deposited in streaks either in the subendothelial layer or in the deeper musculo-elastic layer. Or again, both layers may be affected at the same time and the lesions coalesce, so that a condition of "fatty degeneration" extends from the endothelial layer to the innermost lamella of the media. What process governs the laying down of fat in this or that layer of tissue, or why certain areas in the arteries are the sites of predilection, has not been determined. The main interest that attaches itself to the study is the relation of the fatty streaks of the aorta to the more severe and extensive atheromatous processes.

We have repeatedly observed that as fat accumulated in the subendothelial or musculo-elastic layer, the superficial portion of the intima became thicker and formed a small nodule of flat and parallel cells over it. This was particularly evident in those cases where the process had developed to the degree in which the fat-laden cells were destroyed and liberated their contents into the interstitial spaces. The degree of proliferation varied greatly, but the same compact mass of an almost hyaline-looking tissue was always produced. We were not able to determine whether these new cells were of endothelial origin or were derived from the layer of subendothelial connective tissue immediately beneath this.

As we study these lesions in succession we come upon some in which we can no longer distinguish the sequence of events. The subendothelial thickening forms a definite nodule of long and narrow spindle cells with much hyaline material between them, in which delicate elastic fibrils are found. Below this proliferative nodule is an isolated or diffuse fatty degeneration of the musculo-elastic layer or of the hyperplastic layer. This process, even in the presence of fatty streaks of the intima, makes it difficult to make a positive statement as to whether or no the degenerative condition has been primary. We believe that some, at least, of these lesions have their origin in a primary degeneration of the deeper portions of the intima, with a secondary proliferative reaction close to the endothelial lining.

Our findings are in accord with those of Jores (1903 [4]), Askanazy (1907 [1]), Benda (1909 [2]), and others.

These observers believed that the superficial fatty streaks of the intima were closely associated with advanced arterio-sclerosis. Jores was the first to show that the small white or yellow streaks of thickening found in young individuals were the beginning of arterio-sclerosis of the aorta. He found much of the fatty change in the musculo-elastic layer, where the elastic fibres in particular were altered in the degenerative process. The thickening of the layer on the surface was a condition arising secondarily to the deeper degeneration. Askanazy, while he admitted that the fatty streaks might progress to definite atheromatous processes, believed that many of them disappeared without advancing into the more serious lesion. On the other hand, Thorhorst (1904 [7]) and Hallenberger did not consider the fatty streaks of the intima as important factors in the production of arterio-sclerosis.