

THE ALVEOLAR PORES OF PNEUMONIA.*

By WILLIAM SNOW MILLER, M.D.

(From the Anatomical Laboratory of the University of Wisconsin, Madison.)

PLATES 41 AND 42.

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In a previous report (6) some of the factors leading to the formation of alveolar pores were discussed. It was there shown that in the early inflammatory stage of acute lobar pneumonia there is an engorgement of the blood capillaries followed by a serous exudate into the alveoli in which epithelial cells, leucocytes, and red blood corpuscles are present. These elements provide the necessary ferments by which the blood serum is converted into fibrin and the classic exudate of red hepatization is formed.

The walls of the pulmonary alveoli are made up of a network of capillaries, on each side of which there is a network of branching and anastomosing fibrils (the reticulum), reinforced here and there by elastic fibers, an occasional bundle of collagenous fibers, and covered on the outside by a layer of epithelial cells.

The epithelium which covers the walls of the alveoli is frequently described as being of two types; one made up of large non-nucleated plates, the other of small nucleated cells which are often arranged in groups. In other words, the epithelium is said to be discontinuous. This, however, is not the case.

At birth, before respiration is established, the epithelial cells are more or less cuboidal in shape and completely line the walls of the alveoli. With the establishment of respiration and the full distention of the lung, they are stretched out into thin squamous cells and their nuclei are often indistinct; as soon, however, as atelectasis takes place, they return to their non-distended condition and the alveoli are found to be lined with cuboidal cells which, not infrequently, give to the collapsed alveoli the appearance of small bron-

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chioli, from which they can be distinguished by the absence of smooth muscle in their walls.

When from any cause a serous exudate is poured out into the restricted space beneath the epithelium the cells are pushed off by the *vis a tergo*. Occasionally it happens that this is so equally applied that the epithelium lining an alveolus is pushed off as a continuous sheet. In Fig. 1 such is the case, and various degrees in the conversion of squamous cells into nearly detached spheroidal cells can be recognized. In the alveolus in the upper right quadrant of Fig. 1 what appears to be a large non-nucleated plate can be seen closely applied to the alveolar wall. Sections situated immediately above and below the one from which the photomicrograph was taken show the flattened nuclei belonging to the individual cells. The opposite is true of the alveolus in the center of the photomicrograph; sections immediately above and below the one photographed show what appear to be long non-nucleated plates where nuclei appear in Fig. 1 and by careful examination the boundaries of each cell can be made out.

Shedding of the epithelium constitutes but one step in the formation of a pore. If the epithelium on the opposite side of the alveolar wall remains intact, then no pore can result from the shedding of the epithelium on one side of an alveolar wall. The epithelium on the opposite side of a given alveolar wall may be pushed off by the accumulation of serum behind it, but this must take place from diametrically opposite sides of an alveolar wall to play a part in the formation of a pore.

When there is an extensive shedding of the epithelium on one side of an alveolar wall a free exit is provided for the serum through the sieve-like openings in the network of the reticulum. Should this occur on both sides of an alveolar wall, as so frequently happens in the mechanical edema of mitral stenosis associated with insufficiency of the valves, then the alveoli on each side of the alveolar wall are filled with serum in which free epithelial cells can be seen assuming a more or less spheroidal form. It is on account of this extensive shedding of the epithelium, and the absence of fibrin threads, that pores are so difficult of demonstration in a mechanical edema, although they may be as numerous as in a lobar pneumonia.

The clinical history of the case from which the sections used to illustrate this paper were taken, is unknown to me. The lung was one of a number of specimens sent to me. It proved to be from an early case of lobar pneumonia, and from numerous sections three were selected for photomicrographs to illustrate steps in the formation of alveolar pores.

In Fig. 2 there has been an extensive exudate poured out from two sides of an alveolus and converted into fibrin. Near the angle formed by the union of the two alveolar walls, on its lower side, an epithelial cell can be seen somewhat swollen but still attached by its extremities to the wall of the alveolus. Behind this cell only clear serum is present, for as yet it has not found its way into the lumen of the alveolus. An earlier stage in desquamating epithelium can be seen at the right of this cell on the upper surface of the alveolar wall, and still further to the right three cells can be seen slightly raised from its surface and somewhat swollen. The condition of the exudate at this point is exceedingly interesting. If the photomicrograph be examined by means of a strong reading glass it can be seen that extending through the alveolar wall, into the space beneath the middle epithelial cell, there is a delicate film of fibrin. Had these three cells been completely detached, a pore would have been formed at this point and a thread of fibrin would have extended through the alveolar wall from the lower alveolus into the upper alveolus.

Fig. 3 presents quite a different picture. Here the exudate has escaped from a very definite point. One can see where the epithelium has given way and allowed the exudate free exit into the alveolus. The epithelial cells themselves can be seen attached to the strands of fibrin as they leave the alveolar wall. If the epithelium on the opposite side of the alveolar wall, from that through which the exudate has escaped, be examined it will be found that, although in some places the epithelium has been shed off, directly over the place where the exudate found its exit the epithelium is present although the cells are slightly swollen. Although the amount of exudate is large, the injury to the alveolar wall is of such a nature that no pore has been formed.

In Fig. 4 opposite sides of the alveolar wall have been denuded of

epithelium and a well marked pore has been formed with a thread of fibrin passing through it. As in Fig. 3, epithelial cells can be seen attached to the fibrin thread close to the point where it passes through the alveolar wall.

RECAPITULATION.

Evidence is seen in Fig. 1 that the alveoli are lined with a continuous epithelium.

In Fig. 2 there has been extensive shedding of the epithelium and broad sheets of fibrin are attached to the alveolar wall. On the right the exudate has escaped from both sides of the alveolar wall and the network of fibrin has begun to appear beneath the epithelium on its upper side. Owing, however, to the fact that the epithelium is still intact, though slightly raised from the surface, no pore has been formed.

In Fig. 3 the exudate has found its exit from a very definite point and has been converted into fibrin which radiates into the alveolus. No pore has resulted because the epithelium on the opposite side of the alveolus is still intact.

In Fig. 4 the epithelium has been pushed off from diametrically opposite sides of the alveolar wall and a pore has been formed with a thread of fibrin passing from one alveolus to an adjoining alveolus.

Pores are as numerous in edema due to mitral stenosis with insufficiency of the valves as in pneumonia, but there are no threads of fibrin to direct attention to them.

This study confirms my previously expressed opinion—pores are *not* normal, preformed openings.

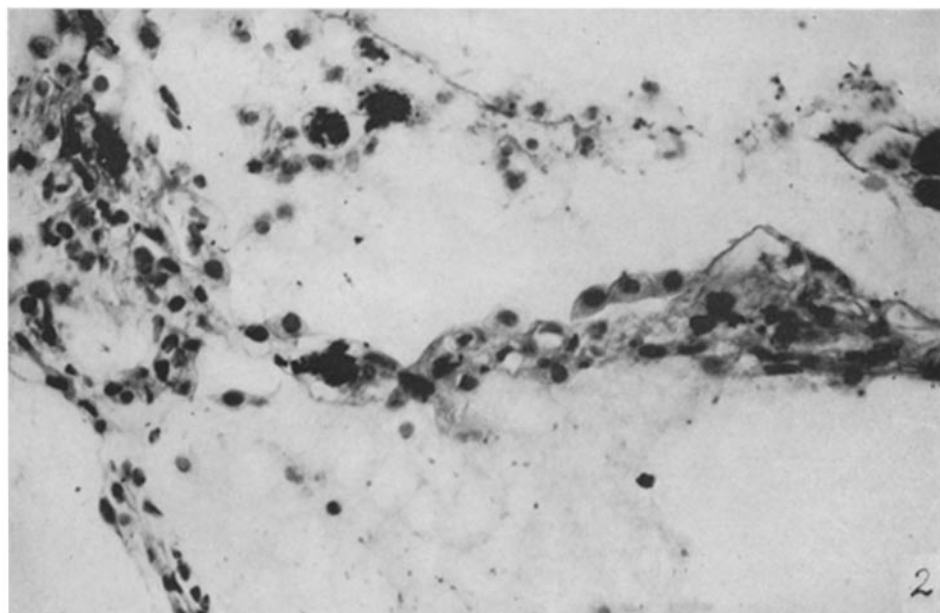
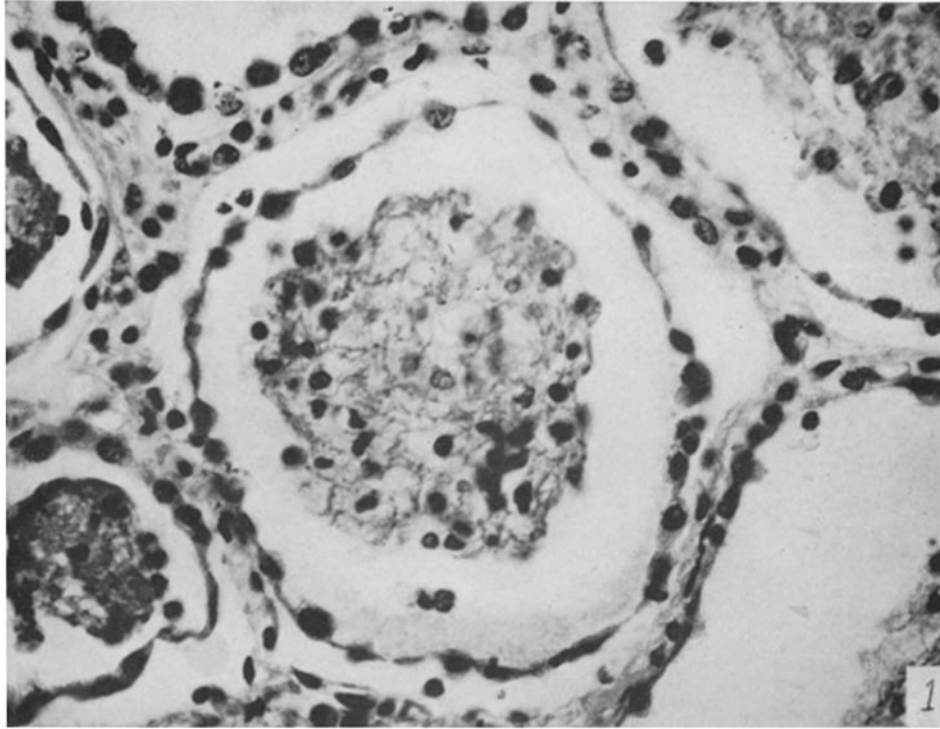
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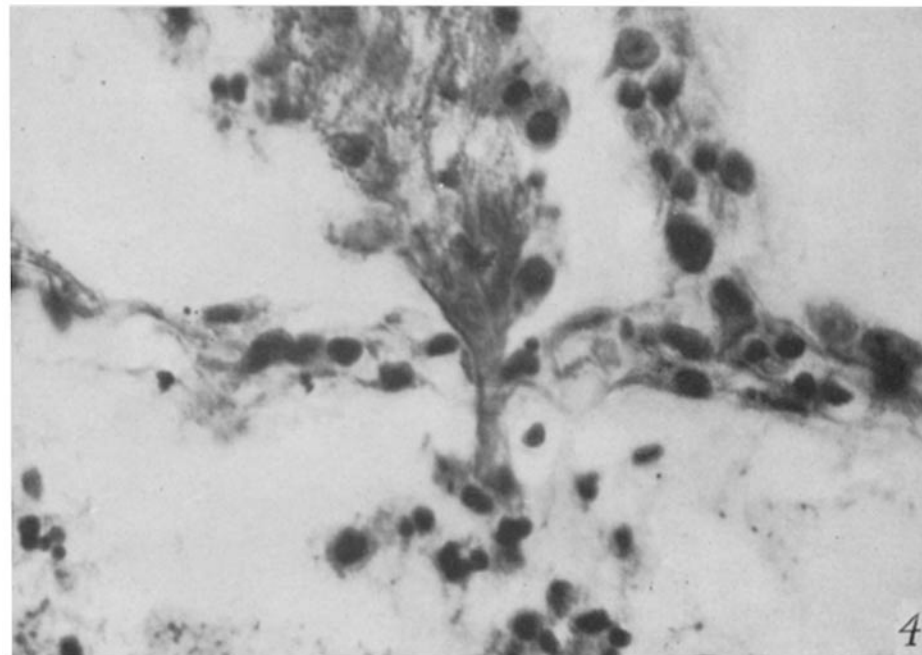
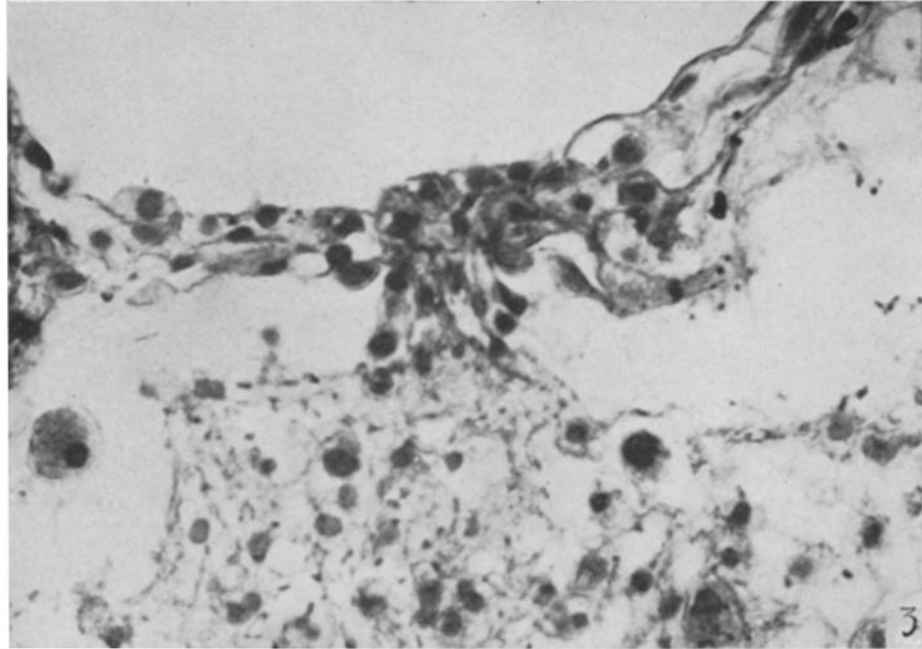
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EXPLANATION OF PLATES 41 AND 42.

All the photomicrographs are from untouched negatives. For explanation of the figures see text. Figs. 1 and 2, $\times 500$. Figs. 3 and 4, $\times 800$.



(Miller: Alveolar pores of pneumonia.)



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