with regard to his sudden death, however, was the presence of a deep, annular, punched out ulcer, situated at the height of the arch where it crosses and is intimately connected with the trachea. The ulcer was nearly circular, its edges somewhat undermined and irregularly thickened. The base of this ulcer was formed by the underlying and involved trachea. It showed a perforation, four millimetres in diameter, through which the fatal hæmorrhage had occurred (Fig. 2).

The aorta itself showed no special aneurysmal dilatation of its arch, and measured only 8.5 centimetres in its interior circumference. Of particular notice is the fact that the rest of the aorta was free from disease. The stomach was filled with semiclotted blood and the lungs were in a state of acute emphysema and filled with aspirated blood. The liver showed a typical large gumma and very deep scars, but smooth atrophy of the base of the tongue was missing.

Microscopical examinations of sections taken from the arch of the aorta showed a characteristic granulomatous aortitis. Throughout the thickened gelatinous adventitia were, partly diffuse, partly localized circumvascular lymphocytic infiltrations. The vasa vasorum themselves showed endarterial proliferation, frequently leading to obliteration of their lumen (Fig. 3). In some places, circumscribed granulomatous formations, made up of lymphocytic and immature fibroblastic and some Langhans giant cells, were plain. (Gummata; Fig. 4). There was an irregular tendency to cell fusion and necrosis, although definite caseation did not occur. This granulomatous infiltration freely invaded the media, leading, particularly in its lower and middle portions, to circumvascular inflammatory foci with patchy tearing and disorganization of its elastic and muscle tissue, which was replaced by cicatrices. On the other hand, the intima, which was well separated and distinct from the media, showed moderate fibrous and delicate fibrillar elastic thickening, and in places degenerating fibres (Figs. 5 and 6).

The case, therefore, may be regarded as a typical specific granulomatous aortitis, which commences in the form of circumvascular infiltrations of the adventitia, becoming more diffuse and extending into and destroying the media, leads to the formation of cicatrices. The intima is not attacked. For, as I will discuss in a moment, the thickening and hyperplasia of the latter cannot be brought in direct causal relation to the specific aortitis. Over the trachea the process had produced extensive loss of