lesion. It is true that vessels with medial calcification not infrequently show a condition of endarteritis, but this endarteritis is found to bear no direct or secondary relation to the medial disease, save that both may be produced by the same injurious agent.

What is true of medial degeneration and dilatations of the peripheral vessels also holds true of the aorta. In the diffuse though not severe degenerations of the media in the aorta in old age, there is sufficient reason to believe that the arterial wall is materially weakened and that the vessel may be locally or diffusely stretched. Nevertheless, from this medial process alone we cannot predict what changes have occurred in the intima. Occasionally in such vessels the intima is without endarterial change.

Saccular aneurysms of the aorta not uncommonly show some thickening of the intima, but, as has been shown by others (Osler, Chiari), these aneurysms are most frequently the result of a syphilitic mesaortitis, and the intimal overgrowth is associated with the syphilitic process before an actual weakness or stretching of the vessel wall has shown itself. The mesaortitis is essentially an inflammatory process to which the neighboring intima reacts.

From our observations on the medial lesions occurring in the human blood vessels, we are unable to make any general statements associating these with the thickening of the intima, nor does there appear to be any "compensatory" reaction on the part of the intima due to medial weakening.

The experimental lesions in the arteries, too, have been of considerable value in the study of intimal hyperplasias. Some have contended that, as spontaneous disease may occur in the arteries of the experimental animals, or that the lesions experimentally produced were not comparable to the disease in man, no conclusions could be drawn from the work in experimental arteriosclerosis. Without discussing the right of this opposition, we need only point out that the study of the arterial lesions, as they occur in animals, has assisted to a great extent our knowledge concerning progressive and retrogressive changes of the various coats of arteries in general.

Various investigators studied the arterial lesions following mechanical injury (D'Anna, Malkoff, Israel). In all of these, varying grades of inflammatory reaction were obtained. The inflammation