dary line between the vascular and non-vascular areas of the arteries. We are forced, I hold, to regard the intima as nourished from the blood circulating within the arteries.

The diseases to which the arterial walls are subject are closely comparable with those of the heart. There can, for example, be undoubted inflammation; we may even have collections of pus cells separating the intima from the media, although this is very rare and is always secondary to a purulent mesarteritis, the pus cells wandering into the intima from the vessels of the media. Even in cases of septic embolism or thrombosis, necrosis is the first noticeable change in the intima, and the invasion of leucocytes appears to be associated with the later inflammation of the media and adventitia. Rather more frequent is an acute productive inflammation, seen especially in the first portion of the aorta. This appears to be secondary to similar verrucose, subacute, and ulcerous inflammation of the aortic valvules. It is characterized by the development of almost papillomatous or warty processes projecting into the lumen of the aorta, and these are richly cellular and also vascularized from the vasa vasorum. They are often covered by a layer of coagulum.

But these obviously inflammatory conditions are relatively rare. The most common form of arterial disease in the larger arteries is that termed by Virchow endarteritis chronica nodosa sive deformans, the arterio-sclerosis of Lobstein, or atheroma. I need not here enter into statistics concerning its frequency, or take up your time by details concerning the forms that it may assume. I will accept Dr. Councilman's classification,[1] simply modifying his terminology to indicate my doubts as to the endarteritic or inflammatory na-

[1] Councilman: "On the Relations between Arterial Disease and Tissue Changes." Trans. Association American Physicians, vi., 1891, p. 179.