

In completely collapsed, rabbit lungs, the lumina of the alveolar ducts, alveolar sacs and alveoli, the walls of which become shortened, thickened, and folded on themselves, are represented as narrow, branching clefts. At the junction of the terminal and respiratory bronchioles with the alveolar ducts, the bronchial epithelium and the reticular, basement membrane on which it rests cease abruptly. The connective tissue and blood vessels of the lamina propria of the smallest terminal and respiratory bronchioles continue on and form the walls of the alveolar ducts and alveoli. The free surfaces of the alveolar ducts between the mouths of the adjoining alveoli are covered with capillary loops, which are seen in thin sections to arise on one side of a septum and continue over the connective tissue knob and onto the adjacent septal wall.

Each alveolar septum contains a network of capillaries which course back and forth from one side of the septum to the other. The capillaries lie in the meshes of the reticular and elastic fiber networks and form loops which bulge into the almost obliterated, former, air spaces. The reticular and elastic fibers in and on which the capillaries rest are separated from the alveolar spaces by the capillary network. The septal cells do not form a continuous membrane over the capillary loops in the alveolar walls following collapse of the lung, but remain in the intercapillary spaces intimately associated with the connective tissue as isolated cells. In the alveolar walls the capillaries with their reticular and elastic tissue support are enclosed in an amorphous, ground substance, which can be demonstrated with the Mallory-Azan stain. In short, a continuous, histological epithelium is not demonstrable in the respiratory portion of the completely collapsed, rabbit lung.

An embryogenic study of the rat lung is being made to determine the origin of the septal cells.

7822 P

Pathogenesis of "White Bile."*

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White bile is a relatively rare but clinically important condition on account of its grave prognostic significance. My own previous work on this subject (experimental production of white bile in

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rabbits) gave the following results; that one might expect to find it when there was present an active infection in the bile ducts, and that it was also dependent upon the activity of the liver cells.

Ligations of the common duct were made in the dog and 4 weeks later bacteria were injected into the bile tracts in order to see what influence infection had on the production of white bile.

Eleven dogs were operated upon of which 4 died from 2 to 20 days after the operation (Group 1). In 6 dogs 4 weeks later bacteria† were injected into the gall-bladder. One dog was kept as a control without the second operation. Three of the 6 dogs survived the second operation for only from one to 13 days (Group 2). The remaining 3 and the control dog remained alive and were sacrificed after 4½ to 5½ months (Group 3). All 3 dogs of Group 3 had white bile. In one dog of Group 2 there was very light colored bile. Both of the other dogs of Group 2, the control dog of Group 3 and all the dogs of Group 1 had frank green bile. Chemical analysis of bile in Group 3 showed a marked diminution or a complete lack of the main constituents of bile (bilirubin, cholesterol and bile salts). In the cases with white bile from Group 3, the following common characteristics were noted. There was the clinical picture of a severe infection lasting several months. The dogs were apparently very ill and bacteriologically there was a virulent infection in the bile accompanied by numerous leucocytes in the sediment. In the control dog these phenomena were absent. It is interesting that in Group 2 one of the dogs with all the signs of infection for 32 days had a bile that was very light colored while the other 2 dogs without signs of infection had green bile.

It appears, therefore, that we find white bile in the common duct in cases of obstruction which had been accompanied by severe infection after a considerable time. A review of the few cases in the literature in which the bile has been studied after relief of the common duct obstruction (Walters and Greene, Andrews³) gives further support to this theory. Clinically, much clear fluid is discharged from the fistula the first few days, to be replaced finally by colored bile from the liver. The presence of pigment in the liver and in the liver cells in generalized icterus speaks against the conception that this colorless bile has been produced by the liver cells

† *Streptococcus hemolyticus*, *Staphylococcus albus* and *S. aureus*.

¹ Andrews, Edmund, Hrdina, L., and Dostal, L. E., *Arch. Surg.*, 1932, **25**, 1081.

² Aronsohn, Hans G., *Brunn's Beitr. zur klin. Chir.*, 1932, **156**, 63.

³ Walters, Waltman, Greene, Carl H., and Frederickson, Clyde H., *Ann. Surg.*, 1930, **91**, 686.

themselves. Furthermore it appears hardly conceivable that the function of the liver cells in a space of 6 to 8 days after relief of the obstruction could change itself from that of a colorless secretion to normal bile. My theory of the white bile in relation to the clinical observations noted after relief of the common duct obstruction is as follows: First, the decolorization of the stagnant bile in the bile duct is caused by infection. The liver cells produce for a considerable period after obstruction a green bile and this secretion is finally stopped on account of the rise in pressure in the liver or it may flow back into the blood stream. After the relief of the obstruction, the white bile already present in the dilated bile ducts pours out first. The fluid discharged in the first few days one might expect to be the secretion of the bile tract epithelium in which a further recurrent infection has been stirred up by the operation. Finally the liver function itself returns, provided that the liver cells have not been too severely damaged, and true bile appears.

7823 C

Thyroid Hypertrophy in the Rat with Reference to the Effect of Light.*

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In describing the thyroid hypertrophy and colloid loss in rats exposed to cold and darkness for 10 to 25 days the writer¹ noted that he had not seen any striking changes due to darkness alone. Bergfeld² had previously stated that in 4 to 6 weeks, absence of ultraviolet light caused thyroid hypertrophy irrespective of the environmental temperature and that in his small series no effects attributable to cold were found if the room were illuminated. While students using the rat have not agreed on this effect of darkness, conclusive evidence that in the chick absence of ultraviolet light for 55 to 105 days results in marked thyroid hypertrophy and colloid loss has been provided by Turner and Benedict.³ The interesting possibility thus suggested that light may play some vital rôle in

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¹ Kenyon, A. T., *Am. J. Path.*, 1933, **9**, 347.

² Bergfeld, W., *Endokrinologie*, 1930, **6**, 269; *Strahlentherapie*, 1931, **30**, 245.

³ Turner, K. B., and Benedict, E. M., *J. Clin. Invest.*, 1932, **11**, 761.