Significance of High Degree Atrioventricular Block in Acute Posterior Myocardial Infarction

The Importance of Clinical Setting and Mechanism of Block

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SUMMARY
This report evaluates the morbidity and mortality, during hospitalization and follow-up, of a subgroup of patients with posterior or diaphragmatic myocardial infarction (PMI) who developed high degree A-V block via a type I mechanism and in the absence of power failure (pulmonary edema or cardiogenic shock). This subgroup was not at any higher risk of hospital morbidity, hospital mortality, or 1-year mortality than three other groups: (a) patients with PMI but neither high degree A-V block nor initial power failure; (b) patients with other infarct sites who developed high degree A-V block in the absence of power failure; and (c) patients with other infarct sites but neither high degree A-V block nor initial power failure. The significance of subgrouping patients with high degree A-V block by the quantity of clinical heart failure is exemplified by a review of the literature and the present study.

Additional Indexing Words:
Power failure Type I second-degree A-V block Third-degree A-V block

The improved survival of hospitalized patients with acute myocardial infarction has resulted primarily from an increased understanding of cardiac arrhythmias and their successful management and prevention. The precipitating factors, natural history, and modes of therapy for manifestations of ventricular irritability in acute infarction are well established. A comparable understanding of disturbances leading to bradycardia, more specifically high degree atrioventricular (A-V) block, has not yet been accomplished. One reason for this is that most previous studies have considered the whole spectrum of high degree A-V block as an entity without characterizing homogeneous clinical subgroups that are at high or low risk of specific target events. This report evaluates the morbidity and mortality, during hospitalization and follow-up, of a subgroup of patients with posterior high degree A-V block or diaphragmatic myocardial infarction who developed high degree A-V block via a type I mechanism and in the absence of power failure (pulmonary edema or cardiogenic shock). This subgroup is compared and contrasted to other subgroups with and without A-V block and power failure.

Methods
Five hundred thirty-nine consecutive patients with acute myocardial infarction who had continuous electrocardiographic monitoring on our coronary care unit were evaluated. The diagnosis of acute infarction was based on (1) a typical clinical history and (2) electrocardiographic changes consisting of either new Q waves or typical evolutionary ST-T changes, and (3) characteristic serial changes in serum enzymes including CPK, SGOT, and LDH. Continuous electrocardiographic monitoring was carried out in all patients during their stay on the coronary care unit with the use of the Hewlett-Packard patient monitoring systems. The monitoring equipment was supervised continuously by trained ECG monitor attendants. Furthermore, daily conventional 12-lead electrocardiograms were taken on all patients and additional electrocardiograms were taken as necessary. The hospital course of each patient and the full composite of the electrocardiograms taken during the hospitalization were carefully reviewed. High degree A-V block was defined as the presence of either second- or third-degree A-V block occurring via a type I mechanism. The presence or absence of power failure and its relation to the development of high degree A-V block was noted. Power failure was defined as pulmonary edema (respiratory distress due to the
presence of pulmonary congestion) or cardiogenic shock (a systolic blood pressure less than 90 mm Hg, urine output less than 20 cc per hour, peripheral vascular constriction, and an altered sensorium). The site of old and acute infarction was evaluated and defined according to criteria as outlined by Lipman and Massie and McConahay et al.7 Posterior myocardial infarction includes patients with characteristics of a diaphragmatic and/or posterior infarction (PDMI). Anterior infarction includes patients with anterior and/or lateral infarction. Anterior-posterior infarction includes patients with electrocardiographic characteristics of both anterior and posterior infarction, and a "nontransmural" infarct was identified when there was a compatible history accompanied by serial enzyme and ST-T changes but without the appearance of new QRS changes. An indeterminate site of myocardial infarction was noted in those cases where there was enzymatic evidence of necrosis occurring in the presence of left bundle-branch block. A careful review of all available electrocardiograms was carried out in order to determine the mechanism of development of high degree A-V block. The width of the QRS complex in the setting of complete heart block was determined.

Type I second-degree A-V block was defined by a changing P-R relationship in the presence of a changing R-P interval occurring on successive beats until a P wave failed to reach the ventricles.8-10 Type II second-degree block was diagnosed by the presence of a constant P-R interval despite a changing R-P interval preceding the failure of conduction to the ventricles.11,12 Complete or third-degree A-V block was diagnosed when none of the P waves propagated to the ventricles, and the ventricular rate was maintained by an escape mechanism in either the junction or the ventricle at a rate less than 60 beats/min.9,10,13

During the period of this study, patients had a temporary pacemaker inserted on development of high degree A-V block. A no. 5 or 6 bipolar (USCI) pacing catheter was inserted into an antecubital vein, advanced to the right ventricular apex, and connected to a Medtronic 5580A demand external pacemaker. In all patients, the pacemaker was set at low ma (<2.0 ma) and at a low rate (50 beats/min) on the demand mode. A need for an increase in rate was determined by the patient's clinical state.

During the period of hospitalization, all patients were followed to time of discharge to determine morbidity, i.e., a worsening clinical state, and hospital mortality. After discharge, all patients were followed to a period of 1 year. In those patients who expired, a careful evaluation of the cause of death was made by communication with the patient's personal physician, family or, when available, by review of the autopsy material.

Results

The overall hospital mortality in the 539 patients was 21% (111 patients). In the group with PDMI there were 181 patients with 32 hospital deaths (18%). Within this group with PDMI, 45 patients developed high degree A-V block. The mean age of the patients in this subgroup was 62.0 ± 8.0 years, and one or more previous electrocardiographically documented myocardial infarctions was present in 23 patients (43%). The overall mortality in these 45 patients was 24% (11 patients).

In the subgroup of 45 patients with high degree A-V block, 13 developed type I second-degree A-V block as the highest degree of block, and 32 developed third-degree block via a type I mechanism. In the group of patients with third-degree A-V block:

1. Thirteen progressed from first-degree to type I second-degree to third-degree A-V block (all 13 subsequently returned to normal A-V conduction).
2. Four progressed from type I second-degree to third-degree A-V block (all except one patient in this group returned to normal A-V conduction).
3. Thirteen were admitted with third-degree A-V block with a narrow QRS (<0.12 sec) and regressed through type I second-degree to first-degree A-V block to normal A-V conduction.
4. In two patients, third-degree A-V block was noted on admission with a narrow QRS, and both returned to normal A-V conduction directly without passing through lesser degrees of A-V block.

In the 30 patients who initially developed type I second-degree A-V block, 17 (57%) were noted to progress to third-degree A-V block. High degree A-V block occurred on admission or within 24-72 hours in over 80% of the patients. The mean duration of high degree A-V block was 3 days, with a range of 1-14 days. In one patient, third-degree A-V block was persistent with the need for permanent pacing at time of discharge.

Within this subgroup of 45 patients with PDMI and high degree A-V block, there were 36 who developed their A-V block in the absence of power failure: 12 with type I second-degree A-V block as the highest degree of block and 24 with third-degree A-V block. In eight (22%) of these 36 patients, the clinical condition deteriorated during the hospital course; five developed mild-to-moderate failure and three developed cardiogenic shock. These last three patients accounted for the only mortality (8%) in this subgroup, and all three patients were from the group with third-degree A-V block. In follow-up of the 33 survivors at 1 year, four (12%) had expired: two following a recurrent myocardial infarction with cardiogenic shock, and
two suddenly of unknown cause. This subgroup without power failure was contrasted to the remaining nine patients with PDMI and type I second-degree or third-degree A-V block which appeared following the onset of power failure. In these nine patients, one had type I second-degree A-V block and eight had third-degree A-V block as the highest degree of block. Eight of the nine patients expired (89% mortality). The one patient who recovered from his power failure and A-V block was alive on follow-up at 1 year.

High degree A-V block did not occur in the remaining group of 136 patients with PDMI. The mean age in this group was 58.0 ± 11.0 years, and one or more previous documented myocardial infarctions was present in 61 patients (47%). The overall mortality in this group was 16% (22 patients).

There were 120 patients with PDMI who were not initially in power failure and who did not have high degree A-V block. In the clinical course of these patients, 19 (16%) deteriorated during hospitalization. From this group of 120 patients, 10 died (9% mortality), six of power failure, two of a rhythm disturbance, and two of unrelated causes. All 110 survivors were followed for a period of 1 year, during which 10 (9%) expired. Death was sudden in six patients, secondary to power failure in the setting of a recurrent myocardial infarction in three, and unrelated to cardiovascular disease in one. Sixteen patients with PDMI and without high degree A-V block were in power failure at the time of hospital admission. Twelve (75%) expired during their hospital course, all of power failure. Two of the four patients who were discharged and followed for 1 year were alive.

When all infarct sites other than PDMI were evaluated, 13 patients with second-degree or third-degree A-V block via a type I mechanism were identified. The overall mortality in this group was 38% (five patients). Nine developed high degree A-V block in the absence of power failure. Five of these nine patients had type I second-degree A-V block as the highest degree of block. Two of these (22%) developed power failure and expired during their hospitalization. Of the seven survivors, three were dead by 1 year: two suddenly and one of power failure with recurrent infarction. In the remaining four patients in this group of 13 in whom high degree A-V block appeared after the onset of power failure, three expired of power failure, and a single patient survived but subsequently died of power failure with a recurrent infarction.

Upon evaluation of infarct sites other than PDMI, a group of 261 patients were noted who did not initially have power failure or high degree A-V block. The clinical state of 44 (17%) patients worsened and 33 (13%) died in the hospital: 20 of power failure, seven of a rhythm disturbance, three suddenly, and three of unrelated causes. Of the 228 survivors, 45 patients (20%) expired during their first year: 15 of power failure with a subsequent infarct, 18 suddenly of unknown cause, and 13 of noncardiovascular causes.

Table 1 contrasts the morbidity and mortality in the seven subgroups discussed by the descriptions of site of infarction and the presence or absence of high degree A-V block and power failure.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Contrasting Morbidity and Mortality in Subgroups of Patients Described by Site of Infarction and Presence or Absence of High Degree A-V Block and Power Failure*</th>
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<tbody>
<tr>
<td>Group descriptions</td>
<td>Posterior and/or diaphragmatic myocardial infarct site</td>
</tr>
<tr>
<td>Patients</td>
<td></td>
</tr>
<tr>
<td>High degree A-V block-present; power failure-absent</td>
<td>N %</td>
</tr>
<tr>
<td>36</td>
<td>100</td>
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<tr>
<td>Hospital morbidity</td>
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<td>8</td>
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<td>Hospital mortality</td>
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<td>Follow-up mortality at 1 year</td>
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<td>33</td>
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*The incidence of hospital morbidity (development of clinical evidence of heart failure including cardiogenic shock), hospital mortality, and follow-up mortality at 1 year is shown. Percentages describing hospital morbidity and mortality refer to percentage of the total number of patients. Percentages of mortality at 1 year refer to the percentage of those patients discharged from the hospital. It should be noted that patients with all other infarct sites and high degree A-V block absent but power failure present are not considered in this comparison.

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Discussion

A recent review on the bradyarrhythmic disturbances occurring in the setting of an acute myocardial infarction noted that the type and consequences of high degree A-V block are highly influenced by the relationship of the conduction system to the coronary circulation and the amount of muscle necrosis. In general, occlusion of the posterior circulation usually involves the right coronary artery and occasionally the left circumflex, and leads to inferior infarction and A-V block in the A-V node. In contrast, occlusion of the anterior circulation (anterior descending coronary artery) leads to conduction disturbances which involve the bundle of His and the bundle-branch system. Pathologic studies of the conduction system in patients with A-V block occurring in the setting of PDMI have revealed necrosis either within or adjacent to the proximal conduction system. The extent of infarction in these patients is not necessarily great, and the necrosis of the proximal conduction system is not extensive. In the setting of anterior infarction, pathologic studies have shown that the extent of infarction is pronounced and necrosis of the distal conduction system prominent.

With PDMI, the development of high degree A-V block, since it occurs in the proximal part of the conduction system, usually has a prodrome beginning with prolongation of the P-R interval with normal QRS morphology and progressing to type I second-degree and subsequently to third-degree A-V block. The escape pacemaker below the site of block usually has a normal QRS morphology and a rate of 45-60 beats/min. Disturbances of conduction in the bundle-branch system or the occurrence of type II second-degree block is rare. The progressive development of high degree A-V block, its time of occurrence in the course of infarction, and the duration of block in our study are similar to those found in the literature.

It is clear from our data that the subgroup of patients with PDMI and high degree A-V block occurring in the absence of power failure was not at any higher risk of hospital morbidity, hospital mortality, or 1-year mortality than three other groups:

1. Patients with PDMI but neither high degree A-V block nor initial power failure.
2. Patients with other infarct sites who developed high degree A-V block in the absence of power failure.
3. Patients with other infarct sites but neither high degree A-V block nor initial power failure.

The follow-up of patients with PDMI who developed high degree A-V block in the absence of power failure during their hospitalization has not previously been recorded in the literature. In our series, this subgroup of patients was not at any higher risk for death during a 1-year period of follow-up than the other groups described.

In a review of the literature, a low mortality (25%) was noted in patients with PDMI, third-degree A-V block, and a junctional escape rhythm (narrow QRS). This is in contrast to a 50% mortality in patients with PDMI, third-degree A-V block and a wide QRS and to 87% and 77% mortality in patients with anterior infarction, third-degree A-V block and narrow or wide QRS, respectively. A composite experience of the literature identifies 147 patients with the onset of high degree A-V block in the absence of power failure. The overall mortality was 11% (16 patients). Since only four patients with anterior infarction were found in this group, it may be assumed that almost all patients reported with little or no congestive heart failure had PDMI.

The literature affords little insight into the natural history of problems of impulse conduction occurring in the setting of acute infarction, because most patients received therapy, and its effect on the patient’s course cannot be evaluated.

On review of the reported therapeutic modalities used in patients with PDMI and high degree A-V block, it is clear that, prior to coronary care units and pacing therapy, these patients did not do well. In the series of 68 patients reported by Cohen, Doctor, and Pick in 1958, there was a 37% mortality. A 40% mortality was noted in 18 patients reported by Courter, Moffat, and Fowler in 1963. In contrast with these results, Jackson and Bashour presented 34 patients with third-degree A-V block; none was paced, and 23 received no therapy aimed at reversal of the block. Many patients had hemodynamic and tachyarrhythmic complications, but the mortality was only 20%. Some investigators have continued to observe patients with high degree A-V block on coronary care units without pacemaker insertion. Norris in 1969 reported a 19% mortality in 26 patients with PDMI and with high degree A-V block (only one was paced). Only nine
of these patients had third-degree block. In 1970 he reported a 38% mortality in 29 patients with PDMI and third-degree A-V block. Still, he states, “few were paced.” The degree of heart failure at the time of onset of high degree A-V block was not noted in these series.

Six series in the literature describe the use of pacemaker therapy for patients with PDMI and third-degree A-V block. In this group of 88 patients, pacing therapy was instituted only after the development of complications, i.e., heart failure, syncope, or arrhythmias. The overall mortality was 35%, with a range of 11–45%. More recently, “prophylactic” pacemaker insertion has been used in patients with acute myocardial infarction, i.e., prior to the development of any of the complications of high degree A-V block. In reviewing three series in the literature and including our patients, a total of 167 patients were found who fit into this therapeutic category. The overall mortality in these patients was 24% (40 patients) with a narrow range of 22–25%. Thus, when the total group of patients with PDMI and high degree A-V block is considered, the facilities provided by coronary care units, including “prophylactic” pacemaker insertion, seem to have significantly influenced prognosis.

In view of these findings, it seems important to subgroup these patients by that most important prognostic parameter: the quantity of clinical heart failure. Though pathologic studies have revealed that the extent of myocardial infarction in patients with PDMI need not be extensive to cause high degree A-V block, all patients with PDMI and high degree A-V block certainly do not have small infarcts. A first approximation of the size of infarction may be attained by observing the quantity of clinical heart failure.

The importance of subgrouping patients with PDMI is exemplified by the remarkably low mortality of 11% in the group of 143 patients reviewed in the literature and the 8% mortality in our 36 patients, none of whom was in power failure at the time of onset of high degree A-V block. This review of the literature and our study leaves a number of questions still unanswered regarding the natural history of patients with high degree A-V block and the effect of pacing therapy. To improve the objectivity in the care of these patients, future studies must identify patients within homogeneous subgroups according to site of infarction, mechanism of development of high degree A-V block, and degree of heart failure at the time of onset of block, and must evaluate clinical therapeutic trials within these groups.

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