Heart size in the elderly: a clinicopathological study[1]

Isabel Hodkinson
Ariela Pomerance MD MRCPath
H M Hodkinson DM FRCP
Northwick Park Hospital, Harrow, Middlesex

Clinical assessment of cardiac size by location of the apex beat or by percussion is very inexact. Information from chest X-ray and electrocardiogram (ECG) is often used to supplement the clinical assessment, the most commonly used measurements being the cardiothoracic ratio and the combined height of the S wave in lead V2 and the R wave in lead V5 (V2 + 5) respectively. Their value has been assessed in younger patients (*see* Masters *et al.* 1963, Friedman 1971, Glover *et al.* 1973, Chikos *et al.* 1977) where they have been shown to be useful though approximate methods. There has been no assessment of their reliability in elderly patients.

The present study sought to establish the value of these methods in old age and at the same time to determine which were the most important pathological causes of cardiac enlargement in the age group.

Patients and methods

The study was a part of a larger investigation of cardiac pathology of the aged and was based on all patients who were admitted to the geriatric department of Northwick Park Hospital and died during the study period September 1971–April 1975 and had a post-mortem examination. Careful clinical records had been kept and the post-mortem examination of the heart was carried out by one pathologist (A P) as described more fully elsewhere (Denham *et al.* 1977, Hodkinson & Pomerance 1977).

The total series comprised 334 patients: all were over 60 years, 64% were women and average age was 81 years. Measurement of V2 + 5 was made where there was a technically-adequate ECG which did not show bundle branch block. Detailed measurements of the chest X-ray were made by one observer (I H) and only where there was a well-centred posteroanterior (PA) film of good technical quality in which there was no obscuring of the cardiac outline by pulmonary shadows or distortion by kyphoscoliosis. Measurements used the methods of Chikos *et al.* (1977). Those allowing calculation of cardiothoracic ratio, perimeter and frontal size are shown in Figure 1. Indices for 'leftness of heart', 'roundness of left ventricle' and 'left ventricular size' were also determined but are not detailed here as they were not significantly correlated with heart size in the series.

Results

The effects of age, sex and body size

The correlations of age, sex, height and weight with heart weight were examined simultaneously using multiple regression analysis. This showed that only age and body weight had significant effects (Table 1), both showing positive correlations with heart weight.

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Requests for reprints to: Dr H M Hodkinson, Geriatric Department, Hammersmith Hospital, London W12 0HS

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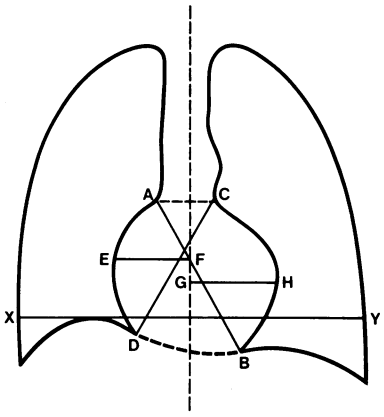

Figure 1. Heart size measurements on the PA chest X-ray: cardiothoracic ratio = (EF + GH)/XY; frontal size = AB × CD; adjusted frontal size = AB × CD/XY; perimeter = length of cardiac outline A–C–B–D; adjusted perimeter = perimeter/XY

Clinicopathological factors affecting heart weight

Age and body weight were then examined together with a number of clinicopathological factors and ECG findings in a step-down multiple regression analysis. Systolic blood pressure, diastolic blood pressure, cardiac amyloidosis, valvular pathology and ECG evidence of ischaemia or infarction made no significant contribution to the prediction of heart weight. Age was also non-significant, suggesting that its previous significant contribution related to the accumulation of relevant pathology with age rather than an age effect proper. The factors making a significant contribution to the prediction of heart weight are shown in Table 2 and comprised body weight, pathological evidence of ischaemia or infarction, V2 + 5, presence of cardiac failure and ECG findings of left ventricular hypertrophy and strain. Atrial fibrillation made a borderline contribution ($P < 0.08$).

Prediction of heart weight from chest X-ray and ECG measurements

The various chest X-ray measurements and V2 + 5 were available as potential predictors of heart weight. However, some measurements would be expected to relate to relative heart weight (e.g. V2 + 5 and cardiothoracic ratio) and others to absolute heart weight (e.g. perimeter and frontal size). To allow comparisons to be made, heart weight, perimeter and frontal size were all converted from absolute to relative quantities by dividing by body weight in the first case and by transverse thoracic diameter in the other two.

Table 1. Multiple regression analysis of heart weight on age, body weight, height and sex (334 cases, R = 0.6230)

Factor	Regression coefficient (*b*)	s.e. (*b*)	*t*	*P*
Age (years)	2.197	0.412	5.34	<0.001
Body weight (kg)	5.346	0.415	12.89	<0.001
Height (cm)	−0.354	0.406	0.87	NS
Sex (0/1)	−15.318	10.571	1.45	NS

Table 2. Multiple regression analysis of heart weight on body weight, pathological evidence of ischaemia/infarction, V2+5, cardiac failure during life, ECG changes of 'left ventricular hypertrophy and strain' and atrial fibrillation (163 cases ●, R=0.7831)

Factor	*b*	s.e. (*b*)	*t*	*P*
Body weight (kg)	4.855	0.4292	11.31	<0.001
Ischaemia/infarction■	55.05	10.99	5.01	<0.001
V2+5 (mm)	1.688	0.4502	3.75	<0.001
Cardiac failure■	32.30	10.94	2.95	<0.005
LVH and strain■	24.21	8.778	2.76	<0.01
Atrial fibrillation■	20.49	11.59	1.77	<0.10

● only those patients with complete results are included
■ qualitative variables are coded 0=absent, 1=present

Only adjusted frontal size, adjusted perimeter, cardiothoracic ratio and V2+5 showed significant correlations with relative heart size (Table 3). Multiple regression analysis showed, however, that cardiothoracic ratio failed to make a significant contribution to prediction of relative heart size in the presence of the other three measurements (Table 4). It still fails to contribute significantly if V2+5 is removed from the analysis. The combination of the two chest X-ray measurements without V2+5 gives a much weaker correlation however ($R=0.5992$ cf. 0.6506). In practical terms, the best single predictor is adjusted frontal size. This involves three simple measurements and takes only marginally more time than the more familiar cardiothoracic ratio but gives a substantially better prediction ($r=0.5463$ cf. 0.4009, i.e. approximately 30% of the variance of relative heart size is explained as compared with only 16%).

Discussion

The very strong correlation of heart weight with body weight is as would be expected on theoretical grounds for any organ. Correction for differences in body weight eliminates differences in heart weight between elderly men and women.

Table 3. Correlations of adjusted frontal heart size, adjusted heart perimeter, cardiothoracic ratio and V2+5 with relative heart size

Measurement	No. of cases	*r*	*P*
Adjusted frontal size	46	0.5463	<0.001
Adjusted perimeter	46	0.4538	<0.005
Cardiothoracic ratio	46	0.4009	<0.01
V2+5	163	0.3850	<0.001

Table 4. Multiple regression analysis of relative heart size on adjusted frontal heart size, adjusted heart perimeter and V2+5 (46 cases, R=0.6506)

Factor	*b*	s.e. (*b*)	*t*	*P*
Adjusted frontal size	36.000	10.560	3.41	<0.005
Adjusted perimeter	−5.144	2.105	2.44	<0.02
V2+5	0.039	0.018	2.16	<0.05

Of the clinicopathological factors showing correlation with cardiac enlargement, ischaemia is the most powerful. This is in keeping with the findings of previous authors that ischaemic heart disease forms a major part of cardiac pathology in the aged (McKeown 1965, Pomerance 1972). It is not surprising to find that ECG evidence of left-ventricular hypertrophy and strain and V2 + 5 contribute to the prediction of heart weight in that both are accepted indicators of increased left-ventricular mass. Severe hypertension is uncommon in elderly patients and McKeown (1965) has noted its relatively small contribution to cardiac pathology in old age. However, as a consequence of such developments as myocardial infarction or severe cardiac failure, elderly patients who have been previously hypertensive may have normal blood pressures during their final illness. This may explain why the presence of left-ventricular hypertrophy and strain pattern on ECG was significantly correlated with heart weight whereas systolic and diastolic blood pressures were not. The contribution of cardiac failure is also in line with expectations and its presence, coupled with the relatively low incidence of valvular lesions of any severity, makes the failure of valvular heart disease as an effective predictor understandable. It is also to be expected that ECG evidence of ischaemic heart disease has no predictive value in the presence of a pathological assessment which must clearly give more information.

The correlations of heart weight with the chest X-ray measurements, though highly significant, are not particularly strong. At best they explain only about a third of the total variance of heart weight. This is not unexpected as the X-ray methods cannot hope to distinguish between cardiac dilatation and cardiac hypertrophy as Chikos *et al.* (1977) have shown. Both will give an enlarged cardiac shadow but only the latter will result in increased heart weight. In contrast, the measurement of V2 + 5 on the ECG, though a far more indirect method, avoids this disadvantage. This can explain why, even though V2 + 5 was a weaker individual predictor of relative heart weight than adjusted frontal size, adjusted perimeter or cardiothoracic ratio, it gave a significantly improved prediction when added to the best combination of these, adjusted frontal size plus adjusted perimeter.

Chest X-ray measurements, of which adjusted frontal size appears to be the most useful, and the ECG are thus seen to be of definite value in the assessment of heart size in elderly patients as in younger age groups.

Summary

Predictors of heart weight have been examined in a prospective clinicopathological study of elderly patients admitted to a department of geriatric medicine. Ischaemic heart disease emerged as the most powerful pathological cause of cardiac enlargement. Heart weight was quite strongly correlated with body weight. After correction for body weight, a combination of electrocardiographic and chest X-ray measurements gave the best prediction of heart size.

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