The Clinical Significance of Bundle Branch Block Complicating Acute Myocardial Infarction

1. Clinical Characteristics, Hospital Mortality, and One-Year Follow-Up

MICHAEL C. HINDMAN, M.D., GALEN S. WAGNER, M.D., MARLENE JA' Ro, A.B., JAMES M. ATKINS, M.D., MELVIN M. SCHEINMAN, M.D., ROMAN W. DESANCTIS, M.D., ADOLPH H. HUTTER, JR., M.D., LAWRENCE YEATMAN, M.D., MELVYN RUBENFIRE, M.D., CHANDRAKANT PUJURA, M.D., MICHAEL RUBIN, D.O., and JAMES J. MORRIS, M.D.

SUMMARY To provide an understanding of the clinical characteristics of patients with acute myocardial infarction (MI) and bundle branch block, experience from five centers was accumulated. Patients in whom bundle branch block first appeared after the onset of cardiogenic shock were excluded. In 432 patients, the most common types of block were left (38%) and right with left anterior fascicular block (34%). In 42% of the patients, bundle branch block was new. Progression to high degree (second or third degree) atrioventricular (AV) block via a Type II pattern occurred in 22% of the patients.

Hospital and first year follow-up mortality rates were 28% and 28%, respectively. Only 46% of the patients developed pulmonary edema or shock (Killip Class III or IV), and hospital mortality was related to the amount of heart failure (8%, 7%, 27%, 83% for Killip Classes I-IV, respectively). Patients with progression to second degree or third degree AV block via a Type II pattern had increased hospital mortality compared with patients without this complication (47% vs 23%, P < 0.001). In the absence of pulmonary edema or shock, patients with Type II second degree or third degree AV block still had a higher mortality rate than patients without advanced AV block (31% vs 2%, P < 0.005), with nearly all the deaths due to abrupt development of AV block.

Thus, in many patients MI with bundle branch block is associated with severe heart failure. However, this was not true for a majority of the patients, in whom therapy aimed at preventing morbidity and mortality due to the bradyarrhythmia of advanced AV block might be beneficial.

ACUTE CORONARY CARE with continuous electrocardiographic monitoring has resulted in a significant reduction in deaths from arrhythmias during acute myocardial infarction.1-3 Whereas the precipitating factors, natural history, and therapy for ventricular irritability and tachyarrhythmias are well-established,4-6 a comparable understanding of disturbances of intraventricular and atrioventricular (AV) conduction has not been achieved. It is recognized that in acute myocardial infarction complicated by bundle branch block, both mortality and the risk of complete heart block are increased. This is presumably a result of large extent of infarction necessary to involve the bundle branches.7-30 Data from several institutions provide information about the variety of conduction disturbances and the resulting hospital mortality.7-30 However, the numbers of patients in most of these studies are small; the groups of patients are not homogeneous, particularly with respect to infarct location and types and timing of onset of bundle branch block; and blocks which occur in the terminal phases of shock have often been included. These shortcomings have not permitted clear identification of patients at risk of progression to higher degrees of heart block or the contribution of the intraventricular conduction disturbances to the risk of dying.

Therefore, a multicenter project involving five institutions was designed to develop a common data bank containing a large number of patients with bundle branch block during myocardial infarction. This study presents the clinical characteristics of the patients and the hospital and first year follow-up mortality. The following paper in this issue defines the indications for insertion of prophylactic temporary and permanent pacemakers.

Methods

Data on all patients admitted to the coronary care units of five hospitals (Duke University Medical Center, Massachusetts General Hospital, Parkland Memorial Hospital, San Francisco General Hospital and Sinai Hospital of Detroit) were retrospectively searched to identify patients with documented acute
myocardial infarction complicated by the presence of bundle branch block. At the institutions where data were prospectively stored in a computerized data bank, patients were identified by searching for both documented myocardial infarction and bundle-branch block. Information was then retrieved from the computer and from the medical records of each patient. At the institutions where no data bank existed, patients were identified from coronary care unit records, and the medical records were examined. A flow sheet was constructed to record information pertaining to each patient’s age, sex, infarct location, presence and dates of old electrocardiograms, electrocardiographic evidence of prior infarct, type and timing of onset of intraventricular and AV blocks, worst Killip Class of heart failure, use of temporary and permanent pacemakers, status of conduction at discharge, causes of death during hospitalization, and follow-up data for a minimum of one year. Extensive efforts were made to render the identification of patients and the subsequent collection of data uniform at all institutions by reference to definition of the variables which had been entered prospectively into the Duke University computerized data bank. In addition, the data collection was completed by one of us reviewing all the primary data. Prior reports of patients with bundle branch block during myocardial infarction had come from the above institutions, but some patients previously included were excluded from this study because of inadequate data to meet the requirements of documented myocardial infarction or bundle branch block as specified in this study, or because of the lack of at least one year follow-up. Additional patients were also added from each of the institutions.

The description of the patients by hospitals and years accumulated is shown in table 1.

A majority of the patients were admitted because of a clinical history of chest pain which suggested acute myocardial infarction. A definite diagnosis of myocardial infarction was made if there were accompanying evolutionary electrocardiographic changes (ST-T wave changes and Q waves), and/or characteristic changes of serum enzymes (SGOT, LDH and CPK; or in recent years, isoenzymes of LDH and CPK) in the absence of other possible causes for elevation of these enzymes. If a patient was admitted more than once for a myocardial infarction, only the first admission in which the infarction was complicated by bundle branch block was analyzed. Subsequent admissions and complications were included with follow-up data. Patients with permanent pacemakers inserted before their acute infarctions were excluded from the study.

All available electrocardiograms for each patient were reviewed, including tracings before the infarction. Patients were continuously monitored in coronary care units during the acute phase of their infarctions, and had daily 12 lead electrocardiograms and frequent rhythm strips taken for at least the first three days. All tracings, including hourly monitoring strips when available, were analyzed for infarct location and the status of intraventricular and AV conduction.

The location of old and new infarctions was noted according to the criteria of Lipman and Massie and included anterior, inferior, posterior and lateral. These criteria plus evolutionary ST-T wave changes in the appropriate leads were required to document and specify the site of infarction. Site was considered indeterminant when there were only ST segment or T wave changes or when the presence of bundle branch block prevented evaluation of the QRS.

The presence and type of intraventricular defect was determined according to the criteria of Rosenbaum and Hecht et al. All patients had at least one electrocardiogram with QRS prolongation ≥ 0.12 sec during AV association. Patients with rate-dependent bundle branch block were excluded. The specific types of blocks and their definitions are as follows: 1) right bundle branch block (RBBB) requires a QRS duration ≥ 0.12 sec with terminal forces directed anterior and rightward; 2) left bundle branch block (LBBB) requires a QRS duration ≥ 0.12 sec with terminal forces directed posterior and leftward; 3) RBBB plus left anterior fascicular block (LAFB) is defined as RBBB with a leftward QRS axis between −30 and −90°, with the axis abnormality not a result of pathologic Q waves; 4) RBBB plus left posterior fascicular block (LPFB) is defined as RBBB with a rightward QRS axis between +120 and +180° in the absence of lateral wall infarction, right ventricular hypertrophy or history of symptomatic chronic lung disease or cor pulmonale; and 5) alternating bundle branch block (ABB) requires the presence of LBBB and RBBB or RBBB plus both LAFB and LPFB on serial electrocardiograms during infarction. The terms unifascicular, bifascicular, and trifascicular block are used as defined by Rosenbaum.

The status of AV conduction was noted in all patients. First degree (1°) AV block requires a PR interval > 0.20 sec. Second degree (2°) AV block is classified as Mobitz types I and II according to standard criteria. Type I block is characterized by the presence of variable PR intervals of conducted beats and Type II block is characterized by constant PR intervals despite variations in PR intervals. Third degree (3°) AV block involves complete AV dissociation with the ventricular rate less than 60 beats/min and non-conducted P waves occurring outside the ventricular refractory period. “High degree AV block” is defined as Type II 2° AV block or 3° AV block which develops after Type II 2° block or without recognized 2° block. High degree AV block does not include 3° AV block preceded by Type I 2° block. Three patients

### Table 1. Breakdown of Patients with Myocardial Infarction and Bundle Branch Block by Hospital

<table>
<thead>
<tr>
<th>Hospital</th>
<th>No. of patients</th>
<th>Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Massachusetts General</td>
<td>128</td>
<td>1967–1974</td>
</tr>
<tr>
<td>Parkland Memorial</td>
<td>85</td>
<td>1969–1974</td>
</tr>
<tr>
<td>San Francisco General</td>
<td>60</td>
<td>1970–1974</td>
</tr>
<tr>
<td>Sinai of Detroit</td>
<td>44</td>
<td>1971–1973</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>494</strong></td>
<td></td>
</tr>
</tbody>
</table>
in whom 2° AV block was manifested by 2:1 AV conduction were handled as follows: In a patient with indeterminant location infarction, 2:1 block and then 3° AV block, the block was coded as Type II; in a patient with an inferior infarction and, at worst, 2:1 AV block, and in a patient with an indeterminant location infarction and 1° AV block which progressed to 2:1 AV block, the blocks were coded as Type I.

The timing of onset of intraventricular and AV blocks was determined by review of old, admission and serial electrocardiograms. The following terminology is used: 1) definitely old — block present on an electrocardiogram antedating infarction; 2) possibly new — block present on the first electrocardiogram with infarction but with no old electrocardiogram available for comparison; 3) probably new — block present on the first electrocardiogram with infarction but absent on electrocardiograms taken within two years; and 4) definitely new — block which develops in the hospital, documented by serial tracings. Bundle branch block which is possibly or probably new is referred to as "indeterminant onset."

The worst class of heart failure for each patient was obtained by review of the clinical record. These were designated Classes I-IV as defined by Killip and Kimball:30 Class I, no heart failure; Class II, mild heart failure manifested by basilar rales and/or an S2 gallop; Class III, pulmonary edema, determined by the presence of dyspnea and S2 gallop, pulmonary rales, and roentgenographic findings compatible with pulmonary edema; and Class IV, cardiogenic shock, manifested by hypotension (systolic pressure < 90 mm Hg), oliguria (< 20 ml/hr), and poor perfusion to the skin and/or central nervous system. The term "power failure" is used to include patients with Class III or IV failure. Patients with bundle branch block or high degree AV block occurring only during the terminal phases of cardiogenic shock have been analyzed separately and are excluded from this data analysis.

Power failure was noted as the cause of death during hospitalization if cardiogenic shock or sudden and progressive pulmonary edema preceded a terminal arrhythmia. Ventricular fibrillation or complete heart block were listed as causes of death only if they occurred in the absence of power failure. Other cardiovascular, unrelated, and unknown causes of death were also noted.

No protocols existed to guide decisions pertaining to temporary or permanent pacemaker insertion. Pacemaker insertion was performed at the physicians' discretion in each case. However, after reports from two institutions in 1973 suggested that long-term survival was improved and the risk of late sudden death decreased if patients received permanent pacemakers after transient high degree AV block,18,30 many patients who experienced high degree AV block and then survived hospitalization were discharged with permanent pacemakers.

In evaluating hospital mortality after myocardial infarction with bundle branch block, a control group of 887 patients who never had bundle branch block and survived their infarctions was obtained from the Duke Computerized Data Bank. Infarct location, worst Killip class of heart failure, and electrocardiographic evidence of old myocardial infarction were noted in these patients. The interaction of these variables in determining hospital mortality was examined and compared to the patients with bundle branch block during myocardial infarction.

Follow-up data were obtained from hospital records, return visits to the hospital, private physicians or clinics, or by verbal and/or written contact with patients, their families, or their physicians. The incidence of recurrent myocardial infarction, power failure or new conduction defects was noted. For the patients dying during the follow-up period, the circumstances surrounding the death were determined from an immediate observer, from an attending physician, or from hospital records. The causes of death were categorized as follows: 1) documented recurrent myocardial infarction, 2) refractory congestive heart failure, 3) other cardiovascular causes, 4) unrelated causes, 5) unknown causes and 6) sudden death. The cause of death was listed as sudden if, in the absence of a gradually deteriorating clinical situation, the time interval from the onset of symptoms to death was less than 2 hours. Follow-up for at least one year after myocardial infarction complicated by bundle branch block was obtained in all patients.

Differences between subsets of patients compared were tested for significance utilizing two-way tables and the standard chi square distributions. P values < 0.05 were considered to be statistically significant.

Results

Criteria for inclusion in this study were met by 494 patients consecutively admitted to the five institutions during the years shown in Table 1. An additional 10 patients met criteria for acute myocardial infarction and bundle branch block but were lost to follow-up before one year after discharge. In 62 of the 494 patients (12%), cardiogenic shock occurred before the development of intraventricular or AV blocks. The remaining 432 patients included 281 males (65%) and 151 females. Their ages ranged from 23–94 years with a mean age of 65 ± 12 years. The infarctions were anterior in 272 patients (63%), inferior or posterior in 79 (18%), indeterminant in 77 (18%) and lateral in four (1%). The four lateral infarctions have been arbitrarily considered with the inferior-posterior infarctions for data analysis. Eighty-one patients (19%) had electrocardiographically documented previous myocardial infarctions.

Hospital, late (during one-year follow-up), and total mortality is summarized in Table 2. Thirty-five percent of the patients died during hospitalization (175 patients). Hospital mortality was 28% in the 432 patients without shock before the development of major conduction disturbance. Of the 62 patients with cardiogenic shock preceding development of bundle branch block or high degree AV block, 52 died in the hospital (84%) and two died during the first year of follow-up. The patients with shock preceding major
conduction disturbances have been excluded from further data analysis.

Bundle Branch Block

Table 3 shows the incidence of the various types of bundle branch block and the timing of onset of the block. LBBB was observed in 163 patients (38%) and RBBB + LAFB in 149 patients (34%). The remaining 120 patients demonstrated one of the other intraventricular conduction disturbances (RBBB, RBBB + LPFB, or ABBB). In 180 patients (42%) bundle branch block was definitely new, and in 91 patients (21%) bundle branch block definitely preceded the infarction.

In 53 patients, unifascicular block involving the anterior or posterior fascicles of the LBBB was the initial intraventricular conduction disturbance. Since the patients were selected by having had a QRS duration 0.12 sec at some time during the course of infarction, RBBB + LAFB or RBBB + LPFB subsequently developed in 34 of these patients, LBBB in 15 patients, and ABBB in the four other patients. Isolated RBBB was initially observed in 58 patients. Twenty-two of these patients developed bifascicular block (RBBB + LAFB or RBBB + LPFB); two patients progressed to ABBB; and 34 patients maintained their RBBB without bi- or trifascicular blocks.

Of 309 patients discharged from the hospital, 72 (23%) had normal intraventricular conduction at the time of discharge and another 29 (9%) were discharged with unifascicular block of the anterior or posterior fascicles of the left bundle branch. Forty-two patients (14%) were discharged with RBBB. The remaining 166 patients (54%) had bifascicular block at discharge.

### AV Block

Only 208 patients (48%) maintained normal AV conduction throughout the course of their myocardial infarctions. First degree AV block occurred in 169 patients (39%). Of these patients, 101 (60%) never progressed beyond 1° AV block, 43 (25%) progressed to high degree AV block and 25 (15%) developed Type I 2° block or 3° AV block preceded by Type I block.

Second or third degree AV block developed in 123 patients (28%). In 19 patients (4%) Type I 2° AV block was the highest degree of block and in another nine patients (2%) 3° AV block was preceded by Type I block. The remaining 95 patients (22%) developed high degree AV block: seven with Type II 2° AV block only, 26 with 3° AV block developing after Type II 2° AV block, and 62 with 3° AV block not preceded by 2° AV block. The course of AV conduction in the 95 patients with high degree AV block is summarized in table 4.

### Heart Failure

Fifty-four percent of patients never developed power failure despite bundle branch block complicating myocardial infarction: 15% remained free of heart failure (worst Killip Class I) and another 39% had, at

<table>
<thead>
<tr>
<th>Type of BBB</th>
<th>Definite Old</th>
<th>Possible New</th>
<th>Probable New</th>
<th>Definite New</th>
<th>Total (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBBB</td>
<td>40</td>
<td>35</td>
<td>26</td>
<td>62</td>
<td>163 (38)</td>
</tr>
<tr>
<td>RBBB</td>
<td>16</td>
<td>13</td>
<td>3</td>
<td>16</td>
<td>48 (11)</td>
</tr>
<tr>
<td>RBBB + LAFB</td>
<td>27</td>
<td>38</td>
<td>27</td>
<td>57</td>
<td>149 (34)</td>
</tr>
<tr>
<td>RBBB + LPFB</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>21</td>
<td>45 (10)</td>
</tr>
<tr>
<td>ABBB</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>25</td>
<td>27 (6)</td>
</tr>
<tr>
<td>Total (%)</td>
<td>91 (21)</td>
<td>95 (22)</td>
<td>65 (15)</td>
<td>181 (42)</td>
<td></td>
</tr>
</tbody>
</table>

*The numbers represent total number of patients with each respective type and timing of onset of bundle branch block. The numbers in parentheses refer to percentage of total number of patients.

Abbreviations: BBB = bundle branch block; LBBB = left bundle branch block; RBBB = right bundle branch block; LAFB = left anterior fascicular block; LPFB = left posterior fascicular block; ABBB = alternating bundle branch block.
worst, mild heart failure (Class II). Twenty-three percent developed pulmonary edema (Class III), and 23% developed cardiogenic shock (Class IV) after bundle branch block occurred.

Patients with RBBB had a 35% incidence of power failure, compared to an average incidence of 47% (range 45–52%) with the other blocks (P < 0.025). Patients with old blocks had a 34% incidence of power failure, compared to 46% with indeterminant onset and new blocks (P < 0.1). Power failure developed in 63% of the 95 patients with high degree AV block, in 55% of the nine patients with 3° AV block preceded by Type I 2° AV block, in 37% of the 19 patients with only Type I 2° AV block, and in 20° of the 309 patients with neither 2° nor 3° AV block. Patients with anterior or indeterminant location infarcts had a 48% incidence of power failure while patients with inferior or posterior infarctions had a 39% incidence (P < 0.1). The average ages of patients with and without power failure were not significantly different.

Hospital Mortality

Seventy-six percent of the 123 hospital deaths were the result of power failure. Nine percent were due to abrupt 3° AV block in the absence of cardiogenic shock (15 patients). These 15 deaths included nine patients who died when pacemakers could not be successfully inserted after sudden progression to high degree AV block, five patients who had had previously functioning pacemakers removed or turned off, and one patient who had pacemaker malfunction (failure to capture). The remaining 25 deaths included primary arrhythmias, other cardiovascular causes, and unrelated or unknown causes. Only seven of these patients died as a result of primary ventricular arrhythmias.

Table 5 summarizes hospital, first-year follow-up, and total one-year mortality in different subsets of patients. Patients with documented previous myocardial infarctions had a significantly higher hospital mortality than patients without previous infarctions (42% vs 25%, P < 0.001). Patients with acute inferior or posterior infarctions had a 17% hospital mortality, while patients with acute anterior or indeterminant location infarcts had a 31% mortality (P < 0.01).

Patients with ABBB or RBBB + LPFB had a higher hospital mortality than patients with the other intraventricular conduction defects (40% vs 26%, P < 0.025). Table 6 shows that this increased mortality is a result of a 41% hospital mortality rate in 46 patients with new RBBB + LPFB or new ABBB. In contrast, 43 patients with old RBBB + LAFB had an 18% hospital mortality. Patients with old, indeterminant, or new onset blocks otherwise had similar mortality rates (26–29%).

Hospital mortality was directly related to the degree of heart failure. Only 7% of patients with no heart failure or, at worst, mild heart failure died, compared to 27% of patients with pulmonary edema and 83% of patients with cardiogenic shock. Thus, the development of power failure subsequent to conduction blocks was associated with a markedly increased hospital mortality (53% mortality with power failure vs 7% without, P < 0.001). The influence of AV block and infarct location on hospital mortality is summarized in table 7. First degree AV block was not associated with increased mortality.

### Table 5. Determinants of Hospital, First-Year Follow-up and Total One-Year Mortality in Patients with Acute Myocardial Infarction and Bundle Branch Block*

<table>
<thead>
<tr>
<th>Determinants</th>
<th>No. of patients</th>
<th>Hospital Mortality (%)</th>
<th>One-year follow-up (%)</th>
<th>Total Mortality (%)</th>
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<tr>
<td>Previous MI</td>
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<tr>
<td>Yes</td>
<td>81</td>
<td>42</td>
<td>38</td>
<td>64</td>
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<tr>
<td>No</td>
<td>351</td>
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<td>Infarct Location</td>
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<td>Ant-Ind</td>
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<td>52</td>
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<td>Inf-Post</td>
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<td>17</td>
<td>17</td>
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<td>Type BBB</td>
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<tr>
<td>LBBBB</td>
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<td>RBBBB</td>
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<td>RBBB + LAFB</td>
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<td>RBBB + LPFB</td>
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<td>Old</td>
<td>91</td>
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<td>47</td>
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<td>Indeterminant</td>
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<td>New</td>
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<td>Killip Class I</td>
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<td>High degree block</td>
<td>95</td>
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*Definitions as in methods.
Abbreviations: MI = myocardial infarction; Ant-Ind = anterior or indeterminant location infarcts; Inf-Post = inferior or posterior infarct locations; BBB = bundle branch block; LBBBB = left bundle branch block; RBBBB = right bundle branch block; LAFB = left anterior fascicular block; LPFB = left posterior fascicular block; ABBB = alternating bundle branch block; AV = atrioventricular.

### Table 6. Hospital Mortality in Subgroups of Patients Described by Type and Onset of Bundle Branch Block*

<table>
<thead>
<tr>
<th>Type of BBB</th>
<th>Definite Old (%)</th>
<th>Indeterminant (%)</th>
<th>Definite New (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBBBB</td>
<td>30</td>
<td>24</td>
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<td>RBBBB</td>
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<td>RBBB + LAFB</td>
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<tr>
<td>RBBB + LPFB</td>
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<td>29</td>
<td>43</td>
</tr>
<tr>
<td>ABBB</td>
<td>100</td>
<td>100</td>
<td>40</td>
</tr>
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</table>

*The numbers refer to the percentage of patients with each respective type and timing of onset of bundle branch block who died during hospitalization. Abbreviations: See table 5.
The influence of degrees of atrioventricular infarct location on hospital mortality (Table 7) showed that patients with AV block or 3° block had higher mortality compared to those without. The numbers represent the number of patients in each subset, and the mortality rates are for the respective subsets of patients.

The numbers in parentheses represent the mortality rates for the respective subsets of patients. Abbreviations: AV = atrioventricular; Inf = inferior; Post = posterior; Ant = anterior; Ind = indeterminant.

Mortality. The 28 patients who developed Type I 2° AV block or 3° block preceded by Type I block likewise were not at increased risk of dying (29% mortality). The majority of the deaths in this group of patients occurred with anterior or indeterminant infarctions (six patients) with 4° AV block (38% mortality).

The 95 patients who progressed to high degree AV block had more than two times the hospital mortality of patients who never progressed beyond 1° AV block (47% vs 23%, <0.001). In these patients, mortality was increased whether the AV block was isolated Type II 2° AV block (43%), 3° AV block preceded by Type II block (50%), or 3° AV block without preceding 2° AV block (55%); or whether the infarct was inferior or posterior (41%) or anterior or indeterminant (55%).

The influence of high degree AV block, power failure and infarct location on hospital mortality is demonstrated in Table 8. Only 2% of the 198 patients who never developed high degree AV block and had, at worst, Killip class II failure, died, whereas 31% of 36 patients with high degree AV block but no power failure died (<0.005). Nine of the 11 deaths in this latter group of patients occurred suddenly as a result of abrupt progression to high degree AV block, and one was due to brain death following successful resuscitation after high degree block. Fifty percent of patients with power failure but without high degree AV block died, and hospital mortality was higher in these patients if the acute infarction was anterior or of indeterminant location (54%) than if it was inferior or posterior (20%) (<0.02). Mortality was highest (65%) in patients who had high degree AV block and power failure, regardless of infarct location. When examined with the above three variables, the presence of a documented prior infarction did not assist in stratifying the risk of hospital death for any of the patients with power failure or for patients with high degree AV block and no power failure. However, patients with a prior infarction, at worst Killip class II failure, but no high degree AV block with their acute infarction, had a higher mortality (16% of 32 patients) than patients with at worst Killip class II failure, no high degree AV block, and no prior infarction (2% of 166 patients) (<0.003).

To examine the influence of bundle branch block on hospital mortality, the Duke University Computerized Data Bank was searched for a control group as described in the Methods section. Between 1967–1976, 887 patients had documented myocardial infarctions not complicated by bundle branch block. The average age of this control group was 62 ± 12 years. Forty-six percent had documented prior infarctions, 42% had acute inferior or posterior infarctions, and 21% developed power failure with acute infarction. Hospital mortality was 1) 9% in patients with inferior or posterior infarctions, compared to 14% in patients with anterior or indeterminant location infarctions; 2) 14% in patients with prior infarctions, compared to 10% in patients without prior infarctions; and 3) 49% in patients with power failure, compared to 2% in patients without significant heart failure. When these variables were examined together, mortality was low (0–7%) in all patients without power failure, and high (40–57%) in all patients with power failure, regardless of infarct location or the presence or absence of a documented prior myocardial infarction. In addition, hospital mortality in patients with Type I 2° AV block or 3° AV block preceded by Type I block was 9% if the infarction was inferior or posterior (78 patients) and 17% if the infarction was anterior or of indeterminant location. Mortality was higher if 3° AV block developed without an intermediate stage of 2° AV block: 83% in six patients with anterior or indeterminant infarction and 40% in five patients with inferior or posterior infarction.

Table 9 compares hospital mortality in patients with and without bundle branch block during acute myocardial infarction. The incidence of power failure was higher in patients with bundle branch block than in those without blocks (46 vs 21%, <0.0001), but the mortality associated with the development of power failure was similar for patients with and without bundle branch block, regardless of infarct location. Although low, mortality in patients with
bundle branch block but no power failure (7%) was higher than in patients with neither bundle branch block nor power failure (2%) (P < 0.001).

**Follow-Up**

The mortality rate during the first year of follow-up was 28% in the 309 patients discharged from the hospital after myocardial infarction and bundle branch block but no shock prior to major conduction defects (table 2). Forty-five patients (15%) died suddenly, 22 (7%) died of power failure and 19 (6%) died from other cardiovascular and non-cardiovascular causes. Nineteen patients had documented recurrent myocardial infarction, and 13 of these patients (68%) died.

First year follow-up mortality was not significantly influenced by the amount of heart failure during infarction (table 10). Twenty-five percent of patients without power failure before discharge died during the year following their infarction, while 33% of patients with power failure died (P < 0.1).

Patients with previous myocardial infarctions had higher first year follow-up and total mortality rates than patients without prior infarctions (follow-up 38% vs 26%, P < 0.001; total 64% vs 44%, P < 0.001).

First year follow-up and total mortality was lower in patients with inferior or posterior infarctions than in patients with anterior or indeterminant infarctions (follow-up, 17% vs 31%, P < 0.05; total, 31% vs 52%, P < 0.001). Mortality rate during the year following infarction was highest in patients with indeterminant location infarcts (46%), reflecting a higher incidence of death during the first year of follow-up in patients with LBBB or ABBB (33%) compared to patients with RBBB, RBBB + LAFB, or RBBB + LPFB (23%) (P < 0.05).

Fifteen percent of the patients with normal intraventricular conduction at the time of discharge from the hospital after myocardial infarction died during the subsequent year, and 14% of the patients discharged with LAFB or LPFB died during the first year of follow-up. Patients discharged with RBBB (24% mortality) or bifascicular block (37% mortality) had a significantly higher mortality than the patients discharged without bundle branch block (P < 0.001).

**Discussion**

Bundle branch block has been reported to be present at some time during hospitalization in 13% of patients with acute myocardial infarction. The frequency of occurrence of the different types of bundle branch blocks in this study is similar to previous reports as reviewed by Mullins and Atkins. LBBB and RBBB + LAFB are the most common, occurring at about the same frequency, and isolated RBBB, RBBB + LPFB, and ABBB are less common. In previous studies which classified bundle branch blocks by the timing of onset, the average frequency of oc-

<table>
<thead>
<tr>
<th>Infarct location</th>
<th>No.</th>
<th>Hospital mortality</th>
<th>Power failure</th>
<th>Type I progression</th>
<th>Type II progression</th>
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<td>One-year follow-up</td>
<td>Hospital incidence</td>
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<td>58</td>
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Type I progression = Type I block or type I + 3° AV block.
Type II progression = Type II block, type II + 3° AV block, or sudden 3° AV block.
Abbreviations: Ant = anterior; Ind = indeterminant; Inf = inferior; Post = posterior.
currence of old, indeterminant, and new onset blocks during myocardial infarction is approximately 33% for each group. This compares to the present study in which 21% of the blocks were old, 37% were of indeterminant onset and 42% were observed to occur during infarction.

The incidence of high degree AV block during myocardial infarction complicated by bundle branch block was 22%, and an additional 2% of the patients developed 3° AV block after Type I 2° AV block. The average incidence of Type II 2° AV block and or complete heart block is 24% (range 8-47%) in previous reports. Over one-third of the patients who developed complete heart block did so suddenly without prior 1° or 2° block, and a majority progressed to 3° AV block without an intermediate stage of 2° block. Thus, when bundle branch block complicates acute myocardial infarction, the intraventricular conduction disturbance may be the only warning of jeopardized AV conduction and impending precipitous complete heart block.

The in-hospital mortality of myocardial infarction, including infarctions complicated by bundle branch block, averages 15-20%. The 28% mortality rate in the 432 patients with bundle branch block is significantly higher than the 12% mortality for a control group of patients without bundle branch block; however, this mortality rate is lower than the 44% mortality (range 19-74%) for bundle branch block during myocardial infarction reported in the literature. The wide range of mortality figures and the difference between this study and those previously reported probably reflects different CCU populations and our exclusion of patients with bundle branch block occurring as a preterminal event in the setting of severe power failure, since complete heart block, asystolic arrest, and ventricular arrhythmias all commonly occur in this setting even in the absence of bundle branch block.

The specific types and timing of onset of bundle branch block have been noted in the literature to influence hospital and follow-up mortality, but the results have been variable. Some studies have demonstrated a lower mortality in patients with LBBB than in patients with isolated RBBB or bifascicular block involving the right bundle branch, however, other studies, including this one, have demonstrated equal or higher mortality with LBBB, often associated with larger areas of infarction and severe three vessel coronary artery disease. In their review of the literature, Mullins and Atkins found that mortality rates were similar for the various blocks (44-57%), and was highest for the small number of reported patients with RBBB + LPFB. This is similar to the results of this study, in which the hospital and total one-year mortality in patients with RBBB and LPFB is exceeded only by the mortality rates of ABBB.

Since chronic intraventricular conduction disturbances are more often the result of fibrosis of the conduction system rather than atherosclerotic cardiovascular disease, the incidence of death due to power failure during myocardial infarction in patients with preexisting bundle branch block might not be expected to be significantly higher than in patients without bundle branch block. Furthermore, patients with preexisting blocks might be expected to have a lower incidence of death from power failure than patients who develop new bundle branch block, presumably because the bundle branch blocks indicate more extensive myocardial damage in the latter patients. This effect of the timing of onset of block on mortality was demonstrated by Lie et al. for patients having RBBB with or without fascicular blocks, and in the present study for patients having RBBB + LAFB. Other investigators have not found that the age of the bundle branch block significantly influences mortality. When bundle branch block complicates acute myocardial infarction, the site of infarction is usually anteroseptal. The relatively small number of patients with inferior or posterior infarctions had a lower risk of dying during or after hospitalization than patients with anterior or indeterminant location infarcts. The reason for this lower mortality rate is not clear. The patients with inferior or posterior infarctions had fewer documented prior myocardial infarctions than the other patients, and although the incidence of power failure was similar for the different infarct locations, mortality was lower in patients with inferior or posterior infarctions and power failure than in patients with anterior or indeterminant location infarcts and power failure. The incidence of high degree AV block and the mortality associated with this complication were similar for the different infarct locations. A Type I pattern of AV block was more common in patients with inferior or posterior infarctions than in patients with other infarct location; however, of the patients with Type I progression, only those with anterior or indeterminant location infarcts and 3° AV block which developed after Type I block were at increased risk of dying. This is in contrast to previous reports of increased mortality associated with Type I progression during myocardial infarction. At least in the high risk subset of patients with bundle branch block and Type I progression, the Type I block may be a manifestation of involvement of the His-Purkinje system, as suggested by Puech et al., rather than of the more proximal conduction system. The incidence of transient bundle branch block was similar for the different infarct locations. These data suggest that either the amount of viable diseased myocardium was greater in patients without anterior wall involvement, or that bundle branch block with inferior or posterior infarctions could be due to involvement of the more proximal portion of the conduction system supplied by the AV nodal artery. The resulting myocardial damage would be less extensive than with the more diffuse involvement of the distal conduction system which occurs with anterior infarctions. Data pertaining to infarct location are summarized in table 9.
result of progressive and irreversible hemodynamic deterioration has been stressed in the literature. This study confirms the common occurrence of pulmonary edema and cardiogenic shock in patients with bundle branch block during acute myocardial infarction, as the incidence of power failure in this study is significantly higher than the incidence in a control group without bundle branch block during infarction. This higher incidence of power failure in patients with myocardial infarction complicated by bundle branch block is probably due to 1) the extensive myocardial damage necessary to cause involvement of the intraventricular conduction system when the bundle branch block is new, and 2) the presence of underlying myocardial and/or coronary artery involvement when the block is old. The risk of dying is slightly higher if both power failure and high degree AV block occur with bundle branch block (65%) than if only power failure occurs (50%); however, the hospital mortality of patients with power failure is similar with and without bundle branch block (53% and 49%). Thus, the poor prognosis for most patients with bundle branch block and power failure is a result of the extent of myocardial damage causing pulmonary edema or cardiogenic shock rather than the result of the conduction disturbance.

The observation that nearly half of the patients with bundle branch in this study had either no heart failure or, at worst, mild heart failure, should be emphasized, for in this group of patients high degree AV block influences mortality independently of power failure. Death during myocardial infarction not complicated by power failure is usually due to ventricular fibrillation, sudden apparent electromechanical dissociation, or non-cardiovascular causes. In patients with bundle branch block, the abrupt development of bradyarrhythmias due to advanced AV block may cause death in the absence of power failure. Indeed, in this study, mortality was higher in patients with bundle branch block and no power failure than in a control group of patients with neither bundle branch block nor power failure. This increased mortality was due to high degree AV block: While only 2% of patients with neither power failure nor high degree block died in the hospital, 28% (10 of 11 deaths in 36 patients) of patients with high degree block but no power failure died as a result of the abrupt development of complete heart block. Thus, while many patients who experience high degree AV block die because of widespread myocardial necrosis even if pacemaker therapy is instituted, some patients without significant heart failure before progression of conduction disturbance might benefit from prophylactic insertion of a temporary pacemaker by avoiding the hemodynamic insult of bradycardia with sudden complete heart block.

Hospital survivors of myocardial infarction complicated by bundle branch block have an increased risk of dying during the first year following infarction compared to patients who survive myocardial infarction not complicated by bundle branch block. Patients with LBBB or ABBB had a slightly higher incidence of death during the first year of follow-up than patients with RBBB or bifascicular blocks involving the right bundle branch, perhaps because of the extent and severity of coronary artery disease and myocardial involvement. The status of discharge intraventricular conduction influenced follow-up mortality. The lower incidence of death during follow-up in patients with normal intraventricular conduction or unifascicular blocks involving the anterior or posterior fascicles of the left bundle branch suggests the importance of permanent and extensive myocardial damage in determining long-term prognosis after myocardial infarction complicated by bundle branch block.

Thus, the occurrence of bundle branch block in acute myocardial infarction is important because it indicates that infarction may be extensive and may result in cardiac failure or death; however, the presence of bundle branch block is also important because it indicates seriously jeopardized AV conduction which could result in sudden extremely slow heart rates or asystolic arrest. Therapeutic intervention with prophylactic temporary and permanent pacemaker insertion might therefore decrease the risk of death during and after hospitalization if high-risk subsets of patients with myocardial infarction complicated by bundle branch block can be identified.

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