A Clinical Appraisal of the Diagnostic Significance of "Peri-Infarction Block"**

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The term "peri-infarction block" was first used by First et al. in 1950 to describe a form of intraventricular conduction defect which develops following myocardial infarction due to involvement of the subendocardial region of the myocardium. Their criteria for this diagnosis were as follows: (1) evidence of subendocardial infarction, or of transmural infarction accompanied by a circumferential region of subendocardial damage, (2) prolongation of the QRS interval to over 0.10 second in the extremity leads, (3) characteristic alterations in the QRS configuration, namely slurring and prolongation of the terminal portion of the QRS complex, in addition to early QRS changes characteristic of infarction. Grant* proposed partly different criteria, stressing that a prolongation of QRS interval was not a prerequisite for this conduction defect. He stated that the basic features of "peri-infarction block" were an abnormality of direction of initial QRS forces of a type characteristic of myocardial infarction and an abnormality of direction of terminal QRS forces so that they point opposite to the initial QRS forces, the angle between them being 100° or more. Grant subdivided peri-infarction block into anterolateral and diaphragmatic, pointing out that the terminal vector is directed in such a way that this conduction defect is seen primarily accompanying only two of the five possible locations of infarction. Burchell and Pruitt drew attention to the value of esophageal leads in the elucidation of postinfarction intraventricular block. Recently, Mayer et al. introduced vectorcardiographic criteria for peri-infarction block.

Though this intraventricular conduction defect had been considered previously as characteristic of a recent or old myocardial infarction, pathologic correlation studies in recent years indicated its occurrence in diseases other than infarction, such as diffuse scarring in the left ventricle, left ventricular hypertrophy and pulmonary emphysema. In the present study, it was therefore attempted to determine the diagnostic significance of "peri-infarction block" in an unselected patient material. This study is based on the clinical evaluation of 24 patients with peri-infarction block who were encountered in a review of all electrocardiographic tracings obtained during the past three and one-half years in our Department.

Material and Methods

All electrocardiograms of patients admitted to our Department between January, 1962 and July, 1965 were reviewed for peri-infarction block. These constituted approximately 3700 tracings of 2100 patients. Peri-infarction block was found in the tracings of 24 patients by applying the following criterion: an angle of 110° or greater between the mean initial 0.04-second QRS vector and the mean terminal 0.04-second QRS vector.

The mean manifest QRS axis in the frontal plane, the mean initial and the mean terminal 0.04-second QRS vectors were estimated by reference to the hexaxial lead system. The net area subtended by the QRS complex, as well as during the initial and terminal 0.04-second of the QRS complex were determined in each case by algebraic summation of the value obtained by multiplying the amplitude by one half the duration at the base. A magnifying glass was used. With an upward QRS deflection, the upper edge, and with a downward

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### Table 1—Salient Clinical and ECG Data in 24 Cases with Peri-infarction Block

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Type of PIB</th>
<th>A&lt;sub&gt;qs&lt;/sub&gt; (degree)</th>
<th>Lead with Q&lt;sub&gt;=0.04&lt;/sub&gt;</th>
<th>Direction of QRS vector</th>
<th>Infarction</th>
<th>Electrical Location</th>
<th>Duration</th>
<th>LVH</th>
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**Group B**

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<th>A&lt;sub&gt;qs&lt;/sub&gt; (degree)</th>
<th>Lead with Q&lt;sub&gt;=0.04&lt;/sub&gt;</th>
<th>Direction of QRS vector</th>
<th>Infarction</th>
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<td>D*</td>
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**Group D**

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<th>Sex</th>
<th>Type of PIB</th>
<th>A&lt;sub&gt;qs&lt;/sub&gt; (degree)</th>
<th>Lead with Q&lt;sub&gt;=0.04&lt;/sub&gt;</th>
<th>Direction of QRS vector</th>
<th>Infarction</th>
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PIB = Peri-infarction Block  AL = anterolateral  D = Diaphragmatic  p = posteriord  a = anteriod  
LVH = left ventricular hypertrophy  *Cases with a QRS interval of more than 0.10 sec.  
†Cases having ventricular premature beats "of infarction type" (10)
deflection the lower edge of the tracing at the beginning of the QRS interval was taken for reference.

Conventional criteria were used for electrocardiographic diagnosis of myocardial infarction and its localization. To determine the extent to which the initial and terminal QRS vectors are directed anteriad or posteriad from the frontal plane, the method proposed by Grant for the interpretation of the tracings by vector methods was used.

**Results**

Pertinent clinical and electrocardiographic data on the 24 patients with peri-infarction block are summarized in Table 1. All but one were men. This sex ratio is partly attributable to the marked male preponderance in myocardial infarction, and in part due to the fact that approximately one-fifth of the beds in our Department are assigned to women. Their ages varied from 26 to 85 years with an average of 56.5 years. Two patients in this series died; necropsy was performed in one of them.

The cases have been grouped according to the presence and relative certainty of myocardial infarction as judged by clinical evidence and electrocardiographic early

**Figures 1A and 1B (Case 4):** A 65-year-old man with anterolateral peri-infarction block with a history of beginning and progressive angina pectoris of ten days’ duration suggested acute coronary insufficiency when first seen on April 29, 1965. The electrocardiogram that day (1A) showed no abnormality except an intraventricular conduction defect with terminal QRS forces directed superiorly. The tracing of May 11, 1965 (1B) was obtained the day he suffered a prolonged pain accompanied by a rise in S-GOT level. Standard and unipolar extremity leads exhibit no Q waves of 0.04-second duration, but indicate again a wide angle between initial and terminal QRS forces. Now, signs of anteroseptal infarction are evidenced in precordial leads.
A 59-year-old man with anterolateral peri-infarction block presented a history of myocardial infarction in January, 1965 and was hospitalized five months later with symptoms of left ventricular failure. The electrocardiogram demonstrates old anterior infarction. While the initial 0.04-second QRS vector points inferiorly and posteriorly, the terminal forces point superiorly. Slurring and notching in the early portion of the QRS complex in various leads are to be noted.

QRS changes characteristic of myocardial infarction.

Group A: Unequivocal evidence of recent or old myocardial infarction by electrocardiogram and by clinical history.

Of 15 cases comprising this group, all patients presented a history consistent with myocardial infarction. Those with a recent history, showed, in addition, the laboratory findings and characteristic evolution of electrocardiographic alterations of infarction. This was recent (less than six weeks in duration with reference to the electrocardiogram studied) in nine patients and
old in six. In two patients with two episodes of infarction, the episode which antedated the development of peri-infarction block was taken for reference. Four patients were mild diabetics. Clinical and roentgenographic evidence of significant left ventricular enlargement occurred in eight cases in this group.

Peri-infarction block was known to be present the first day of infarction in two patients and within two weeks of onset in all nine patients having a recent infarction.

An electrocardiogram preceding the myocardial infarction which led to the presumable development of the conduction disturbance was available in five cases (cases 2, 4, 5, 11, 12). In all five, peri-infarction block was absent in previous tracings. As illustrated in Fig. 4, the conduction defect was transient in case 12.

Of eight cases exhibiting anterolateral peri-infarction block, the electrical location was anteroseptal in two and anterior in six instances (with more or less subendocardial involvement). An electrocardiogram preceding the myocardial infarction which led to the presumable development of the conduction disturbance was available in five cases (cases 2, 4, 5, 11, 12). In all five, peri-infarction block was absent in previous tracings. As illustrated in Fig. 4, the conduction defect was transient in case 12.

Of eight cases exhibiting anterolateral peri-infarction block, the electrical location was anteroseptal in two and anterior in six instances (with more or less subendocardial involvement). In two patients with two episodes of infarction, the episode which antedated the development of peri-infarction block was taken for reference. Four patients were mild diabetics. Clinical and roentgenographic evidence of significant left ventricular enlargement occurred in eight cases in this group.

Peri-infarction block was known to be present the first day of infarction in two patients and within two weeks of onset in all nine patients having a recent infarction.
dial involvement of left ventricular free wall). Left axis deviation of $\Delta_{\text{QRS}}$ beyond $-30$ degrees was noted in five cases. Of seven cases with diaphragmatic peri-infarction block, the electrical location of infarction was posteroinferior in four, predominantly inferior in one and inferoanterior in two patients. The QRS interval did not exceed 0.10 second in any of the patients in this group.

**Group B:** Unequivocal electrocardiographic evidence of old myocardial infarction without an accompanying clinical history.

There were five patients in this group who did not recall symptoms of myocardial infarction in the past. One patient had coronary insufficiency with angina pectoris, two had progressive congestive heart failure not attributable to any other cardiac disease, and two patients (one of whom had mild hypertension) presented symptoms of cerebrovascular insufficiency. All cases in this group exhibited left ventricular hypertrophy except one, in whom this was borderline. Diaphragmatic peri-infarction block occurred in four and anterolateral peri-infarction block in one case. The QRS interval was prolonged in two patients to 0.11 and 0.15 sec., respectively.

**Group C:** Suggestive electrocardiographic evidence of old myocardial infarction without an accompanying clinical history.

This group consisted of two patients, aged over 60 years, both having anterolateral peri-infarction block. Case 21 presented a history of progressive congestive failure of six months' duration. The electrocardiogram demonstrated $R$ waves in leads $V_3,4$ smaller than in $V_1,2$ and a $Q$ wave in aVL of 0.03 sec. duration, a pattern suggestive of an old anteroseptal myocardial infarction which may be considered a cause of his congestive failure. In case 22, electrocardiographic signs were present which suggested old anteroseptal infarction with probable subendocardial involvement of anterolateral aspect without

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**Figure 5 (Case 24):** A 26-year-old man with congenital aneurysm of the sinus of Valsalva and an intraventricular conduction defect of anterolateral "peri-infarction block type." The patient presented symptoms and signs of a recently ruptured aortic sinus aneurysm with a left-to-right shunt. The electrocardiogram shows the initial QRS vector to be perpendicular to lead aVL, while wide $S$ waves in II, III and aVF indicate superior direction of the terminal vector. Early QRS changes characteristic of infarction are absent. Necropsy performed ten days later confirmed the diagnosis and excluded myocardial infarction (see text).
presenting a major abnormality in the mean initial 0.04-sec. QRS vector.

Group D: No evidence of myocardial infarction.

Two cases comprised this group. One was a man, aged 60, with mitral insufficiency, atrial fibrillation and left ventricular enlargement, presenting no clinical or electrocardiographic evidence of coronary insufficiency. The other patient died at 26 years of age from a ruptured congenital aortic sinus aneurysm in whom necropsy showed marked aneurysmal involvement of the interventricular septum. Myocardial infarction was excluded. Both cases had anterolateral peri-infarction block with a QRS interval of 0.11 sec. Not the initial but solely the terminal QRS forces were altered abnormally.

"Peri-infarction block not combined with the initial QRS deformity of infarction" There were seven patients with anterolateral "peri-infarction block" in whom Q waves of 0.04-sec. were absent in leads I and aVL (cases 3-5 and 21-24). In order to evaluate a distinction in clinical significance, these cases were grouped separately from those in whom peri-infarction block was combined with the initial QRS changes characteristic of infarction (Table 2). In the former group, three cases presented conclusive evidence of infarction, two were included in group C (possible or probable infarction), whereas two had no infarction. Left ventricular hypertrophy occurred in all but one of these seven cases.

**DISCUSSION**

In an appraisal of the diagnostic significance of peri-infarction block, electrocardiographic-pathologic correlation on a large patient material is obviously required to arrive at a definitive conclusion. Necropsy findings are, however, available in only one of the two patients who died in our series. Nevertheless, an essentially clinical and electrocardiographic evaluation of this subject is rewarding since the diagnostic accuracy of electrocardiographic early QRS changes supplemented by the clinical history is high in myocardial infarction.

Peri-infarction block has been encountered in 24 of the 2100 cases in whom electrocardiograms were available for review giving an overall incidence of 1.2 per cent. No selection of cases was made in this study except for the natural selection effected by a hospital population in a department of medicine and the fact that an electrocardiogram was obtained. With respect to the total number of cases of recent and old myocardial infarction seen in our Department during the same period, the incidence of this intraventricular conduction defect is approximately 7 per cent. First et al. observed peri-infarction block in 3.7 per cent of 464 patients with myocardial infarction. In an unselected material, O'Reilly and Sokolow found peri-infarction block in 19 patients during a survey of 4,000 consecutive electrocardiograms, an incidence which is closely comparable to that of the present study. Castle and Keane encountered the same conduction defect in over 4.2 per cent of electrocardiograms in deceased patients.

The exact mechanism of peri-infarction block is not known. Three theories have been proposed. First et al. postulated that the normal radial spread of activation toward the epicardium was prevented by subendocardial infarction and that areas of unaffected myocardium lying over this region were activated late through circuitous routes by peripherally located slower conducting fibers. Grant proposed that the basic mechanism involved a block of one of the two divisions (superior and inferior) of the left bundle. A third theory was introduced by Cabrera et al. who postulated that the typical vectorcardiographic picture

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**Table 2—Peri-infarction Block Categorized According to Initial QRS Deformity**

<table>
<thead>
<tr>
<th>Initial QRS Characteristic of Infarction</th>
<th>Number of Cases</th>
<th>Total</th>
<th>Left Ventr. Hypertrophy</th>
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<tr>
<td>Present</td>
<td>A B C D</td>
<td>17</td>
<td>10</td>
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<td>3 - - 2</td>
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is produced by early and late potentials generated by living muscle located within the infarcted area.

Peri-infarction block may occur as a transient phenomenon (Fig. 4) or may be permanent. A permanent peri-infarction block usually develops several months after infarction, but occasionally may occur early, either in transient or permanent form.1 In the present study, this conduction defect was known to be present within the first two weeks in all nine cases with a recent infarction. With the exception of one case, it remained stable for prolonged periods up to nine years.

Among the 24 cases with peri-infarction block in this report, those considered to have definite evidence of recent or old myocardial infarction constituted 15 (62 per cent). The seven cases included in groups B and C may be considered to have probably had myocardial infarction in the past, despite lacking a history. It is also possible that one or more of the cases represented diffuse myocardial scarring pathologically rather than gross scar as seen in infarction. Nevertheless, it is justified to state that in this series infarction was implicated in the development of peri-infarction block in the great majority of cases.

Kossman and colleagues' mentioned their experience based on necropsy controlled studies, that when the pattern of anterolateral peri-infarction block occurs in the electrocardiogram, there is about a 50 per cent chance that there is actually an anterolateral myocardial infarction present. Fibrosis of interventricular septum or free left ventricular wall was frequently encountered in the absence of infarction. Castle and Keane6 recently analyzed post-mortem findings in 55 patients with peri-infarction block. Twelve patients exhibited gross scar from previous infarction. In 26 cases, diffuse scarring with or without concomitant severe coronary sclerosis was found. Left ventricular hypertrophy alone, and an infiltrative process was implicated in three cases each. Ten cases with coronary atherosclerosis and chronic obstructive airway disease with pulmonary emphysema but with a "normal left ventricle" also exhibited peri-infarction block. In our series, the two cases who did not demonstrate any evidence of myocardial infarction or of coronary heart disease, had mitral insufficiency with left ventricular enlargement (case 23) and ruptured congenital aneurysm of the aortic sinus (case 24, Fig. 5). Both had anterolateral peri-infarction block. In the latter patient necropsy disclosed marked aneurysmal involvement of the interventricular septum. Since in this case free left ventricular wall was essentially uninvolved, it is possible that the superior division of the left bundle was damaged near its origin in the anterior portion of the septum rather than more peripherally in the free left ventricular wall. Such a possibility was raised by Kossman and co-workers' based on certain observations.

Anterolateral peri-infarction block due to myocardial infarction may manifest itself without a Q wave characteristic of infarction in the conventional electrocardiographic leads. Grant7 pointed out that in this situation a characteristically wide angle between the initial and terminal 0.04-second vectors would be diagnostic of infarction which would be missed by current pattern criteria. Davies and Evans8 also contended that a Q wave less than 0.04-second in duration, in the presence of the Sr-S2 pattern, can signify anterolateral peri-infarction block. With this consideration we have included in this report also those tracings demonstrating "peri-infarction block" not accompanied by early QRS changes characteristic of myocardial infarction in order to try to assess the significance of this form of peri-infarction block. This form was not in any way specific for infarction, as judged from data given in Table 2. In a pathologic study of 21 patients with selected cardiac diseases, Corne et al.9 also demonstrated that anterolateral peri-infarction block not accompanied by early QRS changes characteristic of infarction may occur in the absence of myocardial infarction. Kossman and associates' commented
that such a pattern may appear after transventricular surgical approaches to the aortic valve and in types of heart disease such as Chagas’ myocarditis and endocardial sclerosis. Conversely, they stated that in anterolateral peri-infarction block, normal direction of the initial 0.04-second QRS vector was encountered in 40 per cent of cases with infarction.

Though “peri-infarction block” with a normal direction of the initial 0.04-second QRS vector has no specificity, it is frequently associated with left ventricular hypertrophy which occurred in six of the seven cases in the present survey (Table 2). Left ventricular hypertrophy was said to be found at necropsy in “almost all” of the cases with peri-infarction block, although only about 25 per cent have met the conventional voltage criteria for left ventricular hypertrophy. Furthermore, patients with peri-infarction block were shown to have a higher incidence of heart disease (84 per cent) and coronary artery disease, and a lower incidence of clinically negative patients than those with left bundle-branch block.8

Based on the observations presented, the following conclusions can be reached: (1) anterolateral “peri-infarction block” not accompanied by early QRS changes characteristic of infarction usually signifies organic left ventricular disease, but is of limited aid in the diagnosis of infarction, since this pattern may also occur particularly as a result of diffuse myocardial fibrosis due to a variety of causes. The term intraventricular conduction defect of the “peri-infarction block type” would be more appropriate for this form, as also proposed by Castle and Keane;9 (2) peri-infarction block combined with the initial QRS deformity characteristic of infarction signifies usually myocardial infarction, but not invariably. As suggested by Corne and associates, the term “peri-infarction block” (or more suitably, “postinfarction block”) should be restricted to the combination of abnormalities of the terminal QRS forces with the initial QRS deformity characteristic of infarction.

More experimental data are needed for a better comprehension of the exact mechanism and more clinico-pathologic correlation data in large series of patients for a more precise appraisal of the diagnostic significance of this intraventricular conduction disturbance.

**SUMMARY**

During a review of approximately 3700 consecutive electrocardiograms in 2100 cases, “peri-infarction block” was encountered in 24 patients. The diagnostic significance of peri-infarction block was evaluated by a detailed analysis of the clinical findings and electrocardiographic early QRS changes. Definite evidence of myocardial infarction was found in 15 patients, while seven patients with cardiovascular or cerebrovascular disease presented electrocardiographic findings consistent with an infarction without an accompanying clinical history. Myocardial infarction was absent in two cases in one of which necropsy disclosed congenital aortic sinus aneurysm with involvement of the interventricular septum.

In accordance with the varying diagnostic significance, it was emphasized to distinguish peri-infarction block from the intraventricular conduction disturbance of the “peri-infarction block type” not combined with the initial QRS deformity characteristic of infarction. The latter is nonspecific, but frequently reflects left ventricular hypertrophy. Pertinent literature on peri-infarction block was reviewed.

**Resumen**

El bloqueo post infarto ha sido observado en 24 casos en un total de 3.700 ECG consecutivos, obtenido en 2.100 pacientes.

La significación diagnóstica del bloqueo post infarto ha sido evaluada mediante el análisis detallado de los hallazgos clínicos y los cambios electrocardiográficos precoces del complejo RQS. En 15 pacientes se observaron signos evidentes de infarto del miocardio, al paso que en 7 sujetos con patología cardiovascular o cerebrovascular se encontraron signos electrocardiográficos compatibles con infarto sin expresión clínica evidente. En dos casos no se comprobó infarto mio-
cardio. En uno de estos la autopsia demostró la presencia de aneurisma congénito del seno aórtico con participación del septum interventricular.

Es preciso distinguir el bloqueo peri-infarto del trastorno de conducción intraventricular del mismo tipo no acompañado de la deformación inicial del complejo RQS característico del infarto. Este último no es específico, y con frecuencia indica hipertrofia ventricular.

Hemos revisado la literatura relativa al bloqueo periinfartico.

**RESUMÉ**

A la suite de l'examen d'environ 3 700 électrocardiogrammes consécutifs dans 2 100 cas, le "Peri-infarction Bloc" a été rencontré chez 24 malades. La signification diagnostique du Peri-infarction Bloc a été évaluée par une analyse détaillée des trouvailles cliniques et des modifications électrocardiographiques initiales de QRS. Une preuve nette d'infarctus myocardique a été trouvée chez 15 malades alors que 7 ayant des maladies cardiovasculaires ou cérébrovasculaires avaient des signes électriques suggérant un infarctus sans aucune histoire clinique associée. L'infarctus du myocarde était absent dans 2 cas, et dans l'un d'eux l'autopsie a montré un anévrisme congénital du sinus aortique, avec atteinte du septum interventriculaire.

En accord avec la signification variée sur le plan diagnostique, il importe de distinguer le Peri-infarction Bloc des troubles de la conducción intraventriculaire du "Type Peri-infarction," sans qu'il y ait en même temps d'anomalie initiale de QRS caractéristique dans l'infarctus. Cette dernière n'est pas spécifique, mais reflète fréquemment une hypertrophie ventriculaire gauche. Les publications antérieures sur le Peri-infarction Bloc ont été revues.

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**REFERENCES**


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