intima showed little or no change over these areas. Macroscopically, and in the relaxed condition, these vessels showed small pouchings and transverse grooves on the inner surface. There were no nodular thickenings of the intima to be seen. When, however, these vessels were subjected to a hydrostatic pressure equal to one hundred and sixty millimeters of mercury and allowed to freeze, the longitudinal section of the artery showed quite a smooth intimal surface. The ridges and pouchings in the vessel wall had been obliterated (Fig. 3). Obviously here the more normal part of the vessel wall had, under pressure, dilated equal to the pouching present in the diseased areas. This is exactly what we should expect in comparing the results with the histological preparations. It has been repeatedly commented upon by various authors that the sclerosed and calcified areas of the media in Moenckeberg's disease diminish the elasticity of the media and fix the muscular tissues in a rigid state. In life this sclerosis takes place while the vessel is under pressure and hence the tissues become fixed and rigid while fully dilated. Under ordinary conditions of life, these sclerosed areas need not be more widely dilated than the adjoining more healthy arterial wall, as is shown in our experiments. Immediately on relaxing the blood pressure at death, the active and more healthy portions of the vessel wall contract, leaving the sclerosed portions as transverse areas to form the pouchings, as is seen at autopsy.

Here, then, we have a process exactly contrary to Thoma's theory. We find that in the areas where the media is diseased there is a pouching of the wall when the vessel is relaxed, and that these areas of medial disease are not covered by a thickened intima of compensatory hyperplasia.

But besides this macroscopic demonstration, we possess clearer evidence that the lesions of the intima are to a great extent independent of medial changes. Dividing the diseases occurring in the intima of arteries into the productive and the degenerative types, we shall, in this paper, confine ourselves to those lesions which lead to the development of new tissues. Nor is it within the province of this discussion to consider the final outcome of intimal hyperplasia, whether this leads to a process of degeneration or continues in the production of new tissue.