Recognition and Significance of Intraventricular Block due to Myocardial Infarction (Peri-infarction Block)

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SUMMARY
Evidence of peri-infarction block was found in 39% of the electrocardiograms of 1,938 patients with definite evidence of myocardial infarction. It most commonly occurred acutely or within several days following infarction and was more common with recurrent infarction. Peri-infarction block infrequently disappeared and was associated with higher values for serum glutamic-oxalacetic transaminase and a greater mortality in the first year following myocardial infarction. There was no autopsy correlation of peri-infarction block and ventricular aneurysmal formation.

The term “peri-infarction block” is appropriate only when abnormal intraventricular conduction occurs in association with definite electrocardiographic evidence of myocardial infarction.

Additional Indexing Words:
Serum glutamic-oxalacetic transaminase
Ventricular aneurysm

The term, “peri-infarction block,” was coined1 in 1950 to describe a specific type of intraventricular conduction disturbance associated with myocardial infarction. The changes were noted to occur months following infarction and, once established, were permanent. Modification of the original criteria2-6 led to considerable confusion in diagnosis with the result that many electrocardiograms were interpreted as showing evidence of “peri-infarction block” even when they did not show QRS changes characteristic of infarction and when prolongation of the QRS interval was absent. In addition, despite the interest of the electrocardiographer in this particular abnormality, there have been no published reports to alert the clinician to its clinical significance. Therefore, the purpose of the present report is threefold: to define again the significant electrocardiographic features of peri-infarction block; to establish its frequency, time of onset, and duration; and to compare the clinical features of the infarction episode in patients with peri-infarction block with those of patients who did not develop this abnormality.

Methods
All the electrocardiograms of patients admitted to the University of Oklahoma Medical Center between 1956 and 1967 (11 years) that were diagnosed as indicating myocardial infarction were reviewed. Standard 12-lead electrocardiograms with the addition of V_{4R} and V_{6} (electrode placed to the left of the spine at the level of V_{3}) were recorded at the University Hospital. Most of the electrocardiograms taken at the Veterans Administration Hospital used the limb lead system.
recommended by Schaffer and associates,7 so that only limb lead I, aVF, and the standard precordial leads were recorded together with V4R and V5. The sensitivity and specificity of this lead system is equivalent to the standard 12-lead electrocardiogram. Every case selected for study displayed significant Q-wave abnormalities (at least 0.04 sec in duration) in appropriate leads. Those electrocardiograms with equivocal evidence of myocardial infarction were not included.

The criteria proposed by First and associates1 were accepted for the electrocardiographic diagnosis of peri-infarction block. They are: (1) alteration of the direction of the initial 0.04 sec portion of the QRS complex to produce a definite Q wave; (2) slurring and prolongation of the terminal QRS forces giving a characteristic QRS pattern in standard unipolar or precordial leads; and (3) QRS complex prolongation in the limb leads exceeding 0.10 sec in duration. These criteria were modified to include those electrocardiograms which showed a marked delay in the time of onset of the intrinsicoid deflection (greater than 0.06 sec) in the precordial leads when there was no prolongation of the QRS interval in the standard limb leads. Each patient had an average of eight to 10 serial electrocardiograms available for study. The time of appearance, the duration, and the time of disappearance of peri-infarction block were noted in each case. When exact data were not available because of infrequent electrocardiographic records, the onset, persistence, or disappearance was classified as unknown.

Clinical charts were reviewed and pathology protocols were analyzed on those patients who died. Age, sex, blood pressure, serum glutamic-oxalacetic transaminase (SGOT) levels, history of recent or previous myocardial infarction, and other associated diseases were sought in each case. Autopsy protocols were studied to note the location of any myocardial infarction and presence of ventricular aneurysm.

**Results**

From the review of electrocardiograms, 1,938 patients had definite evidence of myocardial infarction. Of this group, 756 developed infarction block, giving an overall incidence of 39%. Table 1 summarizes the frequency of occurrence of peri-infarction block and indicates the duration of the follow-up period and the mortality. The occurrence of peri-infarction block did not differ significantly from the electrocardiographic location of the infarction. A slightly higher percentage (54.6%) of the infarctions overall were located anteriorly (anteroseptal, anterolateral, and lateral) with an incidence of peri-infarction block of 37.9%. In those with infarctions located elsewhere (posterior, posterolateral, and inferoseptal or diaphragmatic), the incidence was 40.4%.

The mortality due to cardiovascular causes as indicated in table 1 was clearly higher during the first year in those patients who had peri-infarction block (53.9%) than in those patients without peri-infarction block (22.6%). After the first year there was no difference in the mortality of the two groups. Patients who manifested peri-infarction block had a mean SGOT value of 198 units while the comparable value of 157 units from patients who had no block was slightly lower.

**Table 1**

In incidence and Mortality of Peri-infarction Block

<table>
<thead>
<tr>
<th>Duration of follow-up</th>
<th>No.</th>
<th>Peri-infarction block</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1 mo</td>
<td>157</td>
<td>42</td>
</tr>
<tr>
<td>1-5 mo</td>
<td>82</td>
<td>25</td>
</tr>
<tr>
<td>6-11 mo</td>
<td>58</td>
<td>22</td>
</tr>
<tr>
<td>1-2 yr</td>
<td>136</td>
<td>54</td>
</tr>
<tr>
<td>3-4 yr</td>
<td>176</td>
<td>78</td>
</tr>
<tr>
<td>5-6 yr</td>
<td>113</td>
<td>40</td>
</tr>
<tr>
<td>7-10 yr</td>
<td>53</td>
<td>22</td>
</tr>
<tr>
<td>Inadequate follow-up</td>
<td>1161</td>
<td>473</td>
</tr>
<tr>
<td>Totals</td>
<td>1938</td>
<td>756</td>
</tr>
</tbody>
</table>

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Figure 1

Early onset of peri-infarction block. A. Electrocardiogram recorded a few hours after the onset of symptoms shows characteristic changes of acute myocardial infarction involving the anteroseptal and anterolateral regions. The QRS interval is normal. B. Eleven days later the electrocardiogram displays typical peri-infarction block with prolongation of the QRS interval to 0.12 sec in the limb leads and delayed time of onset of the intrinsicoid deflection (about 0.12 sec) in V₅. Note the marked change in terminal QRS forces from those in A.

Figure 2

Late onset of peri-infarction block. A. Early electrocardiographic changes of inferoseptal or diaphragmatic infarction are present. There is no prolongation of the QRS interval. B. Ten months later, typical peri-infarction block has occurred. The duration of the QRS interval is 0.12 sec. The terminal 0.04 sec QRS vector points toward the blocked region overlying the infarct.
Table 2

Known Time of Onset and Disappearance of Peri-infarction Block

<table>
<thead>
<tr>
<th></th>
<th>Onset (cases)</th>
<th>Disappearance (cases)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 wk</td>
<td>36</td>
<td>10</td>
</tr>
<tr>
<td>1-4 wk</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>1-12 mo</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>1-2 yr</td>
<td>5</td>
<td>1</td>
</tr>
<tr>
<td>2-4 yr</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Totals</td>
<td>52</td>
<td>18</td>
</tr>
</tbody>
</table>

In 52 patients the time of onset of peri-infarction block could be determined exactly by the presence of either a normal electrocardiogram prior to infarction or by the appearance of myocardial infarction prior to the onset of peri-infarction block. In the majority of this group peri-infarction block appeared the first few days following myocardial infarction and was seen less frequently after the first month. In the remainder the exact time of onset of peri-infarction block could not be documented because the features of peri-infarction block were present on the first electrocardiogram obtained at the time of hospitalization of the patient. Hence, it cannot be stated without reservation that some type of abnormal intraventricular conduction was absent prior to myocardial infarction in this group. Figure 1 illustrates the onset of peri-infarction block 8 days after anteroseptal and anterolateral infarction while figure 2 shows the late onset of peri-infarction block associated with inferoseptal infarction.

In one third of the patients in whom the time of onset of peri-infarction block was known, the abnormality proved to be temporary, disappearing in the first week following its onset. In the majority of patients, it tended to persist during the entire period of observation (table 2). In figure 3 is shown an example of transient acute peri-infarction block. Similarly, the onset of peri-infarction block was observed to occur during the first week of infarction in the majority of patients. In only one third did it appear some weeks to months later (table 2).

The occurrence of peri-infarction block in those patients with recurrence of myocardial infarction was 66% or nearly double the overall incidence. From table 1 it is apparent that the incidence of peri-infarction block was lowest in the group of patients followed less than 1 month and increased to a maximum in the group followed from 3 to 4 years. This difference is largely due to a greater number of recurrent infarctions in the groups followed for longer periods of time. Peri-infarction block following recurrence of myocardial infarction is shown in figure 4. Serial electrocardiograms recorded from this patient show the acute appearance of either peri-infarction block or right bundle-branch block in an anteroseptal infarction. It lasted 1 month.

Figure 3

Transient, acute peri-infarction block. A. Peri-infarction block is shown during the early changes of an inferoseptal or diaphragmatic infarction. The QRS interval in aVF is more than 0.12 sec. B. This electrocardiogram recorded 9 days later than A has a normal QRS interval.
Nearly 6 years later following a recurrent bout of chest pain, peri-infarction block was present in a different location (anterolateral and lateral).

Autopsy data were available from 69 patients who had electrocardiographic evidence of peri-infarction block. In 67, there were evidences of recent or old myocardial infarction confirming the electrocardiographic diagnosis of infarction. In two instances, there was no evidence of either recent or old myocardial infarction. In both cases the electrocardiographic interpretation was old anteroseptal myocardial infarction with either right bundle-branch block or peri-infarction block. One patient had chronic lymphocytic leukemia with diffuse myocardial scarring; the other had severe obstructive pulmonary emphysema and cor pulmonale. Twenty-six autopsy protocols with the diagnosis of ventricular aneurysm were reviewed. Eight (44.4%) of the electrocardiograms recorded during the final hospitalization in these cases showed peri-infarction block. This incidence is not different from the occurrence of peri-infarction block in the overall group.

Discussion

Clinical Significance of Peri-infarction Block

Intraventricular conduction defects of the kind described originally by First and associates (peri-infarction block) were present in a surprisingly high number of patients with myocardial infarction—39% in this series. In contrast, the incidence was found to be only 6% in the smaller, original series. Also, in con-
Intraventricular Block

In contrast, the vast majority (69%) appeared during the first week following the onset of infarction rather than several months later as was thought originally. When peri-infarction block occurs several months after infarction, it may be due to pressure atrophy of conducting fibers in the scarred region of muscle as postulated previously; however, the high incidence of peri-infarction block following recurrent myocardial infarction in this series suggests that repeated bouts of ischemia or necrosis may be responsible for its later appearance in many instances.

In view of the higher SGOT values for the patients with peri-infarction block, the increased mortality in these patients, as well as the block itself, may be related to a larger initial area of myocardial necrosis. Regression of the QRS changes characteristic of myocardial infarction is more likely to be observed in patients who have lower SGOT levels; in these patients, peri-infarction block is uncommon.

The higher crude mortality seen in patients with peri-infarction block than in other patients with infarction is impressive. However, the fallacy of attributing cause and effect to a single variable in patients with multiple complications and associated diseases has been pointed out in a previous study. Nonetheless, when discriminant analysis was used to analyze the effects of multiple variables on the immediate mortality of acute myocardial infarction, it was found that the presence of an intraventricular conduction defect, most commonly peri-infarction block, made a significant contribution to mortality. Probably the eventual significance of peri-infarction block in acute myocardial infarction will be determined from further experience accumulated in coronary care units.

Electrocardiographic Manifestations of Peri-infarction Block

Data are available from previously published studies of the activation of the ventricle in experimentally induced myocardial infarction (principally in dogs) which confirm the hypotheses postulated by Bayley from studies of electrocardiograms of human subjects with myocardial infarction and peri-infarction block. Immediately following occlusion, intramural propagation of the access process through the subendocardial regions of the highly ischemic muscle is sufficiently delayed to permit tangential activation of the outermost layers superjacent to the most intense zone of ischemia from surrounding less ischemic muscle. This mode of activation of the subepicardial regions overlying the infarct is known to exist in infarcts studied 4 to 10 weeks after occlusion. Delay in activation of the endocardial surface underlying the infarct has not been demonstrated. Initially an increase in the amplitude of R is observed in the epicardial regions overlying the infarct; later, the R amplitude decreases as significant Q deflections develop. The initial changes are reversible if the occlusion is of short duration. Examples of these temporary changes are seen occasionally in patients during bouts of angina pectoris. The electrocardiograms published by Prinzmetal and associates, designated as a "variant form of angina pectoris" seem to be of this kind. Exploration of a wide area of the precordium at frequent intervals during the early phases of myocardial infarction may show acute peri-infarction block to be a more common occurrence than has been appreciated generally. Because peri-infarction block appears in the majority of patients during the early stages of infarction, the term "post-infarction block," suggested by Castle and associates, may be a misnomer. The diagnosis of peri-infarction block may be qualified by the use of such adjectives as acute or chronic whenever justified. In some instances, the term may be modified to indicate "acute ischemic block" when the electrocardiographic changes are evanescent and significant Q deflections fail to appear.

As pointed out by First and co-workers, peri-infarction block frequently resembles bundle-branch block. In anteroseptal infarctions particularly, the distinction between right bundle-branch block and peri-infarction block usually cannot be made with any degree of certainty. An example is shown in...

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figure 4B. Figure 1B illustrates the similarity of anterolateral peri-infarction block to left bundle-branch block with infarction of the ventricular septum.

The criteria for peri-infarction block proposed by Grant and associates2–6 consisted of (1) an abnormality of the direction of the initial QRS forces of a type accounting for Q-wave characteristics of myocardial infarction, (2) little or no prolongation of the QRS interval, and (3) abnormality in the direction of the terminal QRS forces so that the vector points away from the initial QRS forces with an angle of 100° or more between the two vectors. Using these criteria, a high correlation of left ventricular disease, principally left ventricular hypertrophy and scarring, was found in several clinicopathological studies16–18 and “peri-infarction block” was also diagnosed in chronic pulmonary disease and in the absence of any apparent disease of the heart or lungs. The finding of discrete myocardial infarction varied from 5 to 58%. Thus, these criteria are not specific electrocardiographic indicators of myocardial infarction. It seems to us, therefore, that the use of more stringent criteria for the diagnosis of peri-infarction block is justified. We suggest that those criteria originally proposed are the most valid except in those instances in which the block and dead zone vectors lie perpendicular to the RLF electrode plane. In these cases there may be no prolongation of the QRS interval in the standard limb leads, but there will be a marked delay in the time of onset of the intrinsicoid deflection indicating a significant degree of intraventricular block associated with the infarction. Obviously, QS deflections, no matter how broad, cannot alone be interpreted as evidences of peri-infarction block.

References


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