Left Ventricular and Coronary Angiographic Anatomy

Relationship to Ventricular Irritability in the Late Hospital Phase of Acute Myocardial Infarction


SUMMARY  Late hospital phase ventricular arrhythmias in acute myocardial infarction (MI) have been associated with a high incidence of sudden death following hospital discharge. Thirty-eight patients were studied 10-24 days following onset of symptoms of MI. Each patient had a 24-hour ambulatory ECG tape recording and left ventricular and coronary angiography performed. Patients with complicated ventricular arrhythmias (multiform, coupled, R on T VPCs or ventricular tachycardia), when compared to those with uncomplicated ventricular arrhythmias (unifocal or no VPCs), had a greater number of proximally narrowed major coronary arteries 

VENTRICULAR ARRHYTHMIAS in the late hospital phase of an acute myocardial infarction have been associated with a poor prognosis and a high incidence of subsequent sudden cardiac death. These arrhythmias have been relatively refractory to pharmacologic therapy and suppression, when achieved, has been associated with a high incidence of drug side effects. While late hospital phase ventricular irritability following myocardial infarction (MI) has been correlated with left ventricular dysfunction, large infarct size, location of infarct by electrocardiography (ECG), development of intraventricular conduction defects, and ST-segment abnormalities on ECG. The present study was undertaken to determine whether there is a correlation between the location and severity of coronary artery obstruction and extent of abnormal segmental left ventricular wall motion and the prevalence of serious ventricular arrhythmias in the late hospital phase of acute myocardial infarction.

Methods

Between September 1974 and October 1975 all patients admitted to the Coronary Care Unit of our hospital with subsequently documented acute myocardial infarction (MI) were considered for inclusion in this study. A diagnosis of acute myocardial infarction was made in each patient on the basis of a typical history of chest pain, serial ECG changes, and serial cardiac enzyme elevations. For the purpose of this study all three criteria were required. ECG changes in leads I, aV_L, V_4-V_6 were considered to represent anterior MI; those in leads II, III, aV_F were considered to represent inferior MI. Development of Q waves of 40 msec duration was considered characteristic of transmural MI. Absence of 40 msec Q waves, but presence of serial ST-T wave changes, was considered characteristic of subendocardial MI. Blood was obtained for creatine phosphokinase (CPK) measurement on admission and every four hours thereafter for 48 hours. Peak CPK was used as an estimation of myocardial damage for each patient as previously described.

Patients greater than 65 years of age, with life threatening illness other than coronary heart disease, with ventricular septal rupture or severe papillary muscle dysfunction uncontrolled with medical therapy, or with primary physicians who preferred that their patients not undergo cardiac catheterization prior to hospital discharge, were excluded from the study population.

There were 98 patients with a mean age of 52 years who met the above criteria from an original group of 154 patients with acute MI. Fifty of these 98 patients agreed to undergo coronary angiography and left ventriculography. Informed consent was obtained from each patient. Thirty-eight of these 50 patients had a 24-hour ambulatory electrocardiographic tape recording done prior to discharge. Twelve patients either were not monitored due to problems with equipment or had ECG tapes that were technically unsatisfactory for analysis. Ten to 24 days following hospitalization for myocardial infarction each patient underwent diagnostic cardiac catheterization including cine left ventriculography in right (RAO) and left (LAO) anterior oblique projections and selective cine coronary arteriography in multiple projections using either the percutaneous femoral (Judkins) or brachial (Sones) technique. There were no complications of catheterization in the 38 patients studied.

Segmental left ventricular wall motion was reviewed in...
consultation by at least two authors on the basis of left ventriculograms obtained in the RAO and LAO projections. Analyzed in this fashion, eight ventricular segments were identified for each patient (fig. 1). Each segment was classified as having either normal or abnormal wall motion. Abnormal segments were further subdivided as hypokinetic (decreased but retained wall motion), akinetic (no wall motion), or dyskinetic (paradoxic outward expansion during systole). Left ventricular end-systolic and end-diastolic volumes were calculated from the left ventricular angiograms according to the modified method of Dodge and a left ventricular ejection fraction (EF) was calculated for each patient.

Coronary artery anatomy was similarly reviewed by at least two authors. Each patient was considered to have three major coronary arteries (right, left anterior descending, and left circumflex). The severity of obstructive disease in a coronary artery narrowing was described as (a) 100% (total), (b) greater than 90% and less than 100% (near total), (c) greater than 50% and less than or equal to 90%, (d) greater than or equal to 30% and less than or equal to 50%, or (e) less than 30%. The location of each recognized coronary narrowing was defined according to the 15 segment classification recommended by a committee of the American Heart Association (fig. 2). Only the most severe narrowing in each coronary segment was recorded. A narrowing of greater than 50% was considered to be hemodynamically significant. A patient was considered to have single, double, or triple vessel coronary artery disease depending on whether one, two, or all three major coronary arteries were narrowed by more than 50%. In this group of 38 patients there were two patients in whom the main left coronary artery was narrowed by greater than 50%; we considered this to represent narrowing of both the left anterior descending and the left circumflex coronary arteries.

Three additional interpretations of the coronary angiogram were performed. A coronary arterial “score” was determined for each patient according to the method previously described by Friesinger et al. This method scores each of the three major coronary arteries on a scale of 0 to 5 as follows: 0 — no abnormalities seen on selective coronary angiography; 1 — a trivial irregularity of the coronary artery lumen; 2 — localized narrowing of > 75% but < 90% of cross sectional area; 3 — multiple narrowings of > 50% but < 90% in the same vessel; 4 — narrowing of > 90% but not total obstruction; 5 — total obstruction of any coronary vessel. The score of all three arteries is then added to obtain a score for the coronary angiogram. In general, a score of 0-4 represents single vessel disease, 5-9 represents double vessel disease, and 10-15 represents triple vessel disease. Patients were also classified according to whether they had proximal coronary artery disease. For purposes of this study, proximal disease was the presence of a narrowing greater than 50% in segments 1, 2, 5, 6 and 11 and the initial third of segments 7 and 12 (fig. 2). Finally, a narrowed artery was considered suitable for bypass graft surgery if it fulfilled the following criteria: (a) a narrowing greater than 50% was present in a proximal coronary artery segment, (b)
the distal coronary artery was judged to have an adequate lumen diameter and to be free of recognizable atherosclerotic plaque at or distal to the usual graft artery anastomotic site, and (c) the myocardial segment perfused by the obstructed coronary artery was considered to have retained ventricular wall motion.

A 24-hour ambulatory ECG tape recording was made within 48 hours of the catheterization in each patient while undergoing routine hospital activities. Tapes were recorded on an Avionics model 450 electrocardiocorder and were played back 60 \times \text{real time on an Avionics model 650 electrocardioscanner. Arrhythmias, detected by trained personnel, were written out at normal paper speed (25 mm/sec), analyzed by a cardiac nurse with review by a cardiologist, and categorized according to the modified criteria of Lown and Wolf. Patients with unifocal or no VPCs were classified further as having uncomplicated ventricular arrhythmias; patients with multifocal, coupled, or R on T VPCs and ventricular tachycardia were classified as having complicated ventricular arrhythmias. The left ventricular and coronary angiograms and the ambulatory ECG tapes were read independently. Statistical analysis was performed using Student’s unpaired t-test, Chi-square analysis, regression analysis, stepwise discriminant function analysis, and analysis of covariance.

**Results**

**I. Clinical Characteristics**

The clinical characteristics of the 38 patients are shown in table 1. Patients were divided into two groups: group I consisted of 23 patients with uncomplicated ventricular arrhythmias, group II consisted of 15 patients with complicated ventricular arrhythmias. There was no significant difference between group I and group II with respect to Killip clinical class on admission, prevalence of ventricular tachycardia/ventricular fibrillation, and peak CPK values. Patients in group II, however, had a significantly increased incidence of previous MI (P < 0.005) and were slightly older than those in group I.

**II. Late Hospital Phase Ventricular Arrhythmias**

Of the 23 patients in group I, 10 had no ventricular arrhythmias, 11 had infrequent unifocal VPCs, and two had frequent (> 30/hour) unifocal VPCs on ambulatory monitoring. Of the 15 patients in group II, eight had multiform VPCs, only six had coupled VPCs or ventricular tachycardia, and one had multiform VPCs with the R on T phenomenon.

**III. Left Ventriculography**

Table 2 shows the total number of hypokinetic, akinetic, dyskinetic, and normal segments in the left ventriculograms of patients in groups I and II. Of 169 segments visualized adequately in the 23 group I patients, 52 were normal. Of 110 segments visualized adequately in the 15 group II patients only 15 were normal (P = 0.001). Group I patients had a mean EF of 52 ± 3 while group II patients had a mean EF of 45 ± 4. This trend toward lower EF in group II patients was not significant at the 0.05 level.

**IV. Coronary Arteriography**

Table 3 depicts the prevalence of single, double and triple vessel proximal coronary artery disease in the patients in group I and group II. All 14 patients with single vessel disease were in group I (P < 0.01) and 10 of the 14 patients with triple vessel disease were in group II. Patients in group II had significantly more diseased vessels than patients in group I, 2.7 ± 0.1 vs 1.6 ± 0.2 (P < 0.001).

Using the scoring system for coronary artery disease as previously described by Friesinger et al., patients in group II had a significantly greater extent of coronary disease than patients in group I, coronary score 10.1 ± 0.7 vs 6.3 ± 0.6 (P < 0.001).

Of the 69 arteries evaluated in the 23 patients in group I, 18 (26%) had a proximal obstruction judged to be total or near total (> 90%). Of the 45 arteries in the 15 patients in group II, 16 (36%) had a total or near total proximal obstruction. Fourteen of the 15 patients in group II had at least one coronary vessel judged suitable for bypass grafting by the criteria outlined previously. Since the patients in group II were older and had a higher incidence of previous myocardial infarction than those in group I, the data were subjected to regression, stepwise discriminant function analysis, and analysis of covariance.

**Table 1. Clinical Characteristics of 38 Patients Studied**

<table>
<thead>
<tr>
<th>Group</th>
<th>Number pts.</th>
<th>Age</th>
<th>Killip class I and II</th>
<th>Anterior MI</th>
<th>Transmural MI</th>
<th>CCU VT/VF</th>
<th>Peak CPK</th>
<th>Previous MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>23</td>
<td>44±2</td>
<td>9</td>
<td>12</td>
<td>13</td>
<td>8</td>
<td>890±270</td>
<td>2</td>
</tr>
<tr>
<td>II</td>
<td>15</td>
<td>52±2*</td>
<td>10</td>
<td>7</td>
<td>9</td>
<td>6</td>
<td>710±210</td>
<td>12**</td>
</tr>
</tbody>
</table>

*P < 0.05.
**P < 0.005.

**Table 2. Functional Anatomy of Myocardial Segments from the Left Ventricular Angiogram**

<table>
<thead>
<tr>
<th>Group</th>
<th>Total segments</th>
<th>Normal</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>169</td>
<td>52 (31%)</td>
<td>15 (14%)</td>
</tr>
<tr>
<td>II</td>
<td>110</td>
<td>95 (86%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Hypokinetic: 74 (44%) vs 58 (55%) <0.005
Akinetic: 33 (19%) vs 32 (29%) <0.005
Dyskinetic: 10 (6%) vs 5 (4%) NS

*P values compare normal to abnormal segments in group I versus group II.

**Table 3. Coronary Angiographic Characteristics of Patients**

<table>
<thead>
<tr>
<th>Group</th>
<th>Proximal coronary artery</th>
<th>Coronary angiographic score</th>
<th>Single vessel disease</th>
<th>Double vessel disease</th>
<th>Triple vessel disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>1.6 ± 0.2*</td>
<td>6.3 ± 0.6*</td>
<td>14**</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>II</td>
<td>2.7 ± 0.1</td>
<td>10.1 ± 0.7</td>
<td>0**</td>
<td>5</td>
<td>10</td>
</tr>
</tbody>
</table>

*P <0.001 compared to group II.
**P <0.01 compared to patients with double or triple vessel disease.
Regression analysis for age vs coronary artery score and age vs the number of abnormal ventricular segments failed to reveal a significant relationship between age and either coronary score or the number of abnormal ventricular segments. In spite of this we corrected for age by analysis of covariance and analyzed the data for coronary artery score. There was still a significant difference \( (P = 0.001) \) between group I and group II in coronary artery score. Stepwise discriminant function analysis revealed that age was only an insignificant factor in separating groups I and II. The most important factor proved to be a history of previous myocardial infarction, a factor which would tend to reflect both abnormal ventricular function and extent of coronary artery disease. The factor indicative of acute myocardial damage, peak CPK release, was insignificant in comparison to the number of abnormal ventricular segments found at left ventricular angiography, possibly reflecting the fact that the number of abnormal ventricular segments is a reflection of both old and acute myocardial damage whereas peak CPK release reflects only acute damage. Abnormal ventricular angiography proved to be the second most important variable in separating groups I and II. The next two variables which added to the discrimination between groups I and II in order of importance were the number of vessels with > 50% narrowing and the prevalence of transmural vs nontransmural infarction.

**Discussion**

While the prevalence of ventricular arrhythmias has been stated to be related to the extent of coronary artery disease in patients with angina pectoris, this study documents for the first time the nature and extent of coronary artery disease in patients with complicated ventricular arrhythmias in the late hospital phase of acute MI. Complicated ventricular arrhythmias seem clearly linked to extensive coronary artery disease. When compared to patients with uncomplicated ventricular arrhythmias, patients with complicated ventricular arrhythmias had a higher mean coronary score, a higher mean number of diseased coronary arteries, and an increase in the number of total or near total narrowed proximal coronary arteries. There was a greater prevalence of triple vessel disease and no single vessel disease in the patients with complicated ventricular arrhythmias. By contrast 15 of the patients with uncomplicated ventricular arrhythmias (61%) had single vessel coronary artery disease.

In addition, there was a significant increase in LV wall motion abnormalities in the group of patients with complicated ventricular arrhythmias (group II) compared to the group with uncomplicated ventricular arrhythmias (group I). The greater extent of wall motion abnormality is probably a result of more extensive coronary artery disease and a greater number of previous infarcts in group II compared to group I.

Total myocardial damage as reflected by the number of abnormal ventricular segments at angiography appears to be a more important factor in separating patients with complicated from uncomplicated ventricular arrhythmias than the amount of peak CPK release. The importance of the extent of myocardial damage for the development of complicated ventricular arrhythmias was emphasized by stepwise discriminant function analysis which revealed previous myocardial infarction to be the most important factor in separating group II from group I patients.

Ventricular aneurysms, extensive left ventricular akinesis, and other left ventricular wall motion abnormalities have been related previously to the presence of ventricular arrhythmias. Similarly, patients with documented coronary artery disease with ventricular asynergy have been shown to have an increased frequency of VPCs on ambulatory ECG recording compared to coronary patients without ventricular asynergy. 

Herman and Gorlin have described a direct correlation between extent of left ventricular akinesis or paradoxical wall motion and development of left ventricular dilatation or congestive heart failure both of which are felt to predispose to areas of localized ischemic and re-entrant arrhythmias. An inverse relationship has also been demonstrated between the amount of left ventricular wall damage and the threshold for ventricular fibrillation in experimental animals.

Complicated late hospital phase ventricular arrhythmias following myocardial infarction have been associated with an increased incidence of sudden death following hospital discharge. While suppression of these arrhythmias following infarction presumably should improve prognosis, they have proven to be relatively refractory to presently available antiarrhythmic drugs and suppression, when accomplished, is often associated with a high incidence of drug toxicity. The refractoriness of these arrhythmias may well be related to the presence of extensive coronary arterial disease and multiple areas of abnormal left ventricular wall motion which may represent ischemic or infarcted myocardium.

Previous studies from our institution suggest that group II patients have a one year survival < 50% despite standard antiarrhythmic therapy. It is of interest to compare the catheterization data of our group II patients with another high risk group, the survivors of out-of-hospital ventricular fibrillation reported by Weaver et al. Eighty-one percent of their patients had significant coronary artery narrowing on angiography and 84% some abnormality of left ventricular function. In those with recurrent ventricular fibrillation there was a high incidence of triple vessel disease (93%) and low left ventricular ejection fractions. These findings are similar to those in group II in the present study, although the comparison is not entirely valid since our patients were all post myocardial infarction at the time of study and had not had an episode of ventricular fibrillation following transfer from the coronary care unit, although presumably they were at an increased risk to do so subsequently. The implications of the findings in the present study and that of Weaver et al. for future therapy must await further confirmation on a larger number of patients. It should be noted, however, that all but one of the patients in group II in the present study and all of the patients with recurrent ventricular fibrillation in the study by Weaver et al. had at least one coronary vessel suitable for coronary bypass graft surgery. Should medical therapy prove inadequate in these patients, despite the use of beta adrenergic blocking agents as suggested by the findings of Wilhelmsson et al., surgery, aneurysmectomy and/or coronary artery bypass grafting might be considered in these patients with a known propensity for sudden death.
Acknowledgment

The authors wish to acknowledge the assistance of Myron L. Weisfeldt, M.D. for review of the manuscript, Sally Bowers, R.N., Nancy McDonald, R.N., and Jean Keruly, R.N. in the recording and interpretation of the ambulatory ECG tapes and Lisa Grue in the preparation of the manuscript.

References

17. DeMaria AN, Vera Z, Amsterdam EA, Mason DT, Massumi RA: Disturbances of cardiac rhythm and conduction induced by exercise. Am J Cardiol 33: 732, 1974
Left ventricular and coronary angiographic anatomy. Relationship to ventricular irritability in the late hospital phase of acute myocardial infarction.

R A Schulze, Jr, J O Humphries, L S Griffith, H Ducci, S Achuff, M G Baird, E D Mellits and B Pitt

_Circulation_. 1977;55:839-843
doi: 10.1161/01.CIR.55.6.839

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/55/6/839

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org//subscriptions/