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## Vascular Changes in Chronic Experimental Atelectasis.

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Microscopic pathology of massive atelectasis in man has received little consideration due to its associated low mortality. Reports on this subject have usually been made on autopsies where death was due to some other cause. Bergamini and Shepard[1] reported 2 cases of bilateral massive atelectasis causing sudden death. Microscopic sections of the collapsed lungs revealed cloudy swelling and indistinct outline of the cells; with a dilatation of the capillaries, arterioles and venules which were filled with blood, presenting in parts an angiomatous appearance. From experimental studies it has generally been contended that there appears to be an increased vascularity of the atelectatic tissue; that the capillaries become dilated and engorged with blood simulating a passive congestion of the lung lobe.

Recently we have studied microscopic sections of experimental atelectasis at various stages of duration. Massive atelectasis was produced in dogs by stenosis of the bronchi with a solution of silver nitrate.[2] The dogs were sacrificed at irregular intervals ranging from a few days to several months.

Atelectasis of only a few days' duration revealed complete collapse of the alveolar spaces and smaller bronchioles with some swelling of the septal cells whose outlines were indistinct. There was an apparent increase in the vascularity of the tissue due to the capillary spaces being brought much closer together. These vascular channels appeared to be engorged with blood, giving it the appearance of a passive congestion. Anthracosis was present, scattered pigment appearing throughout the tissue.

By the end of 4 months the alveolar walls were much less closely packed together and the intervening greatly dilated blood spaces more clearly outlined at this time. The blood spaces gradually become larger so that at the end of 9 and 11 months, they were found enormously dilated; some being 2 or 3 times as large as an ordinary alveolus. Accompanying this, the alveolar septae were found fewer in number and widely separated, in contrast to the closely packed

[1] Bergamini, H., and Shepard, L. A., *Ann. Surg.*, 1927, **86**, 35.

[2] Adams, W. E., and Livingstone, H. M., *Ann. Surg.*, in press.

alveoli of earlier stages. The tissue in some parts took on the appearance of a cavernous haemangioma. Red blood corpuscles found in these spaces appeared to be in good condition with blood pigment nowhere to be seen.

*Conclusions.* In the earlier stages of atelectasis the apparent increased vascularity was partially due to collapsing of the air spaces thus bringing the capillary sinuses in closer proximity. The changes exhibited in the more chronic stages were less easily explained. The process was a gradual one, marked changes being present as early as 4 months which were found to increase with the duration of the disease. That the blood in the dilated spaces was in an active state was strongly suggested by the normality of the red blood cells and the lack of blood pigment. The gradual increase in the size of the blood sinuses was in favor of their being dilated capillary spaces rather than alveoli through which blood was flowing. This last factor is under investigation.

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## Production of Intestinal Ulcers by Active Gastric Juice.*

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Gastric mucosa has[1] a strong natural immunity against corrosion by gastric juice, and wounds made artificially in it tend to heal readily. Mann,[2, 5] Dragstedt and Vaughan,[3] Morton,[4] and others have shown that when other tissues such as kidney, spleen, mucosas of various portions of the intestinal tract with an intact blood supply are implanted into defects made in the stomach wall, they tend to remain sound and healthy without erosion or ulceration, even though exposed to the normal stomach contents. Mann and Williamson,[6, 7] after draining the duodenal juices into the

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1 Ivy, *Arch. Int. Med.*, 1920, **25**, 6.

2 Mann, *J. Med. Res.*, 1917, **35**, 289.

3 Dragstedt and Vaughan, *Arch. Surg.*, 1924, **8**, 791.

4 Morton, C. B., 1927, **85**, 207.

5 de Takats and Mann, *Ann. Surg.*, 1927, **85**, 698.

6 Mann and Williamson, *Ann. Surg.*, 1923, **77**, 409.

7 Mann, *S. Clin. N. Amer.*, 1925, **5**, 753.