HEART SIZE IN PRIMARY MYOCARDIAL DISEASE*

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SUMMARY

Heart volume and cardio-thoracic ratio were compared statistically with the haemodynamic and angiocardiographic parameters measured at cardiac catheterization in 38 patients with primary myocardial disease. Three patients had mild cardiomyopathy, 17 had classical cardiomyopathy, in 6 valvular incompetence dominated the clinical picture and 12 were children. Heart volume and cardio-thoracic ratio were related to each other. Heart volume, but not cardio-thoracic ratio, were related to all the abnormal haemodynamic parameters suggesting that measurement of heart volume alone could be of predictive value in assessing the severity of the underlying haemodynamic abnormality.

There is the need in clinical practice to make a quantitative assessment of the degree of haemodynamic abnormality and to have some reproducible index which can be used as a measure of serial changes in a patient's clinical status. Physical incapacity is a subjective phenomenon[1,2] while objective tests of exercise capacity are time-consuming in routine practice.[3-7] On the other hand, a simple parameter such as heart size can be measured on a standard 6-foot postero-anterior and lateral chest radiograph which is made as part of the clinical evaluation of each patient. We have used in the present study, films made in the erect position as part of the day-to-day routine evaluation of the patient, recognizing that there are changes induced by posture, phasing of the cardiac cycle and occasionally by imperfect inspiration.[8]

Enlargement of the heart is a consequence of dilatation of one or more cardiac chambers—this may be a result of an intracardiac shunt, valvular incompetence or ventricular failure. In primary myocardial disease the over-all size of the heart is related to the size of the individual chambers and therefore to the degree of heart failure. Valvular incompetence is absent or insignificant in patients with mild or moderate lesions although when there is great ventricular enlargement with papillary muscle dysfunction it may dominate the clinical picture. Cardiomyopathy is an ideal model in which to test the hypothesis that heart size is related to the underlying haemodynamic abnormality.

The present study was undertaken in patients with primary myocardial disease to determine whether the over-all size of the heart as measured on the standard, erect, postero-anterior and lateral chest films corresponds with the haemodynamic abnormalities as measured at cardiac catheterization and to see whether such films would be of predictive value in estimating the underlying haemodynamic abberation.

PATIENTS

Thirty-eight consecutive patients with primary myocardial disease were selected for study. The clinical diagnosis was made on the basis of ventricular dysfunction in patients in whom there was no evidence of systemic hypertension, coronary vascular disease, underlying rheumatic carditis or a generalized systemic disorder.

The patients were divided into 4 clinical groups:

Group I consisted of 3 patients with mild cardiomyopathy. They had a history of shortness of breath on exertion but were not in cardiac failure at the time of investigation. The clinical, cardiographic and radiological features were in keeping with primary myocardial disease. Other causes of left ventricular hypertrophy were excluded by cardiac catheterization and coronary angiography was performed in one to exclude underlying coronary atherosclerosis. The range of haemodynamic abnormality is summarized in Table I.

Group II consisted of 17 patients with classical cardiomyopathy.[9-12] They gave a history of cardiac disability with several episodes of cardiac failure. Again the clinical features were characteristic with palpable, electrocardiographic and radiological enlargement of the left ventricle, palpable and audible left ventricular third and fourth heart sounds, and in 11, severe pulsus alternans. One patient had repeated pulmonary embolism and also had right ventricular enlargement. The abnormal haemodynamic profile is given in Table I.

Group III consisted of 6 patients who had cardiomyopathy and had additional moderate to severe atrioventricular valvular incompetence. Mitral incompetence was present in all and tricuspid incompetence in 4. In each patient the degree of ventricular dysfunction was out of keeping with the amount of valvular incompetence and on this basis it was possible to exclude underlying rheumatic valvular disease. Acute rheumatic carditis was excluded by serial serological tests. Autopsy proof of underlying cardiomyopathy was obtained in the 2 patients who subsequently died.

Group IV consisted of patients with childhood cardiomyopathy. Two patients had a clinical picture indistinguishable from endocardial fibro-elastosis, 2 had co-existent nephritis with mild hypertension which may have been an underlying aetiological factor, 1 had evidence of a generalized viral infection and another had associated kwashiorkor.

METHODS

Chest radiography was undertaken immediately before cardiac catheterization. Films were taken in the postero-anterior and lateral positions using a standard 6-foot tube-film distance. Barium was placed in the oesophagus to outline the posterior surface of the left atrium and facilitate measurements made in the lateral position. Heart volume was measured by the use of the ellipsoid technique as modified by Jonsell and allowing for magnification.[8,13-17] By taking into account magnification, the formula could be expressed as:

$$V = 0{\cdot}41 \cdot d_1 \cdot d_2 \cdot d_3$$

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TABLE I. SUMMARY OF RADIOLOGICAL MEASUREMENTS AND HAEMODYNAMIC PARAMETERS

					Pressures (mmHg)					*Flows*			
		Age (years)	*CTR*	*HV*	*RAP (mean)*	*RVEDP*	*PAP (mean)*	*PCWP (mean)*	*LVEDP*	*A-V O_2 diff. (ml/litre)*	*Cardiac index (litre/min/m²)*	*Stroke index (ml/beat/m²)*	*Ejection fraction %*
Group I	Range	21 - 33	49 - 58	460 - 480	0 - 13	2 - 6	19 - 35	2 - 12	2 - 12	18 - 38	1·6 - 3·7	16 - 61	16 - 34
Mild cardiomyopathy (3)	Mean	27	53·8	474	6	4	25	7·4	6	25	2·5	37	25
Group II													
Classical cardiomyopathy	Range	27 - 67	54 - 75	610 - 1 200	1 - 27	1 - 25	18 - 52	10 - 43	16 - 45	22 - 50	1·1 - 3·3	11 - 47	13 - 34
(17)	Mean	46	64	875	13	13	33	26	28	37	2·1	24	22
Group III													
Cardiomyopathy with	Range	13 - 50	54 - 84	580 - 1 360	4 - 20	5 - 25	29 - 54	14 - 35	13 - 28	27 - 48	1·5 - 2·2	15 - 21	17 - 24
valve incompetence (6)	Mean	31	68	1040	12	14	35	27·5	22·6	40	1·8	18	20
Group IV													
Children with	Range	6/12 - 8	55 - 83	295 - 965	1·2 - 13	2 - 13	10 - 45	2·5 - 29	6 - 33	25 - 46	2·1 - 5·1	17 - 46	18 - 30
cardiomyopathy (12)	Mean	4·3	66	542	4	5	26	15	19	37	3·4	32	24

Heart volume was expressed as a volume per unit of body surface area to permit standardization and comparison of data. Cardio-thoracic ratio was calculated by the standard method.

Routine right and left heart catheterization was performed using the mid-chest level as a zero reference. Cardiac output was measured by the direct Fick principle. Selective left ventriculography was performed in a 45° right anterior oblique position and ventricular volumes estimated by the uniplane method.[15] Ejection fraction was calculated from the formula:

$$\text{Ejection fraction} = \frac{\text{EDV} - \text{ESV}}{\text{EDV}} = 100$$

where EDV = end-diastolic volume and
ESV = end-systolic volume.

By using this fraction, errors which were a result of magnification or geometrical distortion were excluded.

Radiological and haemodynamic information were compared using standard methods of regression analysis in the individual groups; groups I, II and IV together to exclude the patients with severe valvular incompetence and finally in the total patient sample. Cardio-thoracic ratio and heart volume were related to the following parameters: left ventricular end-diastolic, pulmonary arterial capillary wedge, mean pulmonary arterial, right ventricular end-diastolic, and mean right atrial pressures; arterio-venous oxygen difference, cardiac index, stroke index and left ventricular ejection fraction.

RESULTS

There was a good correlation between cardio-thoracic ratio and heart volume in the individual groups and in the total population sample (CT = 52 + 0·16 HV ± 6·6; R = 0·5; P<0·1%). The results are statistically significant

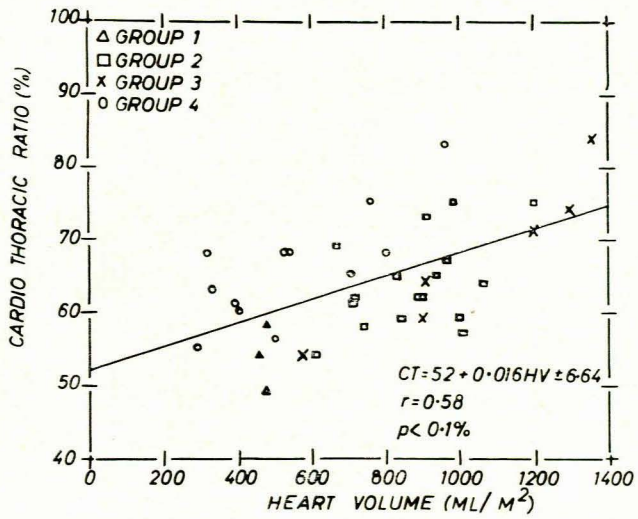

Fig. 1. Relationship between calculated cardio-thoracic ratio and heart volume expressed as volume per m² of body surface area. Group I refers to patients with mild cardiomyopathy, group II to patients with classical cardiomyopathy, group III to patients with valvular incompetence and group IV to the children. The same system labelling is used in all subsequent diagrams. In each diagram the regression equation is calculated by the method of the least squares, and the R and P values are given for the grouped data.

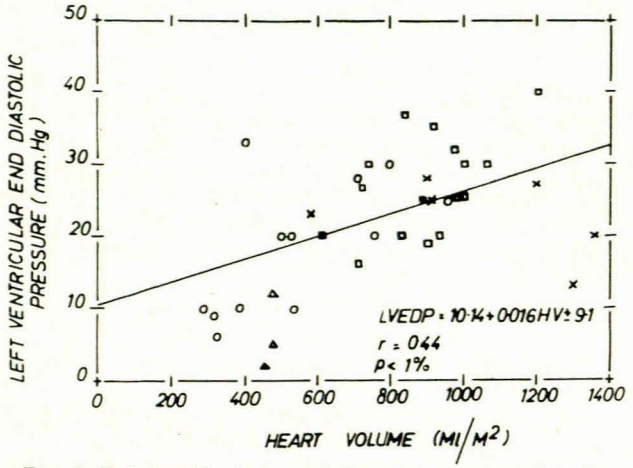

Fig. 2. Relationship between left ventricular end-diastolic pressure (mmHg) and heart volume. Patients with mitral incompetence had disproportionately enlarged hearts for the degree of elevation of left ventricular end-diastolic pressure. The children had a small heart volume.

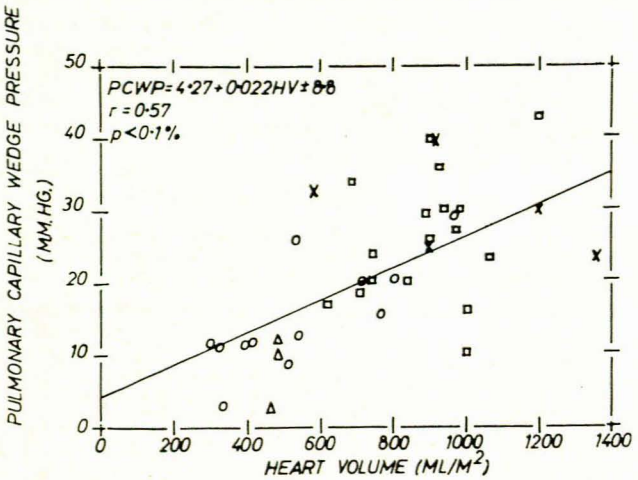

Fig. 3. Relationship between pulmonary capillary wedge pressure (mmHg) and heart volume.

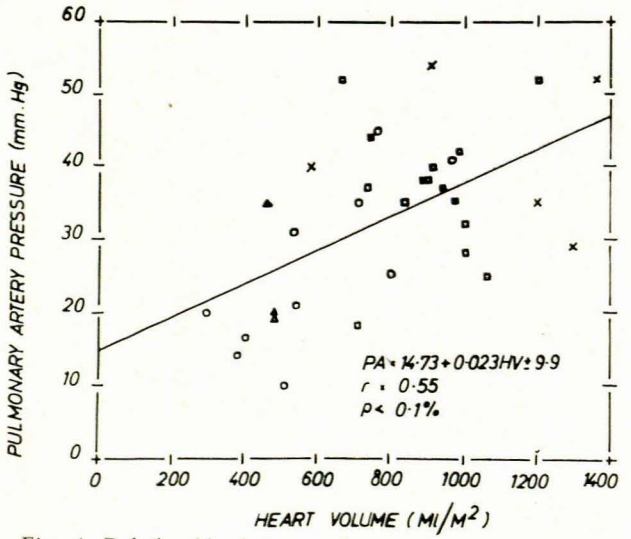

Fig. 4. Relationship between the mean pressure in the pulmonary artery and heart volume.

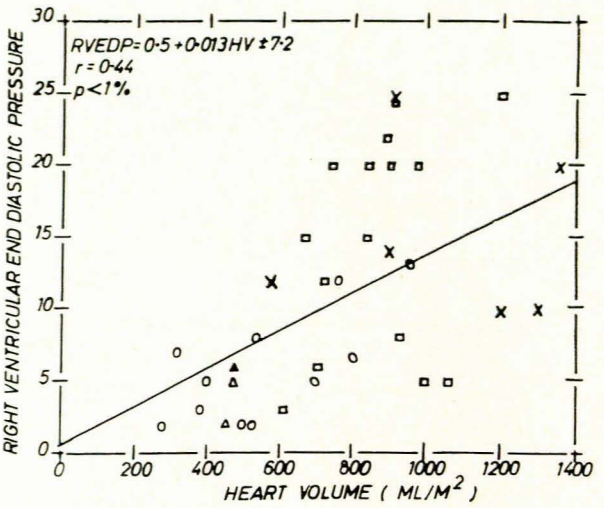

Fig. 5. Relationship between end-diastolic pressure in the right ventricle (mmHg) and heart volume.

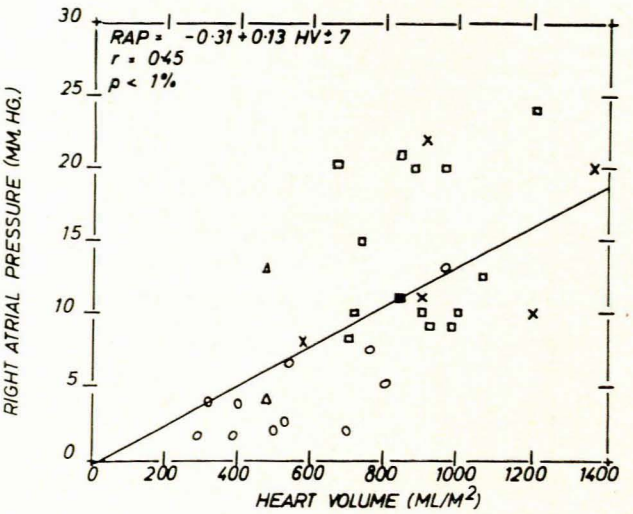

Fig. 6. Relationship between mean pressure in the right atrium (mmHg) and heart volume.

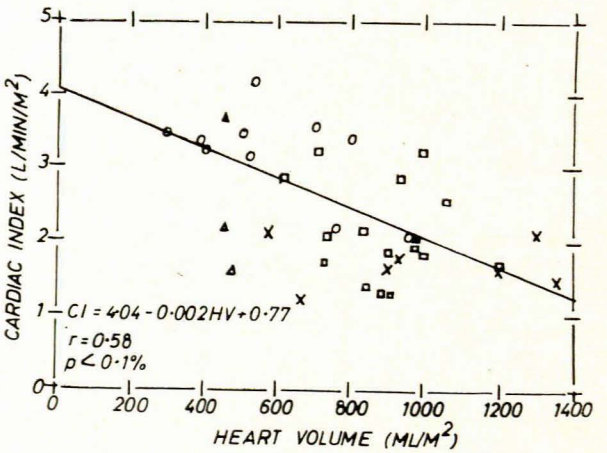

Fig. 7. Relationship between cardiac index (litre/min/m²) and heart volume.

TABLE II. STATISTICAL RELATIONSHIP BETWEEN MEASURED HEART VOLUME (INDEX) AND SELECTED RADIOLOGICAL AND HAEMODYNAMIC PARAMETERS IN PATIENTS IN GROUPS I, II AND IV

Parameter	*N*	*Regression equation*	*Correlation co-efficient*	*P value*
Cardio-thoracic ratio %	32	CT = 54·15 + 0·013 HV ± 6·68	0·45	<1%
RAP (mean)	31	RAP = 3·07 + 0·017 HV ± 6·9	0·51	<1%
RVEDP	31	RVEDP = 2·08 + 0·016 HV ± 7	0·5	<1%
PAP (mean)	28	PAP = 10·4 + 0·029 HV ± 9·2	0·61	<0·1%
PCWP (mean)	32	PCWP = 1·64 + 0·03 HV ± 7·6	0·71	<0·1%
LVEDP	32	LVEDP = 4·11 + 0·026 HV ± 8·8	0·59	<0·1%
A-V O_2 difference (ml/litre)				>5% not significant
Cardiac index (litre/min/m²)	31	CI = 4·22 − 0·002 HV ± 0·82	0·57	<0·1%
Stroke index (ml/beat/m²)	31	SI = 42·55 − 0·02 HV ± 11·3	0·4	<5%
Ejection fraction (%)				>5% not significant

but examination of Fig. 1 shows a considerable range of scatter in the individual patients.

The relationship between the heart volume ratio and the different haemodynamic parameters and measurements of ventricular contractility are shown in Figs. 2 - 9. The statistical analysis is summarized in Table II for patients in groups I, II and IV and for the total patient sample in Table III.

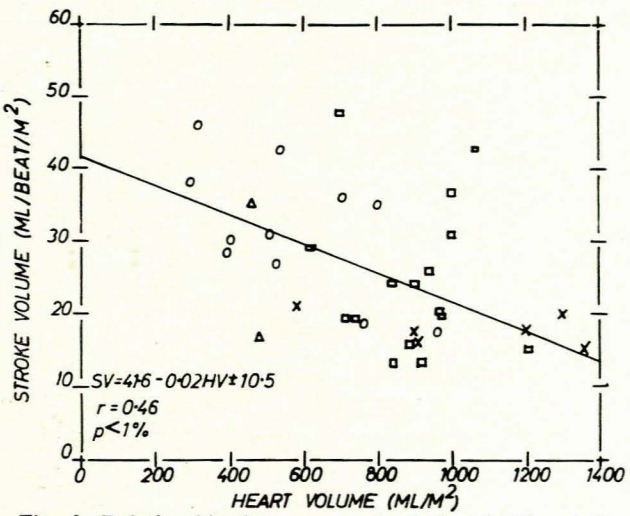

Fig. 8. Relationship between stroke index (ml/beat/m²) and heart volume.

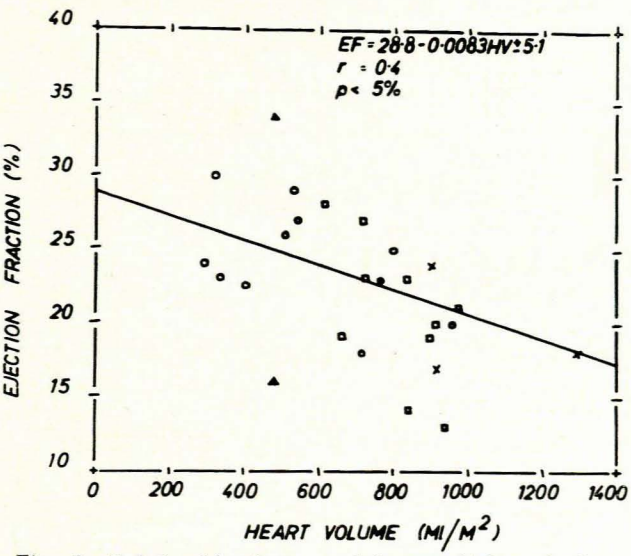

Fig. 9. Relationship between left ventricular ejection fraction (%) and heart volume. Exact measurements of ejection fraction could not be made in all the patients. In all patients, the ejection fraction was very low.

Thus in the individual groups, in the combination of groups I, II and IV and the total population sample heart volume was related significantly to left ventricular end-diastolic pressure, pulmonary arterial capillary wedge pressure, mean pulmonary artery pressure, right ventricular end-diastolic pressure, right atrial pressure, cardiac index, stroke index and ejection fraction. The relationship between heart volume and arterio-venous oxygen difference was not statistically significant. Some patients, the majority of whom were children, had a heart volume which fell within the range of normal (less than 500 ml/m² in males and 450 ml/m² in females).

In general, infants and small children have a relatively smaller heart volume per unit of body surface area than adults, so that in several younger children their volume fell within the adult range of normal.[15] Moreover, the children had been given more vigorous therapy before being submitted to investigation so that the fall in intravascular pressures had preceded a subsequent decrease in cardiac size.

The group of patients with valvular incompetence appeared to have hearts which, for a given degree of haemodynamic abnormality, were larger than those observed in patients in the other 3 groups, and it was obvious that the regurgitant flow was responsible for this additional enlargement. The group was too small for independent statistical analysis and if they were excluded from the over-all patient sample there was little improvement in the degree of statistical correlation (Tables II and III).

By contrast, there was no consistent significant relationship between cardio-thoracic ratio and the individual haemodynamic parameters although graphical examination of the data suggested a similar directional trend. This meant that although cardio-thoracic ratio and heart volume were related, heart volume was of greater discriminant value in the prediction of the underlying abnormality.

Ejection fraction was also related to the abnormal haemodynamic parameters and selected statistical relationships are shown in Table IV. Thus ejection fraction was related to stroke index, arterio-venous oxygen

TABLE IV. CORRELATES OF EJECTION FRACTION

Stroke index (ml/beat/m²)	= −10·58 + 1·65 EF ± 9; r = 0·72; P < 0·1%
A-V O_2 difference (ml/litre)	= 54·7 − 0·82 EF ± 10·3; r = 0·41; P < 5%
PCWP (mmHg)	= 43·24 − 1·00 EF ± 8·9; r = 0·53; P < 1%

TABLE III. STATISTICAL RELATIONSHIP BETWEEN MEASURED HEART VOLUME (INDEX) AND SELECTED RADIOLOGICAL AND HAEMODYNAMIC PARAMETERS IN ENTIRE POPULATION SAMPLE

Parameter	*N*	*Regression equation*	*Correlation co-efficient*	*P value*	
Cardio-thoracic ratio (%)	38	CT = 52 + 0·016 HV ± 6·64	0·58	<0·1%	
RAP (mean)	37	RAP = −0·31 + 0·013 HV ± 7	0·45	<1%	
RVEDP	37	RVEDP = 0·5 + 0·013 HV ± 7·2	0·44	<1%	
PAP (mean)	33	PAP = 14·73 + 0·023 HV ± 9·9	0·55	<0·1%	
PCWP (mean)	38	PCWP = 4·27 + 0·022 HV ± 8·8	0·57	<0·1%	
LVEDP	38	LVEDP = 10·14 + 0·016 HV ± 9·1	0·44	>1%	
A-V O_2 difference (ml/litre)				<5%	not significant
Cardiac index (litre/min/m²)	37	CI = 4·04 − 0·002 HV ± 0·77	0·58	<0·1%	
Stroke index (ml/beat/m²)	37	SI = 41·6 − 0·02 HV ± 10·5	0·46	<1%	
Ejection fraction (%)	27	EF = 28·8 − 0·0083 HV ± 5·1	0·4	<5%	

difference, cardiac output and to the degree of left ventricular failure as demonstrated by its relationship to the pulmonary arterial capillary wedge pressure.

DISCUSSION

Cardiac enlargement is a well known accompaniment of cardiac failure and is commonly encountered as a consequence of cardiomyopathy. The statistical analysis indicates that heart volume appears to be of predictive value in expressing mathematically the degree of deviation from normal of the haemodynamics in patients with myocardial disease but that the use of the cardio-thoracic ratio is of less discriminant value. The latter index takes into account only the diameter of the right atrium, a small degree of right ventricular dilatation and a projection of the long axis of the left ventricle. It does not include left atrial enlargement where it fills in the left middle segment of the heart or where it displaces the oesophagus posteriorly.

The basic disturbance in these patients is a defect in left ventricular contractility which is associated with a mild to moderate increase in muscle mass and compliance, ventricular dilatation, a decrease in ejection fraction and a rise in end-diastolic pressure. There follows a sequence of events in which there is left atrial hypertrophy and dilatation, a secondary and passive rise in pulmonary artery pressure, hypertrophy, failure, dilatation and a rise in end-diastolic pressure in the right ventricle and finally a rise in pressure and dilatation of the right atrium. The net result is generalized dilatation of the heart to which a contribution of varying degree is made by each of the cardiac chambers: it is for this reason that heart volume is related to each in the individual haemodynamic parameters.[19-27] With great cardiac enlargement, the papillary muscles become functionally inadequate to ensure competence of first the mitral, and later the tricuspid valve so that functional atrio-ventricular valve incompetence comes to dominate the clinical picture. Such valvular incompetence with regurgitant blood flow is responsible for slight disproportionate enlargement of the heart.

The ejection fraction measured in the patients was always very low and although the range was small, the degree of correlation with heart volume and other haemodynamic parameters was surprisingly good.

In an analysis of the individual patients, it was evident that some patients had pressures which were disproportionately higher than the measured degree of cardiac enlargement while in others the pressures were unusually low. The patients with disproportionately high pressures probably represented examples of restrictive ventricular dysfunction with ventricular muscle of low compliance. Patients who had unusually low pressures were all studied after a period of vigorous medical treatment. This suggested that the intracardiac pressures had fallen first and that the decrease in heart size was a later and more passive phenomenon.

The change in heart size usually paralleled the change in the patient's clinical status, dilatation was usually associated with an increase in heart failure, and a reduction in heart size followed vigorous treatment in hospital.

The results of this investigation are significant: they allow the physician to assess the degree of haemodynamic abnormality from a simple, routine radiological investigation.

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