Left Ventricular Function and Coronary Artery Anatomy Before and After Myocardial Infarction

A Study of Six Cases

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SUMMARY

Six patients underwent cardiac catheterization before and after occurrence of a myocardial infarction. Results from the two procedures allowed the quantitation of changes in coronary artery anatomy and left ventricular performance associated with myocardial infarction.

Left ventricular biplane or single plane angiography and selective coronary angiography were used to evaluate coronary artery anatomy, left ventricular end diastolic pressure (LVEDP), left ventricular end diastolic volume (LVEDV), end systolic volume (LVESV), and systolic ejection fraction (SEF) under resting conditions.

Four patients had developed occlusion of the artery supplying the area of infarction. In five cases new or progressive contraction abnormalities occurred. One patient had no change in contraction pattern or SEF. Systolic ejection fraction fell in three patients, with no change in LVEDV. In two patients LVEDV rose and SEF fell.

These data demonstrate that a wide spectrum of functional abnormalities is associated with myocardial infarction. Infarction was always associated with significant coronary artery stenosis, but not necessarily associated with occlusion. The SEF and contractile pattern were the indicators of left ventricular dysfunction which most frequently deteriorated.

Additional Indexing Words:
Atherosclerotic heart disease
Coronary arteriography
Left ventricular performance
Myocardial infarction

Recentely Six patients underwent selective coronary arteriography, quantitative left ventricular angiography and maximal exercise testing before and after progression of disease to myocardial infarction.

The purpose of this report is first to demonstrate in a quantitative manner the deterioration in left ventricular performance associated with a single myocardial infarction. The spectrum of abnormalities in left ventricular function in patients with coronary artery disease and the contribution of infarction have been described by Hamilton, Murray and Kennedy.1 In that paper the following hypotheses were made: 1) Transmural myocardial infarction is usually accompanied by a change in contractile pattern and/or by a decrease in systolic ejection fraction (SEF). 2) Infarction-induced decreases in SEF are accompanied by variable changes in end diastolic volume (EDV) and left ventricular end diastolic pressure (LVEDP) when the SEF remains above 40%. 3) When the SEF falls below 40% there is a consistent increase in EDV and LVEDP with clinical manifestations of congestive heart failure. These proposals were made on the basis of cross-sectional data, but paired observations made before and after infarction would be required to substantiate these theses. This paper provides such observations.

Our second purpose is the description of angiographic coronary arterial anatomy predisposing to infarction and the changes in that anatomy associated with infarction. Third, our study facilitates correlations between the changes in angiographic coronary arterial anatomy and left
Comparison of Diagnostic Data of Six Patients Before and Following a Single Myocardial Infarction

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*See Methods section.

Abbreviations: Pre = premyocardial infarction; Post = postmyocardial infarction; ECG = electrocardiogram; WNL = within normal limits; AMI = anterior myocardial infarction; LVH = left ventricular hypertrophy; DMI = diaphragmatic myocardial infarction; LVI = left ventricular ischemia; LAD = percent reduction in lumen diameter of the left anterior descending coronary artery; LC = percent reduction in diameter of the right coronary artery; RCA = percent reduction in diameter of the circumflex coronary artery; EDF = ejection fraction; EDV = end-diastolic volume; SV = stroke volume; LVEDP = left ventricular end-diastolic pressure; FAI = functional aerobic impairment; CHF = congestive heart failure.

Myocardial infarction was documented by typical clinical history and the development of characteristic Q wave changes or evolution of enzyme changes.

Maximal exercise testing was performed on a treadmill until the patient became limited by fatigue, dyspnea, weakness, or chest pain. Exercise performance was evaluated by duration, ST segment change, and heart rate response. Duration of exercise was compared with normal men of the same age and the limitation reported as functional aerobic impairment in per cent (FAI). Left ventricular volumes and mass were determined by the method of Dodge et al. and Rackley et al.

Left ventricular contraction was assessed for symmetry and degree of contraction by superimposing the end systolic film on the end diastolic film. The long axis of the ventricle from the aortic valve to the apex and a bisecting perpendicular were constructed on both the end-systolic and end-diastolic films in the anteroposterior and lateral plane. The end-systolic film was then superimposed on the end-diastolic film with the long axis and its perpendicular as reference points. In the lateral film the long axis was drawn through the posterior aortic valve ring as a landmark. In the anteroposterior film the long axis was drawn through the midaortic valve. These plots were graded in the following manner: I, normal symmetrical contraction; II, borderline abnormal; III localized hypokinesis or akinesia involving more than 25% of the ventricular surface; IV, local dyskinesia involving greater than 25% of the ventricular surface; V, diffuse hypokinesis or akinesia involving greater than 75% of ventricular surface.

Case Reports

Case 1. A 52-year-old construction worker was admitted with a 12-year history of angina pectoris without documented myocardial infarction. Physical examination, electrocardiogram and chest X-ray were normal. Treadmill testing terminated by typical angina
revealed a FAI of 46% without ST segment changes. LVEDV (79 cc/m²), LVEDP (12 mm Hg) and SEF (55%) were normal (table 1). Selective coronary angiography demonstrated an 80% stenosis of the left anterior descending coronary artery (LAD).

Twenty-two months after study, the patient suffered an antero-septal myocardial infarction complicated by congestive heart failure and arrhythmia. Because of recurrent congestive heart failure over the next six months, the patient was restudied. Physical examination revealed cardiac enlargement and an atrial gallop. The electrocardiogram demonstrated an antero-septal myocardial infarction. The treadmill test now showed an FAI of 65% and limitation by dyspnea. Catheterization and angiography demonstrated complete occlusion of the LAD, elevated EDV (123 cc/m²) and LVEDP (25 mm Hg) with a depressed stroke volume (SV) (26 cc/m²) and SEF (21%). A grade III contraction abnormality was present over the anterior left ventricle.

**Case 2.** A 39-year-old commercial diver was admitted with a three-month history of angina pectoris without documented myocardial infarction. His cardiac examination was normal. Arterial pulses were diminished in the lower extremities. An electrocardiogram revealed left ventricular hypertrophy. The chest film was normal. During the treadmill test he was limited by claudication of the lower extremities. Selective coronary arteriography demonstrated an 80% stenosis of the right coronary artery (RCA) and a 90% stenosis of the LAD. EDV (83 cc/m²), LVEDP (8 mm Hg), SV (44 cc/m²) and SEF (53%) were normal. A grade II contraction abnormality was present on the inferior wall.

Six months later, while on medical therapy, he sustained an antero-septal myocardial infarction followed by persistent angina pectoris and congestive heart failure. Restudy four months postinfarction revealed a normal cardiac examination and chest X-ray. The electrocardiogram showed an antero-septal infarction. Selective coronary angiography demonstrated no change in the appearance of either the right or left coronary arteries. EDV (88 cc/m²) was slightly higher. End systolic volume (ESV) (38 cc/m²) and SEF (43%) were both lower. The contraction abnormality had enlarged to involve the anterior wall.

**Case 3.** A 46-year-old fisherman was admitted with a three-month history of angina pectoris. On a previous admission he had had a rise in SGOT and precordial T wave changes suggesting an intramural myocardial infarction. Cardiac examination revealed an apical systolic murmur. An electrocardiogram showed precordial peaking of the T waves. A chest X-ray was normal. Treadmill testing demonstrated an FAI of 30%. He was limited by pain, but had no ST segment depression.

Cardiac catheterization demonstrated a normal LAD, a severely diseased circumflex artery (LC), normal RCA, and normal ventriculogram. During catheterization, the patient sustained an antero-septal infarction with shock associated with dissection of the main left coronary artery. Persistent hypotension one and a half hours following infarction necessitated the institution of full cardiopulmonary bypass. Three hours following infarction, the left anterior descending vein bypass was completed.

Re-evaluation five months later revealed mild angina pectoris and dyspnea on exertion while on digoxin and furosemide. An abnormal precordial impulse was present. Electrocardiogram showed an antero-septal myocardial infarction. The heart was enlarged on X-ray. FAI was now 65%. He was limited by dyspnea. Selective coronary angiography demonstrated an occluded LAD, and open LAD vein bypass and an occluded LC vein bypass. The SEF had dropped from 61 to 37%, associated with an increased EDV (120 cc/m²) and decreased ESV (37 cc/m²). A grade III

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**Table:**

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contraction abnormality was present on the anterior wall.

Case 4. A 47-year-old rancher was admitted with a one-year history of angina pectoris without documented myocardial infarction. Physical examination, electrocardiogram, and chest X-ray were normal. On treadmill testing he was limited by chest pain and dyspnea and had an FAI of 28%. Selective coronary angiography demonstrated a 90% stenosis of the RCA and occlusion of LC. The EDV (71 cc/m²) was normal. The ESV (35 cc/m²) was low and the SEF was slightly diminished (49%). A grade II contraction abnormality was present on the inferior wall.

One month later, the patient suffered an inferior myocardial infarction while awaiting surgery. His angina persisted.

Five months postinfarction his physical examination and chest X-ray remained normal. The electrocardiogram showed a diaphragmatic myocardial infarction. Repeat coronary arteriography revealed occlusion of the RCA and an essentially unchanged EDV (62 cc/m²), ESV (33 cc/m²) and SEF (53%). The contraction plot was unchanged as well.

Case 5. A 44-year-old farmer was admitted with a thirteen-year history of angina pectoris and a myocardial infarction three months prior to admission. Cardiac examination and chest X-ray were normal. An electrocardiogram demonstrated a possible old lateral myocardial infarction. On treadmill testing he stopped with chest pain and had significant ST segment depression. His FAI was 80%. Selective coronary angiography demonstrated diffuse severe three-vessel disease, with a 70% occlusion of the LAD, a 50% occlusion of the LC and an 80% occlusion of the RCA. Only the LAD was suitable for an aortocoronary vein bypass grafting. EDV (66 cc/m²), ESV (39 cc/m²), LVEDP (10 mm Hg) and SEF (59%) were normal.

Two days later, the patient sustained an anteroseptal myocardial infarction followed by persistent chest pain. Restudy two months later demonstrated an occluded LAD and an apical akinetic area. The EDV (65 cc/m²) was unchanged; the ESV (29 cc/m²) had fallen and the SEF had diminished from 59 to 42%. The contraction plot demonstrated an apical contraction abnormality (grade III).

Case 6. A 64-year-old farmer with a previous myocardial infarction seven years prior to admission followed by persistent angina pectoris was studied two days prior to myocardial infarction. On admission physical examination demonstrated an atrial gallop. The electrocardiogram was normal. Due to the severity of his chest pain, no treadmill test was done. Catheterization and angiography demonstrated a 90% stenosis of the LAD and normal ventricular function; LVEDP (10 mm Hg), EDV (68 cc/m²), SV (41 cc/m²), SEF (61%). Two days following study, the patient sustained a subendocardial infarction documented by a classic pain pattern, development of deeply inverted precordial T waves and characteristic enzyme changes. The course was complicated by early congestive heart failure and subsequently by recurrent pain and pulmonary edema on the eleventh post-infarct day without development of further enzyme or electrocardiographic changes. He was discharged without congestion on digoxin and furosemide.

Three months later he returned with increasingly severe nocturnal angina. On restudy he had no change in his coronary artery anatomy since the previous study. His LVEDP (10 mm Hg) was unchanged. EDV (66 cc/m²) was unchanged. Both the ESV (23 cc/m²) and SEF (34%) had fallen significantly. The contraction plot demonstrated a grade III abnormality of the anterior wall.

Results

Four patients had antero-septal infarctions, one had an inferior infarction, and one a subendocardial infarction. Excluding the infarct associated with catheterization (#3) each infarct occurred in an area supplied by a coronary artery which had a stenosis of at least 70% prior to infarction (table 1). In four cases infarction was associated with progressive coronary artery narrowing to total occlusion. In two cases, #2 and #6, no change in the involved coronary artery was seen. Both patients had 90% stenoses of the LAD prior to infarction, which had not changed angiographically when restudied following infarction. Two, #2 and #5, had severe lesions away from the area of subsequent infarction that did not progress over the period between the two studies. In five of six cases infarction was accompanied by a new or progressive ventricular contraction abnormality in the area supplied by a diseased coronary artery (table 1, fig. 1).

Hemodynamic and angiographic results are presented in table 1 and figures 1-5. Changes in ventricular function varied following infarction. Two patients (#1 and #3) with initially normal ventricular function based on EDV, SEF, and contraction pattern developed marked abnormalities in all of these parameters of ventricular function (figs. 1-5) and clinical congestive heart failure. Three patients (#2, #5 and #6) developed moderate abnormalities following infarction. The contraction pattern became definitely abnormal (group III) and there were decreases in SEF unaccompanied by any significant change in EDV or LVEDP. A single patient (#4), in spite of a slightly more abnormal contraction plot, actually decreased both EDV and LVEDP with a slight increase in SEF (49 to 53%).

On exercise testing prior to infarction, the five patients tested had generally mild to moderate limitation by FAI ranging from 29 to 46% in four. A fifth, case #5, had an initial FAI of 80%. Following infarction, the four patients restested all had very poor exercise performance with FAI of greater than

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LV FUNCTION AND CA CHANGE PRE AND POST MI

Figure 1

Pre- and postinfarction contraction plots in each patient. Patients #1, 3, 5, 6 had normal initial contraction plots and post MI developed localized akinesis of more than 25% of the internal ventricular surface. Patient #2 had an initially borderline abnormality that progressed. Patient #3 had no significant change.

56%. Three of the four with marked deterioration in exercise performance with infarction also had marked deterioration of left ventricular performance as measured by the SEF and development of CHF manifested by pulmonary congestion or severe dyspnea and a diuretic requirement. One of the four, case #3, had a marked drop in FAI but no significant change in SEF. Four of five patients were stopped on the treadmill by pain prior to their initial study. One, case #2, was stopped by claudica-
The change in left ventricular end-diastolic pressure (mm Hg) was not a sensitive indicator of change in left ventricular performance following infarction. Only one patient, #1, of the five with pre- and post-MI measurements had a distinctly abnormal post-MI LVEDP. Normal LVEDP ≦ 12 mm Hg.

Discussion

Previous studies from our laboratory and those reported by Herman and Gorlin, Rackley et al., Baxley et al., and Bristow have defined a wide spectrum of ventricular functional abnormalities in patients with coronary artery disease. There is a close relationship between clinical hemodynamic and angiographic abnormalities which occur with progressive coronary disease.

The present study affords a unique opportunity to examine individual patients with progressive disease at two points in time rather than a group of patients at a single point in time. In this sense, this study is a longitudinal rather than a cross-sectional view of ventricular function in myocardial infarction.

Since all patients were selected for study on the basis of clinical indication, there were no uniform time intervals between initial study, myocardial infarction, and the subsequent study. In cases #1 and #2, periods of 28 and ten months elapsed between the two studies. On the basis of clinical evidence we believe that ventricular dysfunction occurred suddenly following a single infarction in each case. However, it is possible that a more gradual deterioration in function on the basis of the slow evolution of coronary heart disease also contributed to the measured changes.

We arbitrarily chose to restudy our patients no sooner than two months following their myocardial infarctions. We are aware of no data in the literature to define precisely the point beyond which no further improvement can be expected following a myocardial infarction. Kumar et al., using the dog model of experimental myocardial infarction, found marked improvement in left ventricular function at six to eight days as compared to initial values. Rahimtoola et al. found that while a majority showed hemodynamic improvement in ventricular performance over initial

The change in left ventricular end-diastolic volume following infarction was marked in two of six patients (#1 and #3). Normal EDV ≦ 70 ± 20 cc/m².

Left ventricular stroke volume fell in five of six patients following myocardial infarction. Patient #4 had no significant change. Normal ESV = 45 ± 13 cc/m².
LV FUNCTION AND CA CHANGE PRE AND POST MI

Figure 5
The SEF fell in five of six cases. In Case 4 a slight increase occurred. Normal SEF = 67 ± 8%.

values at three to five weeks following myocardial infarction in man, a few demonstrated deterioration in cardiac index and a rise in LVEDP. Finally, Feild et al. showed that contraction abnormalities following myocardial infarction persist for at least six to ten months following myocardial infarction. Thus, we cannot rule out the possibility that lev- ventricular function was not in a process of slow dynamic change at the time of study.

Any method of measuring contraction abnormalities yet presented requires an arbitrary superimposition of the end diastolic and end systolic silhouettes of the left ventricular chamber. We have chosen to superimpose films on the basis of internal references—the long axis and its perpendicular. Others have chosen an external reference point, for instance, the center of the X-ray beam. We have found neither method satisfactory in all cases, but in our experience, the method utilizing an internal reference is more often satisfactory.

These data support the hypothesis that myocardial infarction is usually accompanied by a change in contractile pattern and a decrease in SEF (table 1, fig. 5). Five of six patients showed a decrease in SEF and a more severe contractile abnormality. One of six did not significantly change. As noted in the previous study, infarction-induced decreases in SEF were accompanied by variable changes in EDV and LVEDP when the SEF remained in the mildly abnormal range. None of our three patients in this category (#2, #5, #6) developed an elevated EDV (figs. 3, 5). Two of the three (#2, #6) had post infarction attacks of CHF. In the previous study severely abnormal SEF was always associated with increased EDV, LVEDP, and clinical manifestations of CHF (fig. 6). Two patients (#1 and #4) with SEF of 21 and 31% fell in this category of increased EDV and CHF.

These studies of angiographic coronary arterial anatomy are a selected sample of the general population of preinfarct patients in that all patients were sufficiently limited by pain to be considered for vein bypass surgery. In this group, the infarct occurred in an area supplied by a vessel which had at least a 70% reduction in diameter prior to infarction. Infarction was accompanied by progression to total occlusion of the involved vessel in four of six patients. Two of six had no angiographic progression of the previous severe lesion. Total occlusion of the LAD resulted in at least a 17% drop in SEF in three patients. The total RCA occlusion resulted in no change in SEF. Two with no change in LAD anatomy also had a fall in SEF of 10 and 29% suggesting that an AMI may have a similar functional impact with or without vessel occlusion. All changes in contractile pattern occurred in the area of severe coronary artery stenosis.

Though several patients were stopped on the treadmill by dyspnea rather than chest pain following their infarction, all were having typical symptoms of angina pectoris at that time, which was the indication for restudy. The failure to observe significant ST segment depression in four of

Figure 6
The relationship between systolic ejection fraction (SEF), end diastolic volume (EDV), and contraction pattern. The envelope surrounds data reported previously that led to the generalization that patients with SEF ≥ 40 have variable changes in EDV while those with SEF ≤ 40 have consistent increases in EDV. In the six cases reported above only case 4 did not have a more abnormal contraction pattern and a fall in SEF.

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five tested preinfarction and one of three not on digoxin when tested postinfarction is surprising. A possible explanation is that two of the four patients, cases #3 and #4, were having ischemia of the posterior and diaphragmatic areas, when first tested, an area frequently missed by the modified V, lead used in treadmill testing.

References

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DOUGLAS K. STEWART, GLEN W. HAMILTON, JOHN A. MURRAY and J. WARD KENNEDY

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