primarily attacked, and according to the intensity of the intoxication of these drugs, the cells either undergo a fatty degeneration or complete destruction. Along with the death of the muscle cells, the elastic fibers in the media are also affected and, like the former, they either become fatty, or with more severe intoxication, undergo necrosis. However, in each instance the muscle fibers are primarily affected. These lesions, it is obvious, have destroyed the most important tissues in the artery, and have weakened the vessel wall very considerably. Aneurisms are commonly to be found at the sites of medial change while little if any intimal compensation occurs. Thoma's dictum, therefore, that intimal compensatory hypertrophy follows medial weakening is not universally true. This type of arterial disease, in which the media is first destroyed, is spoken of as "Moenckeberg's arteriosclerosis."

Not alone was the medial degeneration with calcification produced by means of drugs, but I have also obtained it by the inoculation of the diphtheria toxin. This is important in demonstrating that the effects of diphtheria are not confined to nervous tissue and heart muscle, but that the muscle elements of the vascular system are also attacked. It may be that the intoxication in cases of diphtheria is an important agent in bringing about Moenckeberg's arteriosclerosis, such as is seen in the radials and other peripheral vessels.

This latter form of medial degeneration with aneurismal pouchings has also its analogy in the peripheral arteriosclerosis in man. The greater majority of the cases of arteriosclerosis which are diagnosed from the condition of the radial arteries are of this type. The beadings so often noted in the radials of old people are the small pouchings in the vessel wall that have become calcified.