Pathologic Features of Atrioventricular and Intraventricular Conduction Disturbances in Acute Myocardial Infarction

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The availability of continuous electrocardiographic monitoring techniques has resulted in recognition that conduction disturbances occur more frequently in patients with acute myocardial infarction than was previously thought to be the case. Complete atrioventricular (A-V) block has been reported in up to 9% of patients who have had acute infarcts,1 and bundle-branch block has been recorded in 13% of cases.2 Complete A-V block is an especially important complication of acute infarction since it is associated with a higher mortality rate. For example, in a recent report by Kostuk and Beanlands, the mortality in 378 patients with acute myocardial infarction was 20%, whereas in those patients who also developed complete A-V block the mortality was 50%.1

Important differences have been described between A-V block occurring during the course of acute posterior as contrasted with acute anterior infarction (table 1). A-V block occurs more frequently in patients with acute posterior wall infarction, but it is associated with a lower mortality rate than A-V block with acute anterior infarction. For example, Norris reported that out of 34 cases of second and third degree A-V block occurring in 276 patients with acute infarctions, 26 had a posterior and eight an anterior infarct.3 The mortality in the patients with A-V block and anterior infarction was 75%, whereas for patients with A-V block and posterior infarction the mortality was only 19%.

In addition to a higher mortality rate, the conduction defect in survivors with anterior infarction tends to be permanent more often than that seen in patients with A-V block complicating posterior infarctions. For example, in a recent survey of 13 patients with chronic complete A-V block studied in our laboratory, most (eight of 13) were not related to a preceding infarct, but of those related to infarction (five of 13), all were anterior in position. In four of these five cases the anatomic damage was found to involve the bundle branches bilaterally rather than the A-V node or common bundle.4 Lenegre similarly found that of 12 cases with chronic bilateral bundle-branch block attributable to coronary atherosclerosis, twice as many were associated with anterior as with posterior infarcts.5

In accord with the clinical observation that conduction disturbances occurring during the course of an acute myocardial infarction are usually transient, anatomic studies report only minor or no demonstrable lesions in most cases of posterior myocardial infarction in which infarct does not extensively involve the septum.6-8 Sutton and Davies, for example, reporting their anatomic findings in the hearts of 29 patients with acute myocardial infarction (24 of which were posterior infarcts) and complete heart block, described the conducting tissue to be normal or to show only minor focal necrosis in 21 cases.7 Extensive necrosis of conducting tissue was present in only eight cases, and in these the bundle...

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branches and not the A-V node or common bundle were involved. Only five cases of anterior infarction were included in the total series of 29, and all five of these were also included in the eight patients who had severe bundle-branch necrosis. These authors pointed out the important fact that anterior infarction complicated by heart block with a widened QRS is frequently related to extensive structural damage to the bundle branches. They also noted that a high risk of permanent conduction damage was present in survivors who had a wide QRS during the acute episode.

Blondeau, Lenegre, and their collaborators have recorded their anatomic findings in patients with heart block complicating acute myocardial infarction. Their observations are essentially similar to those of Sutton and Davies in that most cases (17 of 26) were said to show only partial and probably reversible involvement of the conduction system, and that, in the majority of cases, the sites of demonstrable involvement were the bundle branches. Only two cases showed massive A-V node necrosis, and, in both of these cases, the A-V node artery itself was thrombosed. As an explanation of those cases with no demonstrable lesions, it was hypothesized that with posterior infarction the A-V block was linked to anoxia of the node without necrosis, and that with anterior infarction the block was most often due to damage to the bundle branches.

In relation to these anatomic findings it is interesting to speculate on the possible reasons for the observation that, in some cases, conducting fibers are spared despite massive infarction of adjacent myocardium. An obvious possibility is that diffusion of oxygen from the ventricular cavity directly to the underlying conducting fibers affords protection. An equally attractive explanation lies in the known metabolic differences between contractile and conducting fibers. One study, for example, has shown that the rate of oxygen consumption is one fifth as great in the conducting as in the contractile musculature. It is also of interest to consider the basis for the transient functional defect that arises in the anatomically intact conducting fibers. It is possible that sublethal anoxia may play a role or that there may be an efflux of potassium from the surrounding necrotic tissue. We have suggested that the release of lysosomal enzymes from infiltrated leukocytes could have a transient blocking effect on conduction, and have pointed out that this is supported by the similar time course of arrhythmia development and leukocyte infiltration into the necrotic zone.

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