of intermediate size which passed to the limbs and to the main viscera. It is true that in the majority of cases these moderate sized arteries of the muscular type show no evidence of inflammatory invasion. Nevertheless, as we then indicated, an irregular distribution of the inflammatory reaction may be observed in some of the arterioles when the larger visceral arteries are not involved.

In the cases which we have examined, the simultaneous occurrence of lesions in the myocardium and the arteries has been very constant. The intensity of the reaction in each or both has been varied; at times that in the heart being greater and out of proportion to that in the arteries, at other times again, the reverse was observed. Moreover, we have been able to follow the processes during the various stages of development. From the acute non-suppurative variety with extensive perivascular infiltration of the small arterioles all gradations of chronicity with progressive fibrosis have been found. The amount of fibrosis occurring in the vicinity of the arterioles was dependent upon the intensity of the reaction, and the extent to which the neighboring parenchymatous tissue was affected. From the minute, microscopic fibrous tissue masses to the larger fibrous streaks, such as are observed in the heart and large vessels, all degrees and stages were demonstrated.

It is in association with these particular arterial lesions that hypertrophy of the heart is prone to develop. This hypertrophy, however, does not begin to show itself until the reparative processes about the minute vascular channels become evident. In many cases the heart suffers some dilatation of its cavities during the acute stages, but though the heart at this time is receiving the stimulus for growth through stretching, it is unable to compensate so early by hypertrophy on account of the systemic illness, which offers the explanation in an inadequate nutrition. Hypertrophy does not begin until recovery from the effects of the immediate acute involvement has passed over. Repair of the inflammatory focus does not begin until, in part, at least, the infection is overcome. From this time on not alone is there a repair of the lesion induced during the inflammatory reaction, but also opportunity is given for the compensation of the weakened myocardium sustained in muscular degeneration.

When, now, we suggest a type of kidney lesion ending in chronic interstitial nephritis as commonly associated with this combination of acute and subacute myocarditis and arteritis, we will receive considerable opposition from clinical observers. The constancy of association of myocarditis and mesarterial diseases has