a terminal event, and sections of the lung, stained by the Gram-Weigert method, showed such a massing of the micrococcus lanceolatus about the pneumonic areas that we were forced to conclude that the condition was due to a secondary affection with this germ.

Sections of the Peyer's patches showed, however, bacilli of the morphology of typhoid germs massed in the deeper parts in the characteristic clumps. So that the lowest Peyer's patches may have been the point of origin in spite of the fact that they presented so little divergence from the normal. A further point in favour of this view was the fact that the mesenteric glands were uniformly swollen soft; some beginning to necrose, and others hæmorrhagic. How can we then explain these facts? Observation gives us some information upon this point.

In relapsed typhoid the ulcerative lesions affect those glands which escaped in the [illegible] attack, and are also never so intense as the primary ones. Trou[illegible] indeed goes so far as to say that in the relapse the intestinal lesions are not renewed. However this may be, it seems that the intestinal mucosa having once suffered the action of the typhoid virus can resist the force of a second attack, and thus a local immunity is acquired. So that in these relapses the systemic disease may proceed in the gravest manner and even lead to death, while the intestinal lesions are absent. In the case we record, considering that it was an ambulatory typhoid of six weeks' to two months' duration before admission, it is open to assume that the attack we observed was a reinfection, and that at some earlier period of the illness in the previous attack the Peyer's patches had acquired a local immunity. This primary attack need not necessarily have been a severe one. It is quite probable that in the so-called abortive typhoid the Peyer's patches never get beyond the stage of tumefaction, and yet they will have acquired an immunity for a short time. Cases where a second distinct attack follow shortly after convalescence upon a previous one would, at first sight, appear to negative this view, but we must remember that in experimental animals when this local immunity is attained, it only lasts for a short time, and we have no reason to think that it would be otherwise in the human being. This immunity affects the epithelial and lymphoid elements of the intestine and also the phagocytes, so that the bacilli are carried beyond the first barrier of defence, which remains intact, and are dealt with in the more remote parts of the organism.

We have an analogy to this in the well-known fact that the bacillus of tuberculosis sometimes passes through the intestinal mucosa without affecting the lymphoid elements, and may become localized in the mesenteric glands or in the peritoneum.