Left Ventricular Size and Function and Heart Size in the Year Following Myocardial Infarction

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SUMMARY

Interrelationships among left ventricular (LV) size, LV function, and heart size were investigated in 49 patients studied 2-12 months after myocardial infarction. LV end-diastolic volume (EDV) and ejection fraction (EF) were determined by biplane ventriculography. Heart size was estimated from chest films by the cardiothoracic ratio (CTR) and cardiac volume (CV) methods. Ventricular function (i.e., EF) was related to chamber size (i.e., EDV), but the correlation coefficient was not high (r = 0.74); thus, chamber size was not an accurate predictor of EF. Because of the close linear relation that exists between LV end-systolic volume and EDV (r = 0.95), a hyperbola describes the relation between EF and EDV. In general, EF was depressed (<0.50) when EDV exceeded 70 ml/m², was <0.35 when EDV exceeded upper normal limits (110 ml/m²), and was <0.25 with chamber size >150 ml/m². Thus, relatively small chamber size (<150 ml/m²) was associated with a wide range in ventricular function, while large chamber size was associated with severe dysfunction (EF < 0.30). By either heart size method, cardiomegaly (CTR > 0.50 or CV > 540 ml/m²) was not found consistently until EDV exceeded 150 ml/m². Hence, normal heart size was often associated with moderate EF depression (0.49-0.30), while cardiomegaly was often associated with severe dysfunction. Clinical heart failure (CHF) was usually accompanied by EF < 0.30 but chamber size ranged from 101 to 331 ml/m². Primarily because of this variation in chamber size, both normal heart size and cardiomegaly were at times associated with CHF.

The poor correlations of EF with CTR (r = -0.43) and CV (r = -0.52) preclude use of the heart size indices as accurate predictors of LV function. When the data were analyzed according to the presence or absence of cardiomegaly, the following generalizations could be made regardless of the heart size method used. Cardiomegaly was a reasonable indicator of postinfarction LV function, being associated with depressed function and often with CHF. However, normal heart size was associated with either normal LV function, or commonly, with depressed function, often not clinically apparent.

Additional Indexing Words:
Clinical heart failure Ejection fraction Ventricular volume

It is widely recognized that myocardial infarction involves predominantly the left ventricular (LV) wall and that infarction may produce impairment in LV function and an increase in chamber size. In postinfarction patients the clinician attempts to determine LV size in order to assess the magnitude of myocardial damage and to estimate the degree of functional impairment. A large postinfarction ventricle is thought to be associated with extensive damage and poor function.

Many techniques are available for estimating LV size including the physical examination, chest roentgenography, echocardiography, and angiocardiography. In everyday practice, standard chest roentgenograms are used. Unfortunately, quantitation of LV size from chest films is difficult. The physician commonly describes heart size by the cardiothoracic ratio; occasionally the cardiac volume method is used. In the postinfarction patient, an increase in these heart size indices is thought to represent an increase in LV size.

Little information is available concerning interrelationships among postinfarction LV size, LV function, and heart size. Knowledge of such relationships is important if the physician is to ap-
preciate the usefulness and limitations of roentgenographically-determined heart size in assessing postinfarction LV size and function. Thus heart size, LV chamber size, and LV function data are presented from a group of patients who had sustained myocardial infarction 2-12 months prior to study. Patients who exhibited arterial hypertension or significant valvular disease were excluded from this investigation in order to minimize the number of variables that might affect LV size.

**Methods**

Records of patients who had cardiac catheterization performed at the University of Alabama Medical Center during a 47-month period were reviewed. Forty-nine patients were identified who had: 1) a documented myocardial infarction that had occurred 2 to 12 months prior to catheterization, 2) a technically satisfactory angiographic study in which it was possible to quantitate left ventricular volumes, 3) technically satisfactory postero-anterior and lateral chest films that were obtained within a week prior to catheterization, and 4) no demonstrable arterial hypertension, congenital heart disease, or significant valvular disease.

The criteria for diagnosis of myocardial infarction were: 1) a typical clinical history for an infarct, 2) a prompt rise and fall of the serum glutamic oxaloacetic transaminase (SGOT), and 3) evolutionary electrocardiographic ST-segment changes of myocardial infarction with or without associated Q waves. Seventeen of the patients had been hospitalized in the University of Alabama Myocardial Infarction Research Unit during the acute phase of infarction, and 32 patients had been treated for their infarction at another hospital. Hospital discharge summaries, which included serum enzyme results and copies of serial electrocardiograms, were obtained for patients who were managed at another hospital.

Thirty-two of the patients were hospitalized in the University of Alabama Clinical Research Center for follow-up evaluation; the remaining patients were evaluated elsewhere in the hospital. A detailed history including a description of chest pain and of dysnea was recorded. During physical examination particular attention was directed to the presence or absence of a ventricular gallop sound (S3). Clinical heart failure (CHF) was defined as the presence of an audible S3, or a history of paroxysmal nocturnal dysnea (PND) that occurred after hospital discharge for the acute infarction, or both.

The electrocardiogram (ECG) obtained at the time of catheterization was classified according to the location of ≥0.03 sec Q waves or Q-S complexes present in at least two of the standard twelve ECG leads. The following categories for location of Q waves were used: 1) anterior (includes antero-septal, antero-lateral and lateral Q's), 2) inferior (includes infero-lateral Q's), 3) antero-inferior, and 4) no pathologic Q waves.

Heart size was estimated from standard 6 ft chest roentgenograms by the cardiothoracic ratio (CTR)¹,² and cardiac volume (CV)³ methods, as shown in figure 1. Care was taken to exclude the apical fat pad from cardiac measurements. CTR was expressed as the ratio of the maximal cardiac diameter to the maximal intrathoracic diameter. Maximal intrathoracic diameter was not determined at a fixed thoracic level, but was usually found to be just above the level of the costophrenic angles. CTR values >0.50 were considered to represent cardiomegaly by this method. CV values >540 ml/m² and >490 ml/m² are abnormal for males and females, respectively.⁴ Cardiac volume greater than 540 ml/m² was considered to represent cardiomegaly by this method for purposes of the present study because 46 of the 49 patients were males.

Cardiac catheterization was performed to evaluate chest pain, or abnormal dysnea, or both. Informed patient consent was obtained according to the principles expressed in the Declaration of Helsinki as endorsed by the American Society for Clinical Investigation, Inc.⁴ Coronary

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**Figure 1**

The chest films of patient 1. Cardiothoracic ratio was calculated using the maximal cardiac diameter and the maximal intrathoracic diameter. Cardiac volume parameters are: B, from the right cardiophrenic angle to the base of the main pulmonary artery segment; L, from the junction of the right atrium and superior vena cava to the apex; and D, the greatest cardiac diameter on the lateral film.

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LV size, function, heart size after MI

arteriography was executed by either the Sones's or Judkins' technique. Biplane left ventriculography was accomplished with a filming rate of 6 or 12 frames/sec following injection of 50-75 ml of 76% sodium meglumine diatrizoates into the left ventricular chamber. Ventricular volumes were determined by the Dodge area-length method. Films exposed during a premature ventricular contraction and the beat following were excluded from analysis. Ejection fraction was defined as the ratio of angiographic stroke volume to end-diastolic volume. Ventricular volumes were corrected for body surface area and were expressed in ml/m².

Statistical analysis was performed with the aid of an International Business Machine 370 computer. When correlations were performed, a linear relation was assumed to exist between the two parameters. On two occasions it was apparent that the relation was not linear: (1) A hyperbolic regression equation was used in the correlation of ejection fraction and end-diastolic volume (EDV). The rationale for using a hyperbola is presented in the Discussion and Appendix. (2) When EDV was correlated with heart size indices, it was assumed that EDV increased exponentially with an increase in heart size.

Results

The clinical and laboratory data are recorded in table 1. The patients, 46 males and three females, ranged in age from 30 to 67 years, with a mean age of 47 years. The mean time between infarction and catheterization was five months. A history of hypertension was given by 13 patients. None of the patients was considered to have severe pulmonary disease.

The locations of abnormal ECG Q waves were: anterior in 17 patients, inferior in 15 patients and antero-inferior in eight patients. Abnormal Q waves were not present in eight patients. One patient was not classified because of the presence of left bundle branch block. Trivial mitral regurgitation was apparent during ventriculography in four patients. A coronary artery lesion of ≥50% narrowing in at least one major vessel was identified in each patient.

LV Volumes

Left ventricular end-systolic volume correlated closely with end-diastolic volume (EDV) (fig. 2). Ejection fraction (EF) was related to EDV (fig. 3). EF (range, 0.10-0.69) was depressed (<0.50) in 41 or 84% of the patients. EDV (range 66-331 ml/m²) exceeded the upper limits of normal (110 ml/m²)* in 23 or 47% of the patients.

Clinical Heart Failure

Clinical heart failure (CHF) was detected in 18 patients. Both paroxysmal nocturnal dyspnea (PND) and a ventricular gallop sound (S₃) were present in six patients. PND unaccompanied by an S₃ was present in three patients, while nine patients had an audible S₃ in the absence of PND. EF was <0.30 in 17 of the 18 patients who exhibited CHF (fig. 3). In these 18 patients EDV ranged from 101 to 331 ml/m².

Heart Size

Cardiothoracic ratio (CTR) correlated poorly with cardiac volume (CV) (fig. 4). CTR ranged from 0.375 to 0.577, and was >0.50 in nine patients. CV ranged from 304 to 733 ml/m² and was >540 ml/m² in 17 patients. Each of the nine patients with CTR > 0.50 had CV > 540 ml/m². However, eight patients with CV > 540 ml/m² had CTR ≤ 0.50.

Heart Size and LV Size

The correlation between chamber size (i.e., EDV) and each of the heart size indices was low (fig. 5). Of

Figure 2

Left ventricular end-systolic volume (ESV) and end-diastolic volume (EDV) values for 49 patients.

Figure 3

Left ventricular ejection fraction (EF) and end-diastolic volume (EDV) values for 49 patients. EDV ≤ 150 ml/m² was often associated with normal heart size. Each patient with EDV > 150 ml/m² exhibited cardiomegaly. PND = paroxysmal nocturnal dyspnea, S₃ = ventricular gallop sound.
Table 1

Clinical and Laboratory Data for 49 Patients

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Abbreviations: A = anterior; AI = antero-inferior; CTR = cardiothoracic ratio; CV = cardiac volume; EDV = end-diastolic volume; EF = ejection fraction; I = inferior; LBBB = left bundle branch block; MI = myocardial infarction; N = no abnormal Q waves; PND = paroxysmal nocturnal dyspnea; Sₜ = ventricular gallop sound.

the 43 patients who exhibited EDV < 150 ml/m², 40 or 93% had CTR < 0.50; 31, or 72%, had CV < 540 ml/m². Each patient who exhibited EDV > 150 ml/m² had both CTR > 0.50 and CV > 540 ml/m².
LV SIZE, FUNCTION, HEART SIZE AFTER MI

Dashed lines indicate patients.

Heart Size and LV Function

Ventricular function (i.e., EF) correlated poorly with each of the heart size indices (fig. 6). EF was depressed in all patients with cardiomegaly, regardless of the heart size method used. CV > 540 ml/m² identified a larger number of patients with depressed EF (N = 17) than did CTR > 0.50 (N = 9). CV > 540 ml/m² also identified a larger number of patients with CHF (N = 12) than did CTR > 0.50 (N = 8).

Patients (N = 9) with CTR > 0.50 had a mean value for EF (0.18 ± 0.08) significantly lower than for those (N = 40) with CTR ≤ 0.50 (0.39 ± 0.12) (P < 0.0001). Likewise, patients (N = 17) with CV > 540 had a mean value for EF (0.26 ± 0.12) significantly lower than for those (N = 32) with CV ≤ 540 ml/m² (0.40 ± 0.13) (P < 0.001).

Discussion

This study describes interrelationships among postinfarction LV chamber size (i.e., EDV), LV function (i.e., EF), and clinical heart failure. In addition, two heart size indices determined from chest films are individually correlated with the above mentioned parameters.

Ventricular Function and Chamber Size

A hyperbolic relation was found to exist between EF and EDV. In general, the larger the chamber size the greater was the degree of impairment in ventricular function. EF is dependent on both end-systolic volume (ESV) and EDV. A close linear relation was found to exist between ESV and EDV. As detailed in the Appendix, because of this linear relation, a hyperbola describes the relation between EF and EDV. In addition, it should be mentioned that it is also correct to say that EF varies linearly with 1/EDV.

Although EF was related to 1/EDV, the correlation coefficient was not high (r = 0.74). From the data in this study, postinfarction chamber size cannot be considered to be an accurate predictor of ventricular function. We were intrigued that the correlation between EF and 1/EDV (r = 0.74) was much lower than the correlation between ESV and EDV (r = 0.98). Since the relation between EF and 1/EDV is a function of the relation between ESV and EDV,

Left ventricular end-diastolic volume is correlated with cardiothoracic ratio in (A), and with cardiac volume in (B). Dashed lines designate upper normal limits for the parameter.
one might have anticipated that the correlation between EF and 1/EDV would be higher. The mathematics that explain this apparent discrepancy are complex and are summarized in the Appendix. If the correlation between ESV and EDV had been perfect (i.e., \( r = 1 \)), then the correlation between EF and 1/EDV would also have been perfect. Slight depression in the ESV-EDV \( r \) value produces a relatively large depression in the EF to 1/EDV \( r \) value.

The hyperbolic relation that exists between EF and EDV is of clinical importance. The curve described by the regression equation (fig. 3) was steep when chamber size was within normal limits (<110 ml/m²). The steepness of this portion of the curve indicates that in general, a small increase in chamber size was associated with a relatively large decline in EF. It is because of the steepness of the curve that normal chamber size (<110 ml/m²) was associated with a wide range of LV function, varying from normal to moderate depression.

In contrast, the regression curve was relatively flat when EDV exceeded 150 ml/m². The level of this segment of the hyperbola indicates that EF was, in general, severely depressed (<0.30). The flatness of this portion of the curve indicates that a large increase in chamber size was associated with a relatively small decrease in EF. Thus, because of the flatness of the hyperbola, large chamber size (>150 ml/m²) was associated with a relatively narrow range of LV function.

Further examination of the hyperbolic regression equation helps to emphasize the above mentioned points. The equation indicates that in general, EF was depressed (<0.50) when EDV exceeded 70 ml/m² (mean normal for EDV is 70 ml/m²). When EDV exceeded upper normal limits (110 ml/m² or 2 sd above mean normal), EF was <0.35 and EDV > 150 ml/m² was associated with EF < 0.25. Thus relatively small chamber size (<150 ml/m²) was associated with a wide range in ventricular function, while large chamber size was associated with severe dysfunction.

As will be later discussed, cardiomegaly was not consistently present until EDV exceeded 150 ml/m². Hence normal heart size was often associated with moderate EF depression (0.49-0.30), while cardiomegaly was often associated with severe dysfunction.

The depression in EF when EDV was small contrasts with the levels of LV size and function that may be present in valvular heart disease. For example, a number of patients with aortic regurgitation studied in our laboratory have had EDV > 150 ml/m² and EF > 0.50. Obviously the type of heart disease under investigation might influence the relation between EF and EDV and hence the interpretation of heart size data.

It should be mentioned that in our experience, abnormally contracting segments (ACS) (akinetic or dyskinetic) of the left ventricle can usually be identified in patients who have been evaluated within the year following transmural myocardial infarction. We have found that in such patients, EF was inversely proportional to relative ACS size. In addition, segment size was generally greater than 17% when chamber size exceeded upper normal limits.

Figure 6

Ejection fraction is correlated with cardiothoracic ratio in (A), and with cardiac volume in (B). Dashed lines indicate lower normal limits for ejection fraction, and upper normal limits for the heart size indices. Note that in both (A) and (B) many patients had: EF < 0.50, normal heart size, and no CHF.
Clinical Heart Failure

Ejection fraction was usually <0.30 for patients who exhibited clinical heart failure (CHF). This finding is consistent with results from an earlier investigation in which CHF was also defined as the presence of an audible S3 or a history of PND, or both. A detailed discussion of why the CHF state as defined in this study should be related to EF is beyond the scope of this communication. Both PND and S3 are probably related to LV events that occur in diastole. In our experience with postinfarction patients, depression in LV systolic function (i.e., EF) is accompanied by a reduction in LV diastolic compliance.

Of particular interest was the variation in chamber size in CHF patients, which ranged from 101 to 331 ml/m². Since cardiomegaly was not consistently present until EDV exceeded 150 ml/m², both normal heart size and cardiomegaly were at times associated with CHF.

Heart Size

Heart size was determined by two separate methods, and the merits of each for describing postinfarction LV size and function were assessed. The cardiac volume (CV) method has been widely used in Scandinavian countries and to a lesser extent in the United States. Since this method uses cardiac dimensions from both antero-posterior and lateral films, it might be anticipated that it would be a more sensitive method than the cardiothoracic ratio which uses only a single projection.

The cardiothoracic ratio method was first described by Danzer in 1918. In this country it is probably the method most widely used by clinicians, primarily because of its simplicity. As discussed by Oberman, the value of the CTR as an index of heart size has long been disputed. In determining the CTR, the location of the internal thoracic diameter that is measured is of particular importance. Danzer stated that he “finally decided on taking the thoracic measurement at its greatest diameter which is usually at the level of the apex or one space lower . . . .” Others have used a fixed thoracic level such as the dome of the right diaphragm or the right costocardiocor border. In the present study the maximal thoracic diameter was used and was found usually to be just above the level of the costophrenic angles. This location has also been employed by Meschan.

Heart Size and LV Size

The correlation between EDV and each of the heart size indices was poor. Regression equations revealed that, in general, EDV was >125 ml/m² when CTR exceeded 0.50, and it was >115 ml/m² when CV exceeded 540 ml/m². By both methods, cardiomegaly was not consistently present until EDV exceeded 150 ml/m². Twelve patients with CV > 540 ml/m² had EDV between 90–150 ml/m², while only three patients with CTR > 0.50 had EDV in this range. Thus CV > 540 ml/m² identified a larger number of patients with relatively small chamber size (<150 ml/m²).

There are a number of reasons why heart size indices might not correlate closely with LV chamber size. The most obvious reason would be that heart size indices contain not only the LV chamber, but also the LV wall and other cardiac chambers. Asymmetry of the left ventricle might also influence the correlation. Additional considerations including patient position, phase of respiration, and the fact that the left ventricle might not be at end-diastole during chest roentgenography have recently been discussed in detail.

Heart Size and LV Function

The correlations of EF with CTR and CV were poor. Thus, left ventricular function could not be accurately predicted from either of the heart size indices. The regression equations revealed that, in general, when CTR exceeded 0.50, EF was <0.30; and when CV exceed 540 ml/m², EF was <0.33.

When the heart size data were analyzed according to the presence or absence of cardiomegaly, several observations were noteworthy. By either heart size method, patients with cardiomegaly exhibited a mean value for EF that was significantly lower than the value for those with normal heart size. It has long been the general impression of clinicians that postinfarction cardiomegaly may be associated with poorer LV function than normal heart size. To the best of our knowledge, the present study has demonstrated for the first time that this is indeed the case when LV function is expressed in terms of the EF.

Of interest was the finding that each patient with cardiomegaly (by either method) had EF depression. CV > 540 ml/m² identified a larger number of patients with depressed EF than did CTR > 0.50. Thus CV > 540 ml/m² was a more sensitive indicator. In addition, CV > 540 ml/m² identified a larger number of patients with CHF. It is concluded that cardiomegaly (by either method) was a reasonable indicator of postinfarction LV function since it was associated with EF depression and often with CHF. Normal heart size, however, was associated with normal LV function, or commonly, with depressed function. Often the depressed function was not clinically apparent. The patient whose chest film is shown in figure 1 is one example. Normal heart size was present (CTR = 0.45, CV = 481 ml/m²), CHF was absent, but EF was 0.33.
Several studies have demonstrated that postinfarction cardiomegaly is associated with a poorer prognosis than is normal heart size. It is likely that in these reports the patients with cardiomegaly also had, in general, lower EF than the patients with normal heart size. We have recently evaluated a series of cardiomyopathy patients and found that those with EF < 0.20 had a poorer prognosis than those with EF ≥ 0.20. Thus EF may also be of prognostic value in postinfarction heart disease, although as far as we know this had not been investigated.

It was of interest that in this study the ECG was not helpful in identifying the eight patients who had EF > 0.50. This was because seven of the eight patients with EF > 0.50 had ECG evidence of previous Q wave infarction, while six of eight patients who had no ECG evidence of Q wave infarction had EF < 0.50. A future report will deal with interrelationships between ventricular volume, location and number of infarctions, and coronary artery lesions.

In conclusion, in this select series of patients evaluated 2-12 months after myocardial infarction, roentgenographic heart size indices correlated poorly with left ventricular EDV and EF. Although the indices did provide some useful information, we believe that additional techniques (e.g., echocardiography, ventriculography) should be employed when more precise definition of postinfarction LV chamber size and function is needed.

Appendix

By J. A. Burdeshaw and B. J. Feild

The following analysis explains mathematically why a hyperbola describes the relation between left ventricular ejection fraction (EF) and end-diastolic volume (EDV).

The general equation for a linear relation between two variables expressed in slope-intercept form is:

\[ y = mx + b \]  (1)

where \( y \) = the parameter which is represented graphically on the ordinate or y-axis, \( x \) = the parameter which is represented on the abscissa or x-axis, \( m \) = the slope of the line, and \( b \) = the y-intercept of the line.

As shown in figure 2, the relation between end-systolic volume (ESV) and EDV is linear where \( y = \text{ESV} \), and \( x = \text{EDV} \).

By definition:

\[ \text{EF} = \frac{\text{EDV} - \text{ESV}}{\text{EDV}} = \frac{x - y}{x} \]  (2)

Substituting equation (1) into equation (2) and rearranging:

\[ \text{EF} = \frac{x - (mx + b)}{x} = \left(1 - \frac{m}{1}\right) + \frac{-b}{x}. \]  (3)

Let \( A = 1 - m \), and \( B = -b \). Since \( x = \text{EDV} \):

\[ \text{EF} = \frac{A + B}{\text{EDV}}. \]  (4)

Equation (4) is a formula for a hyperbola. Thus the relation between EF and EDV is hyperbolic, that is, EF varies linearly with 1/EDV.

As mentioned in the Discussion, we were intrigued that the correlation between EF and 1/EDV (\( r = 0.74 \)) was much lower than the correlation between ESV and EDV (\( r = 0.98 \)). As the EF-1/EDV relation is a function of the ESV/EDV relation, one might have anticipated that the EF/1/EDV correlation coefficient would be higher. Using statistical differentials, we derived the following formula:

\[ \rho^* = \frac{1 - Q}{\sqrt{1 - \rho^2} Q^2} \]  (5)

where: \( \rho^* \) = the correlation coefficient between EF and 1/EDV;

\( \rho \) = the correlation coefficient between ESV and EDV;

\( Q = \sigma_{\text{ESV}} \cdot \mu_{\text{EDV}} / \sigma_{\text{EDV}} \cdot \mu_{\text{ESV}} \); \( \mu_{\text{ESV}} = \text{mean of ESV}; \mu_{\text{EDV}} = \text{mean of EDV}; \sigma_{\text{ESV}} = \text{standard deviation of ESV}; \) and \( \sigma_{\text{EDV}} = \text{standard deviation of EDV}.

Equation 5 estimates the behavior of \( \rho^* \) as a function of \( \rho \) and or the means and standard deviations of ESV and EDV. In the present study Q is 1.45. As shown in figure 7, if the correlation between ESV and EDV had been perfect (e.g., \( \rho = 1 \)), then the correlation between EF and 1/EDV would also have been perfect. Slight depression in the ESV-EDV \( \rho \) value (e.g. from 1 to 0.9) produces a relatively large depression in the EF-1/EDV \( \rho^* \) value.

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