Thoma's localization of the connective tissue in the thoracic aorta below the ductus arteriosus, and in the abdominal aorta, would, according to his dictum, place the frequency of arteriosclerosis in this region away above that in the ascending limb and arch of the aorta. Such a relationship, however, does not exist. One of the commonest sites of change of the intima in the aorta is in the convexity of the arch immediately opposite the origin of the vessels of the neck, and above the entrance of the ductus arteriosus. In this region, the aortic wall is thrown into folds which fibrose and frequently undergo fatty and calcareous degeneration.

There is occasionally a small sclerotic patch at the site of the opening of the ductus arteriosus into the aorta. This plaque represents the remains of the fibrosis which closed off the ductus arteriosus, and is only visible as a pucker in the wall, 2.5 mm. in diameter. I have never been able to trace any spreading fibrosis from this centre, and I do not believe that this plaque plays any role in the production of arteriosclerotic changes, or leads to any weakening of the vessel wall.

The musculo-elastic layer of the intima is a rather uncertain layer as regards the aorta in general. Its presence is fairly uniform at the mouths of the arteries where it spreads over the inner wall of the aorta as a flat disk. The peculiarity, however, exists that the muscle fibers in it are, to a great extent, derived from those in the media of the smaller artery. This layer spreads out for some distance in the deep layer of the intima so that a plaque of tissue exists here, which is not found in other sites. As this layer is common to the insertions of all the larger vessels, no significance can be attached to its presence at the opening of the ductus.

I would, therefore, be unwilling to grant that the closing of the ductus arteriosus throws any light upon the origin of arteriosclerosis that from my observations the obliteration of the ductus arteriosus results as a fibrosis subsequent to a contraction of the vessel walls. This contraction is dependent on two main factors: (1) An exceptionally muscular vessel wall acting on (2) a diminished blood pressure.