the inflammatory exudate. There was no evidence of abscess formation in any part of the tissue. On the surface of the intima, close to the aneurysm, there was some adherent blood-clot. The intima appeared slightly œdematous and thickened beneath this attached clot.

Bacteria were demonstrated in the tissues showing the acute inflammatory reaction. These were of the nature of Gram-positive cocci, appearing singly, sometimes in pairs, and rarely in short chains.

The above case is clearly one of an acute saccular aneurysm, occurring at an unusually early age (6 years). The clinical course of this disease was that of acute rheumatism in which the heart had suffered severe valvular disease. The inflammatory process in the aorta simulated in every respect the lesions as we had described them for acute rheumatic fever in a previous article. In this case, however, the destructive feature and the acuteness of the process in the arterial wall was much greater than any we had previously observed.

The development of aneurysm in acute rheumatic fever simulates the process in syphilis in as far as the inflammatory invasion begins in the adventitia and the vasa vasorum of the media. Syphilis was not to be thought of in this case, in that the arterial process was too rapid in its development. The inflammatory exudate was, in part, of a polymorpho-nuclear character, and there was no evidence of fibrosis in the vicinity of the damaged areas in the artery. Moreover, the secondary intimal reaction with its nodular thickenings as it occurs in syphilis was not present, and a type of streptococcus was demonstrated in the affected tissues.

When it is appreciated that acute rheumatic fever attacks the arterial system as an inflammatory process of varying degree, one may readily understand the finding of arterial damage of all grades of intensity. It is probably more common to meet with the more simple inflammatory processes than the more intense ones, which act so severely upon the artery as actually to weaken its walls. Nevertheless, it is more than probable that the subsequent fibrosis plays some part in bringing about the progressive changes in later life.

The repeated observation that systematic bacterial invasion is prone to attack the ascending limb of the aorta still remains without adequate explanation. It has repeatedly been impressed upon us that when the bacteria themselves are making inroads on the vascular tissues, they do this by attacking them by the way of the vasa vasorum. It is only when bacteria are carried in larger emboli or are caught in thrombotic masses that they attack an artery from the surface of its lumen. Moreover, it is much more common to observe this bacterial invasion in the ascending aorta, than it is in any other portion of the arterial system. In confirmation of this we need only refer to the processes induced by syphilis (Heller, 1903 [35]) (Chiari, 1903 [4]), influenza (Marmorstein, 1908 [20]), rheumatism (Feytaud, 1906 [9]), typhoid and streptococcal septicæmia.

It is possible that in a number of cases the ascending aorta may