The Conduction System in Acute Myocardial Infarction Complicated by Heart Block


SUMMARY

Detailed histological examination of the conduction system in 29 cases of acute myocardial infarction complicated by heart block has been carried out. In 24 cases posterior infarction was present and occlusion had occurred in the artery from which the blood supply to the A-V node was ultimately derived. In these cases major structural damage to the conduction system was rare, but when present, involved the bundle branches. A-V block in posterior infarction is therefore usually due to hypoxia or other reversible factors rather than structural damage to the conduction system. These findings are consistent with the high incidence of transient heart block in survivors.

In five cases anterior infarction alone was present. The blood supply of the A-V node was not compromised, but massive infarction had caused major damage to both bundle branches.

Since major structural damage to the conduction system is uncommon in myocardial infarction, only a minority of survivors will have permanent heart block. In those cases in which there is extensive damage to the bundle branches there is a risk of high mortality and permanent block particularly when the infarct is anteroseptal.

Additional Indexing Words:
Bundle-branch block    Coronary arterial occlusion    Pathology of A-V node
ECG findings          Nodal artery occlusion

With the introduction of intensive care and electrocardiographic monitoring for acute myocardial infarction, the incidence of arrhythmias has been observed to be very high.1-3 Complete atrioventricular (A-V) block complicates approximately 5% of cases of acute infarction,2,4 with a mortality varying from 37% to 48%5-7 in recent reports. Great attention has been paid, therefore, to the clinical and pacemaking problems in such cases, but the only detailed morphological study of the conduction system in more than isolated cases is the series that Blondeau and co-workers8 reported in 1961.

Methods

Hearts from 29 patients have been studied in the Pathology Department of St. George’s Hospital. Nineteen of these patients were treated in the Cardiac Department of the same hospital and came from a clinical series of 55 patients treated by endocardial pacing for acute heart block. After death the hearts of the remaining 10 patients were referred from other hospitals for detailed postmortem examination. All the patients of both groups had ECG evidence of acute myocardial infarction and complete heart block. No case is included without definite evidence of complete heart block for some hours. Death occurred from 24 hours to 6 weeks after admission to hospital.

The conduction system was studied by a serial section technique.9 The coronary arteries were studied by postmortem angiography10 and serial tissue blocks were taken to include the artery to the A-V node.

Results

The site of infarction and the arterial occlusions judged to be responsible are shown in table 1. In all 24 patients with postero-septal (diaphragmatic) infarction, the dom-
Table 1

Site of Infarct and Arterial Occlusion: 29 Cases

<table>
<thead>
<tr>
<th>Site of infarct</th>
<th>Total</th>
<th>Coronary artery obstructed*</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>R</td>
</tr>
<tr>
<td>Posteroseptal (alone)</td>
<td>21</td>
<td>16</td>
</tr>
<tr>
<td>Posteroseptal and anteroseptal (combined)</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>Anteroseptal (alone)</td>
<td>5</td>
<td>0</td>
</tr>
</tbody>
</table>

*Coronary arteries: R = right; L = left; AD = anterior descending; LC = left circumflex.

Dominant coronary artery from which the blood supply to the A-V node arose was occluded by thrombosis complicating atherosclerosis. Three of these patients had, in addition, anteroseptal infarction with a second occlusion in the anterior descending coronary artery and two had coincidental thrombosis of the left circumflex artery. In most instances the right coronary artery supplied the A-V node, but in three patients the nodal artery arose from the circumflex branch of the left coronary artery. In five patients extensive anteroseptal infarction only had occurred, and in those five the nodal artery did not arise from the occluded artery.

Histological Findings

Table 2 details the histological findings in the 29 cases. In eight, the conduction system as judged by morphology under the light microscope was normal. In 13, the predominant lesion was focal necrosis affecting the node, or the bundle branches or both, but in such cases only a small proportion of conduction fibers was involved. Massive necrosis of the A-V node has not been observed in this series, and in 22 cases the node was entirely normal.

Extensive necrosis of conduction tissue was seen in only eight cases and in each case involved the bundle branches. The five cases of isolated anteroseptal infarction associated with heart block all showed this severe degree of bundle-branch damage.

Edema and inflammatory cell infiltration of the A-V node, reported by other observers, were not seen in our material.

Hemorrhages into and around the bundle branches were frequently seen, but such hemorrhages are also found in hearts from patients without myocardial infarction or conduction disturbances and were therefore not judged to be significant.

Histological study of the coronary arteries confirmed the presence of recent thrombosis complicating atherosclerosis in all cases. In no case of this series was recent or old occlusion seen in serial sections of the nodal artery itself.

Clinicopathological Correlation

The site of infarction, as diagnosed by ECG in the patient treated at St. George's Hospital (table 3), corresponded to the autopsy findings with one exception. In case 3, a recent posterior infarction and old anterior subendoocardial scarring were misinterpreted as recent anterior infarction. All the patients who had recent anterior infarction confirmed at autopsy had bundle-branch block in the ECG and histological evidence of severe damage to the bundle branches.

Table 2

Pathology of the Conduction Tissue in 29 Cases at Autopsy

<table>
<thead>
<tr>
<th>Cases</th>
<th>Normal conduction tissue</th>
<th>Minor damage to conduction tissue</th>
<th>Focal A-V node necrosis (FAVN)</th>
<th>Focal A-V node necrosis plus partial left bundle-branch necrosis (FAVN + PLBN)</th>
<th>Partial left bundle-branch necrosis (PLBN)</th>
<th>Partial bilateral bundle-branch necrosis (PBBN)</th>
<th>Major damage to conduction tissue</th>
<th>Massive left bundle-branch necrosis (MLBN)</th>
<th>Massive bilateral bundle-branch necrosis (MBBN)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8</td>
<td>13</td>
<td>3</td>
<td>2</td>
<td>4</td>
<td>4</td>
<td>8</td>
<td>2</td>
<td>6</td>
<td>29</td>
</tr>
</tbody>
</table>

Circulation, Volume XXXVIII, November 1968
infarcts and in two of these minor histological damage was present in the bundle branches. While there is good correlation of ECG evidence of bundle-branch block with the histological appearances in most cases, in some, divergence does occur. In case 13 considerable left bundle-branch damage was present without a wide QRS complex in the ECG, and in case 10 there was left bundle-branch block with a normal conduction system.

In the clinical series of 55 patients with acute infarction and heart block, from which this autopsy series derives, the presence of bundle-branch block carries a worse prognosis and a higher risk of permanent conduction defects than a narrow QRS complex in the ECG, does (table 4).

**Discussion**

Posterior rather than anterior myocardial infarction has been associated with A-V block in this postmortem series as in other clinical series.\(^5\)\(^7\)\(^11\) James and Burch\(^12\) have related

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**Table 3**

<table>
<thead>
<tr>
<th>Case</th>
<th>ECG site of infarct</th>
<th>QRS width</th>
<th>Axis</th>
<th>Pathological site of infarct</th>
<th>Pathological damage to conduction tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Diaphragmatic</td>
<td>Narrow*</td>
<td>Normal</td>
<td>Posteroseptal</td>
<td>PLBN</td>
</tr>
<tr>
<td>2</td>
<td>Anterior; diaphragmatic</td>
<td>RBBB</td>
<td>R-axis</td>
<td>Anteroseptal; posteroseptal</td>
<td>PBBN</td>
</tr>
<tr>
<td>3</td>
<td>Anterior</td>
<td>Narrow</td>
<td>Normal</td>
<td>Posteroseptal</td>
<td>PBBN</td>
</tr>
<tr>
<td>4</td>
<td>Diaphragmatic</td>
<td>Narrow</td>
<td>Normal</td>
<td>Posteroseptal</td>
<td>FAVN</td>
</tr>
<tr>
<td>5</td>
<td>Diaphragmatic</td>
<td>Narrow</td>
<td>L-axis</td>
<td>Posteroseptal</td>
<td>FAVN</td>
</tr>
<tr>
<td>6</td>
<td>Anterior</td>
<td>LBBB</td>
<td>R-axis</td>
<td>Anteroseptal; posteroseptal</td>
<td>MBBN</td>
</tr>
<tr>
<td>7</td>
<td>Diaphragmatic</td>
<td>Narrow</td>
<td>L-axis</td>
<td>Posteroseptal</td>
<td>PBBN</td>
</tr>
<tr>
<td>8</td>
<td>Anterior</td>
<td>RBBB</td>
<td>L-axis</td>
<td>Anteroseptal</td>
<td>MBBN</td>
</tr>
<tr>
<td>9</td>
<td>Anterior</td>
<td>LBBB</td>
<td>L-axis</td>
<td>Anteroseptal</td>
<td>MBBN</td>
</tr>
<tr>
<td>10</td>
<td>Diaphragmatic</td>
<td>LBBB</td>
<td>Normal</td>
<td>Posteroseptal</td>
<td>FAxN + PLBN</td>
</tr>
<tr>
<td>11</td>
<td>Diaphragmatic</td>
<td>LBBB</td>
<td>Normal</td>
<td>Posteroseptal</td>
<td>MBBN</td>
</tr>
<tr>
<td>12</td>
<td>Anterior</td>
<td>LBBB</td>
<td>L-axis</td>
<td>Anteroseptal; posteroseptal</td>
<td>MBBN</td>
</tr>
<tr>
<td>13</td>
<td>Diaphragmatic</td>
<td>Narrow</td>
<td>Normal</td>
<td>Posteroseptal</td>
<td>MLBN</td>
</tr>
<tr>
<td>14</td>
<td>Diaphragmatic</td>
<td>RBBB</td>
<td>L-axis</td>
<td>Posteroseptal</td>
<td>PLBN</td>
</tr>
<tr>
<td>15</td>
<td>Diaphragmatic</td>
<td>Narrow</td>
<td>Normal</td>
<td>Posteroseptal</td>
<td>FAxN + PLBN</td>
</tr>
<tr>
<td>16</td>
<td>Diaphragmatic</td>
<td>Narrow</td>
<td>L-axis</td>
<td>Posteroseptal</td>
<td>PLBN</td>
</tr>
<tr>
<td>17</td>
<td>Anterior</td>
<td>Wide</td>
<td>Normal</td>
<td>Anteroseptal; posteroseptal</td>
<td>MLBN</td>
</tr>
<tr>
<td>18</td>
<td>Anterior</td>
<td>RBBB</td>
<td>L-axis</td>
<td>Anteroseptal</td>
<td>MBBN</td>
</tr>
<tr>
<td>19</td>
<td>Anterior</td>
<td>LBBB</td>
<td>L-axis</td>
<td>Anteroseptal</td>
<td>MBBN</td>
</tr>
</tbody>
</table>

*Narrow QRS = less than 0.12 sec.

Abbreviations: For those for "Pathological damage" see table 2; RBBB = right bundle-branch block; LBBB = left bundle-branch block; L axis = QRS frontal vector to left of −30°; R axis = QRS frontal vector to right of +90°.

**Table 4**

<table>
<thead>
<tr>
<th>QRS</th>
<th>Total no.</th>
<th>Deaths*</th>
<th>Mortality (%)</th>
<th>Survivors</th>
<th>Permanent A-V block</th>
<th>Permanent BBB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wide</td>
<td>22</td>
<td>17</td>
<td>77</td>
<td>5</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Narrow</td>
<td>33</td>
<td>8</td>
<td>24</td>
<td>25</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*Autopsy permission was refused for six patients.
the high incidence of conduction disturbances in posterior infarction to the blood supply of the A-V node. When A-V block complicates posterior infarction, the coronary artery occluded is the one that ultimately supplies the node; this is confirmed in the present study. The nodal artery itself is, however, not commonly occluded and massive necrosis of the A-V node is similarly rare; neither has been seen in this series of 29 cases.

In this study many cases of block complicating posterior infarction are associated with a normal conduction system or show small foci of necrosis in the node or more commonly the bundle branches. Such areas are tiny in relation to the whole conduction system and in our opinion are not likely to affect function permanently. In patients who survive heart block and posterior infarction major damage to the conduction system may be even more rare. A small number of patients with isolated posterior infarcts will, however, suffer major bundle-branch damage, but in this series this was seen in only one of 21 cases.

The mechanism of production of A-V block in posterior infarction remains speculative. Infarction of the node, edema, and inflammatory cell infiltration of the node have all been postulated5,18 as causes but have not been observed in this series. Since the artery occluded in that which ultimately gives origin to the nodal artery, temporary hypoxia of the conduction system must be considered. Ligation of the septal arteries in the dog produces a similar electrocardiographic pattern of heart block to that in acute posterior infarction in man.14 In the dog, however, block appears within minutes of ligation, whereas in man the onset of block in acute infarction may be delayed by 12 to 24 hours after the onset of symptoms. The conduction system, while not showing any major damage as judged by morphological criteria, lies immediately adjacent to infarcted muscle and substances released from such areas may inhibit nodal function. In most cases of posterior infarction, block is certainly not caused by major structural damage, a pathological finding supported by the transient nature of the block with sinus rhythm returning in less than 7 days in the great majority of survivors according to clinical reports.6,7,11,15,16 Seven of the 19 patients listed in table 3 returned to sinus rhythm prior to death.

The mechanism of production of A-V block in anterior infarction is more controversial. James37 has put forward the view that such an association must imply additional arterial lesions in the blood supply of the posterior wall and A-V node. In the present series this was found in the three cases with combined anterior and posterior infarction. In five cases, however, isolated anteroseptal infarction was associated with heart block. In all five cases severe damage was present to both bundle branches in extensive septal infarction. The anterior descending coronary artery supplies the distal bundle branches and anterior myocardial infarction must be very extensive to cause ischemia or necrosis of both branches at this level. This fact may explain both the rarity of anterior infarction causing block and the poor prognosis for survival when it does occur. The association of severe bundle-branch damage with anterior infarction is also observed in the combined anterior and posterior infarcts of this autopsy series.

In view of the pathological findings of this series, it is of interest to consider whether the degree of damage to the conduction system can be assessed from the ECG in acute myocardial infarction. In anterior infarction complicated by heart block, bundle-branch block was almost always present and associated with structural damage. A less constant relationship between bundle-branch block and structural damage was present in posterior infarction. Transient bundle-branch block may occur in acute infarction18 and may be due to reversible ischemia rather than structural damage. There are also many practical difficulties in assessing the degree of bundle-branch damage by microscopy. Hemorrhage into the conduction system is a hypoxic phenomenon19 and is commonly present obscuring underlying cellular detail. Myocytolysis is common in the conduction fibers of the left branch and may or may not indicate irreversible cell death.

Circulation, Volume XXXVIII, November 1968
damage. In addition structural damage to the conduction system short of total destruction may not necessarily be associated with an abnormal ECG. We conclude, however, that, in myocardial infarction complicated by complete A-V block, bundle-branch block is more often associated with severe conduction tissue damage than if there is no prolongation of the QRS complex.

Very few patients who survive acute myocardial infarction complicated by A-V block have permanent conduction defects. Studies of patients with chronic A-V block show that ischemic disease is only rarely responsible. In the series from this hospital the majority of cases of chronic A-V block due to ischemic heart disease showed destroyed bundle branches in areas of old infarction. Since the site of major damage, when present, to the conduction system in acute infarction is the bundle branches, it is reasonable to conclude that it is these patients who are at risk of permanent conduction defects. This is in accord with the clinical series of 55 cases (table 4). The table indicates that a wide QRS complex in the acute episode is associated with a high risk of permanent conduction defect in the survivors whereas a normal QRS complex carries a much smaller risk. The exception to this latter category is the patient who has an occlusion of the nodal artery itself. This will produce destruction of the A-V node with production of A-V block associated with a narrow QRS complex which will be permanent. We have seen only one such case in a long-term survivor of acute myocardial infarction followed immediately by permanent A-V block for several years until death from further myocardial infarction.

Our pathological findings are very similar to those of Blondeau and associates who also showed that major damage to the conduction system was rare in acute infarction but if present involved the bundle branches. They pointed out that while posteroseptal infarction rarely causes major damage to both branches, when it does it need not be large to involve the proximal portions of both branches close to their origin. Permanent block may therefore follow a small posterior infarct. In contrast, anterior infarction must be very extensive to destroy the distal portions of both branches.

Acknowledgment

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References

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