bolism, it was held by some that both the heart and arterial lesions were the result of a chemical irritation. The retained substances, it was claimed, had a direct effect upon the musculature of the heart as well as upon the arteries. It was also suggested that besides this the direct toxic effect of the retained excretions caused a persistent high blood pressure induced by arterial spasm. More recently it has been suggested that chronic kidney disease is accomplished by an abnormal function of the adrenal glands, associated with a greater production of adrenalin. This, it is claimed, leads to a tonic spasm, or contraction of the arterial walls, materially raising the blood pressure to which the cardiac hypertrophy is a response. Thus a variety of factors have been suggested as initiating the hypertrophy of the heart secondary to other diseases.

Even under the circumstances where cardiac hypertrophy is recognized clinically, the heart condition may not have reached the final stage of the process. Insufficiency of the myocardium may yet develop, particularly in the presence of a subsequent disease, as myocarditis, or with a progressive sclerosis of the coronary arteries. These changes, however, are rather to be viewed as complications and sequelæ which do not assist in clearing up the nature and process of the primary disease causing the hypertrophy.

Undoubtedly, when a definite sclerosis of the large and small arteries has occurred, the increased resistance rapidly leads to an alteration in the circulation. The maintainance of an equal supply of blood in the peripheral arterioles demands greater activities on the part of the heart, and whether the heart may properly compensate for this increased demand depends, in the individual case, upon the reserve activities of the musculature. An adequate nutrition, using the term in its broadest sense, will permit the myocardium to compensate by hypertrophy. It may be, as suggested by some, that prior to the arteriosclerosis, the heart may show no evidence of enlargement, but it would be going too far to say that in the absence of recognizable hypertrophic changes, the heart had not previously suffered myocardial lesions. It may be, as some have indicated (Hasenfeld, Romberg), that though the heart be damaged by degeneration, the hypertrophy does not arise until the musculature is given the stimulus for growth by suffering undue stretching.

Hirsch examined a series of cases with cardiac hypertrophy and found that where this hypertrophy was associated with arteriosclerosis, the left ventricle was mainly or alone involved. He also observed that when diseases of the lung and pleura had an effect