Are we, then, to exclude the clinician from diagnosing arterio-sclerosis? The answer can but be, No! And this for the adequate reason that such is not the sense in which Lobstein applied the term arterio-sclerosis in 1833. Let us preserve the broader meaning, and regard all scleroses or hardenings of the arteries as included under this general term, recognizing, if need be, distinct varieties.

Thus I would point out that arterio-sclerosis is not a simple disease. Although, in some instances, a single coat of a vessel is found affected by a fibrous or other allied change, in others several tunics of the same artery are involved. Again, we may find that in a certain form of 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 find that 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