overgrowth not so much as an inflammatory as a compensatory process. Whichever view be accepted, or be correct, or whether, as would seem to be truly the case, we encounter both conditions. it is still an open question whether the newly-developed cells in the intima are of endothelial or of connective tissue origin; it may be again that both tissues take part in the overgrowth. At all events, few or many layers of cells, which are very like endothelial cells, are produced immediately beneath the endothelium, and it was the character of these cells which led Virchow to name it "endarteritis chronica deformans."

When a similar intimal thickening, by the proliferation of connective tissue or endothelial cells, occurs in the smaller arteries, so that the lumen of the vessel becomes distorted, or even wholly obliterated, the condition is spoken of as "endarteritis deformans sive obliterans." I may, however, mention that seldom if ever is a vessel occluded by the overgrowth of its intima alone. The usual result is that after a vessel has been partly obstructed by the thickened intima, complete blockage is brought about by thrombosis.

Now although we have ample opportunity to study the damage that has been done by the various noxae to the human arterial system, we are as yet largely without the means of recognizing which lesion has been produced by a particular irritant. It thus becomes evident that the histological changes in the arteries must be investigated by experimental means, for it is only in this way that the changes in the arteries produced by insults of different kinds can be followed step by step, and that a decision can be reached regarding the influence of the various injuries.

It is the common fault of experimenters that, having been able to reproduce a disease in whole or in part by experimental means, the conclusion is drawn that all the features of this disease are due to this one cause. To avoid this common mistake we must advance very cautiously towards our conclusions.

The Earlier Experiments

In the earlier experimental attempts undertaken to produce arterio-sclerosis and aneurysms mechanical means were employed. Thus, Malakoff, and also Fabris, injured the vessel wall directly, either by forcibly pinching it through the skin or by laying it bare and crushing it, or applying corrosive substances to its outer walls. That damages of all kinds were obtained in this manner we can readily understand, but that neither true aneurysms nor arterio-sclerosis resulted is just as clear. Thromboses and inflammation of the arteries were the most frequent results of these violent measures, but these studies have thrown little light on the process of arterio-sclerosis. Malakoff, however, made another interesting experiment, in which he laid the end of the carotid artery bare, and, ligating the vessel about an inch or so away, he