isolated to the thoracic portion of the vessel and did not advance beyond the diaphragm.

Fischer's experiments, too, of producing arterial lesions by the intravenous inoculation of digitalin, were also repeated, and I agree with his findings that the arterial lesions isolated in the aorta are similar to the milder adrenalin destructions.

It was further found that, if the pressure-raising effect of adrenalin be abolished by the use of nitroglycerin, although the arterial lesions were not as extensive as when adrenalin alone was used, nevertheless, tissue degeneration in no way differing from that produced by adrenalin did still occur in the vessel walls.

In such cases where the arterial lesions were just beginning there was change to be noted in the vessels macroscopically. I might point out, too, that in none of the vessels that I have obtained from animals treated with adrenalin was I ever able to make out any naked eye changes in the intima. This coat was at all times stretched smoothly over the damaged media. The earliest damage was always found in the muscle cells of the middle zone of the media. Here patches of homogeneous tissue were met with, where the muscle nuclei were lost, but where the elastic fibres passed through these areas unaffected. With the loss of the muscle cells the parallel elastic fibres were crowded closely together by the blood pressure within the vessel.. This crowding of the elastic fibres from within outwards naturally led to a small dimple at this point and this was the beginning of a saccular aneurysm.

The loss of the muscle cells takes place by a form of necrosis, as was pointed out by Erb and Fischer. The elastic fibres later become affected, losing their elasticity and contractile power. This degeneration of both muscle and elactic fibres occurs through a process of fatty change, which is in some cases difficult of demonstration, but which is, however, readily brought out out in those cases where the metamorphosis is slower. With the high calcium content of the rabbit's blood these areas of fatty degeneration in the media of the aorta and other vessels are converted into calcified plaques by the process described by me elsewhere. Microscopically, no connexion could be linked between the positions of the vasa vasorum and the arterial degenerations, and a true mesarteritis, as noted by Fischer, was not met with.

In no instance have I found a primary change occurring in the intima after any of the above treatments, though in one or two specimens I did note the slight thickening of the intima at the margin of the aneurysm.

It is to be noted, too, that with the abolition of the physiological effects of adrenalin, the arteries are still affected, though more slowly