Stress, Deformation, and Atelectasis of the Lung

By Yuan-Cheng Fung

ABSTRACT
The lung parenchyma as a tissue has a rather unusual stress-strain relationship. A theoretical derivation of this relationship is presented which connects the surface tension and the tissue elastic stress in the alveolar septa with the alveolar geometry. The mathematical expression contains a few meaningful physical constants which can be determined by in vitro and in vivo experiments. With this stress-strain relationship, the general equations of lung mechanics are formulated, and solutions to some simpler problems are presented. First, the equilibrium of a lung subjected to a uniform inflation pressure (definition: alveolar air pressure − intrapleural pressure − pleural tension × mean curvature of pleura) is analyzed, and the stability of the equilibrium states with respect to small perturbations is examined. Second, an exact solution for a lung in a chest under the influence of gravity is presented; the solution is "exact," of course, for only a particular lung, but it can serve as a standard to check numerical procedures being developed in many laboratories. Finally, three types of possible atelectasis—planar, axial, and focal—are analyzed. The planar type can exist in a normally inflated lung, provided the layers of alveoli are forced to collapse toward a plane by some external agent. But axial atelectasis (alveoli collapse into a cylinder) can occur only if the dimension of all alveoli in the direction perpendicular to the axis is smaller than the resting dimension (at which the elastic tension in the alveolar septa vanishes). Similarly, focal atelectasis can occur only if the entire lung is smaller than the resting volume.

When a lung is inflated, the alveolar septa are stretched. This stretching of the liquid-air interface as well as the connective tissues induces stresses in the alveolar septa. The collective interaction of these stretched septa can be described macroscopically in terms of tractions, i.e., forces per unit area acting on any imagined small plane cross-sectional area in the lung. If the cross sections are chosen parallel to the coordinate planes, then the components of the tractions in the directions of the coordinate axes are called stresses or, more specifically, macroscopic stresses. This definition of stress rests on the assumption that tractions can be defined, i.e., that the limiting value of the force-area ratio exists in any cross-sectional plane. When the limit is approached from the vantage point of the alveoli, the macroscopic stresses can be defined only if the cross-sectional area is sufficiently large to include a fairly large number of alveoli so that the force-area ratio can be statistically stabilized. Limiting the analysis to such a level of scale (with linear dimensions larger than several alveolar diameters, e.g., on the order of 1 mm for human lung), the macroscopic stresses can be related to the macroscopic strain of the lung, and the global equilibrium and the stability of the lung can be evaluated. Information gained from such a global analysis can be supplemented by an analysis of the microscopic structure, in which the interaction of individual alveolar septa is considered.

The macroscopic stress-strain relationship of the lung parenchyma depends on the geometry of the alveoli and the surface tension and elasticity of the alveolar septa. I have previously presented a theoretical derivation of the stress-strain relationship of the lung parenchyma (1), but I did not consider the stress due to surface tension in detail. In the present paper, the macroscopic stress due to surface tension is analyzed, and its relationship to deformation and atelectasis is considered. Furthermore, an explicit formula is proposed for the elastic stress in the alveolar septa. This formula is based on the assumption that the constitutive equation for the alveolar septa is similar to those for other connective tissues tested in my laboratory. Synthesizing these results leads to an explicit stress-strain relationship for the lung parenchyma. The constants involved in this relationship can be determined from in vitro and in vivo experimental results.

With the constitutive equation known, the general equations of lung mechanics are formulated. Many problems await solution, and the solutions to three are presented: the uniform inflation of a lung,
the nonuniform stress distribution in a lung due to its own weight, and three possible modes of atelectasis.

Pulmonary mechanics have been studied extensively by many authors over many years. Clements and Tierney (2), Mead (3), Mead et al. (4), and Radford (5) have all presented critical reviews of the literature and outlines of their own contributions. At first, attention was directed mainly at the pressure-volume relationship, but since the paper by Mead (3) was published details of stress distribution have attracted attention. Recently, West and Matthews (6) have presented an approximate numerical analysis of the effect of gravity and thoracic wall deformation on the stress distribution in the lung. Lambert and Wilson (7) have analyzed small perturbations of an inflated lung and applied them to the problem of expiratory flow. Other analyses are rapidly appearing. It seems useful, then, to clarify the fundamental features of the mechanics of the lung, which as a material has no counterpart in conventional solid mechanics.

In this paper, a general description of the method of approach and the principal results obtained is presented and then a detailed analysis is given. It was necessary to present the detailed analysis in mathematical form, because otherwise it would have been too wordy and boring.

**General Description**

**LIST OF PRINCIPAL SYMBOLS**

<table>
<thead>
<tr>
<th>Reference state</th>
<th>Resting state of lung at zero inflation pressure when surface tension is abolished.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A, A_max, A_min</td>
<td>Surface area and maximum and minimum surface area in surface tension tests.</td>
</tr>
<tr>
<td>a, a_1, a_0, a_2</td>
<td>Constants in Eqs. 11, 13, 29, and 30.</td>
</tr>
<tr>
<td>b, b_1, b_2</td>
<td>Constants in Eqs. 11, 13, and 14, respectively.</td>
</tr>
<tr>
<td>e_1, e_2</td>
<td>Two-dimensional strain in membranes (see Eq. 11).</td>
</tr>
<tr>
<td>g</td>
<td>Gravitational acceleration.</td>
</tr>
<tr>
<td>k</td>
<td>Constants defined under Eq. 30, n = 1, 2, 3 ...</td>
</tr>
<tr>
<td>n</td>
<td>Unit vector normal to an alveolar septum, with components n_1, n_2, and n_3.</td>
</tr>
<tr>
<td>N_1, N_2, N_3</td>
<td>Tensile force per unit length acting in alveolar septa and pleura, respectively; N acts in the x direction in septa parallel to the x_3 axis.</td>
</tr>
<tr>
<td>p, P</td>
<td>Pressure and work done by inflation pressure, respectively.</td>
</tr>
<tr>
<td>r, θ, φ</td>
<td>Radius vector, polar angle, and azimuth angle, respectively.</td>
</tr>
<tr>
<td>s, ds</td>
<td>Unit vector tangent to the trace of intersection between an alveolar septum and a cross section; ds is the length of an infinitesimal element tangent to s.</td>
</tr>
</tbody>
</table>

**FUNG**

T_1, T_2, T_3 = Lagrangian stress components (see Eqs. 12, 15, and 17). 

u_1, u_2, u_3 = Displacements from a uniformly inflated state. 

V = Potential energy = strain energy - work done by inflation. 

W, ρ_0W = W is strain energy per unit mass of lung tissue and ρ_0W is that per unit volume of tissue in the reference state. 

x_1, x_2, x_3 = Coordinate axes in a rectangular Cartesian system. 

α_{12}, β_{13} = Constants defined under Eq. 30, n = 1, 2, 3 ... 

B_i = The vector s_i × x_i (see Fig. 8). 

γ, γ_max, γ_min = Surface tension (force/length) and the maximum and minimum surface tension in cyclic experiments. 

Δ = Linear dimension of an alveolus in the reference state. 

e_r, e_p, e_t = Strains in polar coordinates (see Eq. 34). 

κ = Sum of principal curvatures of the pulmonary pleura. 

λ_1, λ_2, λ_3 = Stretch ratios in the directions of x_1, x_2, and x_3, respectively. 

ξ = Dimensionless variable for area variation in surface tension experiments (see Eq. 8). 

D = Density of the lung in the deformed and reference states, respectively. 

σ_11, σ_22, σ_33 = Principal Cauchy stresses (macroscopic). 

σ_12, σ_23, σ_31 = Shear stresses. 

Subscript o = Refers either to a uniformly inflated state or to the reference state. 

Subscripts 1, 2, and 3 = Refer to directions of x_1, x_2, and x_3, respectively, or to planes parallel to x_1, x_2, and x_3. 

Subscript A = Alveolar air. 

Subscript PL = Pulmonary pleural surface. 

Superscript (e) = Elastic tissue. 

Superscript (s) = Surface tension. 

**STRESS-STRAIN RELATIONSHIP**

If one imagines a plane cutting a small piece of lung parenchyma, the interalveolar septa beneath the plane will appear (to an observer above the plane) as they are illustrated in Figure 1. Each interalveolar septum has two moist surfaces which have surface tension. The stretching of these septa due to the inflation of the lung creates elastic tension in the wall. At the cut surface of the cross section, each septum tries to pull away. The sum total of the forces acting on the cross section per unit area of the cross section is called the traction. The traction can be computed by adding together the tensions from all of the septa in the unit cross-sectional area. Now, let x_1, x_2, and x_3 be a set of rectangular Cartesian coordinates. Let the traction acting on a plane perpendicular to the x_3-axis be denoted by T_3. T_i is in general not parallel to the x_i-axis. Let the three components of the vector.
LUNG STRESS, DEFORMATION, AND ATELECTASIS

T. I in the direction of the coordinate axes $x_1$, $x_2$, and $x_3$ be denoted by $\sigma_{11}$, $\sigma_{12}$, and $\sigma_{13}$, respectively. Then, $\sigma_{11}$ is the normal stress acting on a plane section perpendicular to the $x_1$-axis, $\sigma_{12}$ is the shear stress acting in the direction of $x_2$ on a plane perpendicular to the $x_1$-axis, and $\sigma_{13}$ is the shear stress acting in the direction of $x_3$ on a plane perpendicular to the $x_1$-axis. Similarly, the traction acting on a plane perpendicular to the $x_2$-axis (or the $x_3$-axis) has the three components $\tau_{21}$, $\tau_{22}$, and $\tau_{23}$ (or $\tau_{31}$, $\tau_{32}$, and $\tau_{33}$). It can be shown that $\sigma_{12} = \sigma_{21}$, $\sigma_{23} = \sigma_{32}$, and $\sigma_{31} = \sigma_{13}$. Hence, at any point in the lung there are three independent normal stresses, $\sigma_{11}$, $\sigma_{22}$, and $\sigma_{33}$, and three independent shear stresses, $\sigma_{12}$, $\sigma_{23}$, and $\sigma_{31}$.

As I have said before, the stresses $\sigma_{11}$, $\sigma_{12}$, etc. must be related to the deformation of the lung. To describe the deformation, one must refer it to a reference state. I choose to define the reference state of a lung as the configuration taken up by the lung when the surface tension is abolished and there is no inflation pressure. In a real lung, this state can be approached if the airspace is infused with saline and the alveolar pressure is equal to the pleural pressure. Obviously, such a lung exists only in mathematics. It is so simple that the necessary calculations can be carried out to bring out the functional form of the stress-strain relationship. The proposed form can then be checked against experimental results on the real lung.

For this idealized lung, the alveoli are cubes of dimension $\Delta$ in the reference state. When the lung is inflated, the edge lengths become $\lambda_1\Delta$, $\lambda_2\Delta$, and $\lambda_3\Delta$ in the directions of the coordinate axes $x_1$, $x_2$, and $x_3$, respectively, which are parallel to the edges of the cubes. These $\lambda$ values are the principal stretch ratios. The spacing of the membranes (interalveolar septa) is thus increased. In each of these membranes, a surface tension, $2\gamma$ (dynes/cm, two surfaces to each membrane), and an elastic tension, $N$ (dynes/cm), act. To be specific, the tension in the membranes parallel to the $x_1$-axis in a cross section perpendicular to the $x_1$-axis is denoted by $2\gamma_{11} + N_{11}$ (dynes/cm). Similarly, $2\gamma_{12} + N_{12}$ is the tension in the $x_2$-direction acting in the membranes parallel to the $x_2$-axis.

Now, imagine a small cubic element in the reference state with edge length $\Delta$ (Fig. 2). When the lung is deformed, the cubic element becomes a parallelepiped with lengths $\lambda_1\Delta$, $\lambda_2\Delta$, and $\lambda_3\Delta$. The numbers $\lambda_1$, $\lambda_2$, and $\lambda_3$ are called stretch ratios.

It can be proved that at any point in an arbitrarily deformed body there exists at least one cubic element in the reference state which is deformed into a rectangular parallelepiped. The directions of the edges of this rectangular parallelepiped are called the principal directions. The stretch ratios $\lambda_1$, $\lambda_2$, and $\lambda_3$ in the principal directions are called the principal stretch ratios. If the principal directions and the principal stretch ratios are known, the stretch ratios of line elements in any other direction can be computed. Hence, the principal directions and stretch ratios define the deformation.

Now, to derive a relationship between stress and deformation for the simplest idealized case, imagine a lung that is composed of an aggregation of alveoli which are cubes in the reference state and rectangular parallelepipeds in the deformed state with their edges parallel to the principal axes of strain. This lung is made of rectangular alveolar septa arranged in an array of parallel planes and stretched in the direction of the planes. Obviously, such a lung exists only in mathematics. It is so simple that the necessary calculations can be carried out to bring out the functional form of the stress-strain relationship. The proposed form can then be checked against experimental results on the real lung.
Now, consider a square cross section of unit area perpendicular to the \( x_1 \)-axis. This cross section cuts \( 1/(\lambda_2 \Delta) \) membranes in a unit distance in the \( x_2 \)-direction and \( 1/(\lambda_3 \Delta) \) membranes in a unit distance in the \( x_3 \)-direction. Each membrane contributes a tension as mentioned earlier. The sum of the tensions from all of the membranes cut by this cross section of unit area is, therefore, the macroscopic stress, \( \sigma_{11} \):

\[
\sigma_{11} = \left[ 2\gamma + N_{12}^{(e)} \right] \frac{1}{\lambda_2 \Delta} + \left[ 2\gamma + N_{13}^{(e)} \right] \frac{1}{\lambda_3 \Delta}
\]

If it is known how \( \gamma \) and \( N^{(e)} \) vary with the stretch ratios \( \lambda_1 \), \( \lambda_2 \), and \( \lambda_3 \), then Eq. 1 defines the macroscopic stress-strain relationship of the lung material. Stresses \( \sigma_{22} \) and \( \sigma_{33} \) can be obtained from the same formula by a cyclic permutation of the subscripts 1, 2, and 3.

Although Eq. 1 is derived for an idealized lung, it can be applied to a real lung by expressing \( \gamma \) and \( N^{(e)} \) in appropriate mathematical forms which contain a few undetermined constants and using Eq. 1 to solve some boundary-value problems which can be simulated in the laboratory either in vivo or in vitro. The experimental results and the mathematical solution can be compared to determine the unknown constants. These constants are then regarded as the physiological constants of the lung.

**STRESS DUE TO SURFACE TENSION**

Separate \( \sigma_{11} \) into two terms, \( \sigma_{11}^{(e)} \), the stress due to elastic tissue, and \( \sigma_{11}^{(s)} \), the stress due to surface tension:

\[
\sigma_{11} = \sigma_{11}^{(e)} + \sigma_{11}^{(s)},
\]

\[
\sigma_{11}^{(e)} = \frac{1}{\Delta} \left[ N_{12}^{(e)} \frac{1}{\lambda_2} + N_{13}^{(e)} \frac{1}{\lambda_3} \right],
\]

\[
\sigma_{11}^{(s)} = \frac{2}{\Delta} \left[ \frac{\gamma_{12}}{\lambda_2} + \frac{\gamma_{13}}{\lambda_3} \right].
\]

These two terms can be considered separately.

Consider the surface tension term. If \( \gamma_{12} \) and \( \gamma_{13} \) are constant, then according to Eq. 2c, \( \sigma_{11}^{(s)} \) is independent of the stretch, \( \lambda_1 \), and an increase in the transverse stretch, \( \lambda_2 \) or \( \lambda_3 \), decreases the stress, \( \sigma_{11}^{(s)} \). Such a material is certainly new to the theory of elasticity and structures; it is roughly equivalent to a material with a Young’s modulus of zero and a Poisson’s ratio that is negative. In reality, the surface tension does increase with increasing area, so the Young’s modulus of \( \sigma_{11}^{(s)} \) is positive. But the effect of alveolar size through the inverse ratios \( 1/\lambda_2 \) and \( 1/\lambda_3 \) is significant and unmistakable.

It is well known that (1) the surface tension vs. area curves of pulmonary surfactants have large

**FIGURE 2**

Deformation of a group of idealized alveoli. At the reference (or resting) state, the alveoli are assumed to be cubic, with edge length \( \Delta \). When they are deformed, the lengths of the edges become \( \lambda_1 \Delta \), \( \lambda_2 \Delta \), and \( \lambda_3 \Delta \) in the direction of the coordinate axes. \( \lambda_1 \), \( \lambda_2 \), and \( \lambda_3 \) are defined as the stretch ratios.
hysteresis loops, (2) the response to a periodic change of area can reach a steady state (i.e., the loops become unique) only after many cycles, and (3) the values of the surface tension in cyclic changes of area depend on the limits of the area changes and, to a minor degree, on the frequency of the oscillations. An example of a surface tension vs. area loop is shown in Figure 3a for dog lung surfactant on a spherical bubble (8).

Once the surface tension is known as a function of area, it can be given a convenient mathematical expression, and the macroscopic stress, \( \sigma_{ls} \), can be obtained according to Eq. 2c. A difficult question must be answered: how is the area of the surface of the testing bubble or the Wilhelmy balance related to the area of the lung? Since a clear-cut answer is not available, two different cases are illustrated in Figure 3b. Consider a uniformly inflated lung \( (\lambda_1 = \lambda_2 = \lambda_3 \text{ and } \sigma_{11} = \sigma_{22} = \sigma_{33} ) \) so that the stress can be simply written as \( \sigma \). For the figure to the right, it was assumed that 25% of the area in Slama’s bubble corresponds to the reference state of the lung, at which \( \lambda = 1 \), and that 100% of the area corresponds to \( \lambda = 2.0 \), doubling the length. In the figure to the left, it was assumed that 25%, 62.5%, and 100% of the bubble area correspond to lung stretch ratios of \( \lambda = \sqrt{2} \), \( \sqrt{2} \), and \( \sqrt{2} \), respectively. The \( \lambda^2 \) scales are shown on the abscissa in Figure 3a. Dividing \( \gamma \) by \( \lambda \) according to Eq. 2c and multiplying by \( 4/A \), assuming that \( A = 10^{-2} \text{ cm} \), gives the macroscopic stress, \( \sigma_{ls} \), due to surface tension. Note the difference in the shape of these curves due to the difference in the association of surfactant testing bubble area with the lung stretch ratio \( \lambda \).

**Elastic Tissue Stress**

The elastic tissue stress can be measured in a saline-filled lung. The central question is how to derive a general, three-dimensional stress-strain relationship applicable to arbitrary deformations from data obtained from simpler experiments. This question is dealt with in the detailed analysis later in this paper. Generally speaking, the elastic stress, \( \sigma^{(e)} \), increases rapidly with increasing strain, at such an accelerated rate that the stress is an exponential function of the strain. This situation is illustrated in Figure 3c for the case of uniform expansion. The strain-rate effect is minor, but a small hysteresis loop exists.

One aspect remains speculative: would the elastic tissue be able to sustain a compressive stress when the strain becomes compressive (at a lung dimension less than that at the reference state, when \( \lambda < 1 \)?) At the present time, I can only speculate, because none of the testing machines can impose a compressive stress (9, 10). One can reason that, since the alveolar septa are extremely thin and because they are extremely soft when the stress level is low, i.e., they have a very small Young’s modulus when \( \sigma^{(e)} \to 0 \), they will buckle under compression and that the critical buckling load will be so small as to be insignificant. If this is true, then the stress-strain curve should become a horizontal line at \( \sigma^{(e)} = 0 \) for \( \lambda < 1 \).

But if the alveolar dimension is small and the interalveolar septa are thick, the critical buckling load may be high enough to enable the tissue to take up compression without buckling. Then, a negative stress will be obtained for a stretch ratio, \( \lambda \), of less than 1, as sketched in Figure 3c. The
experiments of Stemmler and Du Bois (11) on rabbit lung seem to show that rabbits belong to this category. The length-thickness ratio of the interalveolar septa of the rabbit is probably in the range of 6 to 12, small enough to be qualified as thick-walled in the theory of structures. For the human lung in an upright position, the length-thickness ratio is larger near the apex, smaller near the base, larger in older or emphysema patients, and smaller in the young and healthy. The smaller the ratio, the higher is the critical buckling load, and the more able is the tissue to take compressive stress.

GENERAL LUNG MECHANICS

Adding the curves of Figure 3b and c together yields the stress-strain relationship shown in Figure 4a. In the range \( \lambda < 1 \), the broken curves in Figure 4a (left) represent the case in which the alveolar septa are capable of taking compressive stress without buckling and the solid curves correspond to the case in which the alveolar septa will buckle at a negligible compressive load whenever \( \lambda < 1 \), corresponding to the horizontal curve for \( \lambda < 1 \) in Fig. 3c. For Figure 4a (right),

the region \( \lambda < 1 \) cannot be examined because the minimum area chosen for the surface tension tests corresponds to \( \lambda = 1 \).

Having determined the stress-strain relationship for the lung tissue as a material, the lung can be treated as a continuum enclosed in the visceral pleura and attached to larger blood vessels and airways. The problems of statics and dynamics of the lung are then reduced to the usual equations describing conservation of mass, momentum, and energy and appropriate boundary conditions.

STATIC UNIFORM INFLATION

The mathematical solution to the equations describing a uniform inflation of the lung, ignoring its weight and the local effects of large blood vessels and airways, is simply that \( \lambda_1 = \lambda_2 = \lambda_3 = \text{constant} \). The stress in the lung balances the inflation pressure which is equal to the airway pressure \( (p_A) \) minus the pleural pressure \( (p_{PL}) \) minus the product of the membrane tension in the visceral pleura \( (N_{PL}) \) and the mean curvature of the pleura \( (K_{PL}) \), i.e., \( p_A - p_{PL} - N_{PL}K_{PL} \).

The nature of the solution is illustrated in Figure 4a. The solution to the equilibrium problem is given by the intersections of the stress-strain curves with a horizontal line representing the equation \( \sigma = p_A - p_{PL} - N_{PL}K_{PL} \). Such a horizontal line intersects the \( \sigma \) vs. \( \lambda \) curve for inspiration (or expiration) at either two points, one point, or no point (when the stress is less than \( \sigma_{cr} \) and the alveolar wall buckles at infinitesimal compressive stress, as represented by the solid curve in Fig. 4a, left). The first two cases yield the inflation (deflation) pressure-deformation relationship. Since lung volume is equal to \( \lambda^3 \) times the lung volume in the reference state, the curves of Figure 4a can be replotted as pressure-volume relationships as shown in Figure 4b, where the volume is plotted on the ordinate and the pressure on the abscissa, as is the usual practice for such curves.

The case of subcritical inflation at a pressure less than \( \sigma_{cr} \) shown in Figure 4a for the zero-buckling load case (solid curves) needs more attention. Since a horizontal line representing the boundary condition when \( \sigma < \sigma_{cr} \) does not meet the stress-stretch curve, the equilibrium problem has no solution. In other words, the lung cannot be kept at a uniformly inflated condition in this case. The deformation must become nonuniform. One possibility is for some portion of the lung to become atelectatic so that the number of alveolar surfaces open to air is reduced, decreasing the stress \( \sigma^{*\prime} \) in the remaining part of the lung, which can remain
open and in equilibrium. Three types of atelectasis will be considered later in this paper.

STABILITY

The word instability is like the word disease: it has no unique meaning. There are many kinds of instabilities, each requiring a separate method of investigation. The atelectasis at subcritical inflation pressure discussed at the end of the previous section is a form of instability which can be analyzed by considering the specific deformation patterns. A different type of investigation is concerned with small perturbations of an equilibrium state. For example, will a uniformly inflated lung perturbed in a small but arbitrary way have a tendency to return to the equilibrium condition? If it does, the equilibrium state is stable.

In the section on stability in the second part of this paper, it is shown that the inflated configuration is stable, except possibly when the lung dimension is subcritical. The central idea of the proof consists of three steps. First, the equilibrium condition is solved. Second, all possible infinitesimal perturbations (virtual displacements) from the equilibrium which are consistent with the boundary conditions are considered, and the change in strain energy, \( \delta(\rho_0 W) \), and the work done by the inflation pressure, \( \delta P \), are computed. Third, if the difference \( \delta(\rho_0 W) - \delta P \) is always positive and is zero only when the perturbation vanishes, then the system is stable; otherwise, it is unstable.

The basic argument of this approach is a thermodynamic one. The difference \( \delta(\rho_0 W) - \delta P \) is the change of potential energy due to an arbitrary small perturbation from equilibrium. The system is stable when this potential energy is a minimum. The key to the argument is the arbitrariness of the small perturbations; no restriction is imposed other than the physical boundary conditions. The execution of this proof requires an expression for the strain energy function, which is derived later in this paper.

EQUILIBRIUM OF THE LUNG UNDER ITS OWN WEIGHT

A realistic problem of lung mechanics is to determine the distribution of intrapleural pressure, stress, and strain when the lung is confined within its boundary (chest wall) and subjected to the gravitational force due to its own weight. It turns out that the solution to this simple problem is not easy. Because of the nonlinear stress-strain relationship and the finite deformation, numerical methods must be resorted to. It is highly desirable to have a simple, exact, analytical solution which can serve as a basis for checking any numerical procedures and computing programs. Such a solution is given in the second part of this paper in the section on states of equilibrium. It is merely a lung in hydrostatic equilibrium. The necessary geometry of the lung at the reference state is computed afterwards.

THREE POSSIBLE FORMS OF ATELECTASIS

A different problem is to find whether other equilibrium states exist which can be identified with atelectasis. For this purpose, consider the following possible types of localized perturbations of an inflated lung: (1) atelectasis of the focal type, in which the alveoli collapse toward a central focus (Fig. 6), (2) atelectasis of the axial type, in which the alveoli collapse toward a line (Fig. 6), and (3) atelectasis of the planar type, in which the alveoli collapse toward a plane (Fig. 7).

In each case it is assumed that at the core a number of the alveolar septa are coalesced. When two alveolar septa touch each other, their liquid coverings will fuse, thus eliminating surface tension. Pressure, tension, and shear stress can be transmitted through the coalesced alveolar septa. Immediately next to these coalesced septa are the open alveoli, whose walls are moist, on which surface tension acts. The dimensions of the alveoli next to the coalesced region must be reduced one way or another because of the necessary continuity of the membranes (septa). The closeness of these membranes increases the macroscopic stress due to
surface tension and decreases that due to elastic response. Farther away from the atelectatic core, the alveoli are less and less influenced by the localized perturbation; they ultimately are like those of the normally inflated lung.

There are, however, considerable differences in details with respect to the three types of atelectasis. For the planar type, it can be shown that the transition from the coalesced region to that of the normally inflated alveoli can be immediate. In other words, a planar atelectasis can exist in an inflated lung. Since an inflated lung is stable with respect to all infinitesimal disturbances, such a planar atelectasis cannot occur by itself but must be introduced by some finite disturbances, such as an obstruction in the airway, a compression by a tumor, a pressure by a surgeon's finger, or a temporary local reduction of stress, \( \sigma \), to a level below the critical stress, \( \sigma_{cr} \), by a decrease in either alveolar air pressure or an increase in intrapleural pressure. Because it can coexist with an inflated lung, a planar atelectasis can be quite persistent.

For atelectasis of the axial and the focal type, three regions must be distinguished (Fig. 6): (1) the zone of collapsed alveoli, \( r \leq a \), which is filled with liquid and tissue, (2) the zone of reduced alveoli, \( r < b \), in which the alveolar dimension is smaller than that of the reference state and the elastic tension in the alveolar septa either goes out of action or becomes compressive, and (3) the normally inflated zone, \( r \geq b \). The equations of equilibrium for the first two zones are easily solved, but a peculiar result, which is surprising at first sight but reasonable when you think about it, is obtained. The result is that the stretch ratio in the zone of reduced alveoli is uniform everywhere; as a consequence, the radius \( b \) cannot be found inside the lung. There is no gradual transition out of this zone into the normally inflated zone within the lung. It follows that for the axial atelectasis to
occur the alveolar dimension in planes perpendicular to the axis must be smaller than the reference dimension, \( \Delta \), for the entire lobe. (Recall that the dimension in the reference state is that of a saline-filled lung at zero inflation pressure.) The elastic tissues in alveolar septa are stressed only in the axial direction. Similarly, a focal atelectasis to occur the entire lung volume must be so small that all alveoli are smaller than their dimension in the reference state.

Finally, note that a focal atelectasis is embedded in a uniform stress field. Therefore, if one focus exists, there can be another, and many foci can appear simultaneously under the same condition. A similar comment applies to the axial atelectasis in the case of axially symmetrical compression. It follows also that focal and axial atelectasis can be removed by inflating the lung to a larger size.

How can a planar atelectasis be pulled out? It is not effective to pull the planar coalesced region which can transmit tensile stress. But the edges of a plaque of planar atelectasis must behave somewhat like half of an axial atelectatic core. Since there is no axial symmetry, the condition there is quite complex, but it is clear that to remove a planar atelectasis one should work on the edges. Overinflation will pull the alveolar septa at the edges out like an accordion. One needs to overcome, in this process, not only the surface tension of the newly created interfaces but also the viscosity and friction between the septa because of their relative motion. Therefore, the duration of overinflation should not be too brief. But excessive positive airway pressure compresses the alveolar capillaries and decreases the blood flow; hence, the duration of overinflation should not be too long. Intermittently applied positive-pressure breathing at a suitable frequency or negative pressure applied to the chest or abdomen are logical procedures. When a surgeon massages a lung after an operation to remove signs of atelectasis, he is applying this principle. The same principle tells why it is difficult to open an atelectatic plaque which borders on a pleura where the peeling action cannot be introduced.

**Discussion**

In this investigation, I explicitly used the simplified stress-strain law (Eq. 1) only in the stability problem. The question arises of whether the conclusions on atelectasis are valid beyond the original simplifying assumption. The answer can be obtained by considering a more realistic geometry for the alveoli. For example, if the ensemble mean of the alveoli at the resting state is a sphere, the method of reference 1 can be used to show that the conclusions remain qualitatively correct.

When the equilibrium conditions of atelectasis are not satisfied, motion ensues. To investigate the dynamics of atelectasis, one must study the nonequilibrium conditions.

It appears that of the three types of atelectasis considered, the planar type is the only one that can exist in the normally inflated lung. Perhaps the planar type is the most prevalent of all atelectases, but the literature has been rather vague about the details of atelectasis. These theoretical results are so suggestive that a detailed experimental investigation is warranted.

I did not consider the possible collapse (for one reason or another) of bronchus and bronchioles. Alveoli in the neighborhood of such collapsed vessels may be considered atelectatic also. Perhaps focal and axial atelectasis are associated with these events.

Fleischner (12) first described *platelike* atelectasis in roentgenograms of the lung. The linear shadows in the lung, variously called Fleischner's lines, platter atelectasis, or discoid atelectasis, have been discussed in detail by Fraser and Paré (13, p 301). These lines are undoubtedly shadows of planar atelectasis. Fraser and Paré (13, pp 196–239) following Robbins and Hale (14) and Lubert and Krause (15, 16) have presented detailed patterns of lobar and total pulmonary collapse. They have shown that when atelectasis is approached a collapsed lobe or segment tends to look like a curved plate whose edges tend not to retract from the chest wall and the mediastinum. Thus, an atelectatic lobe is also planar. No other shadows of atelectasis are mentioned in these references.

**Analysis**

**Macrosopic Stress and Alveolar Geometry**

Consider a plane cross section of unit area of the lung parenchyma as shown in Figure 1. It intersects a number of alveolar septa. Tension acts in these septa; the summation of this tension is the macrosopic traction. To account for this summation, consider an infinitesimal element of an alveolar septum as shown in Figure 8. Let \( \mathbf{n} \) be a unit vector normal to the septum. Let the cross section be perpendicular to the coordinate axis \( x_1 \), and let \( x_1 \), \( x_2 \), and \( x_3 \) be a set of rectangular Cartesian coordinates. Let \( x_1 \), \( x_2 \), and \( x_3 \) be unit vectors along the coordinate axes. The line of intersection of the planes normal to \( \mathbf{n} \) and \( x_1 \) has a unit tangent vector \( \mathbf{s} \), and a length of intercept \( ds \). On the line
of intersection the membrane tension in the septum can be resolved into two components, \( N_n \), normal to \( S_i \), and \( N_t \), tangential to \( S_i \). The resultant force \( N_n \cdot ds_i \) acts in the direction of \( n \times S_i \), whereas the resultant \( N_t \cdot ds_i \) acts in the direction of \( S_i \). (Boldface letters denote vectors, \( \times \) denotes vector products, and \( \cdot \) denotes scalar products.) The traction on a cross section perpendicular to \( S_i \) is, therefore,

\[
\int N_n(n \times s_i)ds_i + \int N_t s_ids_i,
\]

where the integration extends over all of the traces of the alveolar septa within the unit area cut by the plane perpendicular to \( S_i \). The components of this traction in the direction of the coordinate axes are the stresses

\[
\sigma_{11} = \int N_n(n \times s_i) \cdot x_ids_i,
\]

\[
\sigma_{12} = \int N_n(n \times s_i) \cdot x_2ds_i + \int N_t s_i \cdot x_2ds_i,
\]

\[
\sigma_{13} = \int N_n(n \times s_i) \cdot x_3ds_i + \int N_t s_i \cdot x_3ds_i.
\]

The integration extends over a unit area. Note that \( s_i \) is perpendicular to \( S_i \), so that \( N_n \) contributes nothing to \( \sigma_{11} \). Similarly, by a permutation of the subscripts 1, 2, and 3, the other components of stress, \( \sigma_{22}, \sigma_{23}, \sigma_{31}, \) and \( \sigma_{33} \), can be obtained.

A somewhat more explicit expression can be given to the stress \( \sigma_{11} \). Note that \( (n \times s_i) \cdot x_i = n \cdot (s_i \times x_i) \) and that \( s_i \times x_i \) is a vector \( B_1 \) lying in the plane of \( x_2, x_3 \) (Fig. 8). Hence, \( n \cdot (s \times x_i) \) is the component of the unit normal vector \( n \) along \( B_1 \). But \( n, B_1, \) and \( x_i \) are all perpendicular to \( s_i \); hence, they lie in the same plane. The component of \( n \) in the direction of \( x_i \) is the direction cosine \( n_i \). It follows that the component of \( n \) along \( B_1 \), which is perpendicular to \( x_i \), must be \( (1 - n_i^2)^{1/2} \). Thus,

\[
\sigma_{11} = \int N_n(1 - n_i^2)^{1/2}ds_i.
\]

An alternative expression for \( \sigma_{12} \) is

\[
\sigma_{12} = \int (N_n \cos \theta_{12} + N_t n_i \sin \theta_{12})ds_i,
\]

where \( \theta_{12} \) is the angle between \( s_i \) and \( x_2 \). This relationship can be seen in Figure 9, by noting that \( s_i \times x_2 \) is parallel to \( x_i \), with magnitude \( \sin \theta_{12} \), whereas \( n \cdot x_1 = n_i \).

Thus, it becomes evident that the macroscopic stresses in the lung parenchyma depend on the length of the traces per unit area of the cross sections parallel to the coordinate planes (integral of \( ds_i \)) and on the average values of the inclination of the alveolar septa to the coordinate planes, \( n_i, \theta_{12}, \) etc.

If \( x_1, x_2, \) and \( x_3 \) are the principal axes of the macroscopic stress tensor \( \sigma_{ij} \), then \( \sigma_{12} = \sigma_{23} = \sigma_{31} = 0. \) If \( N_n \) and \( N_t \) are statistically isotropic, then the integrals of \( n, \sin \theta_{12}, \) and \( \cos \theta_{12} \) as shown in Eq. 6 must vanish. This situation is possible if the lung structure is initially isotropic and if \( x_1, x_2, \) and \( x_3 \) are also the axes of the principal macroscopic strain. This fact suggests that the principal axes of the macroscopic stress and strain may be considered to coincide statistically, i.e., the lung parenchyma is statistically isotropic (the constitutive equation relating stress and strain is isotropic on the average). One must be careful, however, not to apply this argument to too small a region in space, because a small, individual sample may not have the statistical uniformity.

Apply these formulas to the idealized lung shown in Figure 2, and consider the cross section ABCD perpendicular to \( x_1 \) axis. The tension \( N_n \) is the tension in the septa AA'B'B and DD'A'A, etc. The direction cosine \( n_i \) is zero for all septa intersected by the plane ABCD. Hence, if \( N_n \) is written as a
LUNG STRESS, DEFORMATION, AND ATELECTASIS

sum of the elastic tissue stress, $N_n^{(e)}$, and the surface tension $2\gamma$ (two faces on each septum), then

$$\sigma_{11} = \int N_n^{(e)}(\lambda_1, \lambda_2, \lambda_3) ds_1 + \int 2\gamma ds_2.$$  (7)

This equation reduces directly to Eq. 1.

MACROSCOPIC STRESS DUE TO SURFACE TENSION, $\sigma_{ii}^{ms}$

To derive an explicit formula for $\sigma_{11}^{ms}$, a mathematical expression for the surface tensions $\gamma$, is needed. To formulate such an expression, use data such as those illustrated in Figure 3a. A difficulty to be resolved is to relate the area of the surface in the Langmuir-Wilhelmy trough or the test bubble to the stretch ratio of the lung. What stretch ratios of the lung correspond to the 100% area and the 25% area of the curves in Figure 3a? Each decision decides a curve of macroscopic stress, $(\sigma_{11}, \sigma_{22})$ vs. $X$, different decisions may lead to very different $\sigma_{11}^{ms}$ vs. $X$ relationships, as is amply demonstrated in Figure 3b. This difficulty can be resolved by comparing the theoretical results with experiments on the whole lung, such as the volume-pressure relationship.

Let $\gamma_{min}$, $\gamma$, and $\gamma_{max}$ be the surface tension at the minimum area ($A_{min}$), intermediate area ($A$), and maximum area ($A_{max}$) of a cycle, respectively. Define a dimensionless variable $\xi$

$$\xi = (A - A_{min})/(A_{max} - A_{min}).$$  (8)

Then the curves as shown in Figure 3a can be represented by the equation

$$\gamma = \gamma_{min} + (\gamma_{max} - \gamma_{min}) \left( \xi + \sum_{n=1}^{m} c_n \sin n\pi \xi \right).$$  (9)

The first two terms on the right represent a straight line joining the extremities of the hysteresis loop; the rest (Fourier series) gives the loop about this line. One set of constants, $c_n$, applies to inspiration, and another set applies to expiration.

Apply Eqs. 8 and 9 to the membranes of the alveolar model shown in Figure 2. Let $A$ correspond to $\lambda_1\lambda_3$, $A_{min}$ to $\lambda_1\min\lambda_3\min$, and $A_{max}$ to $\lambda_1\max\lambda_3\max$. Denote $\xi$ as $\xi_{12}$ when $A$ is substituted by $\lambda_1\lambda_3$ in Eq. 8 and $\gamma$ by $\gamma_{12}$ when $\xi$ in Eq. 9 is replaced by $\xi_{12}$. Similarly, define $\gamma_{12}$ as a function of $\lambda_1\lambda_3$. Then, a substitution into Eq. 2c yields the desired result:

$$\sigma_{11}^{ms} = \frac{2\gamma_{12}(\lambda_1\lambda_3)}{\lambda_3\Delta} + \frac{2\gamma_{12}(\lambda_1\lambda_3)}{\lambda_3\Delta}.$$  (10)

ELASTIC STRESSES $\sigma_{ii}^{el}$

The tension $N_n^{(e)}$ arises from the stretching of the connective tissues and is expected to be governed by an expression pertinent to such tissues. I would like to suggest a form similar to the one presented by Tong and Fung (17) for the skin:

$$N_{n_{13}}^{(e)} = \frac{c_1}{\lambda_2} (a_1e_1 + a_4e_2) \exp (a_1e_1^2 + a_2e_2^2 + 2a_4e_1e_2)$$  (11a)

$N_{n_{13}}^{(e)}$ has the dimension of force/length. $e_i$ is the two-dimensional strain in the direction of $x_i$ in the alveolar septa:

$$e_1 = \frac{X_1}{\lambda_1^2} - 1.$$  (11b)

$e_2$ is the two-dimensional strain in the transverse direction in septa parallel to the $x_2$-axis, such as AA'B'B in Figure 2:

$$e_2 = \frac{1}{2}(\lambda_2^2 - 1).$$  (11c)

c, $a_1$, $a_2$, and $a_4$ are constants to be determined. A similar expression with 2 replaced by 3 should be used to calculate $N_{n_{13}}^{(e)}$.

If $e_1$ or $e_2$ becomes negative, the alveolar septa may buckle. This question has been discussed earlier in this paper. If buckling occurs in one direction but not in the other, then it may be assumed that a tension field has developed, in which case $e$ equal to zero in the buckled direction in the preceding formulas.

Recently, Lee and Frankus (9) have derived empirical expressions relating $\lambda_1$ and $\lambda_2$ to the elastic stresses $\sigma_{ij}^{el}$ from the distortion data for dog lungs obtained by Hoppin, Lee, and Dawson (unpublished observations). They inverted these expressions to obtain a function $\rho_\theta W(\lambda_1, \lambda_2, \lambda_3)$ which has the property that

$$T_{11} = \sigma_{11}^{el} \lambda_3^2 \lambda_2 = \frac{\partial \rho_\theta W}{\partial \lambda_1},$$  (12a)

$$T_{22} = \sigma_{22}^{el} \lambda_3 \lambda_1 = \frac{\partial \rho_\theta W}{\partial \lambda_2},$$  (12b)

$$T_{33} = \sigma_{33}^{el} \lambda_3 \lambda_2 = \frac{\partial \rho_\theta W}{\partial \lambda_3},$$  (12c)

where $T_{11}$, $T_{22}$, and $T_{33}$ are the Lagrangian stresses. They give

$$\rho_\theta W = \sum_{i=1}^{4} a_i (\lambda_1^{2i} + \lambda_2^{2i} + \lambda_3^{2i})$$

$$+ \sum_{i=1}^{4} b_i (\lambda_1^{2i} \lambda_2^{2i} + \lambda_2^{2i} \lambda_3^{2i} + \lambda_1^{2i} \lambda_3^{2i})$$

$$+ c_1 \lambda_1^{2i} \lambda_2^{2i} \lambda_3^{2i}$$

$$+ \sum_{i=2}^{4} c_i (\lambda_1^{2i} \lambda_2^{2i} + \lambda_2^{2i} \lambda_3^{2i} + \lambda_3^{2i} \lambda_1^{2i} + \lambda_1^{2i} \lambda_2^{2i} + \lambda_2^{2i} \lambda_3^{2i} + \lambda_3^{2i} \lambda_1^{2i})$$  (13a)

where $i$ is 1, 2, 3, or 4, and

$\rho_\theta W$ is the tension per unit area of the alveolar septa.
a_1 = -21.06, a_2 = 19.76, a_3 = -7.88, a_4 = 1.062,
b_1 = 2.673, b_2 = -0.350,
c_1 = 1.324, c_2 = -1.94, c_3 = 0.943. (13b)

On the other hand, if Eqs. 2c and 11 are used to fit the data given by Lee and Frankus (9, Table 3) under the assumptions \( \lambda_2 = \lambda_3, \ a_1 = a_2, \) then

\[ T_{11} = \lambda_2 \gamma_1 \gamma_2 \sigma_{11}(\varepsilon) = 2N_0(\varepsilon) \]

with the factor 1/\( \Delta \) absorbed in the constant \( C \) of \( N_0(\varepsilon) \), i.e.,

\[ T_{11} = 2\lambda_2 \sigma_1 e_1 + \sigma_2 e_2 
   \cdot \exp \left[ a_4 (e_1^2 + e_2^2) + 2a_4 e_1 e_2 \right]. \] (14a)

where \( e_1 \) and \( e_2 \) are given by Eqs. 11a-c, and

\[ C = 19.13 \text{ g/cm}^2, \]
\[ a_1 = a_2 = 0.6137, \]
\[ a_4 = 0.4235 \] (nondimensional). (14b)

The fit is about as good as Eqs. 12a-c and 13a and b. Not only is Eq. 14a simpler (it contains only three material constants), but the physical meaning of each of the material constants is very clear.

**The Strain Energy Function**

For stability studies and for simplicity in experimentally determining the stress-strain relationship, it is convenient to introduce the strain energy function. The usual hypothesis is that in a deformable body the internal energy due to deformation is a function of the strain alone and is independent of strain history. Not all materials have a strain energy function. Those which do are called hyperelastic (18, p 445).

To say that the lung is hyperelastic is incorrect. Large hysteresis exists in the surface tension. The tissue stress also has hysteresis, although it is much smaller. But I have argued that the hysteresis loops of living tissues are rather insensitive to strain rates (10, 19, 20). For a preconditioned specimen, i.e., a specimen that has been subjected to a specified cyclic process enough times so that a steady-state periodic response (homeostasis) has been reached, the stress as a function of strain may change by a factor of two or three when the strain rate is changed 1,000- or 100,000-fold. For such a material, it is convenient to describe the approximate stress-strain relationship for loading (i.e., increasing strain) by a single-valued function and that for unloading (decreasing strain) by another. Thus, the material is described as if it were elastic. The strain-rate effect is ignored except for recognizing the difference between loading and unloading. Such a material is called pseudo-elastic. It is infinitely simpler to analyze the lung as a pseudo-elastic material rather than as an inelastic, history-dependent material.

With this understanding, an approximate strain energy function that will yield the stress-strain relationship in loading can be formulated. This function will be called the pseudo-strain energy function to emphasize that the lung is not hyperelastic. A separate pseudo-strain energy function describes unloading.

In analyzing finite deformations, one must carefully distinguish stresses and strains referred to the deformed state from those referred to the reference state. The stresses defined in the first section of the general description and in the first section of the analysis are the so-called Cauchy or Eulerian stresses referred to the deformed body. The strains \( \varepsilon_i \) and \( \varepsilon_j \) defined in Eqs. 11b and 11c are the so-called Lagrangian, Green, or St. Venant’s strains referred to the reference state. In laboratory experiments, the force acting on a certain surface while the body deforms is often measured and divided by the initial area of the surface in the reference state to obtain a Lagrangian stress, whose numerical value can be very different from the corresponding Cauchy stress. These matters are discussed in reference 18 (pp 91 and 434–456).

Let \( W \) be the strain energy per unit mass of the material. Let \( \rho \) be the density of the material in the deformed state and \( \rho_0 \) that at the reference state. Then, \( \rho_0 W \) is the strain energy per unit volume of the material in the reference state. It is shown in reference 18 (pp 445–451) that the Lagrangian stresses can be obtained by differentiating \( \rho_0 W \) with respect to the stretch ratios. If Eq. 2 of reference 18 (p 449) is specialized to the principal direction \( i = j = 1, \)

\[ T_{11} = \frac{\delta(\rho_0 W)}{\delta\lambda_i}. \] (15)

The corresponding Cauchy stress tensor is given by

\[ \sigma_{ij} = \frac{\rho}{\rho_0} \lambda_i T_{ij} = \frac{\lambda_i}{(\lambda_1 \lambda_2 \lambda_3)} T_{ij}. \] (i, j = 1, 2, 3) (16)

Thus,

\[ T_{11} = \lambda_2 \lambda_3 \sigma_{11}. \] (17)

\( \sigma_{11} \) is the sum of \( \sigma_{11}(\varepsilon) \) from Eq. 10 and \( \sigma_{11}(\varepsilon) \) from Eqs. 2b and 11. Substituting these results into Eqs. 15–17 and integrating yields the final result

\[ \rho_0 W = \frac{2}{\Delta} \left( \gamma_{12_{\text{max}}} - \gamma_{12_{\text{min}}} \right) \frac{\lambda_{12_{\text{min}}} \lambda_{12}}{\gamma_{12_{\text{max}}} - \gamma_{12_{\text{min}}} \lambda_{12}} + \frac{1}{(\lambda_1 \lambda_2)_{\text{max}} - (\lambda_1 \lambda_2)_{\text{min}}} \left[ \lambda_1^2 \lambda_2^2 - (\lambda_1 \lambda_2)_{\text{min}} \lambda_1 \lambda_2 \right] \]

\[ - \sum n \gamma_{12} \left( \lambda_1 \lambda_2 \right)_{\text{max}} - (\lambda_1 \lambda_2)_{\text{min}} \]

\[ \frac{\gamma_{12}}{n!} \]

\[ \text{Circulation Research, Vol. 37, October 1975} \]
LUNG STRESS, DEFORMATION, AND ATELECTASIS

\[
\cos \left( \frac{n\pi}{m_1} \lambda_1 \lambda_2 - \frac{(\lambda_1 \lambda_2) m_1}{m_2} \right) + C \exp \left[ a \epsilon_1^2 + a \epsilon_2^2 + 2a \epsilon_1 \epsilon_2 \right] + \text{symmetrical terms by permutation.}
\]

The symbols have the same meaning as they do in Eqs. 10 and 11, and symmetrical terms by permutation means the sum of all terms obtained by cyclic permutation of the subscripts 1, 2, and \( \epsilon \) by 2, 3 and 3, 1. The resulting function \( \rho W \) is symmetrical in \( \lambda_1, \lambda_2, \) and \( \lambda_3 \). The coefficients \( c_n \) depend on whether the alveolar area is increasing or decreasing. Strictly speaking, \( a_1, a_2, a_3, \) and \( C \) also differ in inspiration and expiration, but generally these differences can be ignored.

STATES OF EQUILIBRIUM

Example 1: Uniform Inflation.—Consider uniform inflation of a lung in vitro (as in Radford's experiment [5]), ignoring the effect of gravity. In this case, the equilibrium equation in a rectangular Cartesian frame of reference \( (x_1, x_2, x_3) \) is simply

\[
\frac{\partial \sigma_{11}}{\partial x_1} + \frac{\partial \sigma_{12}}{\partial x_2} + \frac{\partial \sigma_{13}}{\partial x_3} = 0. \quad (i = 1, 2, 3)
\]

To obtain the boundary condition, consider the balance of forces acting on a small area of the pulmonary pleura. The forces per unit area acting outward from the lung are \( \rho A - p_{PL} - N_{PL} \kappa_{PL} \), where \( \rho A \) is the alveolar air pressure, \( p_{PL} \) is the pressure acting on the outside of the pleura, \( N_{PL} \) is the membrane tension in the pleura, and \( \kappa_{PL} \) is the sum of the principal curvatures of the pleura. The force per unit area acting inward is the normal stress \( \sigma_n \). Hence,

\[
\sigma_n = \rho A - p_{PL} - N_{PL} \kappa_{PL}.
\]

In this case \( \sigma_n \) is constant; hence, the solution of these equations is

\[
\sigma_{11} = \sigma_{22} = \sigma_{33} = \rho A - p_{PL} - N_{PL} \kappa_{PL}. \quad (21)
\]

A substitution of Eq. 21 (with \( \lambda_1 = \lambda_2 = \lambda_3 \) in a uniform expansion) into Eq. 1 yields the pressure-expansion relationship. The shear stresses \( \sigma_{ij} \) \((i \neq j)\) are zero.

This solution has been discussed earlier. The critical condition is given by the point of relative minimum of the curve \( \sigma \) vs. \( \lambda \), i.e., at a point where \( \partial \sigma / \partial \lambda = 0 \). Setting \( \sigma_{11} = \sigma_{22} = \sigma_{33} = \sigma \) and \( \lambda_1 = \lambda_2 = \lambda_3 = \lambda \) in Eq. 1 and differentiating yields the critical condition

\[
\frac{2\sigma^2}{N_{PL}\lambda} - 2\gamma \left( \frac{1}{\lambda^3} - \frac{N_{PL} \kappa_{PL} \gamma}{\lambda^2} + \frac{\partial N_{PL} \kappa_{PL}}{\lambda \partial \lambda} \right) = 0. \quad (22)
\]

It is easy to show that the higher the surface tension, the larger are the critical stretch ratio and the inflation pressure.

Example 2: Effect of Gravity.—If the effect of gravitational acceleration \( (g) \) is not neglected, then in general the directions of the principal stresses are not fixed throughout the lung. In that case, it is necessary to consider the shear stress components \( \sigma_{12}, \sigma_{23}, \) etc. However, it is possible to bring out a special case in which the principal directions are fixed by proper adjustment of the boundary conditions. Consider that special case (Fig. 5). Let the gravitational force \( \rho g \) \((\rho = \text{density}) \) act in the direction of \( x_1 \), and let \( x_1, x_2, \) and \( x_3 \) be the principal axes. Then, \( \sigma_{12} = \sigma_{23} = \sigma_{31} = 0 \), and the equations of equilibrium are

\[
\frac{\partial \sigma_{11}}{\partial x_1} + \rho g = 0, \quad (23a)
\]

\[
\frac{\partial \sigma_{22}}{\partial x_2} = 0, \quad (23b)
\]

\[
\frac{\partial \sigma_{33}}{\partial x_3} = 0. \quad (23c)
\]

The solution depends on the boundary condition. If it is required that there is no shear stress acting on the pleura, as West and Matthews (6) have assumed in their analysis, then the solution is

\[
\sigma_{11} = \sigma_{22} = \sigma_{33} = c - \rho g x_1, \quad (24)
\]

where \( c \) is a constant. The boundary conditions satisfied by this solution are that (1) the tangential stress is zero and (2) the normal stress is

\[
\sigma_{11} \nu_1^2 + \sigma_{22} \nu_2^2 + \sigma_{33} \nu_3^2 = c - \rho g x_1 = \rho A - p_{PL} - N_{PL} \kappa_{PL}. \quad (25)
\]

where \( \nu_1, \nu_2, \) and \( \nu_3 \) are the three components of the unit vector normal to the pleural surface. Eq. 25 defines the intrapleural pressure that must exist for the solution given by Eq. 24 to be correct. The initial lung geometry can then be obtained by substituting Eq. 1 into Eq. 24 and solving for \( x_1, x_2, \) and \( x_3 \). Since Eq. 24 represents isotropic expansion at every given \( x_1 \), the strains are compatible. The solution is therefore exact if the resting configuration of the lung happens to correspond to the strain state \( \lambda_1 = \lambda_2 = \lambda_3 = 1 \). Otherwise, it is not an exact solution.

This solution has only one arbitrary constant, \( c \), which specifies the degree of expansion. Note that no restriction such as linearity of the stress-strain relationship is imposed. If the reference configuration of the lung is right, this simple solution is exact for a large deformation of a real lung.
STABILITY

According to the ideas discussed earlier, arbitrary small perturbations of an inflated lung will be considered. Let the stretch ratios at equilibrium be \( \lambda_{10}, \lambda_{20}, \) and \( \lambda_{30} \). Let the stretch ratios of the perturbed lung be \( \lambda_1 = \lambda_{10} + \delta \lambda_1, \lambda_2 = \lambda_{20} + \delta \lambda_2, \) and \( \lambda_3 = \lambda_{30} + \delta \lambda_3. \) The \( \delta \) values are arbitrary, compatible, and infinitesimal. The strain integral \( \rho_\alpha W \) is changed to \( \rho_\alpha W + \delta \rho_\alpha W. \) Then, the potential energy, which is equal to the integral of \( \rho_\alpha W \) minus the work done by the external load, is computed. In reference 18 (p. 451), it is proved that the vanishing of the first variation of the potential energy yields the equations of equilibrium and the boundary conditions. Hence, the solution \( \lambda_{io} \) renders the first variation of potential energy zero. Thus, the stability is determined by the second variation of the strain energy, \( \delta^2 \rho_\alpha W. \) The system is stable if \( \delta^2 \rho_\alpha W \geq 0; \) otherwise, it is unstable.

Substituting \( \lambda_i = \lambda_{io} + \delta \lambda_i \) into Eq. 18 and retaining only the second-order terms results in

\[
\delta^2 \rho_\alpha W = \sum_{i,j=1}^{3} k_{ij} \delta \lambda_i \delta \lambda_j, \tag{26}
\]

where \( k_{ij} \) are the values of the second derivatives evaluated at \( \lambda_i = \lambda_{io}: \)

\[
k_{ij} = \frac{\partial \delta^2 \rho_\alpha W}{\partial \delta \lambda_i \partial \delta \lambda_j}, \quad (i,j = i, 2, 3). \tag{27}
\]

The right side of Eq. 26 is a quadratic form. If the equilibrium is to be stable according to the definition laid down earlier in the section on stability in the first part of this paper, the quadratic form must be positive definite, i.e., \( >0 \), for whatever values of \( \delta \lambda_i \) and \( \delta \lambda_j \) and 0 only when \( \delta \lambda_i = \delta \lambda_j = 0. \) The conditions for the positive definiteness are (see ref. 18, pp 29–30):

\[
\begin{align*}
&k_{11} + k_{22} + k_{33} > 0, \quad \text{(28a)} \\
&k_{11} k_{22} + k_{22} k_{33} + k_{33} k_{11} > 0, \quad \text{(28b)} \\
&k_{11} k_{22} k_{33} > 0. \quad \text{(28c)}
\end{align*}
\]

The quantities in Eq. 28b and c are determinants. Eq. 18 leads to

\[
k_{11} = b_l^2 \lambda_l^2 + \sum_{n=1}^m H_{n12} \alpha_{n12} \lambda_l^2 \cos \alpha_{n12} (\lambda_l \lambda_2 - \beta_{12})
\]

where \( a_{ij} = \) constants,

\[
\begin{align*}
&b_l^2 = \frac{2C[(a_1 a_1 + a_4 a_4)] + \lambda_l^2}{2}, \quad \text{Eq. 28} \\
&\lambda_1 = \lambda_{10} + \delta \lambda_1, \lambda_2 = \lambda_{20} + \delta \lambda_2, \quad \text{and} \quad \lambda_3 = \lambda_{30} + \delta \lambda_3.
\end{align*}
\]

Thus, Eq. 28 delineates the stability as a function of these parameters.

**Example:** The Uniformly Inflated State Discussed in the Preceding Section.—Let the stress be above the critical level. The solution corresponding to Eq. 21 is \( \lambda_1 = \lambda_2 = \lambda_3 = \) constant, which will be designated \( \lambda_{io} \). Assume all \( \lambda_{max}, \lambda_{min}, \) and \( \gamma_{max} \) and \( \gamma_{min} \) are the same in all subscripts. In this case, \( k_{11} - k_{22} = k_{33} \) and \( k_{12} = k_{23} = k_{31} = k_{21} = k_{32} = k_{13}. \) Hence Eqs. 28a–c are reduced to

\[
\begin{align*}
&k_{11} > 0, \\
&k_{11}^2 - k_{12}^2 > 0, \\
&k_{11}^3 + 2k_{12}^3 - 3k_{11}k_{12}^2 > 0. \quad \text{(31)}
\end{align*}
\]

Assume that \( \lambda_{min} = 1, \lambda_{max} = 1.8, \gamma_{max} = 40 \) dynes/cm, \( \gamma_{min} = 10 \) dynes/cm, \( c_1 = 1 \) for inspiration, \( c_2 = -1 \) for expiration, \( c_3 = 0 \) if \( n > 1, \Delta = 10^{-3} \) cm, \( C = 1.913 \times 10^4 \) dynes/cm², \( a_1 = a_2 = 0.6137, \) and \( a_3 = 0.4235. \) Then, \( a_2 = -678 \) dynes/cm, \( b_{12} = 926 \) dynes/cm, \( H_{11} = 10.695 \) dynes/cm, \( H_{12} = 0 \) for \( n > 1, \Delta_{12} = 0.9696 \) and \( \beta_{12} = 1. \) If \( \lambda_{10} = 1, \) then \( k_{11} = 52674 \) and \( k_{12} = 11586. \) Eq. 31 is satisfied, and the equilibrium is stable. Similarly it can be verified that all equilibrium states above the critical inflation pressure are stable. On the other hand, an unstable state is obtained if the surface tension is a constant independent of area, and the elastic stress is zero (in which case \( k_{11} = 0 \)).

**PLANAR ATELECTASIS**

Consider an inflated lung in which the stresses are \( (\sigma_{ij})_0 \) and the strains are \( (\lambda_{ij})_0, \) \( i, j = 1, 2, 3; \) call it an inflated state. Consider a perturbation in which all alveoli tend to collapse toward a plane
LUNG STRESS, DEFORMATION, AND ATELECTASIS

which will be identified as the \( x_1 \) plane. Let a point \((x_1, x_2, x_3)\) in the inflated state be moved to a new point \((x_1 + u_1, x_2 + u_2, x_3 + u_3)\). Assume that \(u_1\) is a function of \(x_1\) alone and that \(u_2 = u_3 = 0\). The stresses in the perturbed state must satisfy the equation of equilibrium, which can be expressed in terms of the function \(u_1(x_1)\). The problem is to discover a nontrivial solution.

Recalling the solution of example 1, Eq. 21, and Figure 5, one sees that one possible solution is

\[
\begin{align*}
\lambda_{\alpha} - 1 - \epsilon_{\alpha} &= \frac{\partial u_1}{\partial r} \\
\lambda_{\theta} - 1 - \epsilon_{\theta} &= \frac{u_r}{r}.
\end{align*}
\] (34)

As was discussed earlier, three zones must now be considered. The inner zone is the coalesced alveoli. Next to it is a zone in which the alveoli are smaller than their size in the reference state (region b). First consider the case in which the alveolar septa are so thin that they buckle under insignificant compression. Then, equilibrium is maintained by surface tension alone. Since \(r, \theta\), and \(x\) are the principal directions, appropriate substitutions of \(r, \theta\), and \(x\) can be used for 1, 2, and 3 in Eq. 2c to obtain the stress-strain relationship:

\[
\begin{align*}
\sigma_{\alpha}^{(s)} &= \frac{2\gamma}{\lambda_0} \left( \frac{1}{\lambda_{\alpha}} + \frac{1}{\lambda_{\theta}} \right), \\
\sigma_{\theta}^{(s)} &= \frac{2\gamma}{\lambda_0} \left( \frac{1}{\lambda_{\alpha}} + \frac{1}{\lambda_{\theta}} \right),
\end{align*}
\] (35a, b)

where \(\lambda_0\) is the stretch ratio of the uniformly inflated state relative to the reference state. (The alveolar dimension is \(\lambda_0\Delta\) in the inflated state.) Assuming \(\gamma\) to be a constant, substituting Eqs. 35 and 34 into Eq. 33 yields

\[
\frac{1}{(1 + \lambda_0)} \frac{\partial}{\partial r} \left( \frac{u_r}{r} \right) + \frac{1}{r} \left( \frac{1}{1 + u_r} - \frac{1}{1 + \frac{\partial u_r}{\partial r}} \right) = 0,
\] (36)

whose solution is

\[
u_r = \text{constant} \cdot r.
\] (37)

The integration constant is determined by the condition on the inner boundary, \(r = a\). Assume that all alveoli within a radius of \(k\lambda_0\Delta\) in the inflated state collapse into a cylinder of radius \(a\). Then, \(\nu_r = -a/(k\lambda_0\Delta)\). Hence, the constant is \(1 - a/(k\lambda_0\Delta)\). Thus, the displacement in region b is

\[
u_r = -\left[1 - a/(k\lambda_0\Delta)\right] r.
\] (38)

The integration constant is determined by the condition on the inner boundary, \(r = a\). Assume that all alveoli within a radius of \(k\lambda_0\Delta\) in the inflated state collapse into a cylinder of radius \(a\). Then, \(\nu_r = -a/(k\lambda_0\Delta)\). Hence, the constant is \(1 - a/(k\lambda_0\Delta)\). Thus, the displacement in region b is

\[
u_r = -\left[1 - a/(k\lambda_0\Delta)\right] r.
\] (38)

This solution is valid up to \(r = b\), where the alveolar dimension is \(\Delta\), i.e., where \(\lambda_0\lambda_0' = \lambda_0\lambda_0'' = 1\). But Eqs. 38 and 34 imply that \(\lambda_0'\) and \(\lambda_0''\) are constant throughout region b. It follows that the radius \(b\) cannot be found inside the lung. The consequences of this fact have been discussed earlier.
That $\lambda'$ and $\lambda''$ are constant in region b makes the solution valid even if the hypotheses that $\gamma$ is a constant and that the buckling stress of the alveolar septa is zero are not imposed, because these hypotheses are automatically satisfied by the solution. However, since surface tension has hysteresis and alveolar septa may be able to sustain some compression without buckling in some animals and since the surface tension and the elastic compressive stress are nonlinear functions of strain, solutions other than the simple one just given may exist. However, since the elastic stress will vanish anyway as the alveolar size tends to that at the reference state and $\gamma$ tends to $\gamma_{\text{min}}$ when $\lambda < 1$, it is unlikely that these other solutions are significant.

**FOCAL ATELECTASIS**

Let the focus be chosen as the origin and let a set of spherical polar coordinates $r$, $\phi$, and $\theta$ be used; $r$ is the radius vector, $\phi$ is the azimuth angle, and $\theta$ is the polar angle. Consider a radial displacement $u_r$, which is a function of $r$ alone. The equations of equilibrium for such a symmetrical deformation are

$$\frac{\partial \sigma_r}{\partial r} + \frac{1}{r} \left( 2\sigma_r - \sigma_\phi - \sigma_\theta \right) = 0, \quad (39a)$$

$$\sigma_\phi = \sigma_\theta. \quad (39b)$$

The strains relative to the uniformity inflated state are

$$\epsilon_r = \frac{\partial u_r}{\partial r}, \quad \epsilon_\phi = \epsilon_\theta = \frac{u_r}{r}. \quad (40)$$

Thus, the mathematical problem differs from that of the preceding section only in the factor 2 in the last term of Eq. 39a. The solution, outside the core of collapsed alveoli within $r = a$, is exactly the same (Eq. 38). This fact leads to the conclusions stated earlier in the general description of the three possible forms of atelectasis.

**References**

15. **Lubert M, Krause GR**: Patterns of lobar collapse as observed radiographically. Radiology 56:165–182, 1951
Stress, deformation, and atelectasis of the lung.

Y C Fung

_Circ Res._ 1975;37:481-496
doi: 10.1161/01.RES.37.4.481

_Circulation Research_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/37/4/481

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation Research_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the _Permissions and Rights Question and Answer_ document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation Research_ is online at:
http://circres.ahajournals.org/subscriptions/