Hypertension, hypertrophy and dilatation

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ANYONE who has taken part in joint clinico-pathological investigations will be aware of the discrepancies that often exist between the clinical and functional estimates of disease and the histopathological findings. In cardiovascular disease the pathologist encounters cases in which the morphological changes in the myocardium at necropsy are minimal, but the functional changes in life were gross. Conversely, the morphological changes may be gross and yet the functional changes were slight. In the present context both clinician and pathologist are concerned with measurements of cardiac dilatation (volume change) and of hypertrophy (weight change). There is good evidence that the size of a heart at necropsy (displacement volume) is considerably less than the size measured in life by radiographic techniques: in general the former volume is about 33% less than estimates made in life (Linzbach, 1972). These discrepancies should be kept in mind in contrasting the three hearts that I am now going to describe.

The first, from a case of essential hypertension, shows typical concentric hypertrophy of the left ventricle which has a thick wall and a small cavity. The second is from a case of severe long-standing anaemia due to hookworm disease, a common problem in Africa. This shows marked cardiac dilatation but the heart is not increased in weight. The myocardial fibres contain stainable lipid and the wall of the ventricle is thinner than normal. The last case is an example of bilateral ventricular dilatation and hypertrophy. The weight, about 500 g, is similar to that of the hypertensive heart but the volume is greatly increased as a result of the dilatation and the wall thickness is therefore within the normal range. Macroscopic and microscopic examination of this heart failed to reveal any specific pathology apart from the evidence of dilatation and hypertrophy. In the absence of clinical information, for the patient was admitted *in extremis* and died immediately, this might either be idiopathic cardiomegaly or a dilated hypertensive heart.

In the first case, typical concentric hypertrophy due to hypertension, the increased work-load must develop very slowly and initial dilatation is soon followed by hypertrophy. In the case of gross

anaemia the anoxic damage to the myocardium and the haemodynamic state have resulted in gross dilatation without hypertrophy. Perhaps the myofibre response to dilatation in this case with persistent gross anaemia is impaired, for some workers report hypertrophied hearts in chronic anaemia, though this is seldom gross (Brockington & Edington, 1972).

The appearances in the last case could be interpreted in one of three ways. The first possibility is that this is idiopathic cardiomegaly, due initially to a myocardial insult which resulted firstly in cardiac dilatation, and the resulting increase in muscle tension then led to cardiac hypertrophy. The second possibility is that this patient may have had long-standing and undiagnosed hypertensive cardiac disease in which gross dilatation has been superimposed on the initial hypertrophy. Dr John Fowler (p. 775) has indicated that Ugandan Africans with severe essential hypertension, which is not accompanied by significant coronary atherosclerosis, may go for many years without developing cardiac failure, and that many of the cases who die of hypertensive cardiac failure in Uganda have underlying chronic renal disease and therefore a complex pathogenetic background to account for their failure. The difference between these two groups has also been emphasized from Ibadan by Brockington & Edington (1972).

Finally, it is possible that these distinctions are artificial and that most cases of idiopathic cardiomegaly diagnosed by the pathologist are a form of hypertensive heart disease in which dilatation of the heart occurs even though the initiating hypertension may disappear.

It seems unlikely that a single theory of aetiology or pathogenesis will explain all cases of 'idiopathic cardiomegaly'. These appearances may result from a number of different factors affecting the myocardium either directly through some toxic or infective agent and/or through changes in haemodynamics and work load.

References

- BROCKINGTON, I.F. & EDINGTON, G.M. (1972) Adult heart disease in Western Nigeria. *American Heart Journal*, **83**, 27.
- LINZBACH, A.J. (1960) Heart failure from the point of view of quantitative anatomy. *American Journal of Cardiology*, **5**, 370.