Atrioventricular block in posterior acute myocardial infarction: a clinicopathologic correlation

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ABSTRACT The specialized conducting systems of 44 hearts with posterior-inferior acute myocardial infarction were studied to clarify the anatomic basis of atrioventricular (AV) block. The results showed a lack of correlation between the block and the lesional pathology of the specialized conducting system. On the other hand, an evaluation of the atrial prenodal myocardium revealed strong clinicopathologic correlation between the block and the necrotic damage to these fibers. Twenty-nine or 97% of patients with AV block showed acute necrosis of the prenodal atrial myocardium. Considering the conducting prenodal septal atrial myocardium as a suprahisian structure, the necrosis at this level would provide an anatomic basis of the block in posterior-inferior acute myocardial infarction. Analysis of the behavior of the AV block after pharmacologic treatment further established a relationship between the block and acute lesions in the central conduction system. Circulation 75, No. 4, 733-736, 1987.

BETWEEN a fifth and a third of all patients suffering posterior acute myocardial infarction (P-AMI) develop grade II to III atrioventricular (AV) block.1-2 In the majority of patients surviving the acute phase, the block is generally transient and its prognostic significance is variable and rarely grave.3 Most electrophysiologic studies favor a suprahisian origin of the AV block in P-AMI.2,4,5 However, the block has also been ascribed to the His bundle level.6 Although clinical and electrophysiologic studies generally agree on the transient nature and suprahisian location of the block, the anatomic basis of the electrical disturbance remains a matter of controversy.2 Nonanatomic mechanisms have also been advocated to explain the AV conducting disturbance. Likewise, temporal hypoxia,1 cholinergic reflexes,3 and a local increase in extracellular hyperkalemia have been implicated in the genesis of the block.7

The anatomic basis of the block may be hidden in topographic sites not previously studied in this context, such as the conducting septal prenodal atrial myocardium. In this morphologic study we sought to clarify the anatomic basis of the AV block in P-AMI.

Materials and methods
We studied the hearts of 44 patients with P-AMI. A grade III AV block was recorded in 26 cases and grade II block was found in four.

Routine histopathologic examination of the infarcts included postmortem coronary angiography and step sectioning of the coronary arteries. Camera lucida drawings were obtained from four tetrazolium hydrochloride-stained, whole heart slices taken at regular intervals from base to apex. Each slice was then further divided into an average of 10 to 12 mapped and numbered sections comprising the entire slice. The sections were stained with hematoxylin and eosin, and the extent of the infarct was then evaluated morphometrically.

Study of the specialized AV conducting system and the atrial prenodal myocardium was carried out systematically in all cases. The Koch triangle was cut into eight tissue blocks, as shown in figure 1. Each block was then serially sectioned at 200 μm intervals. An average of 10 to 12 microscopic 5 μm tissue sections obtained from each block were examined. Anatomic examination of lesions was carried out with special emphasis in the septal atrial prenodal myocardium, central node, and bundle of His. Staining was carried out with hematoxylin and eosin, Masson trichrome, and Van Gieson-Weigert elastic.

Coagulation necrosis of the myofibers, manifested by intense differential eosinophilia, and/or polymorphonuclear infiltrates were the only acute lesions recorded as positive findings. Because of the difficulties inherent in objective evaluation, minor degenerative changes such as hydropic cell swelling and/or ede-
The eight women had a mean age of $65.1 \pm 6.2$ years (range 50 to 70).

Table 1 shows extent of coronary artery disease, recent thrombosis, and extension of myocardial infarction. The acute morphologic findings from the 44 specimens are summarized in Table 2 and Figure 2.

Twenty-nine of the 30 hearts with AV block had acute lesions involving the atrial prenodal fibers. Only 11 of the 29 hearts displayed acute changes in the node and/or in the bundle of His. Eleven of the 14 hearts from patients without AV block also showed absence of lesions in the prenodal fibers, whereas only three had acute prenodal necrosis (Table 2). The combination of necrosis in atrial prenodal myocardium and conducting system was observed in 13 cases, 11 of which had AV block, one atrial fibrillation, and one sinus rhythm. Nineteen cases had necrosis of atrial prenodal myocardium and normal conducting system, and in 18 of these an AV block was also recorded. Finally, of the 12 cases in which acute lesions could not be demonstrated, only one developed AV block. Isolated necrosis of the specialized conducting system was not present without associated necrosis in the prenodal fibers (Figure 2).

Table 3 summarizes the therapy of the 30 patients with AV block as well as the presence or absence of acute lesions in the specialized conduction system. Seventy-five percent of the blocks responding to pharmacologic therapy, or 80% including those cases with spontaneous reversion, did not have acute lesions in the conducting system, while 64%, counting the two untreated patients, not responding to treatment showed acute lesions in the system. The six patients undergoing pacemaker implantation as the only treatment were not included in the evaluation of the results.

With regard to the long-term histopathologic findings, 10 cases showed important scarring of the atrial prenodal myocardium; five of these also showed simi-
lar concomitant lesions in the node and in the bundle of His. Significantly, in none of the 10 cases were electrical disturbances recorded on last admission. Finally, two other cases with chronic abnormalities in the node and/or His bundle had AV block, but both also showed acute lesions in the corresponding atrial myocardium.

**Discussion**

Previous investigators tried unsuccessfully to establish a clinicopathologic correlation between the necrosis and the AV block in patients with posteroinferior infarction. Our results show a close relationship between the AV block and the necrosis displayed by the prenodal atrial myocardial fibers; 29 of the 30 cases with AV block (97%) showed concordant necrosis of the prenodal fibers. Moreover, 11 of the 14 cases without AV block did not have acute necrosis of the atrial fibers adjacent to the node. If we consider the fact that the supraventricular block is, from an electrophysiologic perspective, a nodal-bound block, the acute lesions

**TABLE 3**

Pharmacologic therapy of AV block (20 cases)

<table>
<thead>
<tr>
<th></th>
<th>β-Adrenergic</th>
<th>+ atropine</th>
<th>No treatment</th>
<th>CCS necrosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Atropine</td>
<td></td>
<td></td>
<td>Pos</td>
</tr>
<tr>
<td>Reversion</td>
<td>3</td>
<td>0</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>No reversion</td>
<td>4</td>
<td>5</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>5</td>
<td>8</td>
<td>4</td>
</tr>
</tbody>
</table>

Abbreviations as in table 2.
should involve the nodal tissue. But if we consider the conducting prenodal septal atrial myocardium as a supraventricular structure, the necrosis at this level would provide the anatomic basis of the block in posteroinferior acute myocardial infarction.

From the perspective of vascularization, the double anterior and posterior vascular supply of the node contrasts with the adjacent supranodal atrial myocardium irrigated by a single vessel.\textsuperscript{9-11} These vascular considerations—the predominance of necrosis in the prenodal fibers and the relatively scanty positive findings related to the specialized conducting system—strongly favor our results.\textsuperscript{2, 8, 11}

The acute lesions of the central conducting system found in AV block implies a lack of response to therapy. Seventy-five percent of the blocks responding to treatment failed to reveal necrosis in the specialized conducting tissue, the percentage rises to 80% if we include those cases spontaneously reverting to sinus rhythm.

In summary, we conclude that the main cause of the AV block in P-AMI is necrosis of the atrial prenodal myocardium. There is also a strong suggestion that the absence of reversion of the block is mainly related to acute lesions of the central conducting system.

Although some authors related scarring of the specialized system to the AV block in P-AMI,\textsuperscript{8} our data show a total lack of correlation between chronic lesions involving either the conducting system and/or atrial prenodal myocardium and AV block.

Further research of the atrial myocardium will contribute to a better understanding of clinicopathologic correlation between supraventricular electrical disturbances and acute myocardial necrosis.

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\textbf{References}

\begin{enumerate}
  \item Rotman M, Wagner GS, Waugh RA: Significance of high degree atrioventricular block in acute posterior myocardial infarction. Circulation \textbf{47}: 257, 1973
  \item Hackel DB, Wagner G, Ratlif NB, Cies A, Estes AH: Anatomic studies of the cardiac conduction system in acute myocardial infarction. Am Heart J \textbf{83}: 77, 1972
  \item Ohkawa S, Hackel DB: Anatomic studies of the conduction system in right ventricular infarction. Jpn Heart J \textbf{23}(suppl): 184, 1982
  \item Lev M: The pathology of complete atrioventricular block. Prog Cardiovasc Dis 6: 409, 1964
  \item Frink RS, James TN: Normal blood supply to the human His bundle and proximal bundle branches. Circulation \textbf{47}: 8, 1973
\end{enumerate}
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