They point out that minute thickenings may be observed in the intima without fatty change.

Thorel (1910[8]) points out that the small fatty areas produce a change in the character of the inner layer of the artery which alters its elasticity and which must be recognised as a definite manifestation of a progressive tissue degeneration. In recognition of this early and progressive change, which combined with it some characters of sclerosis, Marchand (1904[5]) applied the name of athero-sclerosis.

The frequency of these fatty lesions in the vessels of young individuals with acute diseases, and their absence, microscopically, after a year or two, demands some explanation, if we consider they have any association with true arterio-sclerosis later in life. There can be no doubt but that many of these superficial lesions disappear almost entirely and leave the artery in an elastic condition equal to normal. We do believe, however, that although the débris and fat of the degenerative process may in some areas be fully absorbed, the proliferated cells of the subendothelial layer are permanent, and although they do not form isolated nodules assist in producing a more or less diffuse thickening of the intima. On the other hand, the more severe lesions, which have caused the destruction of cells in the musculo-elastic layer and also in the hyperplastic layer of the intima, have a more extensive reaction in the subendothelial layer in producing localised nodular thickenings which are permanent and indistinguishable from endarteritis nodosa. In them, however, the products of degeneration are not readily absorbed, so that these early thickenings of "endarteritis" always have fat and débris in their deepest portion. Such areas have all the characters of chronic endarteritis with early atheroma, and there is no reason to believe that the process cannot go on to atheromatous ulceration.

It is evident, from our finding, that it would be most difficult to determine the process of development of an atheromatous area after it was fully established. From our observations and those of others it is seen that an atheromatous process may be a degenerative lesion from the very beginning, with more or less proliferative reaction above it. On the other hand, Jores and others have determined that the primary intimal hyperplasia may be followed by fatty and other degenerations. We are not familiar with any definite points by which a well-developed atheromatous process may be recognised as coming from one or other process.

In general, the situation of the fatty streaks about the intercostal vessels is the same as the nodular endarteritis. Each is commonly found distributed on the posterior surface of the descending aorta. The thickening of the actual mouths of the intercostal vessels is a process which not infrequently has the condition of fatty streaks of the intima as a forerunner. In such cases the nodular endarteritis is a secondary result of an irritative process.

It is evident from these observations that there is a direct relation