Site of Heart Block in Acute Myocardial Infarction


SUMMARY

Bundle of His electrograms were recorded in eight patients with acute myocardial infarction and heart block. Three patients with diaphragmatic myocardial infarction (DMI) and one with subendocardial infarction were characterized by slowing or block above the bundle of His and A-V junctional escape rhythms during periods of advanced or complete block. An additional patient with DMI had block in the His bundle itself. Intraventricular conduction in the above patients was characterized by normal H-Q intervals (35 to 60 msec) and absence of widened QRS. In contrast, three patients with anterior infarction (AMI) manifested complete block below the bundle of His and idioventricular escape. P-H intervals were normal (80 to 140 msec) and A-V conduction was considered unaffected. Our electrophysiologic observations coupled with previous clinical, anatomic, and pathologic findings suggest that the heart block in DMI is usually due to an ischemic lesion of the A-V node, while heart block in AMI is due to necrosis involving both bundle branches.

Additional Indexing Words:
Wenckebach periods  A-V node  Mobitz block  His bundle electrogram

In myocardial infarction with heart block, the site of infarction may determine the nature of block.1-3 It is suggested that diaphragmatic infarction (DMI) produces block by virtue of reversible ischemia to the A-V node.4,5 In addition, DMI has been characterized by periods of type I block (Wenckebach), junctional rhythms, and a benign course.1-3

In contrast, anterior infarction (AMI) with block is associated with massive septal involvement with necrosis of the bundle branches.4,5 AMI with block has also been characterized by the occurrence of type II block, idioventricular rhythms, and high mortality.1,2

In the present study, we have attempted to localize the physiologic site of block in patients with acute myocardial infarction by recording His bundle electrograms during the period of conduction disturbance.

Methods

Clinical Material

Eight patients with acute myocardial infarction and heart block were studied. All patients were admitted to the Intensive Coronary Care Unit of Cook County Hospital. Clinical data on the patients are summarized in table 1. Pathologic Q waves were present in six patients, with patterns diagnostic of DMI in four and AMI in two. One patient was considered to have subendocardial infarction because of widespread ST depressions and T-wave inversions without Q waves. One patient had complete heart block (CHB) with an idioventricular rhythm on admission, and the
diagnosis of acute AMI was made at autopsy. Elevations of serum glutamic oxalacetic transaminase, serum lactic dehydrogenase, and creatinine phosphokinase were noted in all patients.

**Arrhythmias**

A diagnosis of first degree A-V block was made when the P-R interval was greater than 0.20 sec. Second degree A-V block was defined as incomplete A-V block with sinus rhythm and dropped ventricular beats. Second degree block was classified into Mobitz type I (Wenckebach) block characterized by increasing prolongation of P-R interval preceding the dropped beat and Mobitz type II block, characterized by dropped beats without change in the preceding P-R intervals. Advanced A-V block was defined as incomplete A-V block with a 2:1 or greater degree of block. Complete heart block was defined as periods of complete A-V dissociation with atrial rates faster than ventricular rate.

The arrhythmias observed are summarized in table 1. The patients with DMI as well as the patient with subendocardial infarction had second degree or advanced A-V block on admission. Three of the patients with DMI and the patient with subendocardial infarction all manifested Wenckebach periods at some time during their hospital stay. Three patients with DMI and the patient with subendocardial infarction survived.

One of the patients with AMI had CHB at the time of admission. The other two were admitted with normal sinus rhythm and right bundle-branch block, one with left (−30°) and the other with right (+120°) axis deviation. In the latter two, sudden CHB developed on hospital day 5 and 7, respectively, without preceding periods of second degree block. CHB was permanent in two of the patients with AMI (patients 5 and 6) and intermittent in one (patient 7) who had short periods of normal sinus rhythm. All patients with AMI and block died with either congestive failure or shock.

**Electrophysiologic Studies**

Studies were undertaken at the time of transvenous pacemaker insertion in all patients except patient 3 who was studied electively during first degree A-V block. His bundle electrograms were recorded by previously described technic.6,7 A 6 or 7F tripolar catheter was percutaneously passed via the right femoral vein and positioned across the tricuspid valve. Bipolar leads from the catheter were led into

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>History</th>
<th>Leads with pathologic Q waves</th>
<th>Electrocardiogram</th>
<th>ST-segment changes</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 A.Jon</td>
<td>70</td>
<td>F</td>
<td>Chest pain</td>
<td>II, III, aVF</td>
<td>Elev. in II, III and aVF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 A.Joh</td>
<td>53</td>
<td>F</td>
<td>Chest pain; dyspnea</td>
<td>III, III, aVF</td>
<td>Elev. in II, III and aVF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 L.B.</td>
<td>55</td>
<td>F</td>
<td>Chest pain; syncope</td>
<td>II, III, aVF</td>
<td>Elev. in II, III and aVF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 G.S.</td>
<td>70</td>
<td>M</td>
<td>Chest pain; dyspnea</td>
<td>II, III, aVF</td>
<td>Elev. in II, III and aVF</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 F.K.</td>
<td>82</td>
<td>M</td>
<td>Abdominal pain; syncope</td>
<td>V1-V6</td>
<td>Elev. in V1-V6</td>
<td>RBBB with LAD</td>
<td></td>
</tr>
<tr>
<td>6 I.W.</td>
<td>62</td>
<td>F</td>
<td>Chest pain; dyspnea</td>
<td>V1-V5</td>
<td>Elev. in V1-V5</td>
<td>RBBB with RAD</td>
<td></td>
</tr>
<tr>
<td>7 A.M.</td>
<td>48</td>
<td>M</td>
<td>Chest pain; dyspnea</td>
<td>None</td>
<td>Dep. in II, III, aVF and V1-V6</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: Elev. = elevation; Dep. = depression; RBBB = right bundle-branch block; LAD = left axis deviation; RAD = right axis deviation; AVB = A-V block; NSR = normal sinus rhythm; PM = postmortem examination.
<table>
<thead>
<tr>
<th>Site of infarction</th>
<th>Rhythms and conduction defects observed</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>DMI</td>
<td>2° AVB (type 1); advanced AVB; CHB</td>
<td>Died in shock; PM: acute diaphragmatic infarction</td>
</tr>
<tr>
<td>DMI</td>
<td>2° AVB (type 1); advanced AVB; CHB</td>
<td>Alive; return to NSR</td>
</tr>
<tr>
<td>DMI</td>
<td>1° AVB; NSR</td>
<td>Alive; return to NSR</td>
</tr>
<tr>
<td>DMI</td>
<td>2° AVB (type 1); 1° AVB; NSR</td>
<td>Alive; recurrence of 2:1 block</td>
</tr>
<tr>
<td>AMI</td>
<td>Advanced AVB (2:1); NSR; CHB</td>
<td>Died in shock; PM; massive anteroseptal infarction Died in shock</td>
</tr>
<tr>
<td>AMI</td>
<td>NSR; CHB</td>
<td>Died in congestive heart failure</td>
</tr>
<tr>
<td>Subendocardial</td>
<td>2° AVB (type 1); advanced AVB; CHB; 1° AVB; NSR</td>
<td>Alive; discharged in NSR</td>
</tr>
</tbody>
</table>

The predominant rhythms in patient 1 noted during the study were 2:1 A-V block and CHB. Atrial rate was about 100/min. During 2:1 block (fig. 1), conducted P waves alternated with blocked P waves which were not followed by His bundle deflections. During CHB (fig. 2), there was a regular ventricular rhythm unrelated to atrial activity. Ventricular complexes were supraventricular in configuration and were all preceded by His bundle spikes, with H-Q intervals of 35 msec. Thus, block occurred above the bundle of His, with junctional escape.

In patient 2, both advanced A-V block and CHB were noted. In advanced block, the predominant rhythm was 2:1 A-V block with junctional escape following the blocked P wave and causing interference with conduction of the third P wave of the sequence (fig. 3A). The blocked P waves were not followed by conducted His bundle spikes. The results during CHB were similar to those in patient 1 except that the junctional rhythm was faster.

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**Diaphragmatic Infarction**

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*Electronics for Medicine, DR8 or Hewlett Packard (with modified frequency response).*
Results of Electrophysiologic Studies

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Site of infarction</th>
<th>Rhythms during study</th>
<th>Rate during advanced or complete heart block</th>
<th>Site of block</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Rhythm</td>
<td>At. (rate/min)</td>
<td>Vent. (rate/min)</td>
</tr>
<tr>
<td>1</td>
<td>DMI</td>
<td>Advanced AVB; CHB</td>
<td>100</td>
<td>48</td>
</tr>
<tr>
<td>2</td>
<td>DMI</td>
<td>Advanced AVB; CHB</td>
<td>120</td>
<td>66</td>
</tr>
<tr>
<td>3</td>
<td>DMI</td>
<td>1º AVB</td>
<td>96</td>
<td>48</td>
</tr>
<tr>
<td>4</td>
<td>DMI</td>
<td>Advanced AVB; NSR</td>
<td>109</td>
<td>36</td>
</tr>
<tr>
<td>5</td>
<td>AMI</td>
<td>CHB</td>
<td>80</td>
<td>&lt;40</td>
</tr>
<tr>
<td>6</td>
<td>AMI</td>
<td>CHB</td>
<td>120</td>
<td>&lt;40</td>
</tr>
<tr>
<td>7</td>
<td>AMI</td>
<td>CHB</td>
<td>100</td>
<td>53</td>
</tr>
<tr>
<td>8</td>
<td>Subendocardial</td>
<td>3:2 W; advanced AVB; CHB</td>
<td>100</td>
<td>53</td>
</tr>
</tbody>
</table>

*During 2:1 A-V block.
Abbreviations: W = Wenckebach conduction; H = bundle of His.

(66/min) (fig. 3B). H-Q interval was 34 msec.

Patient 3 was studied while in normal sinus rhythm with first degree A-V block (fig. 4A). The P-R interval was 0.22 sec. P-H was 180 msec, and H-Q was 40 msec. P-R prolongation was due to prolongation of P-H. With atrial pacing at 100 beats/min, typical Wenckebach periods occurred (fig. 4B). His bundle potentials did not follow the blocked P waves.

Patient 4 was in fixed 2:1 A-V block during the study. Conducted P waves with a P-H interval of 100 msec alternated with nonconducted P waves. Initially, a His bundle spike was seen after all P waves (fig. 5A). However, moving the catheter approximately 5 mm distally eliminated the His bundle spike associated with the blocked P. Instead of the sharp H spike associated with the blocked P, a blunted low voltage complex was seen (fig. 5B). The H-Q interval of the conducted beats was 60 msec, and the QRS was not widened.

The above findings may be explained by locating the site of block in the His bundle itself. With the recording poles placed proximal to the block, all P waves were associated with His bundle spikes. With the poles at or distal to the block, only the conducted P waves had His bundle spikes. The low voltage complex probably represented a decrementally blocked impulse in the His bundle. In the absence of bundle-branch block pattern in the conducted beats, one must postulate either block in the common bundle or simultaneous 2:1 block in both bundle branches. The latter seems unlikely.

In summary, recording of His bundle potentials in three patients with DMI and

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

Simultaneously recorded ECG and His bundle electrogram (HBE) showing 2:1 A-V block above the bundle of His in diaphragmatic infarction in patient 1. P waves and atrial electrograms are labeled P, His bundle spikes are labeled H, and QRS and ventricular electrograms are labeled R. Atrial rate (AR) is 100/min, and ventricular rate (VR) is 50/min. Paper speed is 200 mm/sec, and time lines are at 1 sec. The first and third P waves are conducted with a P-H interval of 155 msec while the second P wave is blocked above the bundle of His. Same labeling is used in all of the figures.
HEART BLOCK IN MYOCARDIAL INFARCTION

Continuous recording showing complete heart block above H in diaphragmatic infarction in patient 1. Paper speed is 200 mm/sec, and time lines are at 1 sec.

Diaphragmatic infarction with advanced and complete heart block in patient 2. (A) Advanced A-V block. The first P wave is conducted with a P-H interval of 215 msec. The second P wave is blocked above H and is followed by a junctional escape with preceding H spike. The third P wave is nonconducted and occurs during the escape beat. An alternate and less likely explanation for this arrhythmia would be type I block with the first and second P waves being conducted with increasing P-H intervals and the third P wave being blocked above H. (B) Complete heart block above H in diaphragmatic infarction. Paper speed 100 mm/sec, and time lines are at 1 sec.

Subendocardial Infarction

Studies in patient 8 revealed an atrial rate of 100/min with periods of 3:2 type I block, advanced heart block, and complete heart block. With both type I (fig. 6) and advanced block, the blocked P was not followed by a His bundle potential. During CHB, all
ventricular complexes were supraventricular in contour and were preceded by a His bundle potential with a normal H-Q interval (45 msec).

In this patient with subendocardial infarction, type I block, advanced heart block, and CHB all occurred above the bundle of His. Intraventricular conduction was normal.

Anterior Myocardial Infarction

Patients 5, 6, and 7 were studied during CHB. Atrial rates varied between 80 and 120. All atrial complexes were followed by His bundle spikes (figs. 7 and 8) with P-H intervals of 104, 140, and 100 msec, respectively.

A slow, unrelated, idioventricular rhythm was present characterized by a wide QRS without preceding His bundle potential. Asystolic episodes occurred in patients 6 and 7, necessitating control by ventricular pacing (fig. 8).

In summary, the three patients with AMI and CHB had block below the bundle of His, probably in the bundle branches. A-V conduction appeared unaffected, with P-H intervals remaining normal.

Discussion

A knowledge of the blood supply of the cardiac conduction system is helpful in understanding the site of heart block in myocardial infarction. In about 90% of patients, the A-V node and His bundle are supplied by a branch of the right coronary artery given off at the crux of the heart. Pathologic studies in DMI with heart block have usually shown occlusion of the right coronary artery proximal to the take-off of the A-V node artery without major structural damage to the conduction system. Thus, heart block has been felt to represent ischemia to the A-V node or His bundle, or both. In AMI, the pathologic lesion has usually been massive septal infarction with necrosis involving both bundle branches.

The results of this study are consistent with the pathologic findings. Three of the four patients with DMI and the one patient with subendocardial infarction had either prolongation of P-H interval or block proximal to the bundle of His. Escape rhythms, when present, were characterized by a narrow QRS preceded by a His bundle spike with normal H-Q interval, indicating normal conduction in...
HEART BLOCK IN MYOCARDIAL INFARCTION

DMI 2:1 BLOCK

(A and B) 2:1 block in diaphragmatic infarction (patient 4). The first and third P waves are conducted, and the second P wave is blocked in the bundle of His. (See text for discussion.) Paper speed is 200 mm/sec, and time lines are at 40 msec.

Subendocardial infarction with type I block in patient 8. Typical 3:2 Wenckebach period with P-H of 220, 310, and then block above H. Paper speed is 100 mm/sec, and times lines are at 1 sec.

the distal bundle of His and the bundle branches. Thus, the usual site of block in DMI would appear to be in the A-V node, although block high in the His bundle cannot be excluded by the technic of His bundle recording. This site would explain the frequency of Wenckebach periods as well as the occurrence of pharmacologic reversibility of block in DMI, since these are characteristic of conduction delay at the A-V node. The patient with subendocardial infarction appeared to have a similar lesion.

In one patient with DMI and 2:1 block with narrow QRS, a His bundle spike was initially seen following the blocked P wave. However, when the catheter was positioned a few millimeters distally into the right ventricle, only a low voltage deflection could be seen.
Continuous recordings from patient 5 showing complete heart block below the bundle of His in a patient with anterior infarction. Note H spikes following every P wave with normal P-H interval. Paper speed is 200 mm/sec, and time lines are at 1 sec.

after the blocked Ps where the sharp H spikes were previously noted, and the H spikes of the conducted Ps remained well-recorded. This low voltage spike probably represented a decrementally blocked potential low in the His bundle. Narula and co-workers\textsuperscript{16} have described a patient without infarction, who had chronic block in the His bundle characterized by block distal to the bundle of His recording site with narrow QRS. The recordings obtained in our patient are similar to those obtained by Narula and associates, with the addition of the decremental spike.

The findings in AMI with heart block were also consistent with previously reported pathologic findings. P-H intervals were not prolonged, suggesting normal function of the A-V node. Block occurred below the bundle of His recording site, and escape rhythms were idioventricular, characterized by wide QRS complexes without preceding His bundle spikes. Two of the three patients with AMI had evidence of bilateral bundle-branch disease prior to the development of CHB, both having right bundle-branch block, one with left anterior, and the other with left posterior hemiblock.\textsuperscript{16–18} These ECG findings, together with the results of the His bundle recordings, suggest complete bilateral bundle-branch block as the site of block in AMI. This site would explain the occurrence of type II block in AMI, a type of block often signifying disease in the His-Purkinje system.\textsuperscript{2, 14} Stokes-Adams attacks occur frequently in AMI with CHB, reflecting the slow heart rate and unreliability of the idioventricular pacemaker.\textsuperscript{1, 2, 12}

In summary, DMI was characterized by block proximal to the His bundle while AMI was characterized by block distal to the His bundle. Difference in clinical behavior

Complete block below H in anterior infarction during ventricular pacing. Pacing spikes are labeled Pi and are followed by paced ventricular beats (R). All P waves have associated H spikes with P-H interval of 140 msec. None of these are conducted with block occurring below H. Time lines are at 40 msec, and paper speed is 200 mm/sec.
between these two conditions can be partially explained by differing sites of block.

Acknowledgment
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Site of Heart Block in Acute Myocardial Infarction
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