experiments the dose was gradually increased. The early injections sometimes resulted in death from acute dilatation of the heart and pulmonary edema. In those animals which survived the early injections by the development of a certain amount of immunity, the doses were increased gradually, and they were killed after periods varying from a few days to eight weeks and a half. Ten animals were chloroformed, and the gross lesions were found in the aortas of six. The earliest change in the vessel wall which is apparent to the naked eye consists of a faint longitudinal or irregular grayish streaking of the intima without thickening. Later, irregular isolated or confluent areas of a pearly gray color are found, almost all of which are calcified. Still later, the aorta is more or less distorted, rigid, and nonelastic; but distinct ulceration with atheroma is not readily demonstrable. Diffuse calcification is not infrequent and small aneurysmal dilatations may be present. Microscopically, primary degenerative lesions are well advanced by the end of the third week, and one or two weeks later advanced calcification may be demonstrated. The destruction of the muscle fibres is the older and probably the primary lesion. In the late lesions, when small aneurysms are found, the elastica is so completely destroyed that only an indistinct mass of fractured granular and fused fibres remains. In two animals which died on the fifth and the ninth days of the experiments, respectively, small longitudinal or occasional irregular, finely granular foci of degeneration were seen in the media of the aorta. In these areas there were no nuclei visible, and the muscle fibres were transformed into a finely granular, almost hyaline material.

There appears to be one discrepancy in the conclusions of the authors. They distinctly state that the lesion "of the muscle fibre is the older, and therefore in all probability, the primary lesion." Later they point out "the strong support afforded Thoma's views, that the primary lesion of arteriosclerosis occurs in the media and is essentially the result of changes in the elastica, and that the alterations in the intima constitute a repair process, the object of which is to compensate for the weakened media and the widened lumen." They probably mean that physiologial disturbances of the elastica result in anatomical changes in the muscle, but the inference is not clear. The experimenters have not succeeded in reproducing arteriosclerosis as seen in man, and this they freely admit. It must be allowed, however, that the resemblance is very close.—*New York Med. Jour.*