The experimental endarteritis chronica deformans has histological characters quite similar to that in the human arteries. The lesion is composed of a heaping up, layer by layer, of the endothelial cells, while the connective tissue underneath the endothelium is also undergoing a proliferation. The result is that a white, pearly plaque is produced, under which degenerative changes of a fatty character may develop in the deeper part of the intima.

Whether these pearly plaques in the human or experimental arteriosclerosis are composed wholly of endothelial cells, or of connective tissue, or both, is quite immaterial at present. The result is the same, the production of a nodular hyaline mass of tissue on the surface of the intima. Such a thickening of the intima has a disastrous effect on the tissue just underneath it. The intima and the inner third of the media derive their nourishment from the lumen of the vessel, and the production of a firm mass of tissue at one point in the intima cuts off the supply of nourishment to the cells underneath it. From this there follows the fatty change in the deep layers of the intima and the inner portion of the media, a condition which is so often seen in the aorta.

The experimental lesions which have of late received the most attention are of a different nature. I have just pointed out that the endarteritis chronica deformans is essentially of a proliferative character, and that degenerative processes, if they occur at all, are secondary to this. We have, however, on the other hand, been able to produce pathological conditions which from the first are degenerative in nature. By the use of adrenalin, digitalin, nicotine and barium chloride, it has been shown that *the muscle cells in the middle zone of the media are*