beadings were distinctly palpable, while the vessels in general were firmer than normal. The amount of change in these arteries diminished after the bifurcation of the common carotids, though it was still apparent in some of the smaller branches.

Thorax and Abdomen.—The lungs were healthy and without change. The heart showed an enlargement of, at least, one and one-half times its normal size. The ascending aorta had its walls thickened, and was larger than normal. This increased size was apparent as far as the middle of the arch, or just beyond the opening of the left brachial. The wall felt firm and nodular, and did not collapse when its contents were removed. Opposite the 6th rib the vessel again dilated to twice its size, forming a fusiform aneurysm as far as the diaphragm. This aneurysmal dilatation had firm and brittle walls, in which concentric rings could be distinguished passing about the vessel. Below the diaphragm the aorta again became smaller, but showed thickening of its coat, which was visible as far as the right renal artery. The beginning of the cœliac axis was also sclerosed, though no changes were noted in the branches of this vessel. The renal arteries were normal in appearance, and below them the aorta, too, was without change. There was no change to be noted in the iliac arteries, nor the vessels of the legs, nor did the viscera of the abdomen exhibit any microscopical lesions.

We have, therefore, produced macroscopical changes in the aorta and its branches above the renal vessels. In these changes the aorta is chiefly involved, while the carotids and the vessels of the neck are also sclerosed. Consequent upon the weakening of the aortic wall by sclerosis, a fusiform aneurysm developed in the thoracic aorta.

Microscopical.

Ascending Aorta.—The aortic wall was hypertrophied, the thickening occurring in the intima and possibly in the media. The media, where it was apparently thickened, was normal in structure and showed the alternating layers of elastic fibres and muscle tissue. The intima, where thickened, showed the hypertrophy to be in the muscle elements (of the musculo-elastic layer). There was no connective tissue proliferation to be found. With the intimal thickening there was everywhere a process of degeneration accompanying it. This degeneration in the mildest form occurred close to the internal elastic lamina, and, in the more advanced types, extended closer to the endothelial surface. The muscle cells themselves were degenerating and disintegrating, leaving areas of non-cellular debris. These areas showed many spicules