Does the Surface Tension Make the Lung Inherently Unstable?

By Yuan-Cheng Fung

ABSTRACT

Many authors regard the human lung as a collection of 300 million bubbles independently connected by cylindrical tubes. Under surface tension such a model is inherently unstable in the sense that the small alveoli would empty into the large ones so that the lung would consist only of collapsed and hyperinflated alveoli. It has been demonstrated that this basic model is wrong. My observation is based on the well-known fact that both sides of each interalveolar septum are exposed to ventilated air. When the topological relationship between the alveolar septa is properly taken into account, it can be shown that each interalveolar septum is a minimal surface and that there is no problem of inherent instability in the sense mentioned earlier. However, the lung structure is flimsy and can become unstable in the same sense that an airplane structure or an Atlas rocket can become unstable. The clarification of lung inflation and atelectasis can proceed in a rational manner when the confusion of an erroneous model is removed.

There is a widely held opinion that the pulmonary alveoli are inherently unstable. Comroe (1, p 109) says: “The lung, with hundreds of millions of bubbles of various size, is inherently unstable. Without some special system to prevent it, the lung would empty its smaller alveoli into larger ones and consist only of collapsed and hyperinflated alveoli.” Lightfoot (2, p 154) states: “In the lung, special surfactants must be produced continuously to prevent the collapse of the alveoli across whose walls O₂, CO₂ and H₂O are transferred.” Ruch and Patton (3, p 742) say: “The problem of keeping parallel bubbles open is critical in the lung, where millions of alveoli exist side by side. If these alveoli were inflated like bubbles on tubes in the simple model, then the lung ought to be an unstable structure with only the large alveoli open. The fact that this is not so implies that other factors are involved.” All of these authors go on to name these other factors: the tissue elasticity, the lung lining layer, the surfactants, the dependence of the surface tension of the lung extract on the surface area, the difference of the surface tension in inflation and deflation, etc. However, they do not question the basic inapplicability of the sphere-on-a-cylinder model of the alveolar structure, on which the instability concept rests.

From another point of view, the lung is essentially composed of a spatial fabrication of interalveolar septa that are exposed to air on both sides. This spatial network can hardly be represented as a collection of bubbles. This fact should not be ignored when the stability of pulmonary alveoli is considered.

In the present paper, I will show that the alveoli in an open lung have no inherent instability. The crux of the matter is a proper account of the topology of the alveolar structure. The question of instability arises from the sphere-on-a-cylinder model of the lung, since it is well known, from such popular books as Boys’ Soap Bubbles (4), that bubbles on interconnected tubes are unstable. The question is whether the model is correct. I will show that it is applicable to the pulmonary pleura but not to the alveoli inside the lung.

Fact 1: All pulmonary alveolar septa in adult mammalian lungs are similar. Each septum contains one single sheet of capillary blood vessels and is exposed to air on both sides. This fact is evident from histological specimens of various animals prepared in a variety of ways. Essentially no controversy exists in the literature in this respect, and a large number of published figures corroborate this assertion (Glazier et al. [5], Krahl [6, Fig. 24 p 250, Fig. 28 p 255, Fig. 31 p 257, Figs. 34 and 35 p 259, and Fig. 40 p 266], Sobin et al. [7], von Hayek [8, p 52], Weibel [9, Fig. 24 p 64, and Fig. 100 p 128], West [10, Fig. 1 p 3, Fig. 2 p 4, and Fig. 7 p 9]). Miller (11, p 77) attributed this observation to G. Rainey (1845). Mead et al. (12) based their analysis of lung elasticity on such a model.

Fact 2: At a condition of equilibrium, both sides of each pulmonary alveolar septum are exposed to air at the same pressure. This fact is plainly true for those septa that protrude into the same alveolar...
sac, duct, or respiratory bronchiole, because the pressure everywhere in a sac or a duct must be the same (Fig. 1, septum a-a). Therefore, only for those septa that form the "roofs" of the alveolar sac can this contention be questioned (Fig. 1, septum b-b). Both sides of such septa are exposed to air, but they face two different alveolar sacs or ducts. If the pressures in these two sacs or ducts are the same, then the two sides of the septum are exposed to the same pressure. In a static condition, such as in breath holding, the air is in equilibrium everywhere, and the pressure is uniform in the entire lung. In this case, fact 2 is obviously true.

Fact 3: The pressure loads on alveolar septa are dynamic and small. It follows from fact 2 that an alveolar septum will be subjected to a net nonvanishing pressure load only if the pressures in neighboring alveolar sacs or ducts are different. This situation can occur in dynamic conditions such as in normal breathing. The maximum pressure load acting on any alveolar septum is the maximum difference in pressures in neighboring alveolar sacs or ducts. In such a nonequilibrium condition, air flows from a high-pressure region to a low-pressure region, tending to equalize the pressures.

If the two sides of a particular alveolar septum face two different alveolar sacs or ducts, an investigator can trace up along the airway tree until a common junction is reached. Along each branch there is a pressure drop from that junction to the septum in question. The difference of these pressure drops is the net pressure load which acts on that particular alveolar septum. Computation of this pressure difference requires a knowledge of the detailed parenchymal structure of the lung. Briscoe (13) has given an estimate of the pressure distribution in the human lung at a flow of 1 liter/sec under the assumption of a laminar flow (Table 1). Now, consider an alveolar septum. Suppose that one side of the septum faces an alveolar sac at generation 23, whereas the other side faces a respiratory bronchiole at generation 19. Then, the septum is subjected to a net pressure of \(3 - 0.16 = 2.84 \mu \text{m H}_2\text{O}\). Again, consider another septum whose two

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**FIGURE 1**

Topological structure of pulmonary alveoli. **Top left:** Topologically wrong model for alveolar mechanics. **Top right:** Schematic sketch of the typical topological arrangement of interalveolar septa. Septum a-a: Both sides are in the same alveolar duct; the net pressure difference between the two sides is zero. Septum b-b: One side is in duct A, and the other side is in duct B; the difference in the pressures acting on the two sides is on the order of a few \(\mu \text{m H}_2\text{O}\). **Bottom left:** Equilibrium of pulmonary (visceral) pleura, to which von Neergaard's model applies.
Human Lung at a Flow of 1 liter/sec, under the Assumption of a Laminar Flow, using Weibel's Regular Dichotomy Model

<table>
<thead>
<tr>
<th>Generation</th>
<th>Terminal bronchiole</th>
<th>Respiratory bronchiole</th>
<th>Alveolar duct</th>
<th>Alveolar sac</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure difference (µm H₂O)</td>
<td>16</td>
<td>17</td>
<td>20</td>
<td>23</td>
</tr>
<tr>
<td>Cumulative pressure difference (µm H₂O)</td>
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<td>5</td>
<td>1.4</td>
<td>0.16</td>
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<td>Pressure difference (µm H₂O)</td>
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<td>1716</td>
<td>1726</td>
<td>1727</td>
</tr>
<tr>
<td>Cumulative pressure difference (µm H₂O)</td>
<td>1722</td>
<td>1725</td>
<td>1727</td>
<td>1728</td>
</tr>
</tbody>
</table>

TABLE 1
Briscoe's (13) Estimate of the Pressure Distribution in the Human Lung at a Flow of 1 liter/sec, under the Assumption of a Laminar Flow, using Weibel's Regular Dichotomy Model

side face two sacs, both at generation 23; these two sacs have their common junction in a respiratory bronchiole at generation 19. Since the pressure difference in each branch is about 3 µm H₂O, the pressures acting on the two sides of the septum cannot differ by much more than 3 µm H₂O. At this pressure, the membrane tension required to maintain equilibrium in a spherical membrane of radius 100 µm is only 3 x 10⁻³ dynes/cm². Such a tension is very small compared with the surface tension of water at 20°C (72.75 dynes/cm²), whole human blood (55–61 dynes/cm²), or normal lung extract (2–45 dynes/cm²). Therefore it is clear that the net pressure load acting on the alveolar septa is dynamic and negligibly small.

Fact 4: If the elastic tissue stress in the alveolar sheet is neglected, then under the influence of surface tension each alveolar sheet is a minimal surface. This fact follows from facts 2 and 3. When the net pressure acting on the sheet is zero, the mean curvature of the sheet must vanish, and, hence, the sheet is a minimal surface. (The Young-Laplace relationship for capillary action states that the pressure difference across a surface with constant tension is equal to the product of the tension and the mean curvature of the surface.)

If a ring is dipped into a bowl of soapy water and lifted out again, the soap film formed on the ring is a minimal surface. If the ring lies in a plane, the soap film is a plane. If the ring is a space curve, the soap film forms a saddle surface. Examples of minimal surfaces are often exhibited in museums. Boys' (4) beautiful book contains many illustrations. The application of this concept to the elasticity of the interalveolar septa is discussed in a paper by Fung and Sobin (14).

Now the question of stability of the alveoli can be discussed. It is easy to show first that, if the edges of an alveolar sheet are fixed in space, then the sheet is stable with respect to all small perturbations, because the sheet is a minimal surface. A minimal surface minimizes the potential energy of the system. For a sheet with fixed edges, the potential energy consists of the sum of the strain energy of the alveolar septum due to elastic stresses, the surface energy of the air-liquid interfaces on the septum, and the potential of the pressure load (integral of the pressure difference on the two sides of the septum multiplied by the displacement normal to the pressure). Mathematical expressions of these energies are available in books on classical mechanics (for example, Szilard [15, p 213]). The surface energy is merely the product of surface tension times area. The procedure of minimizing the potential energy can be done by calculus of variations. It is well known that the first variation yields the equation of equilibrium, whereas the second variation determines whether the equilibrium state is stable or not. A positive-definite second variation implies stability and uniqueness of the equilibrium configuration. It is also well known that for a membrane in tension and bending the second variation of the strain energy is positive-definite. The second variation of the surface energy is also positive-definite, whereas the second variation of the potential of external load vanishes. Thus, the sum is positive-definite. Any accidental disturbance of a minimal surface will cause an increase in the potential energy; hence, there will be a tendency to return to the minimal surface. Thus, the sheet is stable.

Now, it is necessary to consider how the edges of the alveolar septa in the lung behave, because a collection of stable sheets may be rendered unstable by the motion of their edges. In this respect, first consider what would happen if the alveolar septa behave as liquid membranes (as in a bowl of soap bubbles). For such liquid membranes the edges between neighboring septa can distend or shrink, and in the limit the perimeter of a septum can shrink to zero. Then the septa will coalesce until a surface of minimal area compatible with the boundary conditions is obtained. Thus, if the alveolar septa were liquid membranes like soap bubbles, the lung parenchyma would be unstable as long as the surface tension was not exactly zero.

A real lung can be stable, because the alveolar septa do not behave like soap bubbles. The septa contain elastic tissues such as collagen, elastin, and ground substance. They have definite structure. The alveolar sheets have four types of edges: (1) those connected to larger blood vessels, (2) those
connected to bronchi and bronchioles, (3) those joining two alveolar sheets together, and (4) those unbounded free edges. The edges of the first two types are attached to massive elastic tissues. The edges of the last two types also have elastic reinforcements. The sheets themselves have an abundance of collagen and elastin fibers, as is clear from the histological photographs shown in references 6, 16, and 17. Obviously these edges cannot shrink to zero length. They exist even when the lung is collapsed (atelectatic), in sharp contrast to a bowl of soap bubbles, which, when collapsed, becomes a single droplet.

Although each alveolar septum is individually stable within the confines of its edges, the spatial framework of intersecting septa can deform and become unstable as a whole. Hence, the problem is reduced to one of studying the stability of the entire lung, because a change in any single septum will affect its neighbors and ultimately the whole lung. The best way to investigate this problem seems to be the method of continuum mechanics. If the macroscopic Cauchy stresses are defined as force per unit area due to the action of all of the alveolar septa intersected by a small area, then the interaction of the alveoli can be posed in the form of the usual differential equations of equilibrium. Such macroscopic stresses can be explicitly related to the tension in individual septa and the geometry of the alveolar structure (18). Details of this approach are presented in a companion article (19). It has been shown (19) that at a given inflation pressure there are two possible states of equilibrium, one corresponding to a larger lung volume in which the elastic stresses dominate and the other to a smaller lung volume in which the surface-tension stresses dominate. At a critical inflation pressure, these two solutions coalesce. For inflation pressures less than the critical value, there is no equilibrium state (at which elastic stresses in the septa vanish).

This method (19) has also been used to investigate several possible types of atelectasis. It has been shown that atelectasis of the focal type, in which the alveoli collapse toward a point, can exist only if the lung volume is smaller than its resting state (at which elastic stresses in the septa vanish). (Such a situation can be achieved by filling the lung with saline and reducing the inflation pressure to zero.) Atelectasis of the axial type, in which the alveoli collapse toward an axis, can exist if the alveolar dimension perpendicular to the axis is smaller than the resting dimension. But a local atelectasis of the planar type, in which the alveoli collapse toward a plane, can be in equilibrium with an inflated lung.

It is not surprising that a lung can be collapsed. It is similar to an airplane, whose wings and fuselage can collapse if the external load is too great, or to an Atlas rocket, which depends on internal pressure for structural integrity. The point is that atelectasis of the lung is a structural phenomenon and is not an instability among parallel, communicative bubbles. When the lung is locally loaded or compressed or when its volume is reduced too much, atelectasis can happen.

Thus, the surfactants of the lung, which reduce surface tension on the alveolar septa, may be relieved from their oft-claimed responsibility of preventing pulmonary alveoli from coalescing into a big bubble. They remain important, but for other reasons. They reduce the tendency toward atelectasis and edema, they make the inflation of an atelectatic lung easier, and they reduce the tension in the interalveolar septa. The reduced tension in the lung parenchyma reduces the effort of breathing. The increased compliance of the capillary-alveolar sheet reduces the resistance to blood flow (14). They are also relevant to edema formation and lymph flow in the lung, as expounded by Staub (20). But, they are not responsible for keeping the individual alveolar septa stable.

MODEL OF THE LUNG STRUCTURE

Most publications picture a lobule of the lung as a bunch of grapes on a vine. Each alveolus is pictured as a little sphere. This picture has a long historical origin (see Miller [11] pp 162-201). It was an impression gained by a partial filling of the airway with Wood's metal, etc. For the purpose of discussing the airway structure, this model is proper and good. But if attention is focused on the blood flow in the capillary blood vessels in the lung, on the distensibility of the thickness of the alveolar septa, or on the deformability of the lung structure, then the picture is misleading. The erroneous instability concept came from such a picture.

That such a picture cannot be correct morphologically can be seen as follows. If each alveolus is a bubble and 300 million such bubbles are crowded together to form a lung, then on a histological section one should see rounded-up bubbles and concave triangular spaces between the bubbles; however, such images are not seen. If such spaces existed, they would be unventilated dead spaces, which again have never been observed.

Forty-five years ago von Neergaard (21), in a great paper demonstrating the importance of sur-
face tension in the lung, introduced the model of an alveolus as a spherical membrane at the end of a cylinder. This model has become a classic, but it is inapplicable when the forces acting on interalveolar septa are considered. For any discussion of pulmonary mechanics, the topological relationship between the interalveolar septa becomes a primary consideration. When such a relationship is taken into account, it is clear that the problem of inherent instability of pulmonary alveoli—the collapsing of smaller alveoli into the larger ones—disappears.

WHERE CAN von NEERGAARD’S MODEL BE APPLIED?

For von Neergaard’s model to make sense, one must recognize that for every interalveolar septum an “inside” and an “outside” exists. Then, he must put a higher pressure on the inside and a lower pressure on the outside. In the topology of the real lung, this situation is unrealistic except on the outer surface of the lung: the pulmonary pleura. In the interior of the lung, the pressure difference on the two sides of each septum is essentially zero, and, according to the Young-Laplace equation, the mean curvature of the septum must vanish. On the pulmonary pleura, however, the alveolar air pressure acts on the inside, and the intrapleural pressure acts on the outside. The difference is the “transpulmonary pressure,” which is on the order of 10 cm H₂O or approximately 10⁴–10⁵ times larger than the net pressure acting on the interalveolar septa. The pleura, then, fits von Neergaard’s model perfectly. On the basis of the Young-Laplace equation, the pulmonary pleura must be a rather wavy surface like the cobblestone streets of old Europe. von Neergaard considered each bulge (a small area of the pleura bounded by alveolar septa) to be spherical, subtending a solid angle smaller than a hemisphere. Such a bulge is mechanically stable. If the pressure were so increased that the surface became hemispherical, it would become unstable if surface tension acted alone. The bulge would then “blow out” into a large bubble, whose ultimate size would be limited only by the elastic tissues in the membrane. To the pleura, all of the old arguments apply. Excellent summaries have been given by Clements and Tierney (22), Mead (23), and Radford (24).

It may be worthwhile to remark that the surfaces of the alveolar septa themselves must be wavy and look like pebblestone pavement because of the finite difference in blood pressure and air pressure. But both the blood pressure and the air pressure are applied to both sides of the septum, and there is virtually no net force to change the mean curvature of the midsurface of the septum. The principal effect of this distending pressure is to increase the caliber of the capillary blood vessels. The effect of this distensibility on pulmonary blood flow has been elaborated on by Fung and Sobin (14, 25).

Jere Mead (private communication) points out that students of lung mechanics are aware of the inadequacy of the simple picture of alveolar instability depicted in many textbooks. As I said earlier, Mead et al. (12) rightly pointed out that in inflating a totally collapsed gas-free lung (as in the first breath of a new born infant), the parallel-bubble model of von Neergaard and the parallel-balloon model (bubble-with-tissue model) of Mead et al. are both completely appropriate and should hold in every detail as the air is pushed in through the airway into the bronchioles and alveoli.

Therefore, from the point of view of pulmonary mechanics, it is inappropriate to regard the lung as a collection of hundreds of millions of bubbles, each independently connected to a system of cylindrical tubes. Topologically the interalveolar septa form a spatial network for which there is no problem of inherent instability by having the smaller alveoli collapsing into larger ones. The stability of the lung with respect to small or finite disturbances and the collapse or atelectasis of the lung can be analyzed with the lung as a structure under appropriate boundary conditions.

References

1. COMROE JH: Physiology of Respiration. Chicago, Year Book Medical Publishers, Inc., 1972
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