Immediate and Remote Prognostic Significance of Fascicular Block during Acute Myocardial Infarction

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SUMMARY

The electrocardiograms of 538 patients with acute myocardial infarction were searched to identify all instances of atrioventricular (A-V) and intraventricular (I-V) conduction disturbances. Data concerning mode of therapy and clinical complications were obtained by review of the record. These variables were then analyzed for significance in relation to the development of type II A-V block acutely and syncope or sudden death during the first year of follow-up.

The most accurate predictor for both these events was the status of A-V conduction in combination with the status of I-V conduction. At highest risk (50%) for type II progression were patients with acute adjacent fascicular block plus P-R prolongation, i.e., left anterior hemiblock plus right bundle-branch block (RBBB), or left bundle-branch block (LBBB), or patients with acute nonadjacent fascicular block, i.e., RBBB plus left posterior hemiblock or alternating bundle-branch block. The nonpaced survivors from this same group, plus any other patients with transient type II progression, were also at high risk (45%) for syncope or sudden death in follow-up. No syncope or sudden death has occurred in seven patients with type II progression discharged with a pacemaker. All other patients were at lower risk for these acute and chronic complications.

Thus, the electrocardiogram in acute myocardial infarction can identify a high-risk group for acute type II progression in whom prophylactic pacer insertion may be beneficial. Similarly, the electrocardiogram can identify a high-risk group for syncope or sudden death in follow-up and implicates progression to higher degrees of A-V block as an important pathophysiologic mechanism. The possible role of permanent pacemaker therapy in preventing syncope or sudden death in this high-risk group is also suggested.

Additional Indexing Words:
Myocardial infarction  Type II A-V block  Fascicular block  Perinfarction block  Pacemaker therapy  Sudden death

UTILIZATION OF electrocardiographic monitoring facilities has resulted in a significant reduction of deaths from arrhythmias during acute myocardial infarction.1,2 Sudden death outside the hospital, however, continues to be a major cause of premature mortality in patients with arteriosclerotic heart disease.8 Efforts to reduce this mortality will require the identification of subgroups of patients at high risk for sudden death for testing the efficacy of various types of therapy. These efforts include several approaches: epidemiologic surveys,4-7 mobile coronary care units,8 detection of premature ventricular beats,9-12 and treatment with antiarrhythmic drugs.5,13

This report describes the use of the 12-lead electrocardiogram obtained during the acute phase of myocardial infarction to identify risk groups for sudden death in follow-up after myocardial infarction. We also report the use of the electrocardiogram as a predictor of type II atrioventricular (A-V) block14 during the acute infarction, and the intimate relation of acute type II block to sudden death in follow-up. Finally this study reports our experience with pacemakers, both in acute type II block and in the subsequent year. In particular, we wish to note the apparent protective effect of
permanent pacemaker therapy against sudden death during the first year of follow-up in a selected subgroup of high-risk patients.

Methods

The study was designed to include the following: (1) Data on all patients admitted to the Duke Coronary Care Unit with a documented myocardial infarction during the 5-year period of December, 1965 through December, 1970. (2) Analysis of A-V conduction. A-V blocks were coded by degree (first, second, or third) to indicate the amount of block, and higher degrees of A-V block were coded by type. In the presence of changing cycle lengths, a changing P-R interval indicated type I block, while a constant P-R relationship indicated type II block. In all instances of A-V block, no final decision as to type was made until a rhythm strip showing changes in cycle length was seen. (3) Analysis of intraventricular conduction according to the fascicular block concept of Rosenbaum et al. and the perinfarction block (PIB) concept of First et al. (4) Identification of the use of both temporary and permanent pacemakers. (5) Evaluation of the degree of heart failure both initially and subsequently during the hospital course. (6) Complete follow-up of the patient population for 1 year after the acute myocardial infarction.

The majority of these patients were admitted to the CCU because of a clinical history of chest pain which suggested acute myocardial infarction. A definite diagnosis was made if there were accompanying evolutionary electrocardiographic changes and/or characteristic changes of the serum enzymes. The enzymes included the serum glutamic-oxaloacetic transaminase, the serum glutamic-pyruvate transaminase, lactic dehydrogenase, creatine phosphokinase and, more recently, the isoenzymes of creatine phosphokinase and lactic dehydrogenase. Ninety-five percent of the patients in the study had enzyme changes considered diagnostic of myocardial infarction, i.e., transiently elevated values which subsequently fell to normal in the absence of other possible causes for the elevation.

All available electrocardiograms from the 538 consecutive monitored patients with proven myocardial infarction were reviewed. Whenever possible, tracings antedating the infarction were included in the review. Each patient had a daily 12-lead electrocardiogram in addition to frequent rhythm strips while on the CCU (average 5 days). These tracings were interpreted in the following manner:

(1) The status of A-V conduction was noted, and abnormalities were coded as old, acute, and/or present at discharge. Abnormalities were coded as acute only when electrocardiograms taken within the past year demonstrated their absence. First degree A-V block required a P-R interval greater than 0.20 sec. If a higher degree of A-V block occurred, the degree, type, and duration (transient or prolonged) were noted. If a higher degree of A-V block occurred in the presence of atrial fibrillation, it was handled as follows: in the presence of an acute posterior or diaphragmatic infarction and QRS duration of less than 0.12 sec, a type I mechanism was assumed; in the absence of a posterior or diaphragmatic infarction and with QRS duration greater than 0.11 sec, a type II mechanism was assumed. If digitalis toxicity was suspected, or when the A-V block developed as an agonal event, the patient was excluded from this analysis.

(2) The status of intraventricular conduction was analyzed according to previously cited criteria. Left anterior hemiblock (LAHB) or left posterior hemiblock (LPHB) required an acute leftward shift of the QRS axis to $-30^\circ$ or less, or an acute rightward axis shift to $+120^\circ$ or greater, respectively. Axis shifts which could not be documented as acute were coded as old left- or right-axis deviation without the hemiblock designation. Axis shifts secondary to pathologic Q waves were not coded as hemiblocks. If left- or right-axis deviation was present in association with a right bundle-branch block (RBBB), a hemiblock (in addition to the RBBB) was coded even though there was no evidence that the axis shift developed suddenly. Bundle-branch blocks were coded as acute only if an electrocardiogram taken within the past year did not show the conduction abnormality. A diagnosis of RBBB or left bundle-branch block (LBBB) was made if the QRS duration was 0.12 sec or greater with delayed activation over the right or left ventricle, respectively. A diagnosis of PIB was made when there was QRS prolongation to 0.11 or 0.12 sec, with a 180$^\circ$ angle between the initial and terminal 0.04 sec QRS vector in the leads reflecting the infarction. Anterior PIB was not coded, because this cannot be distinguished from RBBB with anterior myocardial infarction. All electrocardiograms showing QR morphologies in the anterior precordial leads, with QRS prolongation to 0.12 sec or greater and with delayed right ventricular activation, were coded as RBBB with anterior myocardial infarction. Intraventricular conduction defects (IVCD), occurring as part of an agonal clinical state or in the absence of A-V conduction, were not included in this analysis. Ten patients had neither PIB nor fascicular block, yet had QRS prolongation to 0.11 sec in association with repolarization abnormalities over the left ventricular leads. They are included separately as incomplete LBBB.

(3) The site of old infarction was noted according to the criteria outlined by Lipman and Massie and included anterior, lateral, inferior (diaphragmatic), posterior, ST-T and, in the presence of an IVCD, indeterminant location. The latter two categories denote no localizing QRS changes but enzymatic evidence of myocardial damage. The criteria of McConahay et al. regarding clockwise initial forces in the frontal plane with a mean QRS axis of $+10^\circ$ or less were added for the diagnosis of diaphragmatic myocardial infarction, and the criteria of Gunnar et al. were added for the diagnosis of anterolateral and lateral infarctions.

(4) Location of the acute infarction required the above criteria plus evolutionary ST-T changes in the appropriate leads.

Analysis of the role of pacemaker therapy was confined to those patients with type II A-V block. During the period of this study, prophylactic pacemaker insertion was not done. For those patients with...
FASCULAR BLOCK DURING MI

fascicular block who had type II progression and were paced, the medical records were reviewed in order to determine the hemodynamic significance of the progression.

Initial and worst clinical classes were obtained by a retrospective review of the clinical record. These included classes I–IV (A–D, respectively) as outlined by Killip and Kimball.1 Where controversy existed in the record, a majority opinion was taken regarding the presence of rales and/or a third heart sound. To be included as class IV, in addition to hypotension there had to be signs of poor perfusion to the skin and kidneys and/or central nervous system.

Follow-up data were obtained from the patient’s hospital records, by return visits to the postmyocardial infarction clinic, or by verbal and/or written contact with the patient’s physician, family, or the patient himself. For the patients dying in the follow-up period, the circumstances surrounding the patient’s death were determined and categorized according to one of the following etiologies: (a) recurrent myocardial infarction and its complications; (b) refractory congestive heart failure; (c) “unrelated” causes such as pneumonia, stroke, or cancer; (d) sudden death from unknown cause; and (e) unknown circumstances.

The group categorized as dying suddenly of unknown cause excluded all patients in whom the death (though sudden) was the end result of a gradually deteriorating clinical situation, e.g., refractory congestive heart failure. No attempt was made to define the status of the patient with regard to angina preceding death, and in no case did the time interval from the onset of symptoms to death exceed 24 hours. Approximately 90% of the follow-up data for patients dying in the study period was obtained by personnel without specific knowledge of the conduction disturbance, although no attempt was made to “blind” this part of the study. All information regarding the circumstances surrounding the patient’s death was from an immediate observer or an attending physician. If enough information was not provided by these observers and the death occurred in a hospital, the records of the hospitalization were obtained. In seven cases (one with PIB, one with fascicular block, and five with no IVCD) all sources of information did not provide sufficient data to determine the circumstances surrounding the death. There was no standard time interval between death and subsequent contact for follow-up information. One patient was lost to follow-up in the first year.

Patients with duplicate admissions were handled in the following manner. For acute admissions, each admission was counted as a separate infarction; for follow-up data in patients with nonfatal infarctions and no IVCD, the first admission only was analyzed; for follow-up of patients with IVCD, the admission showing the greatest degree of nonfatal IVCD was analyzed. Thirty-six patients had two admissions, five patients had three admissions, and one patient had four admissions.

The 538 infarcts in this report occurred in 1,178 CCU admissions for possible myocardial infarction, for a definite infarct-to-admission ratio of 46%.

The difference in the incidence of events between different groups was tested for significance utilizing two-way tables and the standard chi-square distribution. In a few instances, other statistical tests were used. These are noted in relevant sections of the Results section.

Results

The average delay times from onset of symptoms to arrival at the hospital and the incidence of transfer from other hospitals were not available on the entire population. In 223 consecutive patients, however, the average delay time was 10.9 hours, with a median delay of less than 4 hours. Ten percent of these patients were transferred from another hospital. Figure 1 presents the overall clinical class data (both initial and subsequent) and mortality rates for initial clinical categories. Fifty-two percent of the patients had no failure on admission and 42% remained free of failure (i.e., class I initially and subsequently). Thirty-one percent of the patients were in mild failure (class II) initially and 34% subsequently. Eight percent of the patients were in pulmonary edema (class III) initially and 6% subsequently. Nine percent of the patients were in cardiogenic shock (class IV) on admission, and 18% developed this complication subsequently. The mortality rate increased with increasing degrees of failure on admission, with a 6%, 21%, 51%, and 75% mortality for classes I–IV, respectively.

Table 1 summarizes the age and acute hospital mortality for the 538 infarctions grouped by intraventricular conduction status. The table shows that, while there are no significant differences in the mean ages of the various groups, the patients with IVCD’s were at increased risk (P < 0.01) for dying in the hospital. The increased mortality rate did not

![Figure 1](http://circ.ahajournals.org/)

This bar graph presents the incidence of patients in each clinical class both on admission (slanted bars) and subsequently (stippled bars). The cross-hatched parts of the initial class bars indicate the proportion of patients within that class who died in the hospital.
Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>Pt (no.)</th>
<th>Mean age (yrs) ±1 sd</th>
<th>Hospital mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>No intraventricular conduction defect</td>
<td>340</td>
<td>58.6 ± 11.4</td>
<td>55/340 16</td>
</tr>
<tr>
<td>Incomplete left bundle-branch block</td>
<td>10</td>
<td>66.7 ± 13.0</td>
<td>2/10 20</td>
</tr>
<tr>
<td>Perifascicular block</td>
<td>72</td>
<td>60.7 ± 10.2</td>
<td>20/72 28</td>
</tr>
<tr>
<td>Fascicular block</td>
<td>116</td>
<td>63.9 ± 10.7</td>
<td>34/116 29</td>
</tr>
<tr>
<td>Total</td>
<td>538</td>
<td>60.2 ± 11.4</td>
<td>111/538 21</td>
</tr>
</tbody>
</table>

The groupings of these fascicles is termed “adjacent fascicular block” in the remainder of this paper. Twenty-one patients

![Figure 2](http://circ.ahajournals.org/)

(A) Initial class. (B) Worst class. Both are presented relative to conduction status. (C) Contrasts the presence or absence of an IVCD relative to no failure or mild failure (initial and worst class I and II) and to more severe degrees of failure (initial and worst class III and IV).
had adjacent fascicular block plus P-R prolongation (12 with LBBB and nine with LAHB + RBBB), while 16 patients had LPHB + RBBB (14 patients) or alternating bundle-branch block (two patients). Block of these types is termed "nonadjacent fascicular block" in the remainder of this paper. In general, the patients with only one fascicle involved tended to be younger (mean age 59.2 years), but there was no statistically significant difference by the univariate analysis of variance test in comparison to the ages of those with more than one fascicle involved (mean age 65.8 years). Likewise, there was no significant difference in the hospital mortality whether the fascicular block was old or acute, or whether one, two adjacent, or two nonadjacent fascicles were involved.

Table 3 summarizes the incidence of type II A-V block in the hospital. Again, the patients are grouped according to the theoretic extent of fascicular involvement (risk of sudden A-V block vs the status of intraventricular conduction). The lowest incidence of type II progression was noted in the patients with no IVCD and those with PIB (2% and 3%, respectively). In the 33 patients who had one fascicle involved, three (9%) progressed. Of the 46 patients with adjacent fascicular block with a normal P-R interval, five (11%) progressed. At highest risk were the patients with nonadjacent fascicular block and the patients with adjacent fascicular block plus P-R prolongation. Of the 37 patients in this group, 13 (35%) progressed via a type II mechanism. These differences in the incidence of progression are significant \((P < 0.01)\) statistically. In the highest risk group there were 16 patients in whom the fascicular block was a documented consequence of the acute myocardial infarction, and eight (50%) progressed. In 15 patients from this highest risk group, at least one component of the block was new with the myocardial infarction, and four (27%) patients progressed. In six patients from this group of 37, the fascicular block was old and only one of these progressed. Thus, the overall incidence of progression via a type II mechanism in the hospital was approximately 3% in those patients with nonadjacent fascicular block or adjacent fascicular block with prolongation of the P-R interval. The risk was 50% if the block was a consequence of the myocardial infarction, but the incidence of progression was low if the conduction disturbance was known to antedate the acute infarction. The mean age of the patients with fascicular block and type II progression (69 years) was greater than the mean of the total infarct population (60.2 years), but the range of ages extended from 53 to 84 years. An analysis of type II progression in relation to worst clinical class developed by fascicular block patients showed that there was no significant difference in the incidence of progression relative to these patients' clinical class. The records of the 19 patients with fascicular block, type II progression, and acute pacemaker therapy were reviewed and showed that eight of the 19 patients suffered a
cardiopulmonary arrest prior to pacemaker insertion. The other patients had either a relatively stable bradycardia (eight patients) or insufficient data were available to determine the patient's status.

Table 4 summarizes the course of the 386 survivors of acute myocardial infarction who were discharged without a pacemaker and followed for 1 year. This table subgroups the patients according to their risk of sudden death or syncope during the first year of follow-up. It should be noted that this grouping is different from that of Table 3, because grouping on the basis of risk of acute type II progression did not allow identification of a subgroup at intermediate risk during the follow-up year. The low and intermediate risk groups are slightly different from the low and intermediate risk groups of Table 3 (risk of type II A-V block), while the high-risk groups are similar. Note that the increasing mortality (column 2) in the three groups

### Table 3

<table>
<thead>
<tr>
<th>Group</th>
<th>Pts (no.)</th>
<th>Pts progressing (no.)</th>
<th>Mortality of pts with type II progression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk:</td>
<td>No IVCD</td>
<td>340</td>
<td>5 (1%)</td>
</tr>
<tr>
<td></td>
<td>ICLB</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>PIB</td>
<td>72</td>
<td>2 (3%)</td>
</tr>
<tr>
<td>Intermediate risk:</td>
<td>One fascicle</td>
<td>33</td>
<td>3 (9%)</td>
</tr>
<tr>
<td></td>
<td>Adjacent fascicles with normal P-R</td>
<td>46</td>
<td>5 (11%)</td>
</tr>
<tr>
<td>High risk:</td>
<td>Nonadjacent fascicles</td>
<td>16</td>
<td>6 (38%)</td>
</tr>
<tr>
<td></td>
<td>Adjacent fascicles plus P-R prolongation</td>
<td>21</td>
<td>7 (33%)</td>
</tr>
<tr>
<td>Total</td>
<td>538</td>
<td>28</td>
<td>11 (39%)</td>
</tr>
</tbody>
</table>

**Abbreviations:** See text.

### Table 4

<table>
<thead>
<tr>
<th>Groups by risk for sudden death or syncope</th>
<th>Pts (no.)</th>
<th>Dead within 1 year: all causes</th>
<th>Dead of causes other than sudden</th>
<th>Dead suddenly</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low risk: No IVCD</td>
<td>290</td>
<td>33</td>
<td>11</td>
<td>23</td>
</tr>
<tr>
<td>RBBB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAHB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LAHB + RBBB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intermediate risk: PIB</td>
<td>70</td>
<td>19</td>
<td>27</td>
<td>9</td>
</tr>
<tr>
<td>LPHB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LBBB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ICLB</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High risk: Adjacent fascicular block + P-R prolongation</td>
<td>26</td>
<td>13</td>
<td>50</td>
<td>3</td>
</tr>
<tr>
<td>Nonadjacent fascicular block Any other patients with type II progression</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>386</td>
<td>65</td>
<td>17</td>
<td>35</td>
</tr>
</tbody>
</table>

**Abbreviations:** See text.
is accounted for by the increasing incidence of sudden death (column 4).

Some of the subgroups composing the three risk levels were rather small (e.g., ICLBBB had only four patients), and the current categorization is considered temporary pending accumulation of larger numbers of patients. At low risk were the 263 patients with no IVCD, the five patients with RBBB, the 11 patients with LAHB, and the 11 patients with LAHB + RBBB but a normal P-R interval. Of this total group of 290 patients, 33 (11%) died during the first year of follow-up, of which 23 (8%) succumbed from causes other than sudden death, and only 10 (3%) died suddenly of unknown cause. At intermediate risk was the group composed of 45 survivors with perinfarction block, six patients with LPHB, 15 patients with LBBB plus a normal P-R interval, and the four patients with incomplete left bundle-branch block. Of this total group of 70 patients, 19 (29%) were dead by 1 year, of which 11 (16%) were from causes other than sudden, and nine patients (13%) died via a sudden unknown mechanism. At high risk were the 13 patients with adjacent fascicular block plus a prolonged P-R interval, the nine patients with nonadjacent fascicular block, and the four patients who were initially in other groups but experienced transient progression during their infarction and survived to be discharged without a pacemaker. From this total group of 26 patients, 10 died suddenly within the first year and two experienced syncope. The differences in the incidence of syncope or sudden death are significant \((P < 0.01)\) for all comparisons, including the intermediate versus high-risk group. Even when the 10 patients with type II progression (the nonpaced survivors of table 5) are excluded, this remains a high-risk group with five of 16 patients (31%) experiencing sudden death in follow-up. Although late in-hospital sudden deaths (of which there were only three) were not included in this analysis, it should be noted that two of these three patients had high-risk electrocardiographic patterns.

The risk of syncope or sudden death was not significantly different when these events were analyzed relative to the duration of the IVCD (prior to infarction) or to the persistence of the IVCD (following infarction). With regard to the latter point, there were 11 patients in our high-risk group whose IVCD was at least partially transient and four (36%) experienced sudden death (three patients) or syncope (one patient) in the first year. In the 15 remaining patients with a high-risk ECG pattern, the IVCD was permanent, and eight patients (53%) experienced sudden death (seven patients) or syncope (one patient). While these differences are not significant \((P > 0.05)\), it should be noted that the groups are small and the relation of persistence of the IVCD to the risk of syncope/sudden death in follow-up will bear reanalysis pending the accumulation of larger numbers of patients.

Figure 3 is a Venn diagram of the entire population of survivors of acute infarction followed for 1 year which is subdivided according to the presence or absence of three variables: (1) more than one old myocardial infarction, (2) the development of pulmonary edema or cardiogenic shock during hospitalization, and (3) a high-risk electrocardiographic pattern. This is an attempt to relate conduction status to other parameters which reflect the severity of coronary artery disease. Of the 32 incidents of syncope or sudden death, only 11 occurred in the 309 patients who had none of these variables, whereas 21 occurred in the 77 patients with one or more of the three variables. By Bennett's multinomial maximal likelihood technic, we were unable to assign a statistically significant label to any one of the three variables \((P > 0.05)\). It is of interest, however, that, of the 10 patients with a high-risk ECG pattern plus another variable, six (60%) had died suddenly by 1 year whereas, of the 51 patients with some combination of the other two variables, 11 (20%) died suddenly. These differences in the incidence of sudden death are significant \((P < 0.02)\) by the standard T test of proportions. The mean age of patients dying suddenly in follow-up is not significantly different from that of the total population of survivors.

Table 5 summarizes a select subgroup of patients, all of whom progressed to higher degrees of A-V block via a type II mechanism during their acute myocardial infarction. Sixteen of these patients had some form of fascicular block and one patient had perinfarction block. Of the 10 patients who progressed but subsequently regained A-V conduction and had no pacemaker inserted prior to discharge from the hospital, seven experienced syncope or sudden death during the first year of follow-up. (Two patients had syncope and five died suddenly.) This group includes one patient who was discharged with a permanent pacemaker presumed to be in the right ventricle. He died suddenly and at postmortem examination was
SYNCOPE—SUDDEN DEATH IN 386 SURVIVORS OF MYOCARDIAL INFARCTION DURING THE FIRST YEAR OF FOLLOW-UP

The rectangle represents the total population of 386 survivors discharged without a pacemaker, while the circles represent population subgroups with one of the three labeled variables. The areas of overlap represent patients with both variables. The denominator of each fraction indicates the total number of patients in that subgroup, while the numerator indicates the number of patients experiencing syncope or sudden death. Note that 11 sudden deaths occurred in the 26 patients with a high-risk ECG pattern and nearly half (five of 11) of these sudden deaths occurred in patients with a high-risk ECG alone.

found to have the transvenous pacemaking wire within a well-endothelialized tract in the right atrium. In one of the two patients experiencing syncope, the episodes were proven to be related to periods of increased A-V block with asystole. Five patients were alive at the end of 1 year but, during the second year of follow-up, three more of these have died: one of refractory congestive heart failure and two suddenly. The data from this first group are in marked contrast to the second group which includes seven patients who progressed to higher degrees of A-V block via a type II mechanism and,

Table 5

Seventeen Patients with Acute Type II Progression Followed for 1 Year

<table>
<thead>
<tr>
<th>Group</th>
<th>Pts (no.)</th>
<th>Mean age (yrs) ±1 sd</th>
<th>Syncope or sudden death by 1 year</th>
<th>No. alive at 1 year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with transient type II progression</td>
<td>7</td>
<td></td>
<td>(2 syncope + 5 sudden death)</td>
<td>5 t</td>
</tr>
<tr>
<td>and no pacemaker inserted prior to discharge</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients with sustained type II progression</td>
<td>10*</td>
<td>68.2 ± 9.0</td>
<td></td>
<td>5 t</td>
</tr>
<tr>
<td>and permanent transvenous pacemaker therapy</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>prior to discharge</td>
<td>7</td>
<td>70.2 ± 10.2</td>
<td>0</td>
<td>6 t</td>
</tr>
</tbody>
</table>

*Includes one patient discharged with a pacemaker who died suddenly and at postmortem was found to have the transvenous pacing wire within a well-endothelialized tract in the right atrium.
†During the second year of follow-up, three more nonpaced survivors have died, two suddenly and one of refractory congestive heart failure. The paced group continues to remain free of sudden death or syncope.
because of failure to regain A-V conduction, were discharged with a permanent transvenous pacemaker in the right ventricle. During the first year of follow-up, none of the patients died suddenly and six patients were alive. During the second year of follow-up, there are still no sudden deaths in this group.

Discussion

The initial and subsequent clinical classes were analyzed to compare conduction status with an estimate of infarct size, i.e., the severity of hemodynamic compromise. These data show that, although groups with IVCD had a higher incidence of pulmonary edema and shock, the majority had either no failure (class I) or milder degrees of failure (class II). This is in contrast to several reports in the literature in which “nearly all patients were seriously ill...”, “shock or severe failure” occurred in 40 of 68 patients at some time during the hospital course, and 31 of 41 cases of bundle-branch block with myocardial infarction had a “severe infarction” or “cardiogenic shock”. The inclusion of isolated hemiblock does not account for the more favorable hemodynamics in our own series, as there were no significant differences in class data among the patients with unifascicular block and those with more than one fascicle involved.

Overall acute mortality data are intimately related to the patient’s clinical class and, in comparison with the literature, our mortality rate for fascicular block contrasts with reports of 56%, 62%, 56%, 56%, and 73%. Our data are more in agreement with Marriott and Hogan who report a 20% mortality in 40 CCU patients with either isolated LAHB, RBBB and LAHB or LPHB. Similarly, our data correspond with those of Scanlon et al. who report a 36% mortality in 22 patients with RBBB and left- or right-axis deviation. The reasons for these wide discrepancies in mortality rate are not immediately apparent but may relate to differences in CCU populations.

Our patients with fascicular block had an overall incidence of developing type II A-V block of 19% (table 3), and this risk increased with increasing degrees of fascicular involvement. It is of interest that first degree A-V block on the surface electrocardiogram is a sensitive discriminator between intermediate and high risks of type II progression for patients with adjacent fascicular block. Without His bundle electrograms, we cannot comment upon concomitant prolonged His-to-ventricle conduction times but, in these selected patients, first degree A-V block appears to predict involvement of the remaining fascicle, i.e., subsequent type II block. While pacemakers were not placed prophylactically in anticipation of the sudden onset of type II block, several considerations have caused us to change this policy for the future. First, we can identify a very high-risk subgroup for this complication. Second, the mortality rate in those patients progressing is no different from the overall mortality of patients with fascicular block. While our mortality rate is relatively lower than most reports in the literature, it is in agreement with the 33% mortality reported by Scanlon et al. Third, a standby pacemaker is capable of pacing patients through type II progression. Fourth, there is a theoretic rationale for making progression as controlled as possible in order to avoid the trauma of a cardiac arrest situation and the possible extension of infarct size.

Our follow-up data lead to several final observations. The electrocardiogram during the period of acute infarction can identify patients at different risk levels for syncope/sudden death after discharge from the hospital. For the patient with adjacent fascicular block, the presence of first-degree A-V block appears to identify a high-risk group for syncope or sudden death in follow-up (table 4). We believe that, in most instances, the prolongation of A-V conduction reflects disease in the remaining fascicle. His bundle electrograms should allow a more precise identification of the site of block and may allow further refinement of groups at risk for sudden death or syncope in follow-up.

The actual mechanism(s) of sudden death in our high-risk group is unknown. Primary ventricular arrhythmias, recurrent myocardial infarction with attendant arrhythmias, or progression to higher degrees of A-V block are three possibilities. Our data add credence to the third possibility that often, the sequence might be type II progression.
with resultant asystole and/or catastrophic ventricular arrhythmia. Type II progression seems likely in this high-risk group for three reasons: (1) patients who were at high risk for, or developed, transient type II A-V block acutely were also at highest risk for syncope/sudden death after hospital discharge; (2) sudden death or syncope has not occurred in patients surviving permanent type II A-V block who were discharged with permanent pacemakers, whereas this complication is frequent in a similar group of patients discharged without pacemakers; and (3) in one of the two patients experiencing and surviving syncope, the syncope was proven to be due to periodic type II A-V block. Our data are in agreement with those of Atkins et al.28 who studied a mixed population of patients with RBBB and LAHB (P-R interval not designated), including some with myocardial infarction, and found a high incidence of postdischarge sudden death in patients without pacemakers and a lesser incidence of sudden death if permanent pacemaker therapy was instituted.

The data in Figure 3 prompt the following observations concerning the identification of patient populations in whom a change in therapy might be indicated. For the 16 patients with a high-risk electrocardiogram alone, five (31%) experienced syncope or sudden death, and we believe the incidence of these events is high enough to warrant a prospective trial of pacemaker therapy in an attempt to prevent sudden death. For the 10 patients with a high-risk electrocardiographic pattern plus one of the other descriptors, six experienced syncope or sudden death. We cannot say whether or not a trial of pacer therapy is indicated in this group, as the accompanying descriptors generally indicate increasing severity of ASCVD and therefore risk of death from factors which theoretically would not be remediable by standby pacemaker therapy.

In summary, these data show that the electrocardiogram can identify a subgroup of patients at high risk (50%) for type II progression during a myocardial infarction. Based on the ability to identify this subgroup, plus our relatively low mortality data, we currently recommend prophylactic pacemaker insertion in these high-risk patients. These data also indicate that certain combinations of fascicular block can identify groups at high risk for syncope or sudden death in follow-up, implicate type II A-V block as a possible mechanism precipitating sudden death, and suggest that prophylaxis against sudden death may be offered by permanent pacemaker therapy. This last possibility could be evaluated in a prospective therapeutic trial.

Even though the actual mechanism of sudden death remains speculative, the study of the higher risk groups as identified by A-V conduction and intraventricular conduction patterns during the acute infarction may provide an informative focal point for the future study of the enigma of sudden death outside the hospital.

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