Regional Left Ventricular Performance in the Year following Myocardial Infarction


SUMMARY

The relationship of abnormal regional myocardial performance to left ventricular (LV) function 2–12 months following transmural myocardial infarction was investigated in 25 patients by quantitative biplane angiocardiography. Abnormally contracting segments (ACS) (akinetie or dyskinetic) of the LV were identified in 24 patients. Their sites correlated with the electrocardiographic locations of infarction. ACS were expressed as a percentage (ACS%) of the end-diastolic ventricular circumference, and the percentages obtained correlated with ejection fraction (EF) \( r = -0.838, P = 0.0001 \) using a quadratic regression equation. The group of patients \( (N = 8) \) with heart failure (paroxysmal nocturnal dyspnea and/or ventricular gallop sound) demonstrated a significantly lower mean value for EF \( (P = 0.0003) \) and a significantly larger mean value for ACS% \( (P = 0.0041) \) than the group of patients \( (N = 16) \) without heart failure. EF sharply separated the two groups. ACS% was a poor separator because in the majority of patients in both groups it was between 14 and 38%. Since EF sharply separated the heart failure and non-heart failure groups but ACS% did not, a theoretic model was developed to assess the contribution of the remaining myocardium to LV function. The curve described by the model did not differ significantly from the curve derived from the quadratic regression equation. Data from heart failure and non-heart failure patients were generally separated by a point \( (EF = 0.30, ACS\% = 23\%) \) on the theoretic equation. Abnormal function of the nonkinetic myocardium was considered to be present when observed EF was lower than predicted EF for the observed ACS%.

Thus, within the year following transmural myocardial infarction, the relative size of an abnormally contracting region of the ventricle was quantitatively related to impairment of LV function. The spherical model not only provided a framework for relating the clinical status of a patient to both ventricular function and size of the ACS, but also offered a means of estimating the function of the myocardium that appeared angiographically to be nonkinetic.

Additional Indexing Words: Dyssynergy Clinical heart failure Ejection fraction Left ventricular function

ABNORMAL regional myocardial performance has been suspected as a cause of ventricular dysfunction in man at least since 1935, when Tennant and Wiggers ligated a coronary artery in a dog and observed

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paradoxic movement of the underlying myocardium.\(^1\) Harrison\(^2\) later emphasized the importance of abnormal regional myocardial performance and used the term asynergy (later changed to dyssynergy)\(^3\) to indicate uncoordinated ventricular contraction. In 1967 Herman and associates, using single-plane cineangiography, showed that localized ventricular contraction abnormalities exist in man and suggested that they may cause heart failure.\(^4\) A theoretic model has been presented by Klein and co-workers showing that the left ventricle must undergo compensatory enlargement when 20–25% of its surface area becomes akinetic.\(^5\) To date, however, a quantitative relationship between abnormal regional myocardial performance and ventricular function has not been established in man.

Biplane angiocardiography provides a means of accurately defining ventricular wall motion. In the present study, angiocardiography was performed in patients with and without clinical heart failure in the first 2–12 months following an acute myocardial infarction. Localized contraction abnormalities were quantitated and related to left ventricular function.

**Methods**

This investigation was initiated prospectively as part of the University of Alabama Myocardial Infarction Research Unit Program to evaluate left ventricular performance in the months following an acute myocardial infarction. The investigative protocol for evaluating postinfarction patients was approved by the Human Use Committee of this institution. Informed consent was obtained according to the principles expressed in the Declaration of Helsinki as endorsed by the American Society for Clinical Investigation, Inc.\(^6\) Twenty-five patients were studied according to this protocol at the University of Alabama Medical Center during a 21-month period. Each patient had sustained a documented transmural myocardial infarction 2–12 months prior to cardiac catheterization with coronary arteriography and quantitative biplane angiocardiography. Catheterization was performed to evaluate chest pain or clinical heart failure, although three asymptomatic patients were also studied at the request of the referring physician or as part of evaluation for a relatively hazardous job.

The criteria for a diagnosis of a transmural myocardial infarction were: (1) a typical clinical history for an infarction, (2) a prompt rise and fall of the serum glutamic oxaloacetic transaminase (SGOT), and (3) evolutionary S-T-segment changes of a myocardial infarction associated with 0.03-sec or greater Q waves or Q-S complexes in at least two of the standard 12 electrocardiographic leads. Thirteen of the patients were hospitalized in the University of Alabama Myocardial Infarction Research Unit during the acute phase of infarction, but 12 patients had their infarction managed at another hospital. Hospital discharge summaries, which included serum enzyme results and copies of serial electrocardiograms, were obtained for patients who had their infarction treated at another hospital. Eight patients gave a history of previous hospitalizations for “heart attacks” in addition to their documented transmural infarction. For these patients a discharge summary and copies of electrocardiograms were obtained for each hospitalization, and the evidence for an infarction was reviewed. The minimal criteria for an additional myocardial infarction were: (1) a clinical history consistent with an infarction and (2) a prompt rise and fall of the SGOT.

Seventeen 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 dyspnea was recorded. Particular attention was directed to the presence or absence of a ventricular gallop sound during physical examination.

Coronary arteriography was performed by either the Sones' or Judkins' technic. The severest lesion in each of the four main coronary arteries (left main, left anterior descending, left circumflex, and right) was graded by the following system, based on the estimated relevant luminal diameters in at least two projections: 0 = no arteriographic abnormalities identified; 1 = irregularity or stenosis of less than 50%; 2 = 50–90% stenosis; 3 = subtotal stenosis of greater than 90%; 4 = total obstruction.

Biplane angiocardiography was performed at a filming rate of 6 or 12 frames/sec following the injection of 50–60 ml of 76% sodium and meglumine diatrizoate into the left ventricle. Ventricular volumes were determined by the Dodge area-length method.\(^7\) Films exposed during a premature ventricular contraction and the beat following were excluded from analysis. Ejection fraction was expressed as the ratio of ventricular stroke volume to end-diastolic volume.

In order to define localized segments of abnormal ventricular contraction, end-diastolic
and end-systolic outlines of the left ventricular chamber for a single beat were superimposed as follows. The left ventricular chamber and the central X-ray beam marker were outlined on the appropriate frames of the large film angiocardiograms with a soft lead pencil. An 8 x 11-in sheet of transparent paper was placed over the end-diastolic frame, with the inferior margins of the paper and the film superimposed. The chamber silhouette and the center X-ray beam marker were traced on the paper. The paper was transferred to the end-systolic frame and the central X-ray beam markers were overlapped, keeping the inferior margins of the paper and film parallel to each other, but not necessarily superimposed. The end-systolic silhouette was then traced.

Akinetic or dyskinetic segments were identified from the superimposed silhouettes. Akinesis (no movement) was considered to be present when a portion of the two silhouettes shared a common line. Dyskinesis (paradoxic movement) was considered to be present when the end-systolic silhouette extended outside the end-diastolic silhouette. The length of the end-diastolic ventricular perimeter (circumference) and the length of the akinetic or dyskinetic segment of the end-diastolic perimeter were determined with a map measurer (Eugene Dietzgen Co.). Abnormally contracting segments (akinet ic or dyskinetic) were expressed as a percentage (ACS%):

$$\text{ACS(\%)} = \frac{\text{akinetic or dyskinetic length of end-diastolic circumference}}{\text{total end-diastolic circumference}} \times 100$$

The anteroposterior and lateral values were averaged.

Figure 1 shows the composite silhouettes of a patient who sustained a transmural inferior infarction 3 months prior to catheterization. The brackets contain the segments used to calculate the average ACS%.

### Results

The clinical and laboratory data are recorded in table 1. The patients, 23 males and two females, varied in age from 30 to 66 years, with a mean age of 48 years. The mean time between the study and the onset of infarction was 5 months. Episodes of paroxysmal nocturnal dyspnea were described by seven patients following discharge from the hospital for their acute infarction. A ventricular gallop sound was audible in six patients at the time of follow-up study. Only two patients (19, 21) exhibited mild mitral regurgitation during ventriculography.

#### Electrocardiographic Location of Acute Infarction

A single infarction was documented for 17 patients: anterior in 10, and inferior in seven. For the eight patients with a history of multiple infarctions, six had sustained a transmural anterior infarction and two a transmural inferior infarction within the year of study. Additional locations of infarction in these patients were anterior, inferior, or undetermined. The exact locations of Q waves (0.03 sec or greater) or Q-S complexes at the time of infarction and the time of follow-up evaluation are shown in table 1.

#### Location and Type of Dyssynergy

Akinetic or dyskinetic segments of the left ventricle were identified in 24 of 25 patients, and were correlated with the electrocardiographic location of infarction. Segment size ranged from 2.3 to 55.4% of the end-diastolic ventricular circumference.

**Single Infarction: Inferior.** The seven patients with a single infarction, inferior by electrocardiographic location, demonstrated akinesis or dyskinesis inferiorly in both anteroposterior and lateral views. The akinetic or dyskinetic segment extended to the apical region in at least one view in six of seven patients.
### Clinical and Laboratory Data for 25 Patients

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#### Abbreviations:
- A = akinesis
- ACS = abnormally contracting segment
- D = dyskinesis
- EDP = end-diastolic pressure
- EF = ejection fraction
- EDV = end-diastolic volume
- LAD = left anterior descending artery
- LCₜ = left circumflex artery
- LM = left main coronary artery
- LV = left ventricular
- MI = myocardial infarction
- N₁ = not injected
- PND = paroxysmal nocturnal dyspnea
- R = right coronary artery
- S₁ = ventricular gallop sound
- + (under ECG-Q waves) = present during acute infarction and at time of follow-up study
- 0 (under ECG-Q waves) = present only during acute infarction

**Single Infarction:** Anterior. Akinesis or dyskinesis was demonstrated in nine of 10 patients who had a single anterior infarction. The patient without an abnormally contracting segment had sustained an anteroseptal infarction associated with S-T-segment elevation and Q waves in V₁-2. Two additional patients had an anteroseptal infarction, associated with Q waves in V₁-3. Both of these patients exhibited akinesis or dyskinesis of the anterior wall of the left ventricle.
prior projection.

Anterior wall in the lateral view.
In the remaining seven patients, electrocardiographic evidence of anterolateral infarction (Q waves in leads aVL, V_s, and various combinations of other leads) was present. All seven patients had akinesis or dyskinesis of the lateral and/or apical wall in the anteroposterior projection and of the anterior and/or apical region in the lateral projection. Two of the patients also exhibited akinesis of the inferior wall in conjunction with Q waves in the inferior electrocardiographic leads.

Segment size for anterior infarction ranged from 2.3 to 55.4% with a mean value (± standard error) of 21.0 ± 16.2%. Akinetic or dyskinetic segments for inferior infarction ranged from 11.6 to 27.9% with a mean value of 18.3 ± 5.4%. The mean value for segment size was not significantly different for the two locations (P > 0.5).
Multiple Infarctions. The location of akinetic or dyskinetic segments for patients who had multiple infarctions correlated well with the electrocardiographic sites of infarction. Details are given in table 1.

Coronary Artery Lesions

A coronary artery lesion producing from 50 to 100% stenosis was identified in every patient and its site was compared to the electrocardiographic location of infarction. The lesions described in table 1 were located in the proximal or middle one third of the given vessel, except for the left main coronary artery.

The left anterior descending artery or one of its branches was completely occluded in nine of 16 patients who had sustained an anterior infarction within 1 year of study. Among the seven patients without complete occlusion of the left anterior descending vessel, two had lesions producing more than 90% narrowing, while five had 50–90% stenosis. In none of the latter five patients was the left circumflex artery occluded, but 50–90% narrowing existed in one. Thus, 50–90% stenosis in the left anterior descending artery appeared to be the significant lesion associated with anterior infarction in five of 16 patients.

The right coronary artery was completely occluded in six of nine patients who had had an inferior infarction within 1 year of study. It was 50–90% narrowed in the other three patients. One of the latter patients had complete occlusion of the left circumflex vessel. Thus, two of nine patients demonstrated 50–90% stenosis of the right coronary artery that could be implicated as the cause of inferior infarction.

Dyssynergy and Hemodynamic Measurements

The relationship between the relative size of an akinetic or dyskinetic segment and the ejection fraction (range 0.09–0.64) is illustrated in figure 2. Segment size correlated significantly with the ejection fraction \( r = -0.838, P = 0.0001 \), using a quadratic regression equation.

Left ventricular end-diastolic pressure (EDP) (range 4–40 mm Hg) correlated significantly with the size of akinetic or dyskinetic segments by a linear function, \( \text{EDP} = 54.4 \text{(ACS/100)} + 4.11 \) \( (r = 0.697, P = 0.0003) \). Left ventricular end-diastolic volume (EDV) (range 64–331 ml/m²) exhibited a linear correlation with abnormally contracting segments, \( \text{EDV} = 285.7 \text{ (ACS/100)} + 61.9 \) \( (r = 0.651, P = 0.0008) \).

Dyssynergy and Clinical Heart Failure

The ejection fraction is shown relative to the presence or absence of clinical heart failure (paroxysmal nocturnal dyspnea and/or ventricular gallop sound) for the 24 patients who demonstrated akinesis or dyskinesis in figure 3A. The mean value for ejection fraction was significantly lower for patients with heart failure \( (0.19 \pm 0.08) \) than for the patients without heart failure \( (0.44 \pm 0.14) \).

The relationship between akinetic or dyskinetic segments and the presence or absence of heart failure is shown in figure 3B. The mean value for segment size was significantly larger for patients with heart failure \( (31.2 \pm 13.0\%) \) than for those without heart failure \( (16.0 \pm 9.7\%) \). Values are mean \( \pm \) sd.

Discussion

The finding of a highly significant correlation between the relative size of an akinetic or dyskinetic segment and the ejection fraction has broad implications. For the first time in
man, abnormal regional myocardial performance has been shown to be related quantitatively to impairment of left ventricular function. This observation supports the recent work of Pairolero and associates, who found a close and linear correlation between relative size of an akinetic segment and ejection fraction in dogs postinfarction.\(^8\) However, ejection fraction was expressed as a function of area rather than of volume.

We found a positive \(r = 0.69\) and significant correlation between the left ventricular end-diastolic pressure and the relative size of an abnormally contracting segment. Examination of the line described by the regression equation revealed that end-diastolic pressure exceeded 12 mm Hg when the akinetic or dyskinetic segment exceeded 15%. Segment size also correlated positively \(r = 0.65\) with left ventricular end-diastolic volume. The theoric spherical model developed by Klein and co-workers\(^5\) indicated that the left ventricle must undergo compensatory enlargement when 20–25% of the surface area is rendered akinetic. In general, the data in the present investigation lend support to this concept, since a 17% abnormally contracting segment corresponded to an end-diastolic volume of 110 ml/m\(^2\) on the line described by the regression equation. The upper normal value for end-diastolic volume is 110 ml/m\(^2\) (2 standard deviations above mean normal)\(^9\).

Visualization of the left ventricle in two planes is an important feature of this study. The difference between the segment size percentages in the two views was 10 or more in 10 patients. An akinetic or dyskinetic segment was detected in only one view in four patients. This variability in distribution of the
abnormally contracting segments demonstrates the importance of visualizing the left ventricle in two projections.

**Theoretic Spherical Model**

The mean value for ejection fraction was significantly lower in patients with heart failure than in those without heart failure. In addition, ejection fraction sharply separated the two patient groups. The mean value for segment size was significantly larger for the heart failure than for the non-heart failure group. Segment size, however, was a poor separator because in the majority of patients in both groups it was between 14 and 38%.

Ejection fraction is dependent on the function of the ventricular wall, which in turn can be considered to have two components, the akinetic or dyskinetic segment and the remaining myocardium. Since ejection fraction sharply separated heart failure and non-heart failure patients, segment size might have been expected to do likewise. As discussed above, segment size did not separate the two groups as well as did ejection fraction. Thus, the function of the remaining myocardium must be implicated as a factor in determining the presence or absence of heart failure. A theoretic, spherical model $EF = 0.67 \left(1 - \frac{\text{akineti}c\ \text{segment}}{100}\right)^3$ was developed to assess the contribution of the nonakinetiC myocardium to ventricular performance. Details of the derivation of the model are given in the appendix. The model provides a means of determining the ejection fraction in the presence of a given akinetic segment, assuming that the nonakinetiC end-diastolic ventricular circumference (or myocardium) is normal in function. The curve described by the model is shown in figure 4. Also illustrated is the curve derived from the regression equation that describes the relationship of an akinetic or dyskinetic segment to the ejection fraction for patients in this study. One curve did not predict ejection fraction significantly differently from the other.

The clinical states of the patients were related to the curve described by the model in figure 5. Note that the percentage of nonakinetic myocardium is plotted on the ordinate. Patients with heart failure are represented on the steep portion of the curve. The ejection fraction was less than 0.30 for seven of eight patients exhibiting heart failure; the nonakinetiC myocardium was less than 77% in six of eight. Data from patients without heart failure generally fell on the flat portion of the curve. The ejection fraction was greater than 0.30 for 15 of the 16 patients not exhibiting heart failure; the nonakinetiC or nondyskinetic myocardium was greater than 77% in 13 of 16. Thus, the point on the curve described by 0.30 ejection fraction and 77% nonakinetiC myocardium (23% akinetic segment) generally separated the heart failure and non-heart failure patients.

Since the model assumes that nonakinetic myocardium functions normally, the actual function of nonakinetic myocardium in diseased hearts may be assessed by comparing the observed ejection fraction to the predicted ejection fraction for the observed abnormally contracting segment. If the ejection fraction is lower than predicted by the model, then the nonakinetic myocardium is abnormal in function. Consider the cases shown in figure 6 which fall above the curve described by the
myocardium (20% segment) and an ejection fraction of 0.15. The predicted ejection fraction for 80% nonkinetic myocardium is 0.34, as indicated by the broken line. The composite outlines of the ventricular chamber for this patient are shown in figure 7A. Note that the nonkinetic myocardium contracts poorly and that significant disease (50% or greater stenosis) was demonstrated in each of the four main coronary arteries. This patient exhibited clinical heart failure. The patients who fall below the curve in figure 6 are designated as having normal function of the nonkinetic or nondyskinetic myocardium. The ventricular chamber silhouettes for one of these patients is shown in figure 7B. The relative segment size is approximately the same as for the patient in figure 7A, but note how well the nonkinetic myocardium contracts. This patient did not exhibit clinical heart failure.

The method in its present form provides only an estimate of the function of the angiographically apparent nonkinetic myocardium. Visualization of the ventricle in two planes may result in over- or underestimation of the true size of the abnormally contracting segment. In addition, the presence of dyskinesis may necessitate slight modification of the method. In theory, significant dyskinesis would produce greater impairment in ventricular function than would akinesis. Although dyskinesis was demonstrated in 13 patients, in most cases the amount of paradoxical motion was relatively small.

Coronary Artery Lesions: Location of Dyssynergy

The relationships found in this study between the sites of the coronary artery lesions, the electrocardiographic sites of infarction, and the locations of abnormally contracting segments are consistent with current concepts of the pathophysiology of transmural myocardial infarction. The majority of patients demonstrated complete occlusion of a vessel expected to supply the abnormally contracting segment, and a 50–90% narrowing appeared to be associated with the segment in seven of the patients. It was
interesting to note that the ejection fraction was less than 0.40 for nine of 12 patients with a single documented infarction and complete occlusion of the corresponding vessel. The ejection fraction was greater than 0.40 for four of five patients having a single infarction and a 50–90% stenosis. This suggests that the patency of a coronary artery may be related to left ventricular function. A recent autopsy study has demonstrated a similar relationship between coronary artery patency at the time of acute infarction and the presence or absence of the power-failure syndrome.\textsuperscript{11}

For patients with a single infarction, the mean value for relative sizes of abnormally contracting segments associated with anterior infarction was not significantly different from the mean value associated with inferior infarction. Anterior infarction was associated with a broader range of values. A single explanation for this variability was not apparent. The akinetic segments were surprisingly large for the seven patients with a single inferior infarction, none of whom had heart failure. Further study is needed to determine if this is a finding representative of all patients who sustain transmural inferior infarction. The relatively large size of the akinetic area and the relatively small variability resulted in the mean value for the inferior akinetic segments being just below the threshold for clinical heart failure. The addition of another infarction might easily result in the clinical heart failure state.

Finally, it should be emphasized that this investigation has dealt only with akinesis or dyskinesis detected 2–12 months following transmural myocardial infarction. The type of dyssynergy associated with nontransmural infarction is not known. Dyssynergy may also be associated with other types of heart disease.\textsuperscript{12} Nevertheless, this study has demonstrated that akinesis or dyskinesis can be detected in the year following transmural myocardial infarction, and that the relative size of the akinetic or dyskinetic segment is quantitatively related to impairment of left ventricular function.

\section*{Appendix}

by B. J. Feild and J. T. Dowling

The model for relating the size of an akinetic segment to ejection fraction assumes that the left ventricle is a sphere. The normal left ventricle more closely resembles an ellipsoid figure. However, recent evidence suggests that dilated ventricles contract more nearly as a sphere.\textsuperscript{13, 14}

For a sphere of any size, volume is equal to \(4/3\pi r^3\). The circumference (L) of the sphere in a single plane is equal to \(2\pi r\) radius. Therefore, volume equals \(4/3\pi (L/2\pi)^3\).

The myocardium of the normal left ventricle functions with an ejection fraction of 0.67, regardless of the actual end-diastolic volume.\textsuperscript{9}

Thus:

\[
\text{EF}_N = \frac{\text{SV}}{4/3\pi (L_N/2\pi)^3} = 0.67
\]

where: \(\text{EF}_N = \text{normal ejection fraction}\), \(\text{SV} = \text{stroke volume}\), and \(L_N = \text{end-diastolic circumferential length of the ventricle with normal myocardium}\).

Creation of a sharply defined akinetic (nonmoving) segment would mean that only the nonkinetic myocardium could contract and contribute to the process of ejecting volume.

Then:

\[
L_A = L_{NA} + AL
\]
where for a ventricle with akinesis at end-diastole: 
\[ L_A = \text{total circumferential length}, \ L_{NA} = \text{nonkinetic length}, \ \text{and} \ AL = \text{akinetic length}. \]

Akinetic length (AL) can be related to \( L_A \) as a percentage:

\[ AS = \frac{AL}{L_A} \times 100 \quad (3) \]

where \( AS \) = akinetic segment, which is identical to ACS as defined in the Methods section.

Rearranging equation (2):

\[ L_{NA} = L_A \left(1 - \frac{AL}{L_A}\right) \quad (4) \]

Substituting equation (3) into equation (4):

\[ L_{NA} = L_A \left(1 - \frac{AS}{100}\right) \quad (5) \]

From equation (1), the ratio of SV to \( 4/3\pi \left(L_N/2\pi\right)^3 \) equals 0.67, provided the functioning myocardium (nonkinetic) is normal. Substituting equation (5) into equation (1):

\[ \frac{SV_A}{4/3\pi \left(1 - \frac{AS}{100}\right)^3} = 0.67 \quad (6) \]

where: \( SV_A = \text{stroke volume of the ventricle with akinesis}. \)

Rearranging equation (6):

\[ \frac{SV_A}{4/3\pi (L_A/2\pi)^3} = 0.67 \left(1 - \frac{AS}{100}\right)^3 \quad (7) \]

The left-hand expression of equation (7) is equal to the ejection fraction of the akinetic ventricle. Thus:

\[ EF = 0.67 \left(1 - \frac{AS}{100}\right)^3 \quad (8) \]

where \( EF \) is the ejection fraction predicted for a ventricle with a given akinetic segment provided the nonkinetic myocardium is normal in function.

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