Clinical and Anatomic Implications of Intraventricular Conduction Blocks in Acute Myocardial Infarction

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SUMMARY
The electrocardiogram in 480 patients with acute myocardial infarction showed right bundle-branch block (RBBB) with normal QRS axis in 18 patients (3.7%), left bundle-branch block (LBBB) in 31 (8%), RBBB and left anterior hemiblock (LAH) in 23 (4.8%), RBBB and left posterior hemiblock (LPH) in four (1%), LAH alone in 20 (4%), LPH in one (0.2%), and no evidence of intraventricular conduction (I-V) disturbance in 383 (80%). Eighteen of the 97 patients with I-V block showed 1° A-V block, and seven of the 18 (39%) showed abrupt progression to high-grade A-V block, while only six of 79 (8%) without 1° A-V block showed similar progression.

Cause of death in patients with I-V block was cardiac failure and/or shock in 92%; only three instances of primary asystole occurred. The incidence of complete heart block was higher in the I-V disease group (15%) than in the group without block (5%), but not significantly. Patients with LAH or RBBB and LAH usually had occlusion of the left anterior descending artery with extensive septal infarction, while patients with RBBB or LBBB had a more variable pattern of vessel involvement. Presence of I-V block in patients with acute myocardial infarction implies a hectic clinical course with poor prognosis, but does not justify prophylactic temporary transvenous intracardiac pacing except perhaps in the subgroup with associated 1° A-V block.

Additional Indexing Words:
Right bundle-branch block  Left bundle-branch block  Left anterior hemiblock
Left posterior hemiblock  Fascicular block

CONTROVERSY EXISTS concerning the medical management of patients with acute myocardial infarction and electrocardiographic findings of fascicular blocks. The intent of the present study was to review our 4 years of experience with patients who had myocardial infarction complicated by intraventricular conduction disturbances admitted to the Coronary Care Unit (CCU) at San Francisco General Hospital. Specifically we were concerned with the incidence of fascicular blocks and the relationship of these disturbances to the other major complications of acute myocardial infarction. Whenever possible, blocks were correlated with postmortem studies of the coronary arteries and the site of infarction. Based on these observations, recommendations regarding general medical management including the rationale of temporary prophylactic intravenous pacing in this group of patients are reported.

Materials and Methods
All patients (480) with the diagnosis of acute myocardial infarction admitted to the CCU from July 1967 to June 1971 were studied. All patients with symptoms suggestive of coronary insufficiency were routinely admitted directly to the CCU.
and in only very rare instances were patients with documented acute myocardial infarction admitted to the general medical wards. Patients with uncomplicated acute myocardial infarction were kept in the CCU for 5 days, and at least three 12-lead electrocardiograms, taken on different days, were available for review. When the clinical course was complicated by arrhythmias, intraventricular conduction disturbances, or pump failure, coronary care was prolonged and thus more electrocardiograms were available for interpretation. Most patients were monitored with a modified V1 lead (CL4), and nurses were instructed to record and incorporate all strips showing arrhythmias in the medical record. A 12-lead ECG was obtained when prolongation of the QRS was noted on the monitor. In addition, ECG rhythm strips from a 12-sec memory loop were available for review after July 1969.

Patients were managed by the house staff under the supervision of the cardiology attending staff. The vast majority of patients were seen at least once daily by the senior author. Temporary transvenous intracardiac pacing was generally instituted only in cases of advanced or complete atrioventricular (A-V) block. Patients were followed until discharge from the hospital or death (including patients transferred to another hospital). The data were recorded on standard data cards and processed by a specially designed program on an IBM 360/50 digital computer. The chi-square method was used for statistical analyses, and P values of less than 0.05 were considered significant.

**Criteria**

The diagnosis of acute myocardial infarction was made according to modifications of the following definitions proposed by the Rand-California Regional Medical Program's Coronary Care Coordinating Committee: Patients with (1) deep (0.04-sec or greater) Q waves with evolutionary ST-T changes, or (2) with history of constricting, dull, or oppressive precordial pain lasting longer than 30 min (or pain abbreviated by syncope or cardiovascular collapse) associated with a positive enzyme curve and equivocal ST-T changes were diagnosed as having an acute myocardial infarction. For inclusion into this study, serial enzyme determinations produced a curve typical for that enzyme in patients with acute myocardial infarction as far as rapidity of rise and fall. In addition, the highest value exceeded the normal range and was at least 50% greater than the lowest. Serum enzyme levels of creatine phosphokinase, glutamic oxaloacetic transaminase, and lactic dehydrogenase were used in the present study.

The following criteria for major complications, also proposed by the Coronary Care Coordinating Committee, were employed.

**Shock:** A state characterized by hypotension (<80 mm Hg systolic arterial pressure in a previously normotensive person and <100 mm Hg in a previously hypertensive patient) for at least 1 hour accompanied by evidence of poor peripheral perfusion including oliguria (<25 ml/hour).

**Severe Congestive Heart Failure:** A state characterized by severe dyspnea, diffuse pulmonary rales or wheezes, and roentgenographic findings compatible with pulmonary edema.

**Pump Failure:** Severe congestive heart failure and/or shock.

- **First-Degree A-V Block:** (1° A-V): Prolongation of the P-R interval above 0.21 sec for heart rate <100 beats/min.
- **Second-Degree A-V Block** (2° A-V): Both Mobitz I (Wenckebach) and II types of block.
- **Advanced A-V Block:** When half or more of the P waves are not followed by the QRS.
- **Complete A-V Block:** No atrial impulses are transmitted to the ventricle with an atrial rate <100 beats/min.

**Primary Asystole:** Cardiac asystole occurring in a patient without severe congestive heart failure or shock.

Arrhythmias occurring during the terminal phases of infarction were excluded from analysis. Other complications were diagnosed according to the following criteria.

**Fascicular Blocks:** Left anterior hemiblock (LAH) (modified from Rosenbaum) was diagnosed when the electrocardiogram showed narrow (<0.02 sec) Q waves in leads I or aVF, small r waves in leads II and III, and terminal forces directed superiorly and to the left with mean frontal-plane QRS axis between −30° and −90°. (Patients with left-axis deviation due to deep Q waves in the inferior leads were not included in the LAH group.)

Left posterior hemiblock (LPH) was diagnosed when the electrocardiogram showed narrow (<0.02 sec) Q waves in leads III and aVF, small r waves in leads I and aVL, with terminal forces pointing to the right and inferiorly, and frontal-plane QRS axis >120°. (Patients with clinical evidence of pulmonary hypertension or lateral wall infarction were excluded.)

**Complete Left (LBBB) and Right (RBBB) Bundle-Branch Block and Incomplete LBBB:** This diagnosis was made according to the criteria of Goldman.

**Right Ventricular Conduction Delay (RVCD):** (The term RVCD, rather than incomplete RBBB, is used so as not to necessarily imply conduction disturbances of the RBBB.) The electrocardiographic pattern is identical to that of complete...
RBBB except the QRS interval is < 0.12 sec and the ventricular activation time is < 0.06 sec.

Patients whose electrocardiogram showed bundle-branch and/or fascicular block as a rate-dependent phenomenon or transiently after resuscitation or as part of the terminal phase of the illness were excluded from the "blocked" group.

Results

Of the 480 patients with acute myocardial infarction studied, 97 (20%) had I-V block and 383 (80%) had no evidence of intraventricular conduction disturbance. Included in the latter group were 43 patients who showed pathologic (>0.04 sec) Q waves, QRS prolongation, and slurring of terminal QRS forces. There was no statistically significant difference in age, sex distribution, major complications, and mortality between patients with nonspecific QRS prolongation and those without intraventricular conduction disturbances.

Right Bundle-Branch Block (Unifascicular Block)

The electrocardiogram showed RBBB and normal mean frontal-plane QRS axis in 18 patients. Eleven patients showed complete RBBB, while RVCD was found in the remainder. The bundle-branch block developed during the course of acute myocardial infarction in eight patients but proved to be transient (10 hours to 3 days) in five. In the remaining 10 patients, RBBB was present on admission and persisted until discharge or death. RBBB was known to be present prior to infarction in one patient. Changes of acute anterior and/or lateral wall infarction were present in 13 patients, and the evolutionary changes of acute inferior myocardial infarction were seen in five. Temporary transvenous intracardiac pacing was instituted in five patients: Two had Mobitz II block, and three had complete A-V block. Three of the four patients with 1° A-V block probably had bifascicular disease since they ultimately developed either Mobitz II or 3° A-V block. Nine of the 18 patients had evidence of severe heart failure and/or shock, and eight patients died because of shock. Only one of the five patients with transient RBBB succumbed (20%), while seven of 24 with persistent block died (54%). Postmortem findings in five subjects showed evidence of septal infarction in all but one patient. Three of the five showed a fresh right coronary occlusion.

Right Bundle-Branch Block with LAH (Bifascicular Block)

Of the 23 patients who had RBBB and LAH, RBBB developed during the course of acute infarction in 12 patients and LAH in five. All patients but one showed persistence of either RBBB or LAH, or both. A temporary transvenous pacemaker was inserted in five patients who developed Mobitz II or complete A-V block. Two of six patients with 1° A-V block showed progression to higher degrees of block, while complete A-V block developed abruptly and without antecedent 1° or 2° A-V block in two of three patients. Severe pump failure was present in 13 patients, and seven died because of shock. Postmortem examination of five of six patients showed occlusion of the left anterior descending coronary artery with extensive anteroseptal infarction. One patient had thrombosis of the right coronary artery with infarction of the posterior wall of the left ventricle and septum.

Right Bundle-Branch Block and LPH (Bifascicular Block)

Four patients had RBBB and block of the inferior radiation of the left bundle. The conduction abnormality developed during the course of acute infarction in two patients and persisted in all. Complete A-V block developed abruptly in one of the patients. This patient underwent insertion of a temporary transvenous pacemaker but eventually died of severe pump failure. None of the other three patients, however, showed any degree of A-V block. Two of the patients died because of shock, and postmortem examination in one showed occlusion of the left anterior descending artery with septal infarction and perforation together with anterior and inferior wall necrosis.

Isolated Left Anterior or Posterior Fascicular Blocks

Left anterior hemiblock was present in the admitting electrocardiogram of 19 of 20
Left Bundle-Branch Block

Twenty of the 31 patients had evidence of heart failure and/or shock and pump failure was considered the cause of death in eight of 11 patients. Eight of 10 patients who developed LBBB in course expired. One patient was successfully resuscitated after a bout of primary asystole, while two other patients had sudden loss of palpable pulse and systemic hypotension as the cause of death. One patient had a history of chronic alcoholism and a sudden death occurred with a systolic blood pressure of 110/40. Another patient had a history of congestive heart failure and a sudden death occurred with a systolic blood pressure of 90/60.

Postmortem examination of the left posterior descending artery showed occlusion of the main stem of the left coronary artery in one, all six had occlusion of the left anterior descending artery in five and occlusion of the left circumflex in one. The electrocardiogram showed LBBB in all five patients with occlusion of the left anterior descending artery. The electrocardiogram of the patient with postmortem examination revealed severe disease of all three coronary vessels with complete occlusion of both right and left anterior descending coronary arteries.

### Table 1

<table>
<thead>
<tr>
<th>Type of block</th>
<th>No. pt</th>
<th>Mean age (yr)</th>
<th>Sex (%)</th>
<th>Incidence in total group (%)</th>
<th>Incidence for each group (%)</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Developed in course</td>
<td>Severe heart failure</td>
<td>Shock</td>
</tr>
<tr>
<td>RBBD + LAH</td>
<td>23</td>
<td>66†</td>
<td>65</td>
<td>35</td>
<td>4.8</td>
<td>48</td>
</tr>
<tr>
<td>RBBD + LPH</td>
<td>4</td>
<td>55</td>
<td>50</td>
<td>50</td>
<td>1</td>
<td>50</td>
</tr>
<tr>
<td>Isolated RBBD</td>
<td>18</td>
<td>66†</td>
<td>56</td>
<td>44</td>
<td>3.7</td>
<td>42</td>
</tr>
<tr>
<td>LBBB</td>
<td>31</td>
<td>66†</td>
<td>55</td>
<td>45</td>
<td>6</td>
<td>32</td>
</tr>
<tr>
<td>LAH</td>
<td>20</td>
<td>66†</td>
<td>60</td>
<td>40</td>
<td>4.0</td>
<td>5</td>
</tr>
<tr>
<td>LPH</td>
<td>1</td>
<td>36</td>
<td>100</td>
<td>0</td>
<td>0.2</td>
<td>100</td>
</tr>
<tr>
<td>No I-V Block</td>
<td>383</td>
<td>59</td>
<td>76</td>
<td>24</td>
<td>80</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>480</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Each of the groups with intraventricular block is compared with the no-block group. Groups RBBD + LPH and LPH were not included in the statistical analyses because of the small number of patients in each group.
†Includes all I-V blocks known to have developed in course of myocardial infarction.
‡Statistically significant difference in incidence between groups with a P value <0.05.
block. Similarly, the mortality was higher in each of the blocked groups compared with the no-block group, but the mortality was statistically significant only in patients in groups RBBB and LBBB. Although there was no statistically significant difference in the incidence of 2° or 3° A-V block between groups, none of the 20 patients with LAH developed high-degree A-V block. In each group the cause of death in the vast majority of patients was attributed to pump failure; only two patients in the blocked groups died of primary asystole.

The data in patients with I-V block were analyzed with respect to QRS duration. Although mortality and incidence of complete A-V block tended to be higher for groups with QRS duration, >0.12 sec (especially for patients with complete RBBB and LAH), these differences were not statistically significant. Similarly, there was no statistically significant difference in incidence of pump failure between the groups.

Eighteen of the 97 patients with intraventricular block showed 1° A-V block, and seven of the 18 (39%) showed sudden progression to higher degrees of block, while only six of the 79 patients without 1° A-V block (8%) showed progression to higher degrees of A-V block. This difference was statistically significant (P < 0.05). There was no correlation, however, between QRS duration or type of intraventricular block with the incidence of sudden progression to higher degrees of block.

At autopsy the septum was the most common site of infarction in all groups. In the RBBB and LAH groups the most common postmortem finding was occlusion of the left anterior descending branch of the left coronary artery, whereas the RBBB and LBBB groups tended to show more diffuse coronary artery disease with both old and new areas of infarction.

Discussion

Patients with I-V block tended to be older, and had an incidence of pump failure, complete A-V block, and death that was more than twice that of the group without block. These complications occurred whether the block developed during the course of the infarction or was present on the initial electrocardiographic tracing. The unfavorable prognosis for patients with I-V block could not be explained on the basis of age, and there was no statistically significant difference in the incidence of pump failure and mortality between the various groups of patients with block. Death in the vast majority of these patients was due to shock; in fact, only three instances of primary asystole occurred in the blocked groups. In the present series, patients with intraventricular conduction block did not undergo prophylactic insertion of a pacing catheter. Cardiac arrhythmias were treated with the appropriate drug therapy, and a pacemaker was inserted only with the development of advanced or complete A-V block. It was our impression that earlier pacemaker insertion would not have substantially altered the clinical course, except possibly for two of three patients with primary asystole. Thus, our data support the hypothesis that mortality in patients with I-V block is primarily related to the presence of pump failure rather than electrical instability.

The postmortem findings showed that the vast majority of patients with ECG findings of RBBB and LAH or LAH alone have occlusion of the left anterior descending artery with extensive anteroseptal infarction. Somewhat surprising was the occurrence of inferior infarction observed on the electrocardiogram of five of 18 (27%) patients with RBBB. In addition, three of five patients with RBBB examined at postmortem had a fresh right coronary occlusion, and four of these patients had concomitant disease of the left coronary artery with extensive infarction of the septum. The most likely explanation for the occurrence of RBBB with inferior infarction (and right coronary occlusion) is the presence of antecedent disease in the left anterior descending artery with branches of the right coronary artery bringing collateral blood supply to the septum and RBBB. Alternatively, the most proximal part of the RBB may be supplied by
branches of the posterior descending branch of the right coronary artery.6 Conceivably in some patients with right coronary occlusion a small lesion in the vicinity of the proximal right bundle branch may be responsible for the block.7 Unfortunately, detailed pathologic studies of the intraventricular conduction system were not performed and thus the latter point remains a matter of conjecture.

Equally surprising was the finding of RBBB and LPH in a patient with isolated occlusion of the anterior descending branch of the left coronary artery. This patient showed extensive infarction of the septum as well as the anterior and inferior walls of the left ventricle. Sutton and Davies8 reported frequent occurrence of bilateral bundle-branch necrosis in patients with anteroseptal infarction and complete A-V block. These observations imply that at least in some patients the anterior descending branch of the left coronary artery constitutes the major blood supply for both fascicles of the left bundle. In addition, the finding of a fresh right coronary occlusion and inferior infarction in one patient who developed RBBB and LAH again emphasizes the variability of blood supply to the bundle branches.9

The serious clinical implications of complete RBBB or LBBB in patients with acute myocardial infarction has long been recognized.10-12 Comparison of our data with those of three other large series reported14-16 from coronary care units shows a comparable incidence of bundle-branch block. The incidences of pump failure and mortality were correspondingly high in all series, and this appeared to be independent of the bundle branch involved. We could not, however, confirm the greater incidence of primary asystole or complete heart block reported in these series.14-16 Norris et al.,16 for example, recorded a 34% incidence of primary asystole and a 26% incidence of complete heart block in patients with RBBB and, in fact, recommended insertion of a temporary transvenous pacemaker in patients with anterior infarction in whom RBBB develops. The reasons for the discrepancy between Norris' study and ours are not apparent.

In table 2 our data for patients with bifascicular bundle-branch block (RBBB plus LAH, and RBBB plus LPH) are compared with those of two previous studies of bifascicular bundle-branch block in patients with acute myocardial infarction.17, 18 The wide discrepancy in the incidence of complete heart block between series also bears comment. Godman et al. included asystolic cardiac arrests in the complete A-V block group, including those patients in whom asystole developed as part of the preterminal phase of severe pump failure.18 "The higher mortality in our group [compared with Scanlon et al.]7 may be due to the fact that continuous monitoring often detected bilateral conduction disorders as almost terminal arrhythmias in patients who were shocked or in severe failure." We specifically excluded asystolic arrests or complete A-V block occurring as preterminal events since these arrhythmias may occur as a preterminal event in any

### Table 2

**Comparison of Present Study with Previous Reports of Patients with Acute Myocardial Infarction and Bifascicular Disease**

<table>
<thead>
<tr>
<th>Study</th>
<th>Diagnosis</th>
<th>No. pt</th>
<th>Incidence of bifascicular block (%)</th>
<th>Mortality (%)</th>
<th>Complete A-V block (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scanlon et al.15</td>
<td>RBBB + LAH</td>
<td>21</td>
<td></td>
<td>36</td>
<td>27</td>
</tr>
<tr>
<td></td>
<td>RBBB + LPH</td>
<td>6</td>
<td></td>
<td>33</td>
<td>0</td>
</tr>
<tr>
<td>Godman et al.16</td>
<td>RBBB + LAH</td>
<td>51</td>
<td>2.8</td>
<td>59</td>
<td>29*</td>
</tr>
<tr>
<td></td>
<td>RBBB + LPH</td>
<td>20</td>
<td>1</td>
<td>80</td>
<td>45*</td>
</tr>
<tr>
<td>Present study</td>
<td>RBBB + LAH</td>
<td>23</td>
<td>5</td>
<td>35</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>RBBB + LPH</td>
<td>4</td>
<td>0.8</td>
<td>50</td>
<td>25</td>
</tr>
</tbody>
</table>

*Includes asystolic cardiac arrest.
patient with severe pump failure whether complicated by bilateral bundle-branch disease or not. Scanlon's report, on the other hand, for the most part comprises a review of the precoronary care experience and thus transient blocks (which occurred in approximately 25% of our patients) might have been missed. Furthermore, Scanlon and his associates do not specify whether or not the block occurred as part of the preterminal picture in patients with severe pump failure.

In the vast majority of instances I-V block preceded or occurred concomitantly with clinical deterioration. The advent of newer therapeutic modalities for patients with severe pump failure\(^{19-21}\) makes early recognition of high-risk patients especially important. This is not meant to imply that more aggressive measures be initiated solely on the basis of the presence of I-V block, but rather to emphasize that these patients are in a high-risk category (especially those patients developing LBBB in the course of myocardial infarction), and if a successful policy of intervention for coronary shock be developed than consideration should be given to transferring these patients to centers where the appropriate facilities are available. More likely multivariate analyses of the many variables (including fascicular blocks) affecting immediate mortality will lead to greater specificity in selecting high-risk patients.

Our study confirms the earlier reports of the serious prognostic import of complete bundle-branch block in patients with acute myocardial infarction, and extends these observations to patients with the other I-V blocks. In addition, we have defined a subgrouping of patients with I-V block (those with 1° A-V block) who are particularly prone to the abrupt development of high-degree A-V block. Medical management of this subgroup would certainly be simplified by insertion of a prophylactic pacing catheter, but whether this would result in decreased mortality is still uncertain. Furthermore, we question the rationale of instituting prophylactic temporary transvenous cardiac pacing in all patients with bilateral bundle-branch block since the overwhelming majority of these patients die as a consequence of pump failure. In the present series temporary transvenous pacing was instituted only for those patients (14) showing advanced or complete A-V block, and 10 of these 14 died because of cardiogenic shock. While there were no deaths associated with insertion of the catheter, serious ventricular arrhythmias developed at the time of catheter insertion in 19% of the patients. Other investigators have also reported serious complications resulting from catheter insertion and deaths attributable to pacing.\(^{15, 22}\) In our view, potential benefits of prophylactic insertion of a catheter electrode pacemaker in patients with bilateral bundle-branch block do not outweigh the risks except perhaps for the small subgroup with associated 1° A-V block.

### Acknowledgments

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