small volumes, the lungs sometimes attempted to float as the retained air volume became a larger fraction of the total lung volume. The rigid tubing cannulating the bronchus prevented flotation by supplying a hilar force. This small, localized loading should not affect the results because it was applied directly to a large, rigid bronchus (12).

The pressure-volume curve remains unchanged over the range 16–40°C. This is consistent with similar findings in rabbits (14) and indicates that data obtained at room temperature are sufficient. However, Inoue et al. (10) did

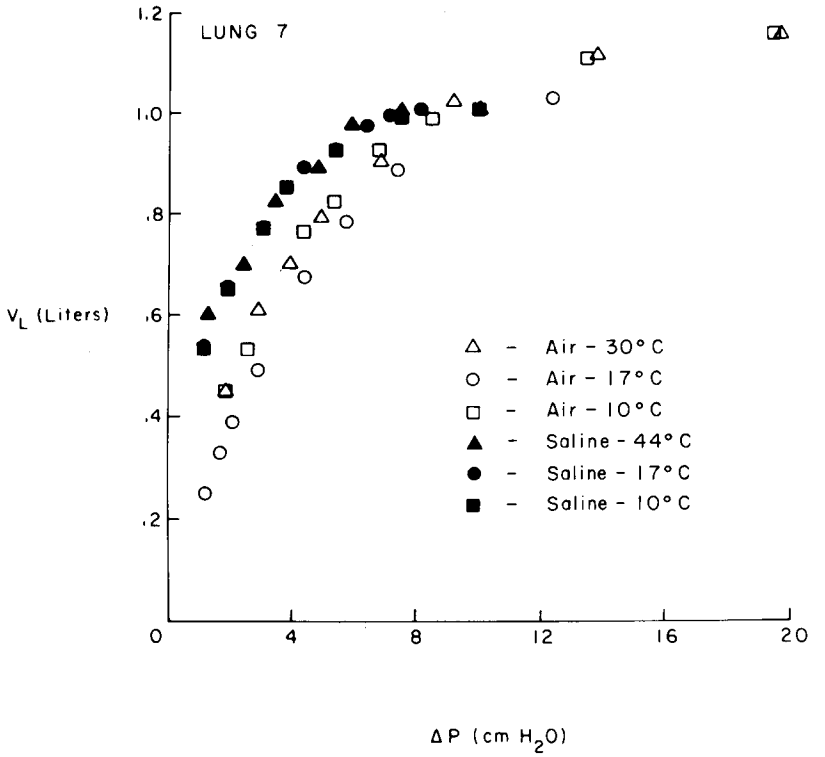

FIGURE 5. Saline and air pressure-volume deflation curves. Effect of temperature.

see a noticeable effect of temperature at very high and low temperatures (52°C and 5°C) in rabbit lungs.

With respect to the maximal sustainable pressure difference in saline, the literature is mute. Other investigators (2,9) have reported pressure-volume curves in saline up to 10 or 12 cm water, but do not reveal what occurred at higher pressures. Frank et al. (5) apparently reached 20 cm water, but report correcting for leaks by assuming that the leak rate was proportional to distending pressure. It is difficult to assess these results without more information. In our experiments, exceeding these limits did not appear to damage the lung despite the seepage described above. We noted total lung capacities in saline ranging from 8 to 15 cc/g of wet lung tissue, which compares well with the 15 cc/g reported by Faridy (2) and the 9.5 to 11 cc/g specified by Hoppin (9) when their data are corrected for lung tissue volume.

As we noted previously, saline appeared to cross from the alveolar spaces into the vessels. We do not know whether this extra compartment represents only the large vessels or whether it consists of the large vessels and the pulmonary capillaries. Frank et al. (5) found that vascular pressures over the range of 0–8 cm water did not significantly affect the saline pressure-volume curves of cat lungs and that vascular volume was less than 5% of lung vol-

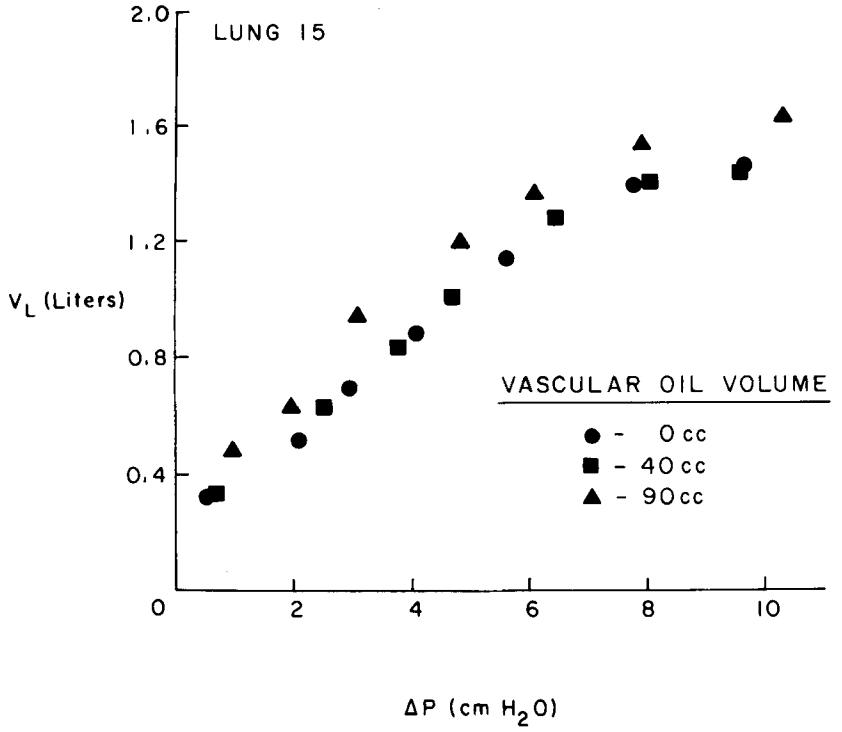

FIGURE 6. Effect of vascular oil on saline pressure-volume curve.

ume over most of the pressure range tested. Therefore, the effects of this extra space are presumably small and safely neglected.

Our data also show that at a given vascular oil volume, the lung has a slightly higher total volume than it would have if the vasculature were permitted to fill with the saline that crosses the alveolar-capillary membrane. Nevertheless, the general curve shape and the local slopes appear unaffected by the presence of oil in the vasculature. Similar results have been shown by Urmey et al. (18) in rat lungs. This is not what we expected. We had anticipated that distention of the capillary wall might introduce a prestress before any transpulmonary pressure difference is applied, altering the pressure-volume curve in the process. Thus, at the transvascular pressures we employed (3 to 7 cm H_2O), which is approximately that found in normal, intact lungs, the vascular space behaved as an extra compartment. The distention of this compartment produced by these pressures did not affect the behavior of the bulk lung tissue. The few experiments attempting similar control of vascular pressure report no effects on the pressure-volume curve (3,5).

Theoretical Aspects

The questions of homogeneity, isotropy, and uniformity of deformation can now be considered. Our data relating surface deformations and volume

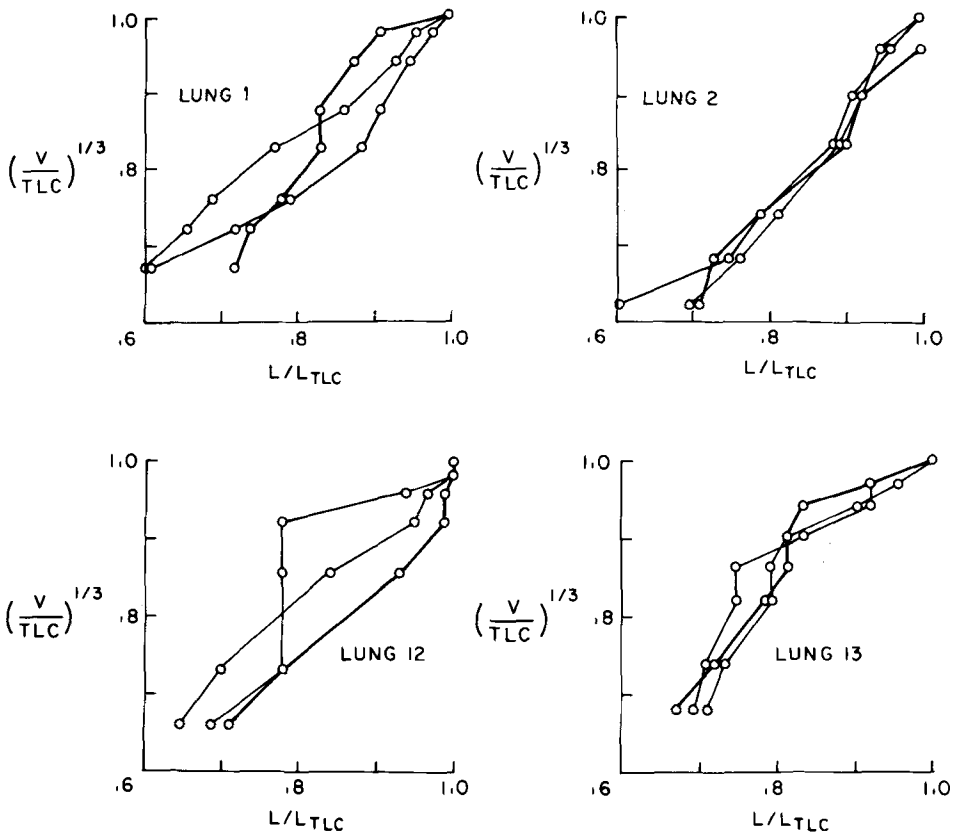

FIGURE 7. Cube root of volume ration versus surface length ratios for both air-filled lungs (1 and 2) and saline-filled lungs (12 and 13).

change indicate that no large consistent deviations from uniform deformation occur in normal lungs with either air or saline filling. The Eulerian force balance showed that gravity could be a significant body force in air, particularly at low lung volumes where the applied stresses are small. In support of this, Kallok and Lai-Fook (11) did find slight differences in lung volume at a given transpulmonary pressure when dog lobes were supported at various orientations to a gravity field during air filling. Katsura (12), however, was generally correct when he felt that the airways provided sufficient support that gravity orientation would not matter and that the airways supported the alveoli "much like the limbs support the leaves" of a tree. Thus, even in air, departures from uniform deflation appear to be small. Furthermore, although morphometric analyses, studies of macroscopic tissue blocks, and observations of pleural surface deformations are variously quoted both to support and to refute pulmonary homogeneity and isotropy, the bulk of the evidence as reviewed by several authors indicates that normal lung tissue is both homogeneous and isotropic (7,17).

The implications of these conclusions for mechanics are severalfold. First, homogeneity and isotropy result in mechanical properties that are relatively

constant throughout the lung, at least for lungs that are not affected by patchy areas of disease. Second, consistent with data from the saline experiments, theory predicts that uniform deformation would occur if body forces were minimized *and* pleural effects were negligible. The occurrence of uniform deformation would greatly simplify the analysis of the pressure-volume data by making sophisticated computational (e.g., finite-element) techniques unnecessary. Third, grossly uniform deformation that occurs during the air experiments implies (at least for normal lungs) that the complicated computational techniques may be needed only to investigate small, secondary phenomena, such as minor distortions caused by lung weight during experiments with excised air-filled lungs.

Finally, for any isotropic body the constitutive relationship depends on certain combinations of the strains known as the strain invariants. When uniform deformation occurs, the strain invariants are such that some of the mechanical properties combine and are indistinguishable. The questions of whether the model parameters can be easily estimated, whether they are well-determined, and whether they differ significantly in health and disease have never been addressed. The complexity of tissue block experiments required to evaluate all the mechanical properties may prove to be unwarranted if (a) those parameters that *can* be obtained from whole lung pressure-volume data discriminate sufficiently between normal and diseased lungs or (b) the lung's behavior proves to be relatively insensitive to the changes in parameter values so that mechanical properties may vary widely among normal lungs. In the latter case, some empirical parameters related to pressure-volume curve shape may be more reliable indicators of disease than the mechanical properties obtained from a sophisticated analysis.

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NOMENCLATURE

A = Surface area
E = Strain tensor
F_i = Applied forces
f = Functional stress-strain relationship
g = Acceleration of gravity
L = Resultant surface load
ΔP = Transpulmonary pressure difference
r = Principal radii of curvature
S = Surface tension
T = Temperature
V = Volume
W = Weight
∇ = Gradient operator
Σ = Stress tensor
ρ = Density
θ = Parameter vector
τ = Pleural tension per unit length