in connexion with the ordinary type of arterio-sclerosis, it has for long seemed to me that we encounter some of the most striking instances of adaptation. The studies made by me some years ago, to which I have referred in the Middleton-Goldsmith Lectures of 1896 upon Fibrosis and Inflammation, led me to support and confirm Thoma's contention that in the commonest type of aortic arterio-sclerosis there is (as in the later experimental researches of Josué, Pearce, Klotz, and so many other workers with adrenalin and allied drugs) a primary degeneration and giving way of the media. To that view, despite Jore's important studies, I still incline, and would harmonize the divergent opinions by laying down that while the primary lesion manifests itself in the media, the primary reaction to that lesion occurs in the intima. It is this reaction—this overgrowth of the musculo-elastic layer—that Jores has so serviceably brought to our notice, an overgrowth which may or may not be accompanied by coincident hyperplasia of the subendothlial connective tissue of the intima. No one nowadays regards this intimal hyperplasia as strictly inflammatory, as a direct reaction to injury; it has none of the characteristics of inflammatory new growth; there is no primary new formation of vessels, no small-celled infiltration; it is a hyperplasia pure and simple, occurring not in the tissue primarily injured, the media, but in another tissue, the intima. But it is secondary to the giving way of the media, and to that extent reactive, and can, I think, only be regarded as adaptive, tending towards a restoration of the original lumen of the vessel and a strengthening of the wall at the region of giving way. I have elsewhere explained this overgrowth as an effect of strain within certain limits. Just as increased strain, up to a certain grade, favours muscular hyperplasia, and as exercise, by causing increased pull upon the tendinous insertions of the muscles is followed by overgrowth of the bony ridges of attachment of those tendons, so I hold that when the media gives way locally to a slight extent the overlying intimal tissue becomes stretched and strained, and as a result exhibits hyperplasia. Too severe and sudden an expansion of the intima arrests any such tendency to overgrowth. Thus, it is significant that in aneurysms intimal thickening is lacking. There is a similar lack of overgrowth in these aortas from rabbits treated with adrenalin and barium chloride, with their aneurysmal pouchings, as, again, in the pouchings of human iliac and femoral arteries of the Moenckeberg type. I shall not be surprised, however, if further observations demonstrate that the common or Jore's arterio-sclerosis and the Moenckeberg or medial degenerative type are the effects of common noxæ acting with