sclerosis particular tissue elements are picked out, while other tissues are unaffected, or that when muscle fibres are degenerating in the media the connective tissue elements of the intima are proliferating. Hence we may have two or more such processes inextricably mixed in a progressive disease of the arterial walls.

Of the more common forms of sclerosis of the arteries I would point out that the hard radial vessels by which the clinician makes his diagnosis of arterio-sclerosis is a widely different disease from that recognized by the pathologist at *post-mortem* examination of the aorta. The sclerosed radial vessels represent a disease which is peculiar to the media; it has its origin in the muscle cells of the middle coat, and the middle coat alone is damaged. The intima and adventitia are not essentially involved in the process, though occasionally a secondary intimal thickening accompanies the medial degeneration. The main changes in the media are a fatty degeneration of the muscle and later of the elastic fibres, both of which become calcified. It is through these calcareous plaques in the media that the beaded character is given to the radials. At these sites of medial degeneration and calcification the vessel wall is perceptibly thinned, so that many small pouchings result. These pouchings, though small, are true aneurysms distributed irregularly in the vessel wall, and when held to the light are seen to be thin and quite transparent. This type of disease, which is most frequent in the vessels of the extremities, I shall later speak of as the Moenckeberg type of arterio-sclerosis, and I shall point out how closely some of the experimental lesions resemble it.

On the other hand, the nodular aorta, which we so frequently meet with at autopsy, is the result of repeated insults telling upon the intima alone. The thickenings of the intima may again be entirely proliferative, and in this case represent a chronic inflammatory production. This I acknowledge is not the view held by all; those who still uphold Thoma's conception of the arterio-sclerotic process see also in the typical nodose sclerotic aorta a primary giving way of the media, and regard the intimal overgrowth not so much as an inflammatory as a compensatory process. Whichever view be accepted, or be correct, or whether, as would seem to be truly the case, we encounter both conditions, it is still an open question whether the newly-developed cells in the intima are of endothelial or of connective tissue origin; it may be again that both tissues take part in the overgrowth. At all events, few or many layers of cells, which are very like endothelial cells, are produced immediately beneath the endothelium, and it was the character of these cells which led Virchow to speak of "endarteritis chronica deformans."