elastic fibres at this time, though themselves not visibly altered, no longer take on the wavy contour which is characteristic of them in a relaxed vessel. It would seem from this that the apparent elasticity, as shown by their undulations, is not an inherent quality, but is due to the contraction of the muscle fibres surrounding them—or, otherwise, that when the artery is in a condition of tonus, its contracted state is due mainly, if not entirely, to the muscle fibres; when dilated it is probable that the elasticity of the elastic fibres comes into play.

A proliferation of the intimal tissue in these cases is to be regarded as secondary to degenerative processes in the media. The proliferation is either of the character of a hypertrophy of the musculo-elastic layer or of the subendothelial tissue. Whether this subendothlial tissue had its origin in connective tissue or endothelial cells we cannot discuss here.

INFECTIVE ARTERIO-SCLEROSIS.

I have also undertaken the production of experimental arterial lesions with infective agents. For this purpose B. typhosus and streptococcus were used in separate experiments, while again in others diphtheria toxins were inoculated. Each of these agents was inoculated intravenously into rabbits.

The results obtained with B. *typhosus* and the streptococcus were of the same order. The first part of the pulmonary artery and the ascending limb of the aorta showed warty thickenings of the intima. There were no anerysmal sacs nor any sign of a calcareous degeneration of the media. Microscopically there was a fatty degeneration of the subendothelial tissue, while there was, however, much connective tissue advancing into the degenerated area. A small-celled infiltration was wanting, as was also any sign of calcification. At the areas of thickening of the intima it was found that the internal elastic lamina had split into several parallel layers, which were stretched between the proliferating cells. The area affected included the intima and the inner layer of the media. Thus we find that these infective lesions (of *B. typhosus* and streptococcus) differ entirely from those produced in our adrenalin series. What we may term the adrenalin group are agents producing destruction of tissue leading to a calcification with little or no local repair to make up for the lost tissue, while the mild infections lead to a slight degeneration of the vessels coats, though the process is followed step by step by the process of repair, and instead of getting a thinning of the vessel wall there is an actual thickening. It becomes self-evident that with the absence of extensive destruction of the muscle fibres in the media no aneursyms were formed.