Elasticity of the Pulmonary Alveolar Sheet

By Y. C. Fung and S. S. Sobin

ABSTRACT

An analysis of experimental data on the pulmonary alveolar sheet of the cat shows: (1) As far as elasticity in the plane of the sheet is concerned, the alveolar sheet may be regarded as uniform; the compliance of the posts is the same as that of the membranes. (2) Within a physiological range of positive transmural pressure, the mean thickness of the sheet varies linearly with the pressure. Also, the stress distribution in the alveolar-capillary membrane is nonuniform and nonisotropic. A theoretical sheet thickness-pressure relationship is derived in which the effect of stress resultants (sum of elastic stress and surface tension) is explicitly linked to the compliance of the sheet thickness.

The sheet-flow theory then shows that average flow is very sensitive to the arteriole pressure. The flow per alveolar sheet is \( \frac{1}{C} (h_a - h_v) \). Here \( h_a \) and \( h_v \) are equal to alveolar sheet thickness at the arteriole and venule, respectively. When \( p_{art} > p_{alv} \), \( h_a = h_0 + \alpha (p_{art} - p_{alv}) \), where \( p_{art} \) is the blood pressure in the sheet at the arteriole, \( p_{alv} \) is the gas pressure in the alveolus, \( \alpha \) is the compliance constant for the sheet thickness, and \( h_0 \) is the thickness at \( \Delta p = 0 \). The constant \( C = 4 \mu f \alpha L / (SA) \), where \( \mu \) is the coefficient of viscosity of blood, \( f \) is a friction parameter (on the order of 4, depending on the post geometry), \( L \) is the average length of the streamlines in the sheet, \( S \) is the vascular space-tissue ratio (on the order of 0.9), and \( A \) is the sheet area. Comparison of this formula with the experimental results of Roos et al., using \( h_0 \) and \( \alpha \) values from Glazier et al., shows reasonable agreement.

KEY WORDS pulmonary blood flow distensibility interalveolar wall

When the idea of sheet flow was developed (1, 2), we reasoned that the pulmonary alveolar sheets were stretched taut in an inflated lung so that they behaved elastically in a linear manner with respect to lateral loading. Therefore, in response to the transmural pressure (the difference in blood pressure in the sheet and air pressure in the alveoli), the alveolar-capillary membrane deflects proportionally. Furthermore, the two alveolar-capillary membranes are so connected by a system of densely spaced posts that the elastic deflection in response to the transmural pressure is localized. Therefore the sheet thickness must be a linear function of transmural pressure:

\[
\bar{h} = h_0 + \alpha (\bar{p} - p_{alv}) = h_0 + \alpha \Delta p, \tag{1}
\]

where \( \bar{h} \) denotes the mean vascular sheet thickness, \( \bar{p} \) denotes the local blood pressure, \( p_{alv} \) is the alveolar pressure, \( \Delta p \) indicates the difference, \( \bar{p} - p_{alv} \), and \( h_0 \) and \( \alpha \) are constants. This formula is expected to work for a moderate range of positive pressure difference, \( \bar{p} - p_{alv} \). Nonlinearity is expected at both ends of the \( \Delta p \) range. At the low or negative end of the \( \Delta p \) range, the nonlinear elasticity of the posts must reveal itself so that
decreases rapidly to zero when $\Delta p \to 0$ or when $\Delta p < 0$. At the high end of the $\Delta p$ range, the kinematic nonlinearity due to finite curvature of the alveolar-capillary membrane and the nonlinear interaction between the nonuniform membrane tension and the curvature of the membrane will make $\tilde{h}$ tend to a constant at very high $\Delta p$. The theoretical curve of thickness vs. pressure must be like the one sketched in Figure 1. The more taut the membrane is, the wider would be the linear range and the smaller the slope of the linear portion of the thickness-pressure curve.

In the accompanying article (3), we have shown that in the cat's lung such a linear relationship indeed holds in the pressure range $6.3 < \Delta p < 27$ cm H$_2$O. On the other hand, data presented in Figure 1 of reference 4 show that when $\Delta p \leq 0$ a good approximation is

$$\tilde{h} = 0.$$  \hspace{1cm} (2)

Further confirmation can be seen in an earlier paper by Glazier et al. (5). Therefore a basic premise of the sheet-flow theory is substantiated, and it seems worthwhile to present some of the consequences of the theory with regard to pulmonary blood flow. In the following, some theoretical results will be compared with the existing experimental data by Roos et al. (6).

But more can be said about the elasticity of the pulmonary alveolar sheet. Both our old (2) and new (3) data on the vascular space-tissue ratio, VSTR,\(^2\) show that VSTR remains constant over a wide range of variations of other parameters. The implication of this finding must be sought. In the following, we shall demonstrate that this means that the posts have the same compliance as the alveolar-capillary membranes with respect to tension in the plane of the sheet.

With these results we can consider the elastic structure of the alveolar sheet in detail. It is essential to agree on a terminology. We shall call each blood-air barrier an alveolar-capillary membrane. An alveolar sheet consists of two such membranes enclosing a vascular space. Each alveolar-capillary membrane is composed of endothelial cells, epithelial cells, and interstitium. The stress distribution in a perfused alveolar vascular sheet, the curvature of the alveolar membrane, and the description of membrane geometry will be presented.

Although our reasoning is simple, the analysis is fairly complex because of the three-dimensional nature of the problem. For this reason a much simpler one-dimensional analogue is presented in Appendix C.

**IMPLICATIONS OF THE CONSTANCY OF THE VASCULAR SPACE-TISSUE RATIO**

A remarkable characteristic of the vascular space-tissue ratio is that it remains essentially the same at different parts of the lung and under different perfusion pressures although the interpost distance and the sheet thickness vary considerably. For example, random sampling of various parts of the lungs of six cats perfused with silicone elastomer (3) yielded the data shown in Table 1.

Note that whereas the standard deviation of VSTR is about 2\%, that of the unit hexagonal area is more than ten times as large, approximately 22\%. The sizable variation in the unit hexagonal area means that the
PULMONARY ALVEOlar SHEET ELASTICITY

TABLE 1
Experimental Results on Sheet Dimensions of the Cat

<table>
<thead>
<tr>
<th></th>
<th>Perfused at 15 mm Hg</th>
<th>Perfused at 25 mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>VSTR (%)</td>
<td>91.43 ± 1.91</td>
<td>91.75 ± 1.95</td>
</tr>
<tr>
<td>Unit hexagon area (μ²)</td>
<td>163.66 ± 37.11</td>
<td>142.06 ± 31.70</td>
</tr>
<tr>
<td>Post diameter (μ)</td>
<td>4.19 ± 0.79</td>
<td>3.82 ± 0.70</td>
</tr>
<tr>
<td>Interpost distance (μ)</td>
<td>9.13 ± 0.85</td>
<td>8.61 ± 0.90</td>
</tr>
<tr>
<td>No. fields analyzed</td>
<td>160</td>
<td>149</td>
</tr>
</tbody>
</table>

Values are means ± sd. VSTR = vascular space-tissue ratio.

alveolar sheets were stretched to different dimensions in different parts of the lung. The small sd for VSTR means that the geometric pattern remained constant as the alveolar vascular sheets were stretched. In other words, the posts expanded at the same rate as the vascular space. This implies that as far as the elasticity of the alveolar wall is concerned, the posts have exactly the same compliance in the plane of the alveolar wall as does the rest of the wall. Thus, as far as elasticity is concerned, the alveolar vascular sheet can be treated as homogeneous.

This empirical simplification is very welcome, because it simplifies the hemodynamic analysis. In spite of the formidably complicated appearance of the alveolar vascular sheet, mechanically it is legitimate to replace it by a double-layered membrane which is initially isotropic and homogeneous.

What does this empirical fact tell us about the structure of the sheet? To appreciate the question let us first look at a rubber membrane. Figure 2a shows a sheet of rubber ("dental dam") with a hole in the center surrounded by a circle marked in ink. Figure 2b shows the same sheet after it was stretched to approximately twice its initial size. Note the great increase in the ratio of the area of the hole to that of the circle (from 0.0695 to 0.280). On the other hand, if the hole was clamped with a rigid disk, the ratio of the area of the disk to that of the circle would be decreased as the membrane was extended (from 0.0695 to 0.0175). Figure 2c and d shows a sheet of the same rubber perforated by a double array of holes; in this case VSTR decreased from 0.931 to 0.796 as the linear dimension of the sheet was increased by 30%, i.e., VSTR was decreased by 17% when the stretch ratio, λ, was increased from 1 to 1.30. On the other hand, if the circular areas were rigid, VSTR would certainly be increased when the sheet is stretched.

If VSTR remains the same as λ changes, it is obvious that the circular regions in Figure 2a and c must be neither void nor perfectly rigid.
Two sheets of rubber glued together at post areas and inflated by air. This model meets the requirement on the elasticity of the posts as demanded by the constancy of the vascular space-tissue ratio.

For a constant VSTR, compliance of these circular regions must be the same as that of the rest of the membrane.

A model of the alveolar sheet that meets such an elasticity requirement is shown in Figure 3. It was made of two sheets of rubber membrane glued together at an array of circular spots and inflated with an internal pressure. It resembles the appearance of an alveolar wall. Such a model is, of course, meant only to be roughly indicative. The alveolar-capillary membrane is composed of epithelial cells, endothelial cells, and basement membranes. The posts have fine structures (3).

STRESS DISTRIBUTION IN A PERFUSED ALVEOLAR SHEET

To describe the stresses in an alveolar sheet, let us consider a small element of the sheet as shown in Figure 4. Each edge of the element is a cross section of the sheet. In each cross section, the stress resultant is a vector which may be resolved into a component perpendicular to the cross section and another component tangent to it. The former is called the normal membrane stress resultant, or simply membrane tension (dyne/cm), and the latter is the membrane shear. Since the stretch may be different in different directions, in general the membrane tension is different in different directions. However, at any given point on the alveolar sheet, two orthogonal "principal" directions can always be found, so that in cross sections made along these directions the membrane shear vanishes. When we vary the direction of the cross sections at a given point, the membrane tension reaches the maximum in one of the principal directions and the minimum in the other. Let these principal membrane tensions be denoted by $T_1$ and $T_2$ and the corresponding radii of curvature of the membrane by $R_1$ and $R_2$ (Fig. 5), then the pressure difference, $\Delta p$, across the sheet is

\[
\Delta p = \frac{T_1 T_2}{R_1 R_2}
\]
For an element shown in Figure 4, at any given point, if the directions x and y are properly chosen the shear resultants, $v_{xy}$ and $v_{yx}$, will vanish. These are the principal directions which are designated as directions 1 and 2. In this figure, the spatial configuration of this element is shown. $R_1$ and $R_2$ are the principal radii of curvature and $T_1$ and $T_2$ are the stress resultants. 

(For example, see Flügge [7].) This equation may be called the generalized Young-Laplace formula (8). When it is applied to an interalveolar sheet with both sides exposed to the same alveolar duct so that $\Delta p = 0$, the sheet must assume such a shape that

$$\Delta p = \frac{T_1}{R_1} + \frac{T_2}{R_2} = 0.$$  

(3)

The particular case in which the two principal membrane tensions are equal, $T_1 = T_2 = T$, so that the tension is the same in every direction, is well known in the theory of soap bubbles. Such a surface is governed by the equation

$$\frac{1}{R_1} + \frac{1}{R_2} = 0$$  

(4)

and is known as a minimal surface in the theory of differential geometry. A soap film, both sides of which are exposed to the same pressure, is a minimal surface. The surface defined by Eq. 3 may be called a generalized minimal surface. Thus most alveolar sheets are generalized minimal surfaces. A special generalized minimal surface is a plane for which $1/R_1 = 1/R_2 = 0$. Alveolar sheets far away from a rigid boundary (pleural surface, blood vessels, trachea, etc.) generally assume such a planar form.

To analyze the elastic deformation of the interalveolar sheet, we must consider not only the deflection of the sheet as a whole but also the deflection of the two alveolar-capillary membranes relative to the middle surface of the sheet. If the deflection surface is known, then we can calculate the strains and stresses in the membranes and deduce the relationship between the sheet thickness and the pressure. With such an analytical relationship, the compliance of the sheet can be expressed in terms of the geometric and physical parameters of the sheet. This derivation is briefly presented below. Our main result is given in Eq. 21.

It is easy to see that when an interalveolar sheet is perfused by blood the alveolar-capillary membranes will be deflected (Figs. 3, 4, and 6). Figure 6 shows a sketch of the deflection surface for a membrane whose plane view is shown in Figure 7. The membrane deflection is governed by the same Young-Laplace equation (Eq. 3) if we interpret $T_1$, $T_2$, $R_1$, and $R_2$ as the principal tensions and curvatures of these membranes. It is intuitively clear that if a flat alveolar-capillary membrane is deflected into a shape as sketched in Figure 6 the stresses and strains will vary from point to point (nonuniform) and from one direction to another (nonisotropic). Some estimation of the magnitudes of $T_1$, $T_2$, $R_1$, and $R_2$, and the degree of nonuniformity and nonisotropy can be obtained by considering the conditions of equilibrium as affected by geometric factors. Appendix A presents such a detailed consideration. Among the results given in Appendix A, the following is of particular interest. The principal curvature at a point O (Fig. 7), which is symmetrically situated with respect to all posts, is estimated to be

$$\left(\frac{1}{R_1}\right)_O = \left(\frac{1}{R_2}\right)_O = \frac{\Delta p}{2T}.$$  

(6)

The corresponding curvatures at the point A
FIGURE 6
Schematic illustration of the equilibrium of the alveolar-capillary membrane in the neighborhood of the posts. See text for abbreviations and details.

FIGURE 7
A pattern of the alveolar-capillary membrane showing the notations of Eqs. 3-8.

(Fig. 8), which lies on the edge of a post, are approximately

\[
\left( \frac{1}{R_1} \right) \approx \frac{\Delta p}{T} + \frac{1}{a}, \tag{7}
\]

\[
\left( \frac{1}{R_2} \right) \approx -\frac{1}{a}. \tag{8}
\]

Here \( \Delta p \) is the transmural pressure, \( \bar{p} - p_{air} \), \( T \) is the average tension in the alveolar-capillary membrane, \( a \) is the radius of the post, and the subscripts 1 and 2 refer to directions normal and tangential to the circumference of the post, respectively. With this information, we can construct an approximate equation for the deflection surface of the membrane as is shown in the section below.

APPROXIMATE MEMBRANE GEOMETRY
To describe the membrane surface analytically, let us consider a basic rectangular array of posts. With a frame of reference shown in Figure 7, we shall let \( w(x,y) \) represent the deflection of the membrane above the top of the posts. The function \( w \) must vanish at the circumference of the posts and should be influenced more by nearby posts than by those...
**PULMONARY ALVEOLAR SHEET ELASTICITY**

\[ \frac{1}{w_0} \left( \frac{\partial^2 w}{\partial x^2} \right) = \frac{B}{A} \left( \frac{\Delta p}{2T} \right) - \frac{1}{w_0} \frac{\Delta p}{2T} \]

(11)

and

\[ w_0 = A \Gamma, \]

(12)

Where \( \Lambda \) and \( \Gamma \) are functions of the ratio of \( a \) to \( c \) (Table 2). Furthermore, in the linear range, a consideration of work and energy (Appendix B) yields the relation

\[ w_0 = K_0 c^2 \frac{\Delta p}{2T}. \]

(13)

Hence,

\[ A = \frac{w_0}{\Gamma} = K_0 c^2 \frac{\Delta p}{2T}. \]

(14)

And

\[ B = \frac{w_0}{2T} \left( 1 - \frac{1}{w_0} \frac{\Delta p}{2T} \right). \]

(15)

A substitution into Eqs. 9 and 10 yields the membrane surface. Figure 9 shows the deflection surface \( w(x,y) \) when \( F(x,y) = 1 \), and \( a/c = 0.1 \).

**RELATIONSHIP OF SHEET THICKNESS TO BLOOD PRESSURE**

The average membrane deflection,

\[ \frac{\Delta h}{\Delta h} = \frac{\int w(x,y) \, dx \, dy}{\int dx \, dy}, \]

(16)

can be computed by integrating over a basic triangular region OMNAO of Figure 7. The mean thickness of the alveolar sheet, \( h \), is \( 2w \) plus the length of the post, \( h_p \).

\[ h = h_p + 2w. \]

(17)

To obtain \( h \), we may substitute \( w(x,y) \) from Eq. 9 into Eq. 16 to obtain

\[ 2w = AK_1 + Bc^2K_2, \]

(18)

where \( K_1 \) and \( K_2 \) depend on the ratio of \( a \) to
Theoretical Values of $\Lambda$, $\Gamma$, $K_1$, $K_2$, $I_1$, $I_2$, $I_3$, and $K_3$ for Several Values of the Geometric Ratio of Post Radius ($a$) to Interpost Distance ($c$)

<table>
<thead>
<tr>
<th>$a/c$</th>
<th>$\Lambda$ (Eq. 15)</th>
<th>$\Gamma$</th>
<th>$K_1$</th>
<th>$K_2$</th>
<th>$I_1$</th>
<th>$I_2$</th>
<th>$I_3$</th>
<th>$K_3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>0.165584</td>
<td>0.935363</td>
<td>1.783013</td>
<td>1.82288</td>
<td>0.45816</td>
<td>0.55625</td>
<td>0.58510</td>
<td>1.705835</td>
</tr>
<tr>
<td>0.2</td>
<td>0.693233</td>
<td>0.763367</td>
<td>1.369105</td>
<td>0.914442</td>
<td>0.40162</td>
<td>0.537862</td>
<td>0.556364</td>
<td>0.770427</td>
</tr>
<tr>
<td>0.3</td>
<td>1.689920</td>
<td>0.538842</td>
<td>0.915354</td>
<td>0.621837</td>
<td>0.231362</td>
<td>0.415689</td>
<td>0.397007</td>
<td>0.243541</td>
</tr>
</tbody>
</table>

$c$ in a minor way, as shown in Table 2. A final simplification is obtained if we assume that in the range of interest the post length, $h_p$, varies linearly with $\Delta p$ and write

$$h_p = h_0 + \frac{\partial h_p}{\partial p} \Delta p. \quad (19)$$

Then a combination of Eqs. 13, 15, and 18 yields

$$h = h_0 + \frac{\partial h_p}{\partial p} \Delta p + \frac{c^2}{4 \Gamma T} (2K_1 K_3 - K_2 + AK_3). \quad (20)$$

A comparison with Eq. 1 yields the slope of the thickness-pressure curve

$$\alpha = \frac{\partial h_p}{\partial p} + \frac{c^2}{4 \Gamma T} (2K_1 K_3 - K_2 + AK_3). \quad (21)$$

This slope, $\alpha$, is a measure of the compliance of the alveolar sheet. It will be called the compliance constant of the alveolar sheet.

Eq. 21 provides an anatomy of the compliance constant, $\alpha$. It shows how $\alpha$ is affected by the parameters describing the membrane geometry and size ($c$, $K_1$, $K_2$, $K_3$, $\Lambda$ and $\Gamma$), the compliance of the posts ($\partial h_p/\partial p$), and the tension in the membranes ($T$). With such a formula, experimental results obtained under different conditions and on different animals can be correlated.

It is interesting to note that according to Eq. 21 the alveolar sheet compliance would decrease when the tension, $T$, increases. Since $T$ is the sum of the surface tension and elastic tension, an increase of surface tension would cause an increase in $T$ and a decrease in the compliance of the alveolar sheet, thus causing a decrease in blood flow. An expansion of the lung also increases $T$ and, if other things remain equal, would also decrease the blood flow in the alveolar sheet.

Figure 9: Theoretical contour lines of the deflection surface. The contours mark the points of equal height. The shaded profiles along the edges and the diagonal are the deflection curves computed from Eq. 9 et sequens. $F(x, y) = 1, c = 1, a = 0.1$ for this figure.

Circulation Research, Vol. XXX, April 1972
BLOOD FLOW IN ALVEOLAR SHEET

We have shown (1) that the mean flow velocity in a pulmonary alveolar sheet is

\[ U = \frac{1}{\mu_f} \frac{h^2}{\text{grad } \Delta p}. \]  

(22)

Here \( \Delta p \) stands for the transmural pressure, \( \bar{p} - \rho_{art} \), \( \mu \) is the coefficient of viscosity of the blood, \( f \) is a numerical factor which depends on the details of the sheet structure and red cell characteristics, \( h \) is the local mean thickness, and the symbol "grad" stands for the gradient operator. The flow per unit width is, therefore

\[ Q_{\text{avg}} = \frac{1}{4\mu_f L} [h_0 + \alpha \Delta p_{\text{art}}]^2 - (h_0 + \alpha \Delta p_{\text{ven}})^2]. \]  

(25)

Let \( L = \int ds \) be the length of the streamline, then assuming \( \mu_f \) to be constant, we obtain the average flow per unit width along a streamline,

Here the subscripts art and ven refer to arteriole and venule, respectively.

Eq. 25 shows that the average velocity of blood flow along a streamline connecting an arteriole and a venule is inversely proportional to the length of the streamline and the viscosity of the blood. This feature is common to all blood vessels. But the dependence of the blood flow on pressure is quite unique in the pulmonary alveoli as compared with that in a rigid tube. Thus, instead of following Poiseuille's law, according to which \( \frac{h_0 + \alpha \Delta p_{\text{art}}}{\mu_f} \) is directly proportional to the pressure drop \( (p_{\text{art}} - p_{\text{ven}}) \), we see that in the pulmonary capillaries

\[ Q_{\text{avg}} = \frac{1}{4\mu_f L} (p_{\text{art}} - p_{\text{ven}})\left\{ (h_0 + \alpha \Delta p_{\text{art}})^2 + (h_0 + \alpha \Delta p_{\text{art}})^2 (h_0 + \alpha \Delta p_{\text{ven}}) \right\} \]

+ \( (h_0 + \alpha \Delta p_{\text{art}})(h_0 + \alpha \Delta p_{\text{ven}})^2 + (h_0 + \alpha \Delta p_{\text{ven}})^3 \]. \hspace{1cm} (26)

The factor in the brackets is the conductance of the flow and depends on the blood pressure.
Since a field of flow can be wholly covered by streamlines, we can sum up all the streamlines between an arteriole and a venule to obtain the flow between these two vessels. Let \( A \) be the area of alveolar sheet in question and \( S \) be the vascular space-tissue ratio, then the total vascular space is \( SA \), and the total flow is

\[
\text{Flow} = SAQ_{ave}/L = \frac{SA}{4\mu fL^2/\alpha} \left[ (h_0 + \alpha \Delta p_{art})^4 - (h_0 + \alpha \Delta p_{ven})^4 \right].
\]

Expressed in terms of sheet thickness, this is

\[
\text{Flow} = \frac{1}{C} \left[ h_0^4 - h_v^4 \right],
\]

where \( L \) is the average length of the streamlines in this field, and

\[
C = \frac{4\mu fL^2/\alpha}{SA},
\]

\[
h_0 = h_0 + \alpha \Delta p_{art}, \quad h_v = h_0 + \alpha \Delta p_{ven},
\]

\[
\Delta p_{art} = p_{art} - p_{ven}, \quad \Delta p_{ven} = p_{ven} - p_{ven}.\]

We may compute \( L \) by associating each streamline with a specific value of the stream function, \( \psi \), and compute

\[
\frac{1}{L} = \frac{1}{\psi_2 - \psi_1} \int_{\psi_1}^{\psi_2} \frac{1}{L(\psi)} d\psi,
\]

where \( \psi_1 \) and \( \psi_2 \) are the dividing streamlines that enclose the whole field of flow between the arteriole and venule in question.

Eq. 27b provides an explicit formula of blood flow in the pulmonary alveoli as related to the blood rheology (\( \mu \)), alveolar area (\( A \)), alveolar structural geometry (\( \alpha, f \)), the vascular space-tissue ratio (\( S \)), the degree of stretch (\( L, c \)), surface tension (\( T, \alpha \)), and the arteriole and venule transmural pressure.

If we write Eq. 27a in the form

\[
p_{art} - p_{ven} = R \cdot (\text{Flow}),
\]

then \( R \) can be called the local resistance. If both \( \Delta p_{art} \) and \( \Delta p_{ven} \) are positive the resistance is given by

\[
R = h_0^4 + h_0^2 h_v + h_0 h_v^2 + h_v^4/\alpha^2.
\]

So far we have not explicitly dealt with the discontinuity of the thickness-pressure relationship at zero pressure at which the governing equation changes from Eq. 1 to Eq. 2. When Eq. 2 is used we see that Eq. 27b holds also for negative transmural pressure.

\[
Q = 0. \quad \text{But if } p_{ven} < p_{art}, \text{ then } h_0 = h_v = 0 \quad \text{and}
\]

\[
Q = \frac{h_0^4}{C}.
\]

Thus, if \( p_{art} < p_{ven} \), then \( h_0 = h_v = 0 \) and

\[
Q = \frac{h_0^4}{C}.
\]

A conceptual difficulty arises in the last named case. If \( h_0 = 0 \), how can there be flow? To answer this question, a detailed consideration of fluid dynamics is necessary, which is presented in reference 4, where it is shown that when \( p_{art} > p_{ven} \) the flow adjusts itself so that the smallest value \( h_0 \) can have is to approach zero at the venule exit very rapidly like a cusp. Interpreted in the framework of the bilinear approximation of the thickness-pressure relationship, this means that if the left atrium pressure is indefinitely decreased, the limiting flow, \( Q = (h_0^4 - h_v^4)/C \), is reached first, but eventually a maximal flow, \( Q = h_0^4/C \), can be obtained.

To display these relationships graphically, we may consider the variation of flow and resistance with independent changes of the
FIGURE 11

Curves associated with the ordinate to the left show the variations of flow and resistance with pressure head, \(p_{art} - p_{ven}\), at a constant venule pressure of 3 cm H\(_2\)O. The alveolar pressures are 23, 17, and 7 cm H\(_2\)O for positive inflation, with pleural pressure equal to 0. For negative inflation, \(p_{art}\) is 0, while pleural pressure is negative. \(Q\)' is the flow when \(C = 1\) and \(R' = \alpha R\) in units of \((\mu m)^4\) and \((\mu m)^{-3}\), respectively. Points associated with the ordinate to the right correspond to experimental data by Roos et al. (6) on dogs with left atrium pressure equal to 3 cm H\(_2\)O, pleural pressure equal to 0, and alveolar pressure equal to 23 (*), 17 (•), and 7 (○) cm H\(_2\)O.

arteriolar pressure, \(p_{art}\), the venular pressure, \(p_{ven}\), and the alveolar pressure, \(p_{alv}\). In anticipation of a possible comparison with experimental data on the pulmonary vascular resistance of the dog, we take \(h_0 = 2.5 \mu m\) and \(\alpha = 0.122 \mu m/cm H_2O\). Figure 11 shows the results for the case in which the venule pressure is fixed at 3 cm H\(_2\)O (in all the figures the pressure is in units of cm H\(_2\)O), the alveolar air pressure is 0, 7, 17, or 23 cm H\(_2\)O, and the pressure difference between the arteriole and venule is varied. The pleural pressure is assumed to be zero in the cases of positive inflation and negative in the cases of negative inflation represented by the curve for \(p_{art} = 0\). The values of \(Q\)' and \(R\)' plotted in Figure 11 are, respectively, the flow and the product \(\alpha R\) in units of \((\mu m)^4\) and \((\mu m)^{-3}\). To obtain flow in ml/min and \(R\) in cm H\(_2\)O ml\(^{-1}\) min, one should divide \(Q\)' by \(10^{16}C/\alpha\), with \(C\) in units of cm minutes and \(\alpha\) in \(\mu m/cm H_2O\).

The features of the positive inflation curves in Figure 11 may be explained as follows. Consider the case \(p_{art} = 23\) cm H\(_2\)O. Since \(p_{ven} = 3\) cm H\(_2\)O, \(p_{art}\) will be smaller than 23 cm H\(_2\)O when the pressure head, \(p_{art} - p_{ven}\), is smaller than 20 cm H\(_2\)O. Hence when \(p_{art} - p_{ven} < 20\) cm H\(_2\)O, there will be no flow and the resistance is infinite. When \(p_{art} > p_{alv}\) there is flow. If \(p_{ven} = 3\) cm H\(_2\)O and \(p_{alv} = 23\) cm H\(_2\)O as assumed, \(Q\) will be represented by the solid curves (\(h_v = 0\)), which is exactly the same as the limiting maximal flow (when \(h_v \to 0\)) that can be extracted from a given \(p_{art} - p_{alv}\). By comparison, if this limiting flow is approached from the right (so that \(h_v \to h_0\)), then the flow is shown by the dash-dot curve in Figure 11.

These curves are useful in furnishing some idea about pulmonary alveolar flow when one...
FUNG, SOBIN

varies the pressures in the alveoli, pulmonary artery, and left atrium. From the arterial pressure and flow, we can calculate \( p_{ae} \). From the left atrium pressure and flow, we can determine \( p_{en} \). Hence the flow and pressure relationship can be plotted in the same way as in Figure 11. Although it was shown in reference 4 that when flow exists, \( p_{en} \) cannot be less than \( p_{ae} \), the flow is still given by the formula \( Q = (h_a - h_v) / C \). If the left atrium pressure is sufficiently low, the limit \( h_v \to 0 \) can be reached. Therefore, the flow is bounded between the solid curve and the dash-dot curve of Figure 11.

The curves in Figures 12 and 13 refer to the same values of \( h_0, a, p_{a}, \) and pleural pressure. Figures 12 and 13 (left) give \( Q' \) and \( R' \) when the arteriole pressure is fixed at 26 cm H\(_2\)O and the venule pressure is varied. On the right, Figures 12 and 13 refer to a constant pressure head, \( p_{ae} - p_{en} = 15 \) cm H\(_2\)O while the venule pressure is varied. As before, points representing \( p_{ae} \) greater than \( p_{ae} \) and \( p_{en} \) less than \( p_{ae} \), though unrealizable, are retained in these figures to show the bounds that would be obtained if \( p_{en} \) is roughly replaced by the left atrium pressure.

**Discussion**

Roos et al. (6) investigated the pulmonary vascular resistance by independently varying the pressures in the pulmonary artery, left auricle, trachea, and pleura in open-chest mongrel dogs. It is not possible to directly compare their results with our theory because their data refer to an entire isolated lung whereas our analysis pertains only to the microvasculature. However, we should expect a qualitative resemblance between their measurements and our predictions.

It is necessary to estimate the values of \( h_0, a, T, \) etc., before we can compare our formula numerically with experimental data. These constants are not presently known for the dog. Fortunately, Glazier et al. (5) have measured the capillary dimensions in rapidly frozen

---

**Figure 12**

The variation of flow with venule pressure. Left: Arterial pressure is 26 cm H\(_2\)O. Right: \( p_{ae} - p_{en} = 15 \) cm H\(_2\)O. \( Q' \) is the flow given by Eq. 27 with \( C = 1 \), in units of (\( \mu \)m\(^3\)).

_Circulation Research, Vol. XXX, April 1972_
The variation of the resistance with venule pressure. Left: Arteriole pressure is 26 cm H₂O. Right: Pressure head, \( p_{\text{art}} - p_{\text{ven}} \), is 15 cm H₂O. To obtain actual resistance, multiply \( R' \) by \( 10^{16} \text{ C/s} \), with C in units of cm minutes, and \( a \) in \( \mu \text{m/cm H}_2\text{O} \).

Figures 11-13 were prepared for \( h_0 = 2.5 \mu \text{m} \) and \( a = 0.122 \mu \text{m/cm H}_2\text{O} \). The trends shown by these curves may be compared with the experimental results of Roos et al. (6). In Figure 11 we have taken a number of points from experimental curves of Roos et al. (6) and plotted against the ordinate shown on the right side of the figures. The diamonds denote alveolar pressure of 23 cm H₂O, the squares denote 17 cm H₂O, and the circles denote 7 cm H₂O. It is seen that the experimental curves
and the theoretical curves show the same trend. A similar general agreement in trends between theory and experiment is obtained if the curves of Figures 12 and 13 are compared with the corresponding curves of Figures 5 and 6 of reference 6.

If we identify the corresponding points on the theoretical curves with the experimental data by Roos et al., we can determine the value of the parameter C (Eq. 28) pertinent to the whole lung. However, since these curves are not identical, the calculated value of C will not be constant along the entire curve. A typical calculation is listed in Table 3. For a capillary sheet the variation of C reflects the combined effect of the changes in the lung inflation, the tension in the capillary membrane, the capillary-sheet surface area, and the average length of streamlines between arterioles and venules. Furthermore since we have lumped the blood flow in the whole lung and compared it with a formula derived for alveoli only, the variation of C must also reflect the difference in elastic behavior of the arteries and veins from that of the capillaries. The interpretation is not simple.

The three negative-inflation cases shown in reference 6 were obtained by fixing the intrapulmonary pressure at 0 and decreasing pleural pressure to —7, —17, and —23 cm H2O while the left auricular pressure was kept at 2 cm H2O. The negative inflation case shown in Figure 11 was calculated by setting \( p_{at} = 0 \) in Eq. 30. The pleural pressure does not appear explicitly in our equations, although it controls the lung inflation and hence the values of \( \alpha \) and C. Identification of the curves of Roos et al. with those of Figure 11 yields the C values for the negative inflation cases. Generally, the lower the pleural pressure, the higher is the tension in the capillary membrane and the larger is the capillary-sheet area; but these two factors influence the flow in opposite directions. The influence of the elasticity of the arteries and veins on flow is another complication.

Finally we must point out that although C is intrinsically independent of the thickness, \( h_0 \), the process of identifying experimental curves with theoretical curves which are based on assumed values of \( h_0 \) makes C dependent on \( h_0 \). Figure 14 shows the calculated values of C for \( h_0 = 2.0, 2.5, \) and \( 3.0 \mu m \) and for both positive and negative inflations. The indicated range shows how C varies for flow in the range between 220 to 800 ml/min. This figure calls attention to the fact that an experimental determination of C requires accurate information on \( h_0 \). Once C is known, it may be used to calculate T or L or other parameters.

### TABLE 3

<table>
<thead>
<tr>
<th>( p_{at} ) (cm H2O)</th>
<th>Pleural pressure (cm H2O)</th>
<th>( \Delta p_{at} - \Delta p_{ve} ) (cm H2O)</th>
<th>Theoretical ( Q' ) (ml/min)</th>
<th>Experimental flow (ml/min)</th>
<th>( C ) (flow/Q')</th>
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<td>10</td>
<td>418</td>
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<td>1.79</td>
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</table>

\[ C = 4 \mu L^2 \alpha/(SA) \text{ ml min}^{-1} \mu m^{-4}; h_0 \text{ was assumed to equal } 2.5 \mu m \text{ and } \alpha \text{ equals } 0.122 \mu m/cm H_2O; \text{ left auricular pressure } = 3 \text{ cm H}_2O; \text{ } p_{at} = \text{ intrapulmonary pressure}; (\Delta p_{at} - \Delta p_{ve}) \text{ = pressure head}; Q' = (h_0 - h_*)^2. \text{ See text for more detailed definitions.} \]
The legitimacy of identifying the curves shown in Figures 11-13 with curves of Roos et al. (6) is questionable, because we have not accounted for the regional differences and for the resistance that occur in the arteries and veins. Two possibilities may account for the resemblance between the resistance characteristics of the entire lung and those of the capillary bed. Either the resistance in the arteries and veins is smaller than hitherto believed or the distensibility of the arteries and veins happens to be such that their resistance characteristics are similar to the capillaries with respect to transmural pressure differences. Further experiments to settle this question should be interesting.

Although the application of the integrated sheet-flow formula (Eq. 27) is quite useful in obtaining gross estimates of flow and resistance, we believe that the value of the theory lies in the opposite direction: it provides a tool to link various separate investigations together, such as the elasticity of the lung, the stress and strain distribution, the surfactant effects, the regional differences, the hematocrit distribution, and the hemodynamics. It provides a handle to quantify and analyze the ideas about the elasticity of the pulmonary blood vessels in a long line of investigations.

Appendix A

ESTIMATION OF STRESS AND CURVATURE DISTRIBUTION IN THE ALVEOLAR-CAPILLARY MEMBRANE

The spatial geometry of the alveolar sheet as a whole is defined by the curvatures of the middle surface of the sheet, whereas that of the (alveolar-capillary) membranes is described by the membrane deflection relative to the middle surface of the sheet. Equation 3 is applicable to the individual membrane as well as to the whole sheet. In this case $T_1$, $T_2$, $R_1$, and $R_2$ refer to that of each membrane alone. For a membrane the pressure difference,

$$\Delta p = P_{blood} - P_{alveol}$$

causes it to deflect. The deflection surface can be computed from Eq. 3 if the tensions, $T_1$ and $T_2$, are known. $T_1$ and $T_2$ are caused primarily by inflation of the lung, but they are disturbed locally by the posts. To see this, we note first that all the load acting on the upper membrane must be transmitted to the lower membrane through the posts. The total vertical load acting on the membrane is $\Delta p \times \text{area}$. The vertical load transmitted to the edge of the post is equal to $T_1 \sin \alpha$ per unit length, where $T_1$ is the maximum principal tension per unit length acting in cross sections parallel to the edge of the post and $\alpha$ is the angle of inclination of the membrane to the middle plane of the sheet (Fig. 6). Let $a$ be the radius of the post. At equilibrium we have

$$2\pi a T_1 \sin \alpha = \Delta p \times \text{area of the membrane per post}$$

Hence

$$T_1 \sin \alpha = \Delta p \frac{\text{area of membrane}}{\text{area of post}} \times \frac{\text{area of post}}{\text{circumference of post}}$$

$$= \Delta p \frac{S}{1 - S} \frac{a}{2}$$

where $S$ is the vascular space-tissue ratio. For example, if $a = 2 \mu m$, $S = 0.9$, $\alpha = 90^\circ$, and $\Delta p$ is in units of cm H_2O, then $T_1 = \Delta p \times 9.0 \times 10^5 \times 1 \times 10^{-4} = 0.9 \Delta p$ dyne/cm. At $\Delta p = 20$ cm H_2O, $T_1 = 18$ dyne/cm.
The other principal membrane tension acts in cross sections perpendicular to the edge of the post. If we isolate a small rectangular element in the neighborhood of the post as shown in the upper left corner of Figure 7, then $T_1$ and $T_2$ are vectors as shown in the figure. We can show that in contrast to $T$, which is proportional to $A_\text{p}$ at the edge of the posts, the circumferential principal tension, $T_\theta$, is affected by the blood pressure only slightly. This occurs because when the blood pressure varies $V_{\text{STR}}$ and the circumferential length of the post remains unchanged; consequently the circumferential strain remains unchanged and the circumferential stress changes only slightly with the transmural pressure, $A_\text{p}$.

Thus, in general, the principal tensions, $T_1$ and $T_\theta$, are unequal in the neighborhood of the posts. The larger the membrane deflection, the more unequal $T_1$ and $T_\theta$ would be.

Next we shall show that the curvature of an inflated alveolar-capillary membrane is nonuniform: it is larger at the post than at the center. For this purpose let us first remark that with regard to curvature there are two kinds of surfaces—elliptic and hyperbolic. An elliptic surface is like one on an ellipsoid or a sphere: the two centers of curvature in the two principal directions lie on the same side of the surface and $1/R_1$ and $1/R_2$ are of the same sign (Fig. 8a). A hyperbolic surface is like one on a hyperboloid: the two centers of curvature in the two principal directions lie on opposite sides of the surface so that $1/R_1$ and $1/R_2$ are of the opposite sign (Fig. 8b). A familiar hyperbolic surface is a saddle. A general surface may be composed of both types of surfaces. The alveolar-capillary membrane is such a surface (Fig. 8c). To see this, we first note that if we take a basic rectangular pattern for the posts as shown in Figure 7, then the condition of symmetry implies that the whole pattern of deflection is a repetition of that in the area $OMNA$ (shaded in Fig. 7), where the line segments $OM$, $MN$, and $AO$ are lines of reflective symmetry. The contour lines of equal deflection, $w_0 = \text{constant}$, are sketched in Figure 9. The center point, $O$, (Fig. 7) is obviously an elliptic point, where the membrane is elliptic. On the other hand, in the neighborhood of the post (Figs. 7 and 8c,d) the surface is hyperbolic. For example, consider the point $A$ in Figure 7. At $A$, one of the principal directions is $OA$, perpendicular to the circle, the other is along $AN$, tangential to the circle. The center of curvature in a cross section passing through $OA$ lies below the surface, and that in a cross section containing $AN$ lies above the surface (Fig. 8d). Thus $A$ is a hyperbolic point.

Now let us assume $T_1 = T_2 = T$ and $R_1 = R_2$ at the center $O$, then at $O$ we have

$$\Delta \rho = \frac{2T}{R_1},$$

or

$$\left(\frac{1}{R_1}\right) = \frac{A_\rho}{2T}.$$

On the other hand, we have shown that at the point $A$, the tension $T_1$, (normal to the post) is larger than $T$, but $T_2$ (tangent to post) is about equal to $T$. Let the curvatures corresponding to $T_1$ and $T_2$ be $1/R_1$ and $1/R_2$. From Figures 6–9, it is seen that $R_2$ is approximately equal to the radius of the post, $a$, in magnitude, but opposite to $R_1$ in sign. Thus, applying Eq. 3, we have

$$\Delta \rho = \frac{T_1}{R_1} + \frac{T_2}{R_2} = \frac{T_1}{R_1} - \frac{T_2}{a}.$$  

For the purpose of estimation we may let $T_1 = T_2 = T$; then we see that

$$\left(\frac{1}{R_1}\right) = \frac{\Delta \rho}{T} + \frac{1}{a} \cdot \left(\frac{1}{R_1}\right) = -\frac{1}{a}. $$

As an example, if $\Delta \rho = 10 \text{ cm H}_2\text{O}$, $T = 10 \text{ dyne/cm}$, and $a = 2 \mu\text{m}$, then we have

$$(R_1)_0 \approx 20 \mu\text{m}, \quad (R_1)_A \approx 1.7 \mu\text{m}.$$  

In this case the curvature at the post is about 12 times larger than that at the center $O$.

Appendix B

DEVIATION OF EQUATIONS 11, 12, AND 13

We identify the curvature,

$$\left(\frac{1}{R_\omega}\right) = -\frac{\partial^2 w}{\partial x^2} \left[1 + \left(\frac{\partial w}{\partial x}\right)^2\right]^{-3/2},$$

evaluated at $x = 0$, $y = 0$ with $1/R_\omega$ of Eq. 6. With Eq. 9, the deflection at the center ($x = y = 0$) is

$$w_0 = A \sum_{k_1, n_1} \frac{1}{k_1 n_1} \left[\frac{(1 + 2n_1)^2 + 4k_1^2 - a_1^2/c_1^2}{(1 + 2n_1)^2 + 4k_1^2}\right],$$

Taking logarithm of both sides of Eq. 9, differentiating twice, and evaluating the result at $x = y = 0$, we obtain

$$\text{Circulation Research, Vol. XXX, April 1972}$$
PULMONARY ALVEOLAR SHEET ELASTICITY

\[
\left( \frac{\partial w}{\partial x} \right)_0 = 0, \quad (B3)
\]

\[
\frac{1}{w_0} \left( \frac{\partial^2 w}{\partial x^2} \right)_0 = 2 \frac{B}{A} - \frac{\Lambda}{c^2}, \quad (B4)
\]

where

\[
\Lambda = \frac{8a^2}{c^2} \sum_{n=-\infty}^{\infty} \sum_{k=-\infty}^{\infty} \frac{(1 + 2n)^2 + 4k^2}{[(1 + 2n)^2 + 4k^2]^2} \left[ (1 + 2n)^2 - k^2 \right] - 2k^2 \right] \frac{s^2}{2c^2}. \quad (B5)
\]

Eqs. B2 and B5 provide explicit expressions for the constants \( \Gamma \) and \( \Lambda \) in Eqs. 12 and 11, respectively.

We shall derive Eq. 13 under the approximation that \( T_1 = T_2 = T = \text{constant} \) and that the bending energy can be ignored. Then when a transmural pressure, \( \Delta p \), is applied to the membrane, the strain energy contributed by the lateral load may be equated to the corresponding change in the strain energy. If we let \( \delta w = \delta \Pi \), where \( \Pi \) stands for the infinite product listed on the right hand side of Eq. 9, i.e., \( w \) with \( F = 1 \), then we obtain the variational equation

\[
\Delta p \int \Pi \, dx \, dy = T \int \left( \frac{\partial w}{\partial x} \right)_0 + \frac{\partial \Pi}{\partial y} \, dx \, dy. \quad (B6)
\]

By a substitution from Eq. 9, the right hand side of Eq. B6 is reduced to a linear function of \( \Lambda \) and \( B \). Combining the result with Eq. 11, we obtain Eq. 13 with

\[
K_3 = \frac{\Gamma(4I_1 + \Gamma I_2)}{\Lambda + 2c^2I_2}, \quad (B7)
\]

where

\[
I_1 = \int \Pi \, dx \, dy
\]

\[
I_2 = \int \left[ \left( \frac{\partial \Pi}{\partial x} \right)^2 + \left( \frac{\partial \Pi}{\partial y} \right)^2 \right] \, dx \, dy
\]

\[
I_3 = \int \int \left\{ 2\Pi \left( x \frac{\partial \Pi}{\partial x} + y \frac{\partial \Pi}{\partial y} \right) + (x^2 + y^2) \left[ \left( \frac{\partial \Pi}{\partial x} \right)^2 + \left( \frac{\partial \Pi}{\partial y} \right)^2 \right] \right\} \, dx \, dy. \quad (B8)
\]

The values of \( I_1, I_2, I_3 \), and \( K_3 \) are computed and listed in Table 2 for three values of \( a/c \) and with \( c = 1 \). \( K_3 \) is, of course, dimensionless.

Appendix C

ELEMENTARY ANALOGUE OF THE THEORY

Although the arguments presented above are very simple, the details might have obscured the issue. If we are permitted to use a one-dimensional analogue, then the analytical problem becomes trivial, as is shown below.

Consider two uniform, parallel, horizontal, elastic strings as shown in Figure 15A. At finite intervals these strings are tied by vertical cross members. If we pull on the horizontal strings with a tension, \( T \), the strings will be stretched uniformly. The ratio of the lengths of segments \( bc \) and \( ac \) will remain a constant. This ratio is an analogue of \( VSTR \), the segments \( ab \) and \( cd \) are analogues of the posts, and \( bc \) and \( de \) are the analogue of the alveolar-capillary membrane.

On the other hand, if the post segments were replaced by springs of different compliance than the strings, then \( VSTR \) \( (bc/ac) \) will change with the tension, \( T \) (Fig. 15B). The constancy of \( VSTR \) observed in the pulmonary alveolar sheets of the cat suggests that the elastic structure of the post area (segments \( ab \), \( cd \), etc.) is represented by the analogue shown in Figure 15A.

Let the strings be loaded by internal vertical loading, \( \Delta p \) per unit length, as shown in Figure 15C. The equilibrium of the string requires that

\[
T \times \text{curvature of string} = \Delta p. \quad (C1)
\]

If the tension remains constant and the deflection is small, then the vertical deflection of the string will be proportional to \( \Delta p \).

The change of the average distance between the strings (analogue of the sheet thickness) is given by the sum of the distention of the posts \( aa', bb' \), etc. and the average deflection of the strings.

Now consider flow in a channel of unit width represented by Figure 15D (replace the strings by channel walls). Let the average speed of flow be \( U \), the volume flow rate per unit width be \( Q \), the local thickness be \( h \), and the pressure be \( p \).
Then
\[
U = -\frac{\eta}{\delta \mu} \frac{dp}{dx}, \quad Q = hU. \tag{C2}
\]
In the range of \( p \) in which \( h = h_0 + \alpha p \), we have
\[
\frac{dh}{dx} = \alpha \frac{dp}{dx}. \tag{C3}
\]
Hence
\[
Q = -\frac{h^3}{8 \mu \alpha} \frac{dh}{dx} = -\frac{1}{32 \mu \alpha} \frac{dh^4}{dx}. \tag{C4}
\]
If the channel length is \( L \), then the mean flow in the whole channel is
\[
\frac{1}{L} \int_0^L Q \, dx = \frac{1}{32 \mu \alpha L} [h^4(0) - h^4(L)]. \tag{C5}
\]
For a one-dimensional channel flow the conservation of mass requires that \( Q \) be a constant. Hence the left side of Eq. C5 is exactly \( Q \). In this case, we can integrate Eq. C4 to obtain
\[
h^4 = -32 \mu \alpha Q x + \text{constant}.
\]
But \( h = h(0) \) when \( x = 0 \). Hence the constant equals \( h^4(0) \), and we have the channel thickness distribution:
\[
h(x) = [h^4(0) - 32 \mu \alpha Q x]^\frac{1}{4}. \tag{C6}
\]
This is shown in Figure 15D. When \( x = L \), we obtain the exact result
\[
Q = \frac{h^4(0) - h^4(L)}{32 \mu \alpha L},
\]
which is the analogue of Eq. 27. In the three-dimensional case, the flow along a stream tube is similar to that in a channel, except that the width of the channel is variable as well as the thickness. Therefore, an additional average over the sheet area is necessary.

Footnotes
1The following example may be helpful for those readers who are not familiar with the mechanical theory of membranes and plates. Ordinary rubber and leather are nonlinear elastic materials; when a strip of such a material is stretched, its elongation is not linearly proportional to the load. A mass-spring system with a rubber cord as the spring will not oscillate harmonically in finite amplitude. A pendulum made by hanging a weight at the end of a rubber cord will exhibit such typical nonlinear oscillations as subharmonic resonance. However, when a drum is made of a tautly stretched sheet of rubber, it will respond linearly with respect to lateral loads. A proof of this linearity can be easily found by listening to the drum when it is struck. If it gives a pure tone, it is vibrating harmonically, and the system is linear.
2For any area on the sheet enclosing a sufficiently large number of posts, \( VSTR \) is defined as the ratio of the vascular lumen space to the circumscribing tissue space of that network. Therefore, the fraction of the volume of the posts in the sheet is equal to \( 1 - VSTR \).
3If we treat the rubber membrane as an alveolar sheet, \( VSTR \) is equal to the area of the white portion of Figure 2c or d divided by the total area.
4In the notations of reference 2 and for the lung data from cats presented therein, \( c \) is \( L \), \( a \) is \( \epsilon/2 \), and the interpost distance equals \( (L \sqrt{3}) - \epsilon \), then \( a \approx 2.1 \mu \text{m} \) at a low perfusion pressure (15 mm Hg), \( c = \frac{1}{\sqrt{3}} \) (interpost distance + post diameter) \( \approx 7.7 \mu \text{m} \), and \( c/a \approx 3.7 \).
5For lack of a better term, we shall call an arterial blood vessel that conveys blood into an alveolar sheet an arteriole. Similarly, one that drains the sheet is called a venule. These terms are used here without the usual connotation about the structure of these vessels as defined for peripheral circulation.
6The regression line of reference 5 was redrawn for the range 0 to 25 cm to obtain this value of \( a \).
References


Elasticity of the Pulmonary Alveolar Sheet
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Circ Res. 1972;30:451-469
doi: 10.1161/01.RES.30.4.451
Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4371

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