

the vessel walls were due to a slowing of the blood current. These pathologists, Rindfleisch, Stronganow, Koester, and Talma, were unable to trace any connection between the vascular media and the non-vascular intima, and, therefore, thought that the latter was seriously affected by a slow blood stream. This led these men to advocate anew Resse's theory of interrupted nutrition.

Durante, Trompeter, and Krafft added to the discussion by showing that the media is always involved about the same time as the intima, and that the vasa vasorum are the real agents in maintaining the nutrition of the vessel walls. By the experiments made by Durante, it was shown that stoppage of the flow in the lumen of the vessel did not affect its nutrition; but that a similar condition in the vasa vasorum at once caused degeneration.

These various theories bring the subject to the position taken by Thoma. His theory has been well named the compensatory process. He divides arteriosclerosis into primary and secondary. In the primary there is a yielding of the vessel from loss of elasticity. The vessel is widened and the blood stream slowed. Connective tissue is formed in the deeper layers of the intima to restore the original relations. As age advances this thickening goes on regularly in keeping with the slowing of the blood current. In this way an adjustment is effected between the heart, the vessels, and the blood. In the secondary form of arteriosclerosis the change has its origin around the vasa vasorum, or in the small arteries. These changes in the small vessels may be nodular and local, or diffuse. When the vessels yield at points they may bend at these points, and in this way the tortuosity noticed in arteriosclerosis is explained.

This very ingenious theory of Thoma, which rests upon an unproved hypothesis of slowed blood stream and a lost vessel elasticity, has been keenly contested by Beneke, Marchand, Fuchs, Huchard, Gibson, Councilman, and others. They think it is pushing the mechanical theory too far, and are strongly inclined to look for the causes among more general and constitutional states and tendencies. These later teachers cannot agree with Thoma that when the blood stream becomes slowed down either by dilatation of and lost elasticity in the vessels, or by resistance to the onward flow of the blood from any change in the tissues, there is established a compensatory endarteritis.

These brief remarks on the history of the subject bring us to what may be called the present and more rational view of the etiology of arteriosclerosis. And I think we may admit that the following are the factors that stand in the relationship of cause and effect:

1. Long continued straining of the coats of the vessels affects nutrition and elasticity. The periods of rest are shortened and those of strain