Morphologic Changes in Pulmonary Emphysema*

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INTENSE INTEREST EXPRESSED BY CLINI-
cians, physiologists, and pathologists in pulmonary emphysema has stimulated the need for establishing some criteria for the normal anatomy and subsequent pathologic change. Regardless of the conflict in the theories of etiology, we must be cognizant of certain changes which take place in the pulmonary parenchyma. The fine relationships between structure, function, and the effects of the disease process previously have been difficult to study. Routine histologic studies on conventional 5μ sections of lung provided earlier morphologists with considerable information regarding certain structural changes. Newer techniques developed in recent years now provide a means for obtaining additional pathologic information which was beyond the capabilities of previous methods. One may now survey overall changes in the gross sections of the lung, as well as investigate additional histologic responses in the alveolar wall, vasculature, and corresponding airway structures. Documentation of these changes, combined with previously known criteria about the pathology of pulmonary emphysema, should provide the clinicians and physiologists with a more accurate concept of this disabling disease.

In order to evaluate the abnormal, it is essential that we understand the normal structure of the lung. The three-dimensional aspects of the newer methods of study permit a more thorough and careful study of the entire lung. This is feasible when the relatively rigid 1 mm. slices of the entire lung are grossly surveyed and the structural formation evaluated. The alveoli are small and uniform in size, and appear to be hexagonal in shape. The sweep of the alveolar membrane can be studied readily with an ordinary hand lens. Changes in the texture and consistency may be visualized due to the semi-transparent state of the normal alveolar wall. Frequently one may also observe the relationships between bronchiolar structures, alveolar ducts, and alveoli. It will also be noted that the alveolar membrane is an intact structure extending from one septum to another without grossly visualized "pores" of Cohn. The latter may be seen occasionally as microscopic rounded spaces.

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Figure 1: "Pores" of Cohn in alveolar membrane.

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in the alveolar membrane that are frequently no larger than the diameter of a mononuclear cell or red blood cell (Fig. 1). Whether these pores are "normal" or actually represent the earliest lesion of pulmonary emphysema is still subject to considerable conjecture.

Microscopically, the uniform and hexagonal structure of the alveoli can be seen (Fig. 2). The septal regions are sharply defined and occasionally contain sparse granules of pigment. These areas of pigmentation in the normal lung usually reflect the environment and occupation of the individual concerned. The alveolar membrane itself does not demonstrate evidences of an inflammatory reaction (Fig. 3).

The walls of the terminal bronchioles, respiratory bronchioles, and alveolar ducts are regular in size, thin, and appear to taper from the bronchiole to the region of the alveolar vestibule. The alveolar ducts are invariably rounded and approximately 50μ in diameter. Thick 300μ sections demonstrate a delicate capillary surrounding the duct and extending as a network throughout the alveolar membrane. The reticulum structures are condensed in the region of the septum and send fibrillary extensions into the alveolar wall.

Pulmonary emphysema is now considered to be a destructive process in the lung despite the literal Greek translation meaning "to dilate" or "to distend." Numerous investigators now consider destruction of
Figure 4: Rent or slit formation on alveolar wall, x 100.

Figure 5: Early slough and fenestra formation.

Figure 6: Fenestra enlarging and in excess of 50 microns, x 100.
the alveolar wall the essential pathologic change. The decompartmentation of alveolar spaces associated with other pathologic changes produces a decreased surface area for gas diffusion as well as uneven distribution of certain gases. The etiology at present cannot be attributed to one cause, but multiple factors are now suspected. The frequent association with chronic bronchitis interjects an inflammatory component to the concepts of bronchial obstruction, idiopathic degeneration, alveolar wall weakness, congenital defects, reaction to pigment deposits, and progressive fibrosis. Despite the single or multiple etiologic theories, certain pathologic changes have been observed in varying stages of this disease. Observations other than functional studies are not available in the early stages of the disease. The preliminary lesions may occur in the region of the terminal bronchiole, alveolar duct, or in the alveolus itself. Based on the study of our cases, we are inclined to feel that the primary lesion probably occurs in the alveolar wall, and that the abnormalities in the adjacent structures are secondary manifestations of alveolar involvement.

The overall pathologic changes in advanced pulmonary emphysema will vary considerably in extent and degree. This is to be expected since it has already been suggested that this disease may be based on degenerative changes associated with several totally unrelated etiologic factors. Vascular changes in the region of the
bronchioles and alveoli have not been sufficiently studied to comment on the role played in the production of pulmonary emphysema. It is certainly possible that many of the visualized changes may be due to the effects of ischemia. Whether these changes were present prior to vascular involvement and merely exaggerated by mechanical capillary compression or obliteration is still unknown.

The gross observation and microscopic formation of alveolar fenestræ is one of the earliest manifestations of lung destruction and probably of major importance in progression of this disease.\(^1\)\(^2\) This change usually occurs on pigmented or non-pigmented portions of the alveolar membrane that has already undergone fibrotic change (Figs. 4, 5 and 6). The first manifestation is usually that of a slit or rent in an area of the alveolar wall which may be considered ischemic secondary to fibrosis. This slit-like opening enlarges into an ovoid-shaped space, greater than \(50\mu\) in diameter, and provides an enlarged communication with the adjacent alveolar space. The edges of the fenestræ are usually devoid of capillary structures in contrast to the alveolar ducts. Later, when the fenestræ have reached a size greater than \(50\mu\) in diameter, they are usually delineated by small capillary structures found in the alveolar wall (Fig. 7). This is probably due to impingement of the expanding edge of the fenestrum on capillary structures that remain in the uninvolved portion of the alveolar membrane. The stresses within the residual portions of the alveolar

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**FIGURE 9:** Fibrosis alveolar wall, x 160. **FIGURE 10:** Extensive destruction and fibrosis, x 40.
membrane usually cause fenestrae to remain ovoid in shape until they fuse into larger spaces. During the process of decompartmentation, we find that the enlarged fenestrae are separated from each other by what grossly appears to be fibrotic strands. Microscopically, these structures are small capillaries which have gradually become partially fibrosed (Fig. 8). Eventually these fibrotic capillaries will rupture and retract, with the formation of one large space where two fenestrae were previously present. The rupture of these capillaries in the final stages of fenestral fusion may be associated with clinical hemoptysis. It can be visualized readily that the continuation of this process will cause considerable destruction of the pulmonary parenchyma and loss in gas diffusion surface area.

The reticulum network also demonstrates a marked condensation in the alveolar wall adjacent to the enlarging fenestra. Frequent foci of degeneration can also be seen in areas of pigmentation. The reaction of pigment upon the alveolar wall is not clearly understood as yet, but may play a role in the production of insidious fibrotic change (Fig. 9). Finally, the progressive destruction of the alveolar structures is seen as extensive rarified areas in the pulmonary parenchyma (Fig. 10).

**SUMMARY**

Certain constant gross and microscopic changes seen in pulmonary emphysema have been presented and contrasted with normal structures of the lung. The etiologic bases for these changes are still unknown, but it is apparent that multiple causes are involved.

**REFERENCES**


For reprints, please write Dr. Hentel at VA Hospital, Albuquerque.

**SWEATING AND CONGESTIVE HEART FAILURE**

Increased sweating is a symptom of a large group of children with congestive heart failure. Such children produce a larger quantity of sweat after pilocarpine iontophoresis than controls. The salt and water loss due to increased sweating in children may, to a limited extent, offset renal sodium retention in congestive heart failure.