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THE SEGMENTAL AND LOBULAR PHYSIOLOGY AND PATHOLOGY OF THE LUNG

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THE introduction of the concept of segmental resection of the lung opened what was referred to at that time as an unwritten chapter in surgical anatomy. This chapter is now quite complete, and gratitude is expressed to Brock, Blades, Huber, Boyden, Scannell, and others for their painstaking contributions to descriptive anatomy. Mastery of the three dimensional intricacies of the interlacing bronchi and blood vessels is now an essential step toward the attainment of the precision required in present-day pulmonary surgery Attention is now directed to another chapter that awaits development—the segmental and lobular physiology and pathology of the lung. Some of the material is available and a firm structural basis has been provided by William Snow Miller, but the parts remain insufficiently related and incomplete. While the concepts that are certain to emerge may not guide our dissections, they will provide us with methods that are based on a more complete understanding of the organ with which we work.

Tribute is paid immediately to the brilliant contribution of Van Allen and Lindskog on collateral ventilation and the aerodynamics of bronchial obstruction presented to this Association many years ago. I shall return to this later on, but wish to comment now on the fact that this discovery was made by surgeons. Physiologists have been slow to accept the results of the easily repeated experiments and the significance of the mechanism has never received the attention it deserves. Possibly one reason is that physiologists are in the habit of dealing with the two lungs as a single organ. It has been their custom to select the trachea as the site to introduce resistance to the entrance and exit of air when experiments are designed to study the aerodynamics of respiration. Similarly the analysis of pulmonary edema has been approached through the mimicry of cardiac failure with abnormal conditions imposed upon the pulmonary circulation

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as a whole, or through the inhalation of irritant gases that are dispersed throughout the entire respiratory tract.

The bronchopulmonary segment with its component lobules is a physiologic unit of the lung just as the segment is a surgical unit of the lung and has become a unit of roentgenologic diagnosis. At the level of the segment or the lobule, obstruction to the outflow of air produced by disease or by neuromuscular action can produce volume and tension changes within a limited lung area at the expense of volume alterations in adjacent areas. The potentialities of this mechanism are lost sight of when all lobules and segments are subjected to identical conditions, which is the case when obstruction or resistance is imposed at the level of the trachea. The discovery of collateral air drift by Van Allen and Lindskog brought emphasis to this important difference.

The increased tension of the air within alveoli that is produced by partial occlusion of the bronchus to an entire lobe is referred to clinically as obstructive emphysema. The mechanism of its production by partial occlusion of the lumen and a widening of the air passages on inspiration and a narrowing on expiration is understood. The basic condition is obstruction to the outflow of air. Similarly, the acute lobar collapse caused by complete bronchial obstruction and absorption of the alveolar air is recognized experimentally and clinically. Both the collapse and the obstructive emphysema that might be expected to occur by the same mechanisms acting within the bronchopulmonary segment are subject to the special conditions imposed by collateral air drift and also by the secretory activity of the bronchial mucous membrane. These will be discussed. I shall, however, begin with the single lobule or a cluster of adjacent lobules and consider the significance of increased tension of the air within the alveoli and a possible relationship to the onset of pulmonary edema.

The several factors that control the passage of exudates and transudates across the wall of the capillary need not be enumerated. They are itemized in a diagram by Drinker (Fig. 1). Attention will be confined to the physical force studied by Landis and Gibbon[1] that assists in the control of filtration edema in the extremities, increasing tissue pressure. These authors measured the effects of the pressure that is built up by an increasing volume of fluid in the tissue spaces as the elasticity of separated tissue elements is encountered. This pressure opposes the filtration pressure within the capillary and slowly brings the leakage from capillaries to a halt. When one attempts to visualize the possibility of such a force acting in pulmonary tissue it is apparent that the unique structure of the lung creates a set of conditions different from those that exist in the tissues of the extremities. The lung tissue may undergo alterations in volume without creating pressure changes of any considerable magnitude. Tissue pressure built up by fluid escaping from the capillaries is a control measure that could be effective in the lung only when carried to the extreme of lobar consolidation, at which point the effect of the limiting membrane of the visceral pleura might be felt. In any event, the control force studied by Landis and Gibbon would be intolerable in the respiratory organs, for leakage from pulmonary capillaries into alveolar spaces is immediately destructive to the primary function of the lung.

In the lung the equivalent of mounting tissue pressure from fluid may be found in increased tension of the alveolar air. As long as the increased tension is confined to a lobule or segment, this portion of lung can distend through encroachment on adjacent lung without appreciable respiratory embarrassment. Were the mechanism activated in all portions of the lung simultaneously, a crisis of labored breathing, anoxia, and, at times, death would be expected, as is the actual fact in bronchial asthma.

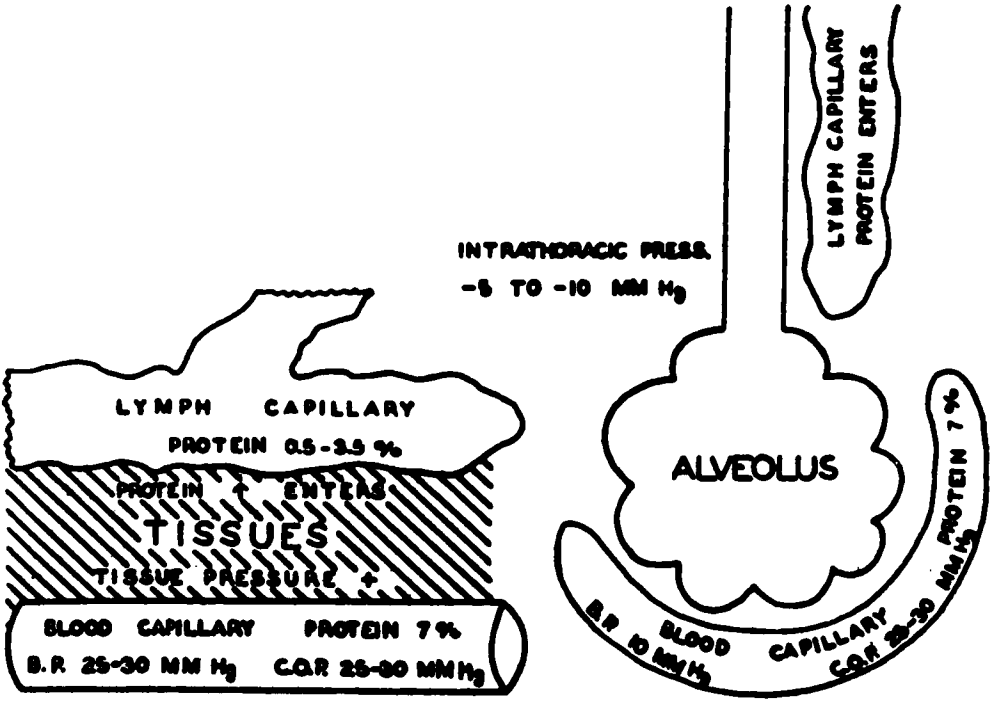


Fig. 1.—Diagrams of contrasting fundamental physiologic relations between blood capillaries and tissues. On the left, a representation of typical relations in the skin; on the right, relations in the lungs. (From Drinker: Pulmonary Edema and Inflammation, Harvard University Press.)

To postulate this happening in a portion of lung threatened by edema requires an explanation for the occurrence of obstruction to the outflow of air from a single area. This is known to occur locally in tension cysts and cavities, but in such instances is usually traced to a valve-like fold of mucous membrane or other "ball-valve" mechanism inherent in the pathologic lesion. In normal lung this action could arise from local reflex activation of the neuromuscular apparatus of the bronchi, for the vegetative neuromuscular apparatus of the human lung appears concentrated in the bronchial tree. It is true that the pulmonary arteries are possessed of smooth muscle but the weight of present evidence indicates that the lesser circulation is regulated in a passive manner except perhaps under certain emergency situations. The elaborate neuromuscular structure that surrounds the air passages has been described in detail (Fig. 2). The constant and vigorous activity of this apparatus has been ex-

pressed by Macklin[2] in the concept of the dynamic bronchus. Far from being rigid tubes, the bronchi undergo inspiratory dilatation with lengthening and expiratory construction with shortening. The walls of the alveoli do little or no stretching and the factor of safety of their elastic tissue is said to be slight.

The bronchial musculature is projected along the bronchiole and respiratory bronchiole to form sphincters about the openings into the atria. Sphincters are also formed about the openings into the alveoli that arise directly from the

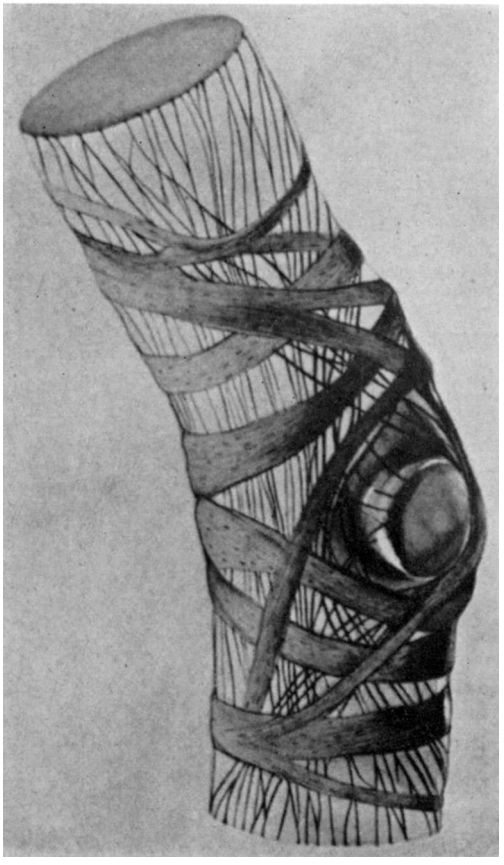


Fig. 2.—Arrangement of smooth muscle bands and elastic fibers. (From Miller: The Lung. Charles C Thomas, Publisher.)

respiratory bronchiole. Trapping of air in the alveoli by obstruction to its outflow may well result from the sphincteric action of the bronchiolar musculature. The increased tension of the trapped air is dependent upon cough or expiratory grunting. Cough is known to have a twofold effect: The forcing of air into alveoli by the increased pressure built up within the air passages, as well as the expulsion of mucus from the upper portions of the tracheobronchial passages that comes on release of the laryngeal sphincter. Fluoroscopists are completely familiar with the increased radiance of the apical segments when they "light up" with cough; also with the dispersion of lipiodol from bronchi into alveoli

with cough. The irritative nonproductive cough that attends the onset of pneumonia or ushers in a paroxysm of asthma thus provides a protective measure that builds up air tension in alveoli to support the capillary walls by external pressure.

The sequence of events may be pictured somewhat as follows (Fig. 3): (1) inspiratory widening of the terminal bronchioles; (2) early in expiration the quick closure of the laryngeal sphincter with elevation of endobronchial air tension by the muscular effort of a cough or grunt; (3) trapping of air in lobules under increased tension; (4) slow reduction in the tension by collateral air drift through the pores so that on the next cough the lobule receives a blast of fresh air with renewed oxygen supply.

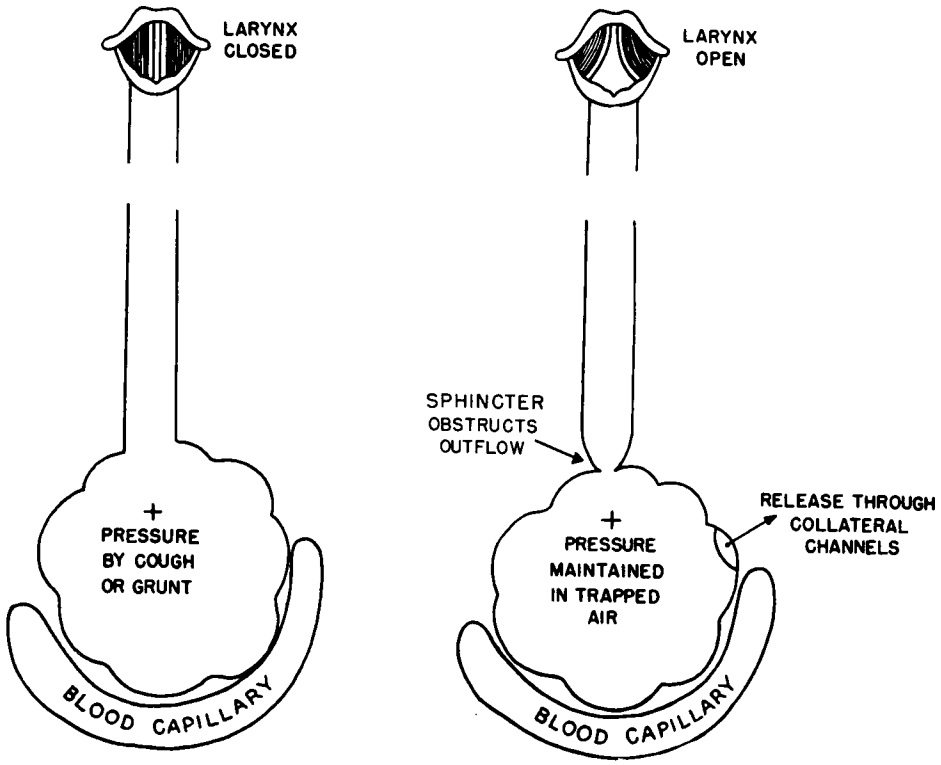


Fig. 3.—Diagram representing mechanism to establish and maintain intra-alveolar air pressure supporting capillary wall in a segment or subsegment of lung.

Surgeons of World War II, faced by localized pulmonary edema resulting from the direct trauma of a chest wound, found that the effective course of management lay in clearing the upper respiratory passages, controlling pain by intercostal nerve block, and aspirating pleural blood and air that might be limiting lung expansion. The cough reflex was not suppressed by morphia nor was the chest wall motion restricted by tight strapping. By these measures the wet massive collapse of the lung seen in World War I was practically unknown.

The use of a gaseous medium under tension as an extrinsic means of applying increased pressure to support the capillary wall provides the theoretical basis for administering oxygen under pressure in the treatment of irritant gas poisoning and other forms of generalized pulmonary edema as recommended by Barach. The intrinsic mechanism was forecast by Keith in 1909[3] and also by Miller,[4] who stated that, "the action of the bronchial musculature in expiration is not passive, but active, and its action in regulating the tensions within air sacs deserves more attention than physiologists have given to it." Surgeons who work daily with the human lung are well aware of the seeming vagaries of lobular and segmental inflation and deflation. Local reflex changes in circulation that are commonly observed in the abdominal viscera as a result of manipulation and exposure are not discernible in the lung. It is possible that the commonplace local changes in ventilation may have a similar origin and significance. Again, the experienced thoracic surgeon holds great concern for the immediate restoration of the cough reflex after it has been temporarily suspended by anesthesia. This is usually rationalized on the basis of clearing the major air passages. Perhaps it is equally important in the protection of traumatized lobules from the hazards of posttraumatic edema.

The level of action for these mechanisms that integrate ventilation and alveolar air tension, and that I suggest are available for edema control, seems to be at the bronchiole and respiratory bronchiole, the site of the sphincters around the necks of the air sacs. The character of the epithelium has already changed before this point is reached and goblet cells have disappeared. At the level of the segmental bronchus, however, the secreting membrane of the respiratory tract is well developed and introduces a factor in obstruction of the air passages that does not occur at the bronchiolar level. Obstruction of the segmental bronchus is followed by retrograde filling or drowning of the bronchioles and air sacs by the pent-up secretions. In the absence of infection this appears to be a relatively benign event. Accurate observations are not available, but presumably water and salts may be removed by the blood capillaries and protein by the lymphatic channels. The lumens of these obstructed small bronchi become filled by cylindrical casts of a tough rubbery substance. The situation is revealed in the pathologic findings of mucoceles and certain cysts but will not be explored at this time. However, certain physiologic and pathologic implications of the secretory activity of the bronchial mucous membrane deserve brief comment if only to display our present state of ignorance concerning them.

As an example, consider the enigma of the so-called Kartagener syndrome-bronchiectasis, situs inversus, and agenesis of the accessory sinuses. The pathogenesis of the bronchiectasis component has always been baffling. When Adams and I[5] tried to develop a hypothesis some years ago we could only postulate as others had done, that the bronchiectasis was a stigma of maldevelopment. Whether the maldevelopment was structural or took the form of some obscure predisposition to bronchiectasis could not be determined. It was pointed out, however, that the bronchiectasis itself took the form of the acquired rather than the congenital type. Since that time I have had the opportunity of observing two children with situs inversus and agenesis of the sinuses but without bron-

chiectasis demonstrable in complete series of bronchograms. Both children displayed an extraordinary increase in the secretory activity of the mucous membrane of the respiratory tract. This was manifested by the production of large quantities of mucous secretions both by cough and profuse nasal discharge. The content of pus was minimal. Both children had been born in the chemotherapeutic era of medicine and both had enjoyed the protective concern of well-to-do families able to provide winters in the southwest under able medical surveillance. Threatened episodes of respiratory infection had been handled by preventive measures including chemotherapy and careful observation for collapse and re-expansion of segments. The stage was set for the development of segmental or lobar bronchiectasis but as yet it had not appeared.

In the light of these observations a careful review of the childhood experience of an intelligent young man with the Kartagener syndrome, including bilateral basal bronchiectasis, brought forth a reconstruction of events that paralleled the story of the two children. The profuse mucoid secretion in his case became purulent only after an attack of bronchitis at the age of 14.

It seems, therefore, that the bronchiectasis of the Kartagener trilogy is a secondary manifestation and that the underlying disturbance may be an altered secretory activity of the respiratory tract epithelium.

Recently Bergstrom, Cook, and Scannell[6] have assembled a family history that not only confirms but throws added light on the subject. In this family the bronchiectasis, or as has been proposed the abnormality of the respiratory tract epithelium that precedes it, occurs in two siblings with situs inversus and in two others without situs inversus. Whether the stigma that affects the mucous membrane has a structural basis, a metabolic basis, or is attributable to some derangement of the neurosecretory mechanism, remains unknown. The question as to whether the respiratory mucous membrane behaves as a single organ in its secretory activity or whether lobar or segmental regional autonomy of control may exist also remains unanswered.

Returning to the pulmonary circulation, I remind you that many studies on minor pulmonary embolism that are now classics have demonstrated a collateral drift of blood in the pulmonary capillary network sufficient to maintain a slowly moving circulation despite occlusion of sizable terminals of the pulmonary artery. Increased back pressure in the pulmonary veins or abnormal slowing of the capillary stream by reduced respiratory excursion may convert a bland infarct into a hemorrhagic infarct. It is likely that this capillary blood drift crosses segmental boundaries. In the immediate situation imposed by the embolism, this drift of blood has no respiratory significance; having passed through functioning alveoli, the drifting blood is arterialized and not mixed venous blood. It may help maintain the integrity of the capillary wall, although there is some appearance of edema even in bland infarction, despite the fact that alveolar ventilation continues.

With the vast margin of safety provided the respiratory organs, the functional loss of a few lobules or even segments might seem to be unimportant. There is some evidence, however, to show that the lodgment of minute emboli of

cells and agglomerations of tissue debris in the lesser circulation may be such a common event that it can be regarded as a physiologic happening. If this is so, the margin of safety would soon be used up if minor embolism in the terminal arteries always spelled hemorrhagic infarction. The healing process would reduce such an occluded area to a thin plate of fibrous tissue. A series of healed hemorrhagic infarctions thus would cause the remainder of the pulmonary tissue to distend with the changes of complementary emphysema and a concomitant reduction in functional capacity. The limited damage that attends bland infarction, in contrast, does not reduce the volume of the lung.

While the loss of a few square meters of diffusion surface from the 140 with which the lung is equipped for its primary function of respiration might be insignificant, the lung appears designed to conserve a *space-occupying* function. This is true even though the respiratory function of a segment is destroyed. It is observed in the series of events that follows bland infarction with the collateral drift of capillary blood. The same preservation of lung volume apparently to occupy space is also provided for by collateral air drift, as will now be shown.

After the demonstration of Van Allen and Lindskog[7] it was expected to find many instances where collateral respiration came into play in conjunction with pathologic lesions of the lung. However, both the original experiments and the postulated situations in which the drift of air might serve a useful function seemed to characterize collateral air drift as an emergency device. The reason it is not seen more commonly is that segmental bronchial obstruction at levels chosen for experimental demonstration and as observed with tumors, strictures, and foreign bodies takes place in the secretory zone of the respiratory tract epithelium. As a result, maintenance of air in the affected alveoli by passage through collateral channels soon is masked by the drowning of those alveoli with retained secretions. One must descend below the mucous cells to the finer ramifications of the air passages to find an example of collateral air drift at play. The example I shall provide is not protective in the sense of maintaining gas exchange function; it does act to conserve what I have referred to as the space-occupying function of pulmonary tissue that protects the remainder of the lung from complementary emphysema.

In describing the techniques of resection of a pulmonary segment,[8] reference was made to identification of the boundaries of a segment by deflating the lobe, clamping the segmental bronchus, and then inflating the lobe. The segment remains deflated. This is readily demonstrated in a normal lung and holds true with certain forms of bronchiectasis. After further study, however, it was apparent that only in the exceptional cases can this procedure be utilized. Either the segment under consideration is airless and shows the fibrotic changes of long-standing inflammation that preclude inflation, or the segment does not deflate when the endobronchial air pressure is reduced. Indeed, certain of these segments do not deflate when removed from the body and left with widely open bronchial apertures. The air in the alveoli is trapped air. It then occurred to us that bronchography not infrequently shows dilated bronchi extending into

air-containing lung segments, but never with lipiodol entering the atria and alveoli. The dilated bronchi resemble the branches of a sea sponge known as "dead-man's-fingers" *(Chalina arbuscula)* and extend into a zone of radiant, air-containing parenchyma but in no way communicate with it. Other observers had commented on this phenomenon and referred to such bronchi as "leafless trees." Robert Klopstock was working with me at the time these observations were made, and using the techniques of Van Allen and Lindskog it was not difficult to demonstrate that the imprisoned air entered or escaped from the area either by way of an adjacent segment with normal bronchi or from an occasional normal branch bronchus within the segment. By more careful preoperative

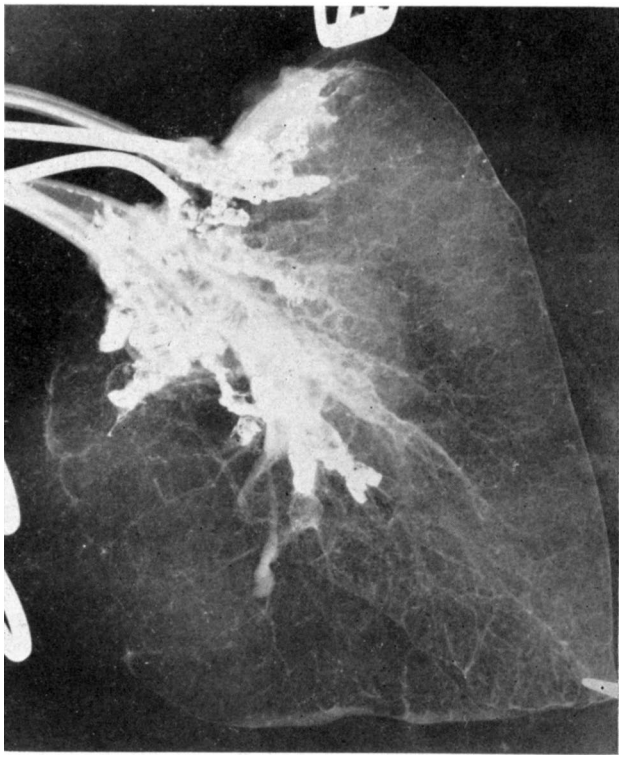


Fig. 4.

fluoroscopic examination it was possible to confirm the fact that the air in such a segment was indeed trapped exactly as in obstructive emphysema. From the standpoint of gas exchange, this area of lung is devoid of function. The air that reaches the alveoli within it has already passed through other alveoli and reached equilibrium with the pulmonary capillary blood. The air-containing segments are simply filling space. But this, as has been suggested, may have a functional significance.

Klopstock studied these surgically removed lobes with lipiodol injections of the bronchi, correlating the observations with those made at operation and those

made with air-flow techniques. Brief reference is made to three examples, using soft tissue roentgenograms as records.

Fig. 4 records the lipiodol injection of the air passages of a lobe excised from a 23-year-old man with bronchiectasis dating to attacks of pneumonia in early childhood. When the chest was opened the right lower lobe appeared emphysematous but with little or no deformity in its architecture. The dorsal segment deflated when the endotracheal pressure was reduced and inflated when it was elevated. The basal segments could not be deflated. The air that filled their

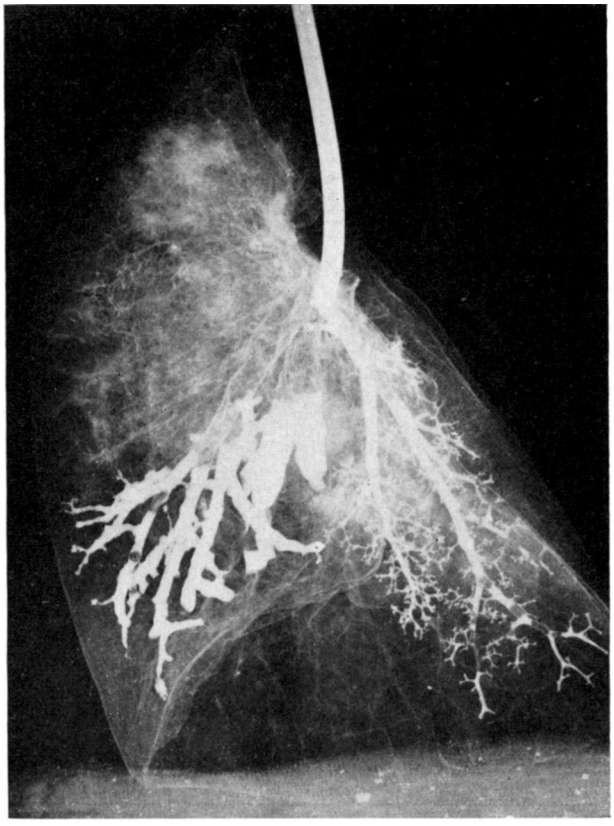


Fig. 5.

alveoli was trapped, having entered through collateral channels by way of the dorsal segment. The lipiodol filling showed no communication between the dilated bronchi of the basal segments and the surrounding alveoli of those segments.

This specimen showed, in addition, a phenomenon that is quite constant and that provides further confirmation. The dorsal segment displayed the usual pattern of anthracotic pigmentation, but this was entirely lacking in the basal segments. It is understandable that airborne particulate matter cannot traverse the tortuous channels of the air-drift filter.

As an example of a more complex situation, reference is made to the lower lobe of a young adult woman (Fig. 5). The dorsal segment is not injected in this surgical specimen. It still contains a cloud of alveolar lipiodol from the preoperative bronchogram showing normal air sac filling. One of the three basal segments appears normal, one is grossly bronchiectatic, and one shows a cylindrical enlargement of the bronchi. In only one or two areas of the segment containing the cylindrically dilated bronchi do flecks of alveolar filling appear. All of these segments were found symmetrically distended with air when exposed at operation, in fact, architecturally the lobe appeared completely normal. The affected segments were being maintained in an inflated form by collateral air drift from the adjacent normal segments.

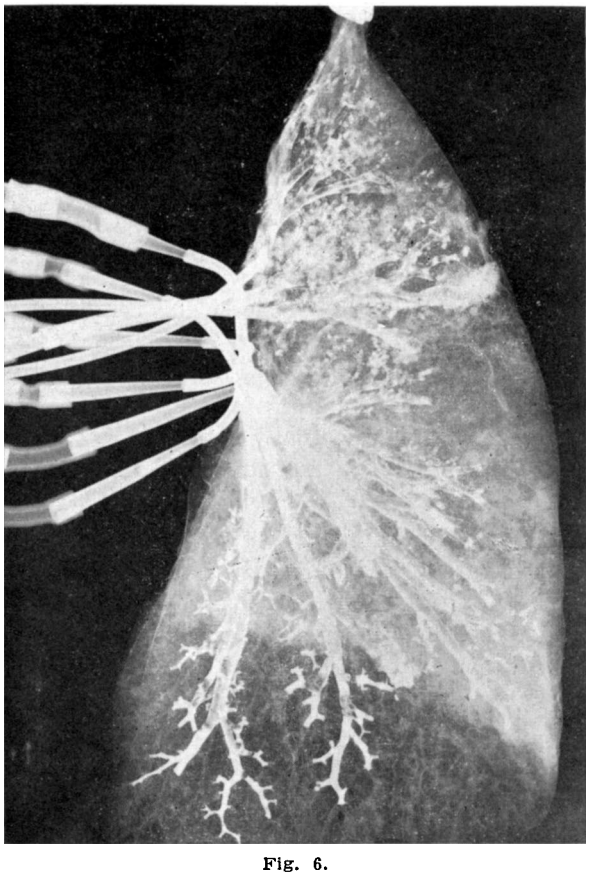


Fig. 6.

From the gross bronchiectasis displayed in these specimens, one may proceed to the state of affairs in the lobe shown in Fig. 6. This is the surgical specimen from an older woman, aged 56 years. Two bouts of pneumonia had been localized to this lobe, then ensued cough, continuing slight fever, lassitude, and disability. This patient was treated for many months with rest, chemotherapy, and climatic change but without improvement. The dorsal segment shows normal

alveolar filling. In the basal segments there are a few areas in which the lipiodol enters the alveoli, but in general it is the picture of the "leafless tree," particularly in the mesial segment. There is little deformity that can be termed dilatation of the bronchi, and in fact it is a misnomer to classify this disease as bronchiectasis. The significant lesion appears to be *obliterative bronchitis* and *bronchiolitis.*

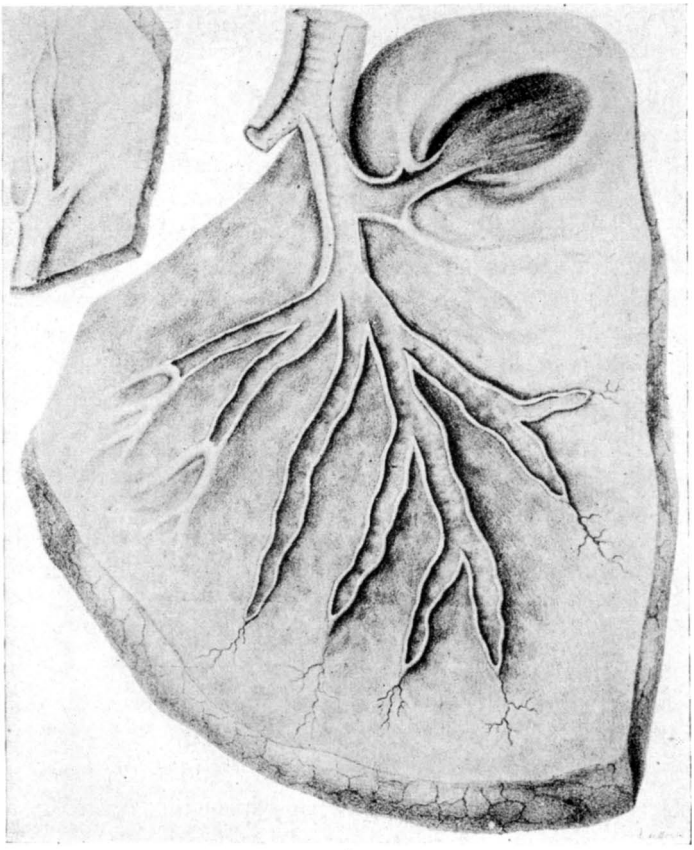
As shown in the preceding illustration this process may exist in one segment and true bronchiectasis in another. In some cases, as those described by Blades[9] as pseudobronchiectasis following virus infection, the obliteration appears to be temporary; in others, when the finer air passages are converted into fibrous cords, it is permanent.

For an adequate description of the morbid anatomy of the changes in the air passages that have been presented, one may go back to 1835.[10] This was the era when lungs were carefully examined in their three dimensional aspects and not chopped up into limp fragments for two dimensional cellular microscopy. In these old illustrations (Fig. 7) of bronchiectatic lobes, the bronchioles and presumably the smaller bronchi are shown reduced to fibrous cords that can be traced to the proximity of the visceral pleura. Observations regarding the state of the surrounding alveoli were not recorded.

I cannot agree with Fleischner[11] that this represents a *preatelectatic* phase in the development of bronchiectasis, although this could be true if the obliterative fibrosis of the bronchi and bronchioles progressed to involve all segments of a lobe. It appears, on the contrary, to represent a static phase of what may initially have been an acute inflammatory process that destroyed and obliterated the finer air passages. It might actually represent a *postatelectatic* phase, if collateral air drift penetrates an area that was atelectatic during the acute phase of infection.

As I have implied, there are, of course, other forms of bronchiectasis in which small dilatations and herniations of the bronchial wall can be traced in the finest bronchial twigs and even in the bronchioles. There are other more common types where inflammation and fibrosis have destroyed the architecture of the air sacs and bronchioles, and converted the lung or a portion of it into an airless, solid organ in which the mechanism of collateral air drift has been forever lost.

Obliterative bronchitis and bronchiolitis with or without atelectasis may be significant in the development of certain types of bronchiectasis and not merely be a by-product of the pathologic process. As a primary lesion it would favor the retention of secretion in the larger bronchi and possibly promote the atrophy of disuse. Basic to an understanding of obliterative disease in the finer air passages is a recognition that their lining membranes are very different from those of the larger divisions. They have a serous rather than mucous secretion. Thus, it is possible for the flames of infection to sweep across the lung and produce permanently obliterated bronchioles without evidence of collapse or fibrosis that is discernible on the x-ray film. Recent studies on lung function tend to confirm this statement.



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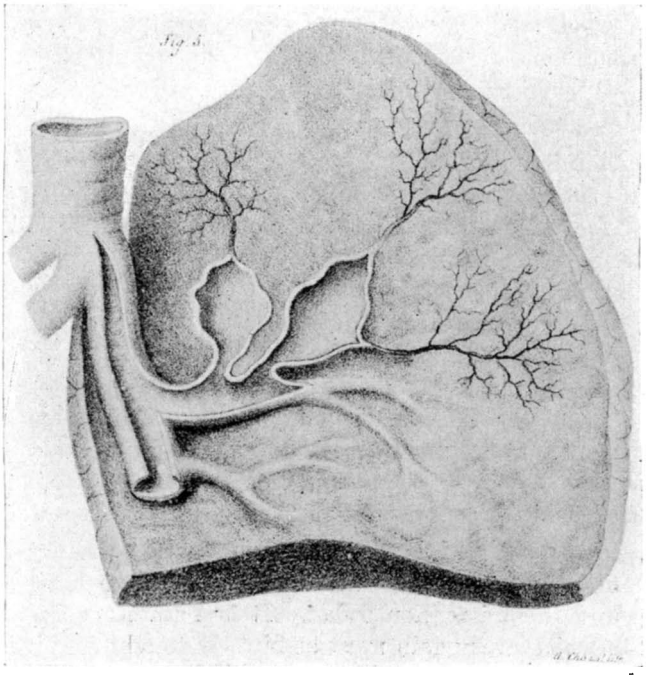

B.

Fig. 7.

Both capillary blood drift and interalveolar air drift are phenomena unique to this organ. Respiratory function depends on ventilation, not on alveoli containing air; it depends on an active capillary circulation, not on capillaries containing blood. Air-containing lung with normal pulmonary circulation as well as blood-containing lung with normal ventilation may be ineffective from the standpoint of respiratory exchange when the air has entered by way of adjacent alveoli and the blood has entered after traversing adjacent capillaries.

The structure of the lung appears to maintain its spatial integrity despite serious functional deterioration. Intricate mechanisms exist that act to maintain its volume and shape. It is unlike parenchymal organs that dwindle in size following the obliteration of ductal systems or occlusion of vascular channels. It does not become small with the changes of senile atrophy, in fact, the atrophic changes of the aging process produce quite the opposite result as recognized in senile emphysema. When normal lung is called upon to fill spatial demands the changes of complementary emphysema ensue, presumably with further reduction of the functional margin of safety with which this organ is so liberally provided. A segment of the kidney or the liver may be removed without significant reduction of the function of the part that is left behind. Not so with the lung. Surgical principles of conservation of function thus center in conservation of volume.

The apparent homogeneity of the structure and function of the lungs is highly deceptive. They are not two gas bags suspended in a container. Morphologically and functionally the lungs are a highly integrated organ system in which the whole must be considered in relation to the parts, and the parts in relation to the whole and to each other. The two lungs, the single lung, the lobes, the bronchopulmonary segment, and the lobules must be studied from this point of view. Only in this manner can the natural laws be disclosed that will ultimately guide pulmonary surgery.

I have made full use of the immunity that a Presidential Address affords to rebuttal and critical discussion. It must be taken for granted that the interrelationships of factual observations that have been presented will be recognized as hypotheses and valued only as such. I shall conclude with the expression of a conviction that is neither a fact nor an hypothesis, but a value judgment. Surgeons who stand on the threshold of the surgery of the lung today will not find prestige and fame in treading the easy pathways that were open to the older members of this Association. We enjoyed for a brief period the heyday of rapid technical development that follows the initial conquest of a new territory. Advance in the surgery of the lung will not be derived from technical experience alone. Anyone who gains the opportunity to operate on enough patients will have had experience, but it does not follow that one learns from it.

The area I have outlined is not only unexplored territory but it is a no man's land. Our allies in the medical sciences seem to have other pressing affairs that occupy their attention today. It is a territory in which surgeons may work but in which every gain must be bitterly fought for; it is remote from

the glamour of scialytic lights and the comforts of lucrative trade. Yet in this area and adjacent ones lie the concepts that are essential if further advance in the surgery of the lung is to be realized.

REFERENCES

1. Landis, Eugene M., and Gibbon, John H., Jr.: The Effects of Temperature and of Tissue Pressure on the Movement of Fluid Through the Human Capillary Wall, J. Clin. Investigation **12**: 105, 1933.
2. Macklin, Charles Clifford: The Musculature of the Bronchi and Lungs, Physiol. Rev. **9**: 1, 1929.
3. Keith, Arthur: The Mechanism of Respiration in Man. Further Advances in Physiology, edited by Leonard Hill, London, 1909, Longmans, Green & Co.
4. Miller, William Snow: The Musculature of the Finer Divisions of the Bronchial Tree and Its Relations to Certain Pathological Conditions, Am. Rev. Tuberc. **5**: 689, 1921.
5. Adams, Ralph, and Churchill, Edw. D.: Situs Inversus, Sinusitis, Bronchiectasis—A Report of 5 Cases, Including Frequency Statistics, J. THORACIC SURG. **7**: 206, 1937.
6. Bergstrom, William H., Cook, Charles D., and Scannell, J. Gordon: (To be published.)
7. Van Allen, C. M., and Lindskog, G. E.: Collateral Respiration in the Lung. Rôle in Bronchial Obstruction to Prevent Atelectasis and to Restore Patency, Surg., Gynec. & Obst. **53**: 16, 1931.
   Van Allen, C. M., Lindskog, G. E., and Richter, H. G.: Collateral Respiration. Transfer of Air Collaterally Between Pulmonary Lobules, J. Clin. Investigation **10**: 559, 1931.
8. Churchill, Edw. D., and Belsey, Ronald: Segmental Pneumonectomy in Bronchiectasis. The Lingula Segment of the Left Upper Lobe, Ann. Surg. **109**: 481, 1939.
9. Blades, Brian, and Dugan, David J.: Pseudobronchiectasis, J. THORACIC SURG. **13**: 40, 1944.
10. Reynaud, A. C.: Mémoire sur l'oblitération des bronches—Mémoires de l'Académie Royale de Médecine, vol. 4, Paris, 1835, Pl. 3 & 5, J. B. Baillière et fils, p. 167.
11. Fleischner, Felix: Pathogenesis of Bronchiectasis, Am. Rev. Tuberc. **42**: 297, 1940.