Quantitative Angiocardiography in Ischemic Heart Disease

The Spectrum of Abnormal Left Ventricular Function and the Role of Abnormally Contracting Segments

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SUMMARY

Appropriate surgical and medical management of the patient with ischemic heart disease depends upon a thorough assessment of the clinical and pathophysiologic derangements in left ventricular function. This study examined the spectrum of abnormalities in ventricular function found in 66 patients with documented coronary artery lesions.

Catheterization and biplane angiocardiography were used to measure end-diastolic and end-systolic volume (EDV, ESV), systolic ejection fraction (SEF), ventricular mass (LVM), end-diastolic pressure (LVEDP), peak systolic and end-diastolic stress (PSS, EDS), and stroke work (SW). The pattern of ventricular contraction was assessed for hypokinesis, akinesis, and dyskinesis and graded according to severity.

The SEF, SW, and contractile pattern were sensitive and interrelated indicators of left ventricular contractile dysfunction or fiber shortening. Measurements of ventricular filling or fiber lengthening (EDV, LVEDP, EDS) were related but less sensitive parameters of dysfunction. However, when contractile function was reduced to about one half of normal, there was an associated marked increase in EDV, EDS, and LVEDP.

A wide spectrum of derangements was found ranging from virtually normal function in 18 patients with angina alone to severe dysfunction in 18 patients with myocardial infarction, mitral regurgitation, or heart failure.

Abnormalities in ventricular function were uniformly associated with myocardial infarction. Angina alone was associated with minimal or no ventricular dysfunction. Most patients with mitral regurgitation and all patients with heart failure had severe ventricular dysfunction manifested by an increase in EDV, LVM, and PSS, a marked decrease in SEF and SW, and a severe abnormality in contractile pattern.

Additional Indexing Words:
Left ventricular function  Contractile pattern

Abnormalities in left ventricular pressure, volume, mass, and contractile function are common in patients with ischemic heart disease. The pathogenesis of these abnormalities is thought to be due to localized myocardial dysfunction resulting from coronary arterial disease. The spectrum of these abnormalities, their relationship to each other, and their clinical presentation are largely undefined. The role played by segmental

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myocardial dysfunction in the overall deterioration of cardiac function in patients with ischemic heart disease is of special concern.

Herman and co-workers\(^1\) have qualitatively characterized ventricular function from cineangiocardiograms and shown that regional and temporal abnormalities in ventricular contraction are closely related to ventricular dysfunction and heart failure. Specifically, patients with asynergy were shown to have diminished stroke volume, cardiac index, maximum rate of left ventricular pressure change, and elevated left ventricular end-diastolic pressure and volume, although there was no correlation between the latter two abnormalities. The areas of asynergy were found to be closely related to sites of occlusive coronary arterial lesions. They postulated that, when akinesia involves more than 25\% of the left ventricular surface, the physiologic limits of contractile-element shortening are exceeded, and ventricular dilatation or depressed stroke volume occurs.

Rackley et al.\(^2\) and Baxley et al.\(^3\) studied patients with coronary artery disease, with biplane quantitative angiocardiography, most of whom had clinically suspected mitral regurgitation and/or heart failure. They noted an elevated left ventricular volume and mass in this group as a whole. Left ventricular end-diastolic volumes (EDV) of greater than 154 cc/m\(^2\) were found predominantly in patients with mitral regurgitation. Systolic ejection fraction (SEF = stroke volume/EDV) was decreased and EDV was increased in virtually all patients with clinical congestive heart failure.

Bristow et al.\(^4\) studied a selected group of patients with ischemic heart disease and relatively normal EDV as determined by single-plane cineangiocardiography. They noted that some patients had reduced ejection fractions and elevated end-diastolic pressures in spite of a normal end-diastolic volume and that mean systolic ejection fraction was significantly reduced for the entire group.

Falsetti et al.\(^5\) studied 75 patients with coronary artery disease with single-plane cineangiocardiography and found that end-diastolic pressure and volume were elevated and SEF was reduced in all patients with clinical heart failure. Close relationships between end-diastolic pressure and volume or between end-diastolic pressure and ejection fraction were not noted, although an elevated end-diastolic volume was associated with a reduced ejection fraction.

The present study was undertaken to define the spectrum of ventricular functional abnormalities found in a group of patients with documented coronary artery lesions. Measurements of left ventricular volumes, mass, pressure, wall stress, stroke work, and cardiac

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**Figure 1**

A normal contraction pattern. The end-systolic (ES) contour has been superimposed on the end-diastolic (ED) contour in the anteroposterior (AP) and lateral planes. The long axis of the ventricle from the aortic (Ao) valve to the apex and its bisecting perpendicular were used as reference points.
output have been made. The relationship of these determinations to each other, to the ventricular contractile pattern, and to the clinical manifestations of disease has been examined.

**Methods**

Sixty-six patients with clinical evidence of ischemic heart disease manifested by angina pectoris, myocardial infarction, congestive heart failure, or a combination of these were studied by selective coronary arteriography using the Judkins technique and left ventricular biplane angiocardiography at filming rates of 6 or 12 frames/sec. In all cases, the angiocardiogram was performed prior to coronary arteriography by injection of 0.75–1.0 cc/kg of iodinated contrast material into the left ventricle so that the presence or absence of mitral regurgitation (MR) could be ascertained. Sequential left ventricular volumes were determined from the angiocardiograms by the length-area method and a volume-time curve constructed. Wall thickness was added to the chamber dimensions of the diastolic films to estimate left ventricular mass (LVM). End-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), and systolic ejection fraction (SEF) were determined from the volume-time curve. Left ventricular pressure, oxygen consumption, and arteriovenous oxygen difference (A-V\(O_2\)D) were determined immediately prior to the angiocardiogram. Regurgitant

**Localized Akinesia or Hypokinesia (III)**

![Borderline Abnormal Contraction (II)](image1)

*Figure 2*

The contraction pattern shows a small area of diminished contraction on the inferior surface in the AP plane.

*Figure 3*

The contraction pattern illustrates absence of contraction (akinesis) involving the apex in the AP plane and the anterior wall in the lateral plane.
flow was quantitated by subtracting the Fick cardiac output (FCO) from the angiographic cardiac output (ACO).

Sequential left ventricular mean circumferential wall stress was calculated from the angiographic and pressure tracings using the thin-walled ellipsoidal model of Sandler and Dodge.10 This method is known to induce a systemic error of 10–15%, but is more easily used than thick-wall models.11 Peak systolic stress (PSS) and end-diastolic stress (EDS) were recorded. Left ventricular stroke work (SW) was calculated by integration of the area enclosed by the pressure-volume loop and expressed as g·m/m² and g·m/100 g of left ventricular mass.12

Cases were excluded when arrhythmia or poor-quality films interfered with the accurate determination of ventricular volumes. Cases with large ventricular aneurysms which distorted the shape of the ventricle were also excluded because they invalidate the method of left ventricular volume measurement. (This eliminated about 15% of cases otherwise suitable for study.) In the eight cases in which the lateral ventricular wall was involved by infarction with or without endocardial thrombus, no attempt was made to measure wall thickness for the calculation of LVM or wall stress.

The pattern of left ventricular contraction was assessed in both planes by an adaptation of the method of Herman et al.1 The long axis of the ventricle from midaortic valve to the apex and a bisecting perpendicular were constructed on both the end-systolic and end-diastolic films in the anteroposterior and lateral planes. The end-systolic film was then superimposed on the end-diastolic film with the long axis and its

**Figure 4**

*Outward motion (dyskinesis) of the apical surface is shown in both the PA and lateral planes.*

**Figure 5**

*The entire ventricle demonstrates decreased (hypokinesis) or absent (akinesis) contraction.*

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perpendicular as reference points (figs. 1–5). In some cases, the posterior aortic valve ring is better visualized on the lateral film than is the aortic valve, and in these cases the long axis was constructed from this landmark to the apex. In theory this method corrects the apparent wall motion due to movement of the entire heart and allows assessment of actual contractile motion toward the geometric center of the ventricle. Abnormalities were graded as follows:

I. Normal. The entire ventricular wall moves inward appropriately toward the geometric center of the ventricle. (fig. 1). Occasional patients with small volumes and normal ejection fractions have asymmetric-appearing ventricles at end-systole. This is due to distortion of the cavity shape by the papillary muscles, and these cases have been included in this category if the long-axis shortening of the ventricle was normal.

II. Borderline Abnormal. These cases demonstrated a minor degree of contraction asymmetry involving less than 25% of the ventricular surface (fig. 2).

III. Localized Akinesis or Hypokinesis. Greater than 25%, but less than 75%, of the ventricular surface showed diminished or no contraction (fig. 3).

IV. Localized Dyskinesis. Greater than 25% of the ventricular surface demonstrated paradoxical outward motion during systole (fig. 4).

V. Diffuse Akinesis or Hypokinesis. Greater than 75% of the ventricular surface showed diminished or absent contraction (fig. 5).

The presence of congestive heart failure (CHF) was based on definite historical, physical, or X-ray evidence of fluid overload either peripherally or in the lungs. Myocardial infarction was judged to be definite if the ECG showed diagnostic Q waves and probable if there was a documented history of myocardial infarction without diagnostic ECG abnormality. The diagnosis of angina pectoris was based on a history of typical exertional chest pain confirmed in most cases by exercise testing with reproduction of pain and ECG changes. None of the patients had severe hypertension (diastolic consistently greater than 110 mm Hg). Hypertension was judged to be present if diastolic pressure exceeded 100 mm Hg on several temporally isolated measurements. Patients who fulfilled the latter criteria were classed as hypertensive even if the pressures prior to or during catheterization were normal.

Normal hemodynamic and angiographic values used in this study were based on normal values previously reported from this laboratory and others using identical methods.

Results

The clinical findings for the entire study

<table>
<thead>
<tr>
<th>Clinical Findings</th>
<th>Mitral regurgitation (% of CHF)</th>
<th>Myocardial infarction (%)</th>
<th>Occasional</th>
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<tr>
<td>Group</td>
<td>Age (yr)</td>
<td>Angina (%)</td>
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<td>Entire group</td>
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<tr>
<td>I. Normal</td>
<td>66</td>
<td>47 ± 11</td>
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<td>II. Borderline Abnormal</td>
<td>16</td>
<td>47 ± 5</td>
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<tr>
<td>III. Localized Akinesis or Hypokinesis</td>
<td>14</td>
<td>46 ± 10</td>
<td>0</td>
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<td>IV. Localized Dyskinesis</td>
<td>11</td>
<td>48 ± 6</td>
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<td>V. Diffuse Akinesis or Hypokinesis</td>
<td>8</td>
<td>52 ± 7</td>
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<tr>
<td>No MI</td>
<td>22</td>
<td>45 ± 9</td>
<td>45 ± 4</td>
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<tr>
<td>Probable or definite MI</td>
<td>44</td>
<td>48 ± 8</td>
<td>50 ± 5</td>
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Catheterization and Angiographic Data

<table>
<thead>
<tr>
<th>Group</th>
<th>EDV (ml/m²)</th>
<th>ESV (ml/m²)</th>
<th>SV (ml/m²)</th>
<th>SEF (%)</th>
<th>LVEDP (mm Hg)</th>
<th>LVM (g/m²)</th>
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<tbody>
<tr>
<td>Normal</td>
<td>70 ± 20</td>
<td>24 ± 10</td>
<td>45 ± 12</td>
<td>67 ± 8</td>
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<td>92 ± 16</td>
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<td>Entire group</td>
<td>41 ± 316</td>
<td>8 ± 282</td>
<td>20 ± 74</td>
<td>11 ± 80</td>
<td>3 ± 45</td>
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<td>Divided on basis of contraction pattern:</td>
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<tr>
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<td>41 - 91</td>
<td>8 - 37</td>
<td>33 - 62</td>
<td>52 - 80</td>
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<td>62 - 118</td>
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<tr>
<td>II. Bord abnormal</td>
<td>69 ± 14</td>
<td>23 ± 9</td>
<td>45 ± 10</td>
<td>67 ± 10</td>
<td>9 ± 4</td>
<td>91 ± 16</td>
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<tr>
<td>III. Local hypokinesia or akinesia</td>
<td>59 - 109</td>
<td>23 - 50</td>
<td>32 - 74</td>
<td>48 - 74</td>
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<td>65 - 141</td>
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<td>IV. Local dyskinesia</td>
<td>82 ± 15</td>
<td>33 ± 8</td>
<td>49 ± 12</td>
<td>60 ± 8</td>
<td>11 ± 4</td>
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<td>V. Diffuse akinesia or hypokinesia</td>
<td>48 - 160</td>
<td>23 - 129</td>
<td>25 - 47</td>
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<td>4 ± 35</td>
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<td>102 ± 29</td>
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<td>165 ± 29</td>
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<td>181 ± 7</td>
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<tr>
<td>No MI</td>
<td>41 - 107</td>
<td>8 - 50</td>
<td>33 - 65</td>
<td>48 - 80</td>
<td>5 ± 15</td>
<td>62 - 123</td>
</tr>
<tr>
<td>Probable or definite MI</td>
<td>78 ± 16</td>
<td>29 ± 11</td>
<td>48 ± 10</td>
<td>64 ± 10</td>
<td>10 ± 3</td>
<td>93 ± 16</td>
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<tr>
<td></td>
<td>126 ± 65</td>
<td>87 ± 66</td>
<td>38 ± 10</td>
<td>38 ± 18</td>
<td>16 ± 11</td>
<td>126 ± 41</td>
</tr>
</tbody>
</table>

Abbreviations: EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; SEF = systolic ejection fraction; LVEDP = left ventricular end-diastolic pressure; LVM = left ventricular mass; CWT = corrected wall thickness; A-Vo.D = arteriovenous oxygen difference; ACI = angio cardiac index; FCI = Fick cardiac index; PSS = peak systolic stress; EDS = end-diastolic stress; and SW = stroke work.

Group are recorded in table 1. The group has also been divided on the basis of the ventricular contraction pattern and the presence or absence of myocardial infarction. Sixteen patients had a normal pattern of contraction (group I), 17 had borderline abnormal contraction (group II), and 33 had definitely abnormal contraction patterns (14 group III, 11 group IV, eight group V). There were no significant differences (P = < 0.01) in age, duration of symptoms, incidence of angina, or hypertension between the five contraction groups, although patients in contraction groups IV and V tended to be older and have a longer duration of symptoms. The percentage of patients with congestive heart failure and myocardial infarction shows a striking increase with progressive abnormalities in contraction, while the percentage of patients with mitral regurgitation was increased predominantly in those patients with diffuse akinesia or hypokinesia (group V).

Hemodynamic and angiocardiographic data are presented in table 2. Patients with ischemic heart disease and normal symmetrically contracting ventricles (group I) demonstrated no significant differences from patients without left ventricular disease in any of the variables measured. Patients with borderline abnormal contraction (group II) showed significant (P = < 0.01) increases in EDV, ESV, LVM, and LVEDP, and decreased SEF, but SV, SW, PSS, EDS, A-Vo.D, angiographic cardiac index (ACI), and Fick cardiac index (FCI) were not significantly different from normal. Patients with definitely abnormal contraction (groups III, IV, and V) show progressive abnormalities in all variables measured except wall thickness.

Abnormalities in contractile pattern were associated with a decrease in SEF. All patients in groups IV and V and most patients (11 of 14) in group III had an SEF of less than 50% (the three exceptions were all 53%), while virtually all patients (32 of 33) in groups I

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and II had an SEF of greater than 50% (Fig. 6).

Left ventricular mass/m² was significantly increased ($P < 0.01$) for the entire group and demonstrated a progressive increase from group I through group V. This increase in LVM was due primarily to an increase in EDV without an associated increase in wall thickness. All patients with LVM/m² of greater than 120 g had a definite contraction abnormality.

Abnormally elevated left ventricular end-diastolic pressures were found in 41% (27 of 66). An increasing prevalence was found with progressive contractile abnormalities: 12.5% (two of 16) of group I, 35% (six of 17) of group II, 21% (three of 14) of group III, 82% (nine of 11) of group IV, and 88% (seven of eight) of group V. Of the group judged to have definitely abnormal contraction (groups III, IV, and V), 68% (19 of 33) had an elevation of LVEDP.

Although PSS and EDS were significantly increased in patients with definite contraction abnormalities (groups III, IV, V), there was considerable overlap among the five groups.

The progressive decrease in SW with increasing contraction abnormality was quite striking. Because of the associated increase in left ventricular mass, the decrease in SW was more pronounced when expressed in terms of work/g of LVM.

Although stroke volume, A-Vo₂D, FCI, and ACI demonstrated increasing abnormalities with increasing ventricular dysfunction, the magnitude of change was less, and the differences between the various contractile groups were much less marked.

Clinical Relationships

Angina-Ventricular Function. Eighty-six percent (57 of 66) of the patients studied manifested angina. The presence of angina without prior myocardial infarction, mitral regurgitation, or heart failure was usually predictive of normal or near normal resting volume, mass, SEF, contraction pattern, wall stress, and stroke work.

Myocardial Infarction-Ventricular Function. Sixty-seven percent (44 of 66) of the total group had a definite (33) or probable (11) myocardial infarction. As noted in Table I, all patients with markedly abnormal ventricular contraction (groups IV and V) had...
sustained a definite myocardial infarction, and 92% of patients with localized akinesis or hypokinesis (group III) had had probable or definite infarction. Eighteen percent of patients with normal ventricular contraction (group I) and 54% of patients with borderline abnormal ventricular contraction (group II) had sustained probable or definite infarction.

Clinical, hemodynamic, and angiographic data for patients with and without myocardial infarction are presented in tables 1 and 2. Patients without myocardial infarction show virtually no abnormalities compared to the normal control group, except for minimal elevation of EDV and ESV. In contrast, patients with myocardial infarction show definite abnormalities in all parameters measured except for wall thickness. There are, however, wide ranges for the latter group, and some patients clearly maintain normal volume, pressure, contraction pattern, wall stress, SW, and cardiac output following infarction.

Mitral Regurgitation-Ventricular Function. Fifteen percent (10 of 66) of the total group demonstrated mitral regurgitation at the time of study. None of this group demonstrated more than mild mitral regurgitation (1-3 liters/min). As a whole, patients with mitral regurgitation demonstrated markedly abnormal ventricular function with EDV of 177 ± 68 cc, SEF of 27 ± 17%, and LVEDP of 26 ± 10 mm Hg, and 90% had definitely abnormal ventricular contraction (groups III, IV, V). Eight of 10 patients with mitral regurgitation also had CHF and fell into the

Figure 6
The relationship between systolic ejection fraction (SEF), end-diastolic volume (EDV), and contraction pattern. Patients with depression of SEF below 40% demonstrated a consistent elevation of EDV and a high incidence of heart failure. CHF = congestive heart failure.
The relationship of end-diastolic volume (EDV), left ventricular end-diastolic pressure (LVEDP), and contraction pattern. The dashed lines indicate the upper limits of normal for pressure and volume. Three patients with CHF had a normal LVEDP, but none had a normal EDV. While patients with only minimal elevation of EDV and LVEDP (<100 cc and <15 mm Hg) demonstrated no consistent relationship of EDV and LVEDP, there was a definite relationship when the entire spectrum of disease was considered. CHF = congestive heart failure.

Congestive Heart Failure—Ventricular Function. Twenty patients (30%) had definite CHF, and all of these had definite abnormalities in ventricular contraction pattern (groups III, IV, V). As illustrated in figures 6–8, all patients with CHF had an EDV of greater than 90 cc/m² and an SEF below 40%. Three patients with CHF demonstrated a normal LVEDP in spite of the elevated EDV and reduced SEF. Only three patients with an SEF of less than 40% did not manifest clinical CHF, and only four patients with an EDV of greater than 110 cc/m² did not have CHF. The most consistent abnormality demonstrated in patients with CHF was a marked decrease in SW to less than 0.3 g-m/g (fig. 8). Sixty-one percent (20 of 33) of patients with definitely abnormal ventricular contraction (groups III, IV, V) had CHF. Of the 20 patients with CHF, 19 had sustained myocardial infarction with a diagnostically abnormal ECG, and eight manifested mitral regurgitation.

Hemodynamic-Angiocardiographic Relationships

The interrelationships between the various hemodynamic and angiographic variables are
The relationship between stroke work (SW), systolic ejection fraction (SEF), and contraction pattern. The combination of an SEF below 40% and an SW below 0.30 g-m/g was unique to patients with CHF. The dotted lines delineate patients with marked abnormalities (lower left-hand corner) and patients with clearly normal values (upper right-hand corner). CHF = congestive heart failure.

Useful in understanding the pathophysiologic changes in ischemic heart disease. Both linear and curvilinear relationships were present.

Linear relationships were present between EDV and LVEDP ($r = 0.74$, $P < 0.01$; fig. 7) between EDV and EDS ($r = 0.72$, $P < 0.01$) and between LVEDP and EDS ($r = 0.95$, $P < 0.01$). Thus, these three parameters of ventricular filling or fiber lengthening (volume, pressure, and wall stress) showed consistent relationships in the population of patients with ischemic heart disease. Similarly, SEF and SW were related parameters of ventricular contraction or fiber shortening ($r = 0.79$, $P < 0.01$) as shown in figure 8. The relationship of contraction pattern to SEF and to SW is also illustrated and was clearly related to both SEF and SW.

Although there is considerable scatter of points about the line of best fit, several relationships are obvious. The combination of an SEF less than 40% and SW of less than 0.30 g-m/g is unique to the group of patients with CHF and abnormal ventricular contraction pattern. Conversely, patients with a clearly normal SEF and SW have normal or only borderline abnormal contraction patterns. Patients with a definite localized contraction abnormality (group III) demonstrated a wide range of ventricular function dependent upon the degree of contraction abnormality.

Less striking linear relationships were also noted between SEF and A-V O$$_2$$D ($r = -0.58$); A-V O$$_2$$D and LVEDP ($r = 0.55$); A-V O$$_2$$D and EDV ($r = 0.42$); FCI and SEF ($r = 0.46$); and between FCI and LVEDP ($r = 0.51$). In
spite of the low correlation coefficients, the following relationships were evident. Patients with a normal SEF all had a normal A-VO₂D (<50 cc/liter). The opposite was not true, and patients with normal A-VO₂D had SEF ranging from 10 to 76%. Patients with an EDV of less than 100 cc/m² all had a normal A-VO₂D. Some patients with increased EDV and LVEDP and reduced SEF had a normal A-VO₂D at rest. Thus, while measures of resting cardiac output (A-VO₂D, FCI) were normal in patients otherwise judged to have normal ventricular function, they did not show consistent changes with deterioration of ventricular function and remained normal in some patients with clearly depressed ventricular function.

When parameters of left ventricular contraction (SEF, SW, contraction pattern) were compared to parameters of left ventricular filling (LVEDP, EDV, EDS), a curvilinear relationship similar to a Starling ventricular function curve was apparent (figs. 6, 9–11). When the SEF was above 40% and the SW was above 0.30 g-m/g, there appeared to be random variations in LVEDP, EDV, and EDS. On the other hand, when the SEF and SW fell below 40% and 0.30 g-m/g, respectively, there were striking increases in LVEDP, EDV, and EDS. All but one of 43 patients with an SEF above 40% had an LVEDP below 15 mm Hg. Conversely, when the SEF was below 40%, the LVEDP was usually elevated (18 of 23).

The relationship of contraction pattern to these parameters of ventricular function and the presence or absence of heart failure are
also illustrated in figures 6–11. In all of these relationships it is evident that normal contraction patterns were associated with normal ventricular function and an absence of CHF, while markedly abnormal contraction patterns (groups IV, V) were associated with marked abnormalities in ventricular function and CHF. Patients with localized akinesis or hypokineses (group III) demonstrated considerably more variability in degree of ventricular function impairment than did patients in contraction groups I and II or groups IV and V.

Discussion

The 66 patients presented were studied for the evaluation of ischemic heart disease manifested by angina, myocardial infarction, or congestive heart failure. Criteria for inclusion in the study were based on proven coronary occlusive disease plus the availability of high-quality hemodynamic and angiographic data. Patients with large ventricular aneurysms were excluded because of the difficulties in the angiographic evaluation of ventricular function in this group. No additional attempts were made to select patients with any particular clinical, hemodynamic, or angiographic abnormality. Despite these factors, there is a wide spectrum of ventricular function abnormalities in this relatively small group of patients.

The significant observations that can be made in this study are the interrelationships of clinical, hemodynamic, and angiographic data. Clinical manifestations of ischemic heart disease were associated with fairly

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specific hemodynamic and angiographic patterns. Angina pectoris alone without a prior history of congestive heart failure or myocardial infarction was nearly always associated with normal resting hemodynamic and angiographic findings. While not all patients with a myocardial infarction demonstrated abnormalities in ventricular function, virtually all patients with abnormal ventricular function had sustained prior myocardial infarction. Clinical congestive heart failure was uniformly associated with marked abnormalities in SEF, SW, and contraction pattern. Indeed, when both SEF and SW were markedly abnormal, the ventricular contraction pattern was also abnormal, and CHF was always present. CHF was not, however, always associated with abnormalities in LVEDP or EDS at the time of study. While EDV was elevated in all patients with CHF, this increase was slight in some patients, and other patients with similar elevations of EDV did not manifest CHF.

Mitrail regurgitation was noted most commonly in the group of patients with severe ventricular function abnormalities and associated CHF. However, in contrast to the study of Rackley et al.,\textsuperscript{2} elevations of EDV greater than 154 cc/m\textsuperscript{2} were not uniformly associated with mitral regurgitation. Since most of their patients had systolic murmurs, it is likely that patient selection accounts for this difference in that some of their patients were studies specifically for evaluation of mitral regurgitation.

Of the various parameters of ventricular function observed, EDV, LVEDP, EDS, SEF, SW, and contraction pattern show progressive
and interrelated abnormalities as ventricular function deteriorated. On the other hand, parameters of cardiac output at rest (A-V_{0.2}, D, ACO, FCO), while normal in patients with good ventricular function, did not show consistent abnormalities with deterioration of ventricular function. They were, thus, much less sensitive parameters of ventricular performance.

In contrast to the studies of Bristow and Falsetti, which included mainly patients with normal ventricular function, and those of Rackley and Baxley, which included mainly patients with abnormal ventricular function, when the entire spectrum of ischemic disease was considered, there was definite relationship between LVEDP and EDV as parameters of ventricular filling or fiber lengthening. EDS combines both end-diastolic pressure, volume, and wall thickness in a single term. While this term should theoretically be the best indicator of ventricular preload, in the ischemic population studied here, EDS was not greatly different from LVEDP. It is uncertain whether this calculation has any practical value.

Although Herman et al. did not measure SEF or SW in their single-plane cine study of ventricular contraction, the close relationship of contractile pattern abnormalities and ventricular function abnormalities which they demonstrated is confirmed by this study. Indeed, the relationship of contraction pattern, SEF, and SW is striking (fig. 8) and suggests that these are related parameters of ventricular contraction or fiber shortening.

For years emphasis has been placed upon the relationship between diastolic fiber lengthening (EDV, LVEDP, EDS) and systolic fiber shortening (stroke volume, SEF, SW). Although the dependent relationship of systolic and diastolic events can be easily demonstrated in animal experiments and acute experiments in man, this relationship has generally been obscure when applied to a population of patients with a particular cardiac disease. Earlier studies from this laboratory showed the absence of such correlation in patients with aortic and mitral valve disease. In patients without valvular heart disease, however, a better relationship might be expected because stroke volume or stroke work are determined primarily by the functional status of the left ventricle rather than the relative severity of the valvular abnormality. The studies reported here have reaffirmed this relationship for a population of patients. Comparisons of parameters of systolic contraction (SEF, SW, contraction pattern) and diastolic filling (LVEDP, EDV, EDS) demonstrate a definite curvilinear relationship. The correlation of systolic and diastolic events is not closely related and does not allow prediction of systolic function from diastolic function or vice versa. However, measurement of several systolic and diastolic parameters in a single patient allows a reasonably good assessment of ventricular function when compared to the curvilinear ventricular function curve of a population of patients. In addition, the curvilinear function curve of the ischemic population allows a better understanding of how ventricular function deteriorates with progressive ischemic disease and provides a framework for evaluation of changes in ventricular function due to medical or surgical interventions. This curvilinear relationship between parameters of ventricular filling and contraction is not unique to this series of patients. In fact, if the data on EDV and SEF from Bristow, Falsetti, Rackley, and Baxley is combined, the relationship is virtually identical to that illustrated in figure 6.

Viewing the entire spectrum of ventricular function abnormalities in ischemic disease, the following postulates would account for the observed data. Patients with ischemic heart disease manifested by angina alone have normal or only slightly impaired ventricular function. Myocardial infarction leads to local contraction abnormalities with diminished SEF, SW, and stroke volume. In patients with borderline contraction abnormalities (group II) or small hypokinetic or akinetic areas (group III), a compensatory increase in EDV (most cases) or LVEDP (few cases) allows maintenance of a normal stroke volume with a moderate decrease in SEF. With contraction abnormalities of greater magnitude (some
group III and all IV and V), the SEF drops below about 40% and, in spite of inordinate increases in EDV, LVEDP, and EDS, stroke volume and stroke work decrease with subsequent congestive heart failure. This overall relationship is illustrated in figure 12.

In addition to progressive contractile abnormalities, the changes in left ventricular mass are unique in patients with ischemic disease. In valvular heart disease, volume and wall thickness increase in a manner which tends to maintain relatively normal left ventricular wall stresses. In ischemic disease, wall thickness does not increase with corresponding increases in volume, and wall stresses increase inordinately. This may be due to the limitation of coronary blood flow. Additionally, the increase in wall stresses lead to increased myocardial oxygen demand, which may explain the deleterious effects of ventricular enlargement in patients with ischemic heart disease.

The role of local abnormalities in left ventricular contraction pattern deserves particular emphasis. With a relatively simple method of grading severity, it was possible to

![Figure 12](http://circ.ahajournals.org/)

**Figure 12**

The idealized relationship demonstrated between the parameters of ventricular contraction (SEF, SW) and parameters of ventricular filling (EDV, LVEDP, EDS). When SEF and SW were below about 50% of normal, there were associated increases in EDV, LVEDP, and EDS. When SEF and SW were above this level, there was little relationship between the parameters of ventricular filling and contraction. The areas of the idealized curves associated with various contraction patterns (I, II, III, IV, and V) are also noted. The general relationship of ventricular function to myocardial infarction is illustrated. CHF = congestive heart failure; EDS = end-diastolic stress; EDV = end-diastolic volume; LVEDP = left ventricular end-diastolic pressure; SEF = systolic ejection fraction; and SW = stroke work.
define groups of patients with normal, mildly abnormal, and severely abnormal ventricular function nearly as well as with the more laborious calculations of EDV, ESV, and SEF. Since contraction abnormalities can be obtained easily by cineangiographic methods, it is readily available to the clinical cardiologist. Although these studies were done with biplane contraction analysis, Herman et al.1 demonstrated that the majority of contraction abnormalities can be visualized by single-plane analysis.

References
Quantitative Angiocardiography in Ischemic Heart Disease: The Spectrum of Abnormal Left Ventricular Function and the Role of Abnormally Contracting Segments

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