Electrocardiographic Poor R-Wave Progression
Correlation with Postmortem Findings

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Electrocardiographic criteria have been derived from vectorcardiographic and angiographic correlation which allow division of patients with electrocardiographic “poor R-wave progression” or “reversed R-wave progression” into the following four etiologic groups: (1) anterior myocardial infarction; (2) left ventricular hypertrophy; (3) type-C right ventricular hypertrophy; and (4) the normal variant. The sensitivity, specificity, and predictive value of this approach to the electrocardiogram with poor or reversed R-wave progression were studied in a series of 33 patients examined at autopsy.

Using the scheme and criteria outlined, 85 percent (11/13) of the pathologic anterior myocardial infarctions were correctly diagnosed. The electrocardiographic criteria correctly identified 75 percent (15) of 20 patients with poor or reversed R-wave progression without postmortem evidence of myocardial infarction, with only 12 percent (2/17) predictive error. The relative risk of autopsy-documented anterior myocardial infarction in patients meeting the specified electrocardiographic criteria was six times that of other patients with poor or reversed R-wave progression.

Classification is based on standard electrocardiographic criteria and simple derived discriminants. When the R wave in lead 1 is less than or equal to 4.0 mm or the R wave in lead V3 is less than or equal to 1.5 mm, anterior myocardial infarction is likely unless criteria for type-C right ventricular hypertrophy are present. In the absence of these findings, classification as left ventricular hypertrophy or normal has a high predictive value for no anterior myocardial infarction.

The purpose of this study is to apply the previously derived approach to the ECG with poor R-wave progression to a group of patients examined at autopsy, in order to test and improve the accuracy of electrocardiographic classification.

Materials and Methods

Postmortem records from 1973 to 1977 were reviewed. The ECGs of patients who at autopsy were found to have had left ventricular hypertrophy, anterior myocardial infarction, anterior myocardial infarction with left ventricular hypertrophy, subendocardial myocardial infarction, or no cardiac pathologic findings were retrieved.

Isolated ventricular weights were not available. The diagnosis of left ventricular hypertrophy was made in female patients when total heart weight equalled or exceeded 350 gm (range, 350 gm to 540 gm) and in male patients when total heart weight equalled or exceeded 400 gm (range, 400 to 700 gm). The minimum weights used to diagnose left ventricular hypertrophy are two standard deviations of the mean greater than the normal heart weight for male and female subjects as determined by Zeek.4 The diagnosis of right ventricular hypertrophy was
made only when total heart weight did not exceed the minimum weight used to diagnose left ventricular hypertrophy and when the thickness of the right ventricular free wall measured at the base of the heart equaled or exceeded 6 mm. In all other cases of anterior wall infarces, there was microscopic confirmation of old (characterized by fibrous scar) infarct, and in most cases the infarcts were visible on gross examination of the hearts. Infarcts involving less than two-thirds the thickness of the left ventricular myocardium were designated nontransmural or subendocardial infarction. Patients with recent myocardial infarction by pathologic or clinical criteria (or both), postmortem findings of probable cardiomyopathy, or bundle-branch block on the ECG were excluded.

Thirty-three patients satisfied the previously specified criteria and possessed poor or reversed R-wave progression on the two ECGs recorded closest to the date of death. Only such cases were used, in order to ensure that the electrocardiographic pattern was not secondary to improper lead placement. Poor R-wave progression was present when the absolute magnitude of the R wave was less than or equal to 3.0 mm in lead V2 and the R wave in lead V6 was less than or equal to the R wave in lead V5 (10 mm = 1 mV). Reversed R-wave progression was defined as the presence of decreasing R waves such that the R wave in lead V5 was less than the R wave in lead V6, or the R wave in lead V6 was less than the R wave in lead V2, or the R wave in lead V2 or lead V6 was less than or equal to 3.0 mm.1,3

The ECGs were interpreted in a blind manner, with the reader having no knowledge of which pathologic abnormality (left ventricular hypertrophy, right ventricular hypertrophy, anterior myocardial infarction, anterior myocardial infarction with left ventricular hypertrophy, or no abnormality) was associated with the pattern of poor or reversed R-wave progression. Abnormalities of repolarization were recorded when present. Voltage criteria used for the diagnosis of left ventricular hypertrophy were as follows: (1) Ungerleider8 (R wave in lead I plus S wave in lead 3 greater than or equal to 25 mm; or (2) Lewis8 (net positive lead 1 plus net negative lead 3 greater than or equal to 17 mm); or (3) Sokolow10 (R wave in lead aVL greater than or equal to 11.0 mm); or (4) Sokolow10 (S wave in lead V1 greater than or equal to 20 mm). Poor and reversed R-wave progression may be present in type-C right ventricular hypertrophy, since most of the QRS vector loop projects onto the negative side of the lead axis of the right and midprecordial leads. The late rightward forces present in this condition, moreover, are responsible for the significant S wave present in orthogonal lead X and standard limb lead I, and allows differentiation of patients with poor R-wave progression (or reversed R-wave progression) secondary to type-C right ventricular hypertrophy from those secondary to anterior myocardial infarction. An electrocardiographic diagnosis was determined from the scheme outlined in Figure 1, which is based upon previous vectorcardiographic11 and angiographic10 diagnostic correlation.

Electrocardiographic correlation with each of the postmortem findings was analyzed according to the following definitions:12 (1) true-positive ECG: ECG abnormal and abnormal findings on autopsy; (2) true-negative ECG: ECG normal and normal findings on autopsy; (3) false-positive ECG: ECG abnormal and normal findings on autopsy; (4) false-negative ECG: ECG normal and abnormal findings on autopsy; (5) sensitivity: true-positive ECGs divided by total autopsies with abnormal findings; (6) specificity: true-negative ECGs divided by total autopsies with normal findings; (7) predictive value of an abnormal electrocardiographic criterion: true-positive ECGs divided by total abnormal ECG; (8) predictive value of a normal electrocardiographic criterion: true-negative ECGs divided by total normal ECGs; (9) predictive error: false-negative ECGs divided by total normal ECGs; and (10) relative risk: predictive value of an abnormal electrocardiographic criterion divided by predictive error.

The extent of myocardial necrosis in an additional 14 randomly selected autopsies of patients with Q waves (QS, qr, or qR complexes) in the right or mid precordial leads (or both) secondary to myocardial infarction was compared with that in patients with poor or reversed R-wave progression secondary to myocardial infarction.

Significant differences between the means of various variables were sought using the two-tailed Student’s t-test. Pearson’s correlation coefficients (r) between certain of the variables were calculated by the raw score method.13 Comparison of proportions in two independent samples was achieved by x² corrected for continuity.14 Where the expected frequencies were less than five, Fisher’s exact probability test was substituted.14

**RESULTS**

**Postmortem Classification.**

The 33 patients with poor or reversed R-wave progression were classified by diagnosis at autopsy.

**Group 1 (Anterior Myocardial Infarction).** There were 13 patients with anterior myocardial infarction, including 15 percent (5/33) of the total population with anterior myocardial infarction alone (group 1A) and 24 percent (8/33) with additional left ventricular hypertrophy (group 1B). In group 1B the degree of left ventricular hypertrophy as assessed by wall thickness was poorly related to the R wave in lead V3 (r = 0.21) but was well correlated with the S wave in lead V2 (r = 0.89). Both by the total weight of the heart (probability not significant) and by the thickness of the left ventricular wall (probability not significant), patients in group 1B were not different from patients in group 2. Deeply inverted, symmetric T waves in right precordial leads V2 and V3 were seen in 31 percent (4/13) of the patients in group 1 but only in one patient in other groups. Persistently elevated S-T segments (≥ 2.0 mm at 60 msec after the J point) were present in the right precordial leads in 31 percent (4/13) of the patients in group 1 but in none from the other groups. None of the patients in group 1 demonstrated depressed S-T segments in the right precordial leads.

**Group 2 (Left Ventricular Hypertrophy).** Patients with left ventricular hypertrophy and normal coronary arteries comprised 21 percent (7/33) of the population under study. The degree of left ven-
tricular hypertrophy as assessed by wall thickness was poorly related to the R wave in lead V₅ (r = 0.21) but correlated well with the S wave in lead V₂ (r = 0.89). Biphasic T waves rightward of precordial lead V₄ occurred in two cases.

**Group 3 (Right Ventricular Hypertrophy).** There were two cases with right ventricular hypertrophy and normal coronary arteries, comprising 6 percent (2/33) of the population under study.

**Group 4 (Normal).** There were 11 patients with normal myocardium and insignificant coronary arterial disease at autopsy, comprising 33 percent (11/33) of the population under study.

**Electrocardiographic Classification**

The results of electrocardiographic correlations with postmortem findings are outlined in Table 1.

**Group 1.** The electrocardiographic criteria correctly identified 11 of 13 patients with anterior myocardial infarction with a sensitivity of 85 percent, a specificity of 75 percent (15/20) and a predictive error (or false exclusion of anterior myocardial infarction) of only 12 percent (2/17). The predictive accuracy of an ECG anterior myocardial infarction was 69 percent. The predictive value for no anterior myocardial infarction of absent electrocardiographic criteria for anterior myocardial infarction was 88 percent. The relative risk of a patient with poor or reversed R-wave progression having anterior myocardial infarction if the electrocardiographic criteria of Figure 1 were satisfied was 5.8 times that of other individuals with poor or reversed R-wave progression.

**Group 2.** The electrocardiographic criteria for left ventricular hypertrophy have relatively low sensitivity (43 percent; 3/7) but high specificity (100 percent; 26/26), with many individuals with a minor degree of postmortem left ventricular hypertrophy being classified as normal. The predictive accuracy of electrocardiographic criteria for left ventricular hypertrophy was 100 percent (3/3). The predictive value for no left ventricular hypertrophy of absent electrocardiographic criteria for left ventricular hypertrophy was 87 percent (26/30). The relative risk of a patient with poor or reversed R-wave progression having left ventricular hypertrophy if the electrocardiographic criteria of Figure 1 are satisfied was 7.7 times that of other individuals with poor or reversed R-wave progression.

**Group 3.** The electrocardiographic sensitivity for autopsy-proven right ventricular hypertrophy was only 50 percent (1/2). Because of the small number of cases of right ventricular hypertrophy, the specificity was high (97 percent; 30/31).

**Group 4.** The electrocardiographic criteria for normal patients have low sensitivity (55 percent; 6/11) and specificity (73 percent; 16/22). The predictive accuracy for identifying autopsy-proven absence of disease was only 50 percent (6/12), largely because of difficulty in overlap with autopsy-documented milder forms of left ventricular hypertrophy, as reflected by low sensitivity in group 2.

**Type of Infarction**

Poor R-wave progression was seen in patients with septal (four), anterior (six), apical (one), lateral (one), anterolateral (one), and posterolateral (one) myocardial infarctions, or combinations of these types. If poor R-wave progression extended from lead V₁ to V₃, then septal or anterior left ventricular free wall myocardial infarction had occurred (6/8). Extension of poor R-wave progression to precordial leads V₄ and V₅ (or both) suggested apical or lateral wall involvement (2/3). Reversed R-wave progression from leads V₁ to V₄ (V₅) was seen in two of two cases of circumferential subendocardial myocardial infarction.

In 13 patients with poor or reversed R-wave progression on the ECG and myocardial infarction of the left ventricular free wall at autopsy, 92 percent (12) had nontransmural myocardial infarction. In five patients with Q waves (QS, qR, or qR complexes) in precordial lead V₂ only and with myocardial and infarction of the left ventricular free wall at autopsy, 80 percent (four) had nontransmural infarction (probability not significant). In seven patients with Q waves (QS, qR, or qR complexes) in precordial leads V₁ to V₃ and with myocardial infarction of the left ventricular free wall at autopsy, only 29 percent (two) had nontransmural myocardial infarction (P < 0.01).

**DISCUSSION**

Poor and reversed R-wave progression are commonly encountered patterns in adult electrocardiograms, occurring in as many as 10 percent of all hospitalized patients. Furthermore, a third of the patients with previous anterior myocardial infarction may have only this finding on the surface ECG. Thus, elucidation of the specific anatomic equivalents of this electrocardiographic pattern by postmortem study is of clinical significance.

The present study provides direct pathologic confirmation of the existence of four subgroups of patients who may have poor or reversed R-wave progression on their ECG. These include (1) anterior myocardial infarction, (2) left ventricular hypertrophy, (3) right ventricular hypertrophy, and (4) normal individuals.
Of paramount importance is the ability to differentiate patients with anterior myocardial infarction from those with poor R-wave progression on the ECG secondary to other causes. An approach to the patient with poor or reversed R-wave progression on the ECG is shown in Figure 1. Although investigations regarding lead placement have shown more variability in placement of left precordial leads than right, at least two ECGs should be recorded with attention paid to lead placement in order to assure that the poor or reversed R-wave progression noted is not artifactual. Having been assured of this, application of the criteria shown in Figure 1 will overdiagnose the presence of myocardial infarction; however, of clinical usefulness, this study demonstrates that 75 percent of the patients with either poor or reversed R-wave progression who do not have an anterior myocardial infarction can be identified by the 12-lead ECG with only 12 percent predictive error (or false exclusion of anterior myocardial infarction). Stated differently, the predictive value of an ECG negative for anterior myocardial infarction is as high as 88 percent (15/17). On the basis of our pathologic data, the relative risk of a patient with poor or reversed R-wave progression having an anterior myocardial infarction if criteria of Figure 1 are met is approximately six times that of other individuals with poor or reversed R-wave progression.

Since patients in group 1B (anterior myocardial infarction with left ventricular hypertrophy) and group 2 (left ventricular hypertrophy) did not differ in their degree of left ventricular hypertrophy, the concomitant presence of myocardial infarction was probably responsible for the R wave in lead V₃ being less than or equal to 1.5 mm in group 1B. Therefore, Figure 1 may be used for the diagnosis of myocardial infarction in patients with poor or reversed R-wave progression regardless of whether or not concomitant left ventricular hypertrophy by voltage criteria is also present.

In the setting of poor or reversed R-wave progression, deep symmetrically inverted T waves in the right precordial leads were a rather insensitive discriminator (31 percent; 4/13) for the detection of previous myocardial infarction but were quite specific in this regard (5 percent false-positives); however, biphasic or flat S-T intervals in these leads were nonspecific and of little diagnostic value regarding the cause of poor or reversed R-wave progression in a given case. Likewise, the presence of significant ST-segment elevation (≥2 mm) in the right precordial leads, while of relatively low sensitivity (31 percent; 4/13), possessed high specificity for the detection of previous myocardial infarction (no false-positives). ST-segment depression was not of diagnostic value, being present in some cases of isolated pathologically proven left ventricular hypertrophy.

Poor and reversed R-wave progression were associated predominantly with nontransmural myocardial infarction. On the other hand, Q-wave infarctions in the same leads were associated with predominantly transmural myocardial infarction. A recent smaller retrospective study correlating postmortem anatomic findings with electrocardiographic changes in patients with myocardial infarction also
Table 1—Postmortem Findings and Electrocardiographic Correlation in Poor R-Wave Progression*

<table>
<thead>
<tr>
<th>Data</th>
<th>Group 1</th>
<th>Group 1A</th>
<th>Group 1B</th>
<th>Group 2</th>
<th>Group 3</th>
<th>Group 4</th>
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<tr>
<td>No. of cases (percent)</td>
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<td>5/33 (15)</td>
<td>8/33 (24)</td>
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*Group 1, all anterior myocardial infarctions; Group 1A, only anterior myocardial infarction; Group 1B, anterior myocardial infarction plus left ventricular hypertrophy; Group 2, left ventricular hypertrophy; Group 3, right ventricular hypertrophy; and Group 4, normal.

revealed that 50 percent (two) of four patients with poor R-wave progression had infarcts confined to the subendocardium, whereas only 13 percent (1/8) of the Q-wave infarcts were similarly confined.17 This is consistent, moreover, with ventriculographic evidence in living subjects, suggesting less marked abnormalities of wall motion in poor R-wave progression than in Q-wave infarctions. Reversed R-wave progression, while also nonspecific for myocardial infarction, when present in cases of pathologically proven myocardial infarction seemed to be limited to cases with circumferential nontransmural myocardial infarction, perhaps implying a more extensive degree of myocardial necrosis than in cases with merely poor R-wave progression.

Patients with poor, or reversed R-wave progression secondary to left ventricular hypertrophy by our criteria may represent a more severe degree of autopsy postmortem left ventricular hypertrophy (mean weight of heart, 495 gm) than those individuals misclassified by the electrocardiographic criteria of Figure 1 as normal, in whom there was autopsy-proven left ventricular hypertrophy (mean weight of heart, 385 gm). This explains the relatively low electrocardiographic sensitivity for the detection of left ventricular hypertrophy (Table 1) and is consistent with previous studies.18,20 The association of left ventricular hypertrophy with left axis deviation21 and the inclusion of individuals with autopsy-documented mild left ventricular hypertrophy in the electrocardiographic group 4 (normal) by our criteria as demonstrated in this study, may contribute to the leftward axis previously described in those subjects with poor or reversed R-wave progression who do not possess quantitative vectorcardiographic criteria for anterior myocardial infarction, type-C right ventricular hypertrophy, or left ventricular hypertrophy.1

Regardless of the mechanisms involved, considering the common occurrence of poor and reversed R-wave progression in clinical practice, more accurate assessment of routine ECGs regarding the presence or absence of organic heart disease and particularly coronary arterial disease has immediate clinical application in the management of patients.

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