Hypertension, hypertrophy and dilatation

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WHEN a patient who has suffered for any length of time from systemic hypertension dies, one usually finds severe hypertrophy, particularly of the left ventricle, best seen on cross-section (Fig. 1). This shows that the hypertrophy is uniform and measurements of the anterior and posterior walls and interventricular septum will be of approximately the same thickness. This is the so-called concentric hypertrophy. Occasionally slight dilatation of the left ventricular cavity can be found (Fig. 2). Injection of the coronary arterial tree usually shows narrowing of the lumen, which may be severe, due to atherosclerosis. The extent of luminal narrowing can be studied post-mortem by injecting radiopaque material into the coronary arteries and/or by dissection of the major vessels.

FIG. 1. Cross-section of a heart from a patient suffering from systemic hypertension. Note the severe concentric hypertrophy and the small cavity of the left ventricle.

Histology shows an increase in diameter of the myocardial fibres and an average of $20\text{ }\mu\text{m}$ may be found (normal $5-12\text{ }\mu\text{m}$). These changes are uniformly distributed throughout the left ventricular wall, but some variation does exist. Collagen tissue, which is normally present between individual or groups of myocardial fibres in very small amounts, increases and non-inflammatory fibrous replacement of myocardial fibres takes place. Another feature commonly found in patients who die as a

FIG. 2. The left ventricle from a patient with systemic hypertension has been opened to show some dilatation of the left ventricle and hypertrophy of the ventricular wall, which as a result of dilatation does not appear to be prominent.

result of hypertension is an increase in the thickness of the medial coat of the small arteries and intimal thickening. The degree of medial thickness is difficult to assess quantitatively as there is usually only a well-developed internal elastic lamina but no external elastic lamina present (in contrast to medial wall thickening in pulmonary arteries where there are always well-defined internal and external elastic laminae).

It is also difficult to assess whether hypertrophy or hyperplasia or both have taken place. This problem has been investigated and it has been found that there is a critical heart weight beyond which hyperplasia takes place, but below which physiological hypertrophy occurs (Linzbach, 1947). If one takes the whole heart, this weight is 500 g but if one takes the ventricle separately, 200 g for each ventricle is the critical heart weight. Under pathological conditions the critical heart weight may be exceeded and hyperplasia then takes place,

irrespective of the underlying cause. Macroscopically one or other ventricle may be affected, for example the left ventricle in systemic hypertension, the right ventricle in pulmonary hypertension, but histologically no distinction can be made in the respective ventricles.

Whenever a patient has hypertrophy the heart may, for known or unknown reasons, fail. Dilatation then takes place, which again can be best assessed by examining cross-sections of the heart (Fig. 3). Dilatation of both right and left ventricular cavities is at times very striking. Histologically the picture is again one of attenuation of myocardial fibres, particularly in the chronic cases, and nuclear changes typical of hypertrophy are also evident. In addition dilated capillaries and small blood vessels are present in these areas. The attenuation of myocardial fibres in dilatation (as in the patients suffering from congestive cardiomyopathy) is more marked near the cardiac cavity compared with the epicardial surface, and there one may see thick myocardial fibres measuring well above $12\text{ }\mu\text{m}$ in diameter.

In conclusion the heart can only react to a variety of stimuli in a certain limited way. If increased work is required, the increase takes place by physiological hypertrophy, but once the critical level has been reached there is evidence to suggest that hyperplasia takes place, and that an increase of nuclei, probably by amitotic division, may occur. When the heart dilates the myocardial fibres become attenuated, which is much more notable nearer the left ventricular cavity than at a distance from the cavity. Histologically, hypertrophy, due to whatever cause, has a non-specific appearance, but when dilatation supervenes, myocardial and endocardial changes will allow a diagnosis of dilatation.

FIG. 3. Cross-sections of the heart of a patient with congestive cardiomyopathy showing dilatation of both ventricular cavities.

Reference

- LINZBACH, A.J. (1947) Mikrometrische und histologische Analyse hypertropher menschlicher Herzen. *Virchows Archives of Pathology and Anatomy*, **314**, 534.