

DISSEMINATED NECROSIS OF THE PULMONARY CAPILLARIES IN INFLUENZAL PNEUMONIA *

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One of the difficulties in attempting to find an explanation of either the hemorrhage or the huge edema¹ of the lungs in influenzal pneumonia is that many different stages are encountered, most of them well along or late in the disease.

Very early in this attempt, attention was directed to the presence of a layer of fibrin close to the lining of the alveoli and ducti alveolares, often a very thin layer and in some instances the only fibrin present. With lobar pneumonia (by this term I mean a disease which is lobar pneumonia not only anatomically but also clinically), such a limitation of the fibrin deposit to the alveolar lining is not common.

Another outstanding condition is the necrosis of the alveolar lining epithelium, which appears early as a hyaline layer in which the outline of the separate cells is entirely lost, thus becoming a layer of necrotic cells sometimes with fibrin on its inner surface or in it and in some instances with very little fibrin anywhere else in the alveoli. Later in the disease these cells are reproduced and the large new cells may be found with mitotic nuclei in them.

DISSEMINATED CAPILLARY NECROSIS

While studying these as well as other conditions, I encountered a feature of the changes in influenzal

is readily found in places markedly changed, where the alveoli are already filled with blood or plasma, with the addition at times of the comparatively slight leukocytic exudate so characteristic of influenzal pneumonia. But with such changes it may well be inferred that necrosis of the interalveolar walls and of the capillary meshwork they contain is altogether secondary to alter-

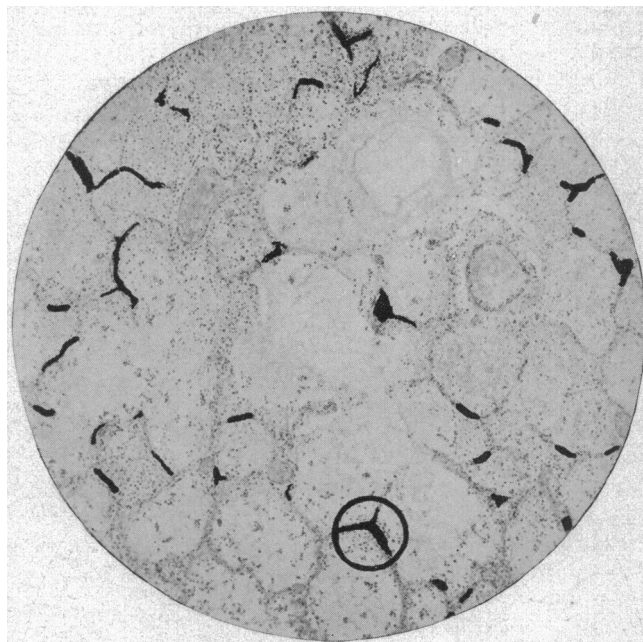


Fig. 2.—The necrotic regions are made black with india ink to show their number and the amount of the interalveolar capillary meshwork and alveolar wall involved; \times about 60.

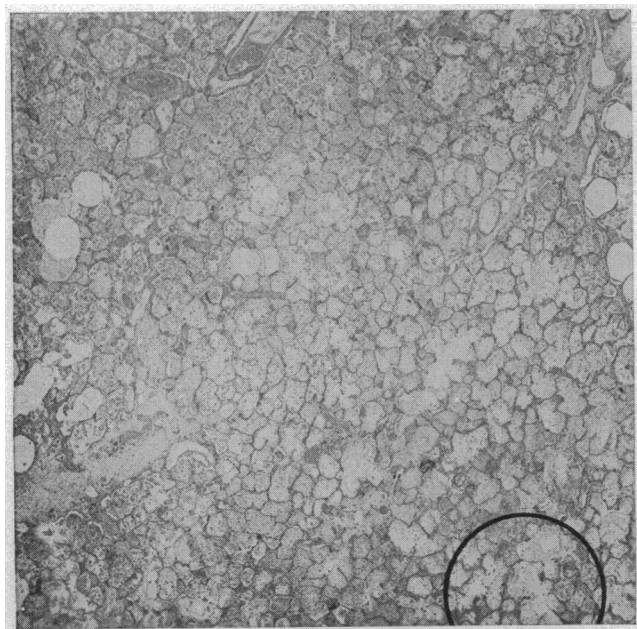


Fig. 1.—Most acini in this section of about 7 sq. mm. of lung tissue from a patient who died from influenzal pneumonia contain some red blood corpuscles, some none; all contain some leukocytic exudate. Edema is more widespread than either of these alterations. The necroses indicated by Fig. 2 are everywhere as abundant as in the small part of the section selected for Fig. 2; \times about 14.

pneumonia which may throw some light not only on the hemorrhages but also on the edema. This feature, a disseminated necrosis of the interalveolar capillaries,

ations within the air sacs. But examination of many sections from many postmortem examinations has revealed some conditions in which such an inference of necrosis secondary to the pneumonia may well be doubted, and the accompanying photomicrographs are an attempt to set these lesions clearly forth with the hope that others, too, will search for them.

Although present in a few lungs seen in other postmortem examinations, the best examples of this disseminated necrosis were found in some of the small regions of pneumonia in the lower lobe of the right lung (weight 1,350 gm.) of a soldier, aged 23, who became acutely ill after having had a slight cold for six days and who died four days later.

Figure 1 represents about 7 sq. mm. (magnification 14 diameters) of a section in which the pathologic condition is chiefly edema. Scarcely any alveoli are jammed full of red blood cells. A little fibrin is peripherally disposed in the air sacs, and a little exudate of leukocytes is present in most of them, but at no place is there any highly cellular consolidation. The pale oblong space jutting in from the lower half of the left edge is a hugely dilated perivascular lymph channel. Part of the circle in the lower right-hand corner represents the region shown in Figure 2 (magnification 60 diameters); in this the portions of the interalveolar meshwork of capillaries which are necrotic have been made black with india ink, as accurately as possible. The region included in the circle near the lower margin is shown in Figure 3 (magnification 470 diameters).

The changes that are illustrated by these figures, such as fragmentation of nuclei and "nuclear dust" in tissue taken a few hours after death, and, moreover, in which there is no suggestion of postmortem change; a

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1. LeCount, E. R.: The Pathologic Anatomy of Influenzal Bronchopneumonia, *J. A. M. A.* 72: 650 (March 1) 1919.

widely disseminated, sharply outlined focal necrosis of the minute blood vessels and with so little of alteration except edema in the lung tissue, goes a long way toward at least suggesting an explanation for a number of things, such as the early hemorrhages in the lung, the escape of such large amounts of fluid from the blood into the lungs and from them into the pleural cavities, also the "button-like" firm peripherally located regions of consolidation compared by many observers to hemorrhagic infarcts.

They also suggest that the disease if not very early a systemic infection may become so later, because the nature of these lesions is best explained by embolism. In such sections as these no organisms other than those commonly present have been found.

CONCLUSION

In conclusion it may be well to emphasize that the 7 sq. mm. of Figure 1 is but part of a section, and that the necrotic regions represented by india ink in Figure 2 may be found not only in all parts of the 7 sq. mm. of

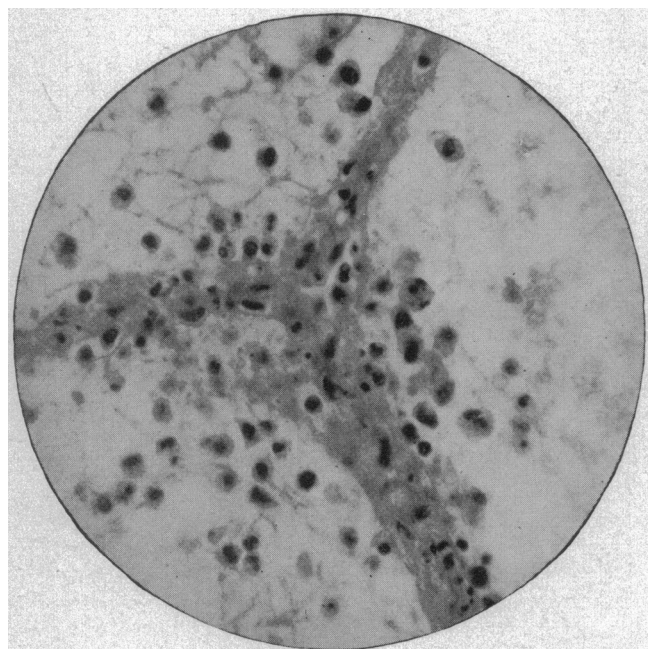


Fig. 3.—No red blood corpuscles or very few are present in the necrotic places, more commonly at junctions of adjacent alveoli, and their walls are replaced by leukocytes, fragmented nuclei and fibrin, for the channels and circulation are interrupted. The capillary meshwork is continued at the ends of these necrotic regions and the blood circulates in them as usual; \times about 470.

Figure 1, but also in all parts of the section containing the 7 sq. mm., and in other sections as well; also, as stated, in sections of some of the other lungs examined. It may well be that such discrete necroses as these, with so little of other change except edema, represent a phase of the disease which is of short duration.

Influenza has been compared with the exanthematic fevers such as measles. Prodromal engorgement of the conjunctival blood vessels is referred to by many observers,² also the early appearance of lividity of the pharynx, described as a red fringe or crescent bordering the hard palate, with minute hemorrhages and a papulovesicular rash in the livid mucosa (Alexander). Moreover, Oberndörfer³ has defined the disease as a

bacteriemia with localization of the virus in the pulmonary blood vessels and secondary infection of the lung tissue so involved. For the foregoing reasons it has seemed best to attempt some description of these necroses at this time.

STUDIES IN STREPTOCOCCIC INFECTIONS AT CAMP CUSTER, MICHIGAN

WITH SPECIAL REFERENCE TO INFLUENZA AND OTHER
ANTECEDENT INFECTIONS

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Sixty-five per cent. of the deaths from acute infections occurring in this camp since its establishment and coming to necropsy have been due to the streptococcus. If the month of October, 1918, is excluded, when influenza was epidemic here, the streptococcus has been responsible for 75 per cent. of the fatal acute infections. For more than a year the attention of this hospital has been focused on the diseases caused by this extremely prevalent and virulent organism, and the facts which this experience has brought to light are here assembled.

TABLE 1.—BACTERIOLOGIC FINDINGS IN ACUTE INFECTIONS
AT NECROPSY

	Deaths from Streptococci		No. Deaths from	
	Number	Per Cent.	Pneumococci	Other Acute Infections
Prior to the influenza epidemic...	37	70	10	6
During the influenza epidemic....	142	62	78	8
Since the influenza epidemic.....	26	86	1	3
Total	205	65	89	17

Reference to the accompanying chart will show the total incidence of streptococcus, compared to its closest competitor, the pneumococcus. The incidence of measles has also been charted. It will be seen that streptococci, except for a period in the spring and summer of 1918, have always outnumbered pneumococci. During the summer months, living conditions peculiar to military establishments are less productive of carriers, and cross infections by such organisms as hemolytic streptococci appear to be less frequent. Certainly during the summer months the relative incidence of these two infections more nearly approached what is commonly observed in civil life.

This camp was several months old before streptococcal infections began to appear. It was in January, 1918, that acutely fulminating pleuritis appeared associated with bronchopneumonia, terminating fatally in a high percentage of cases. Irons and Marine¹ were among the first to recognize the relation of these fatal infections to such antecedent factors as exposure and fatigue, or to other diseases, such as measles. Throughout the winter, streptococcal invasions con-

2. Alexander: Berl. klin. Wchnschr., 1918, No. 38, abstr., Deutsch. med. Wchnschr., 44: 1171, 1918. Bloomfield and Harrop: Bull. Johns Hopkins Hosp. 30: 1, 1919.

3. Oberndörfer: München. med. Wchnschr. 65: 810, 1918.

1. Irons, E. E., and Marine, David: Streptococcal Infections Following Measles and Other Diseases, J. A. M. A. 70: 687 (March 9) 1918.