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Poor R Wave Progression in the Precordial Leads: Clinical Implications for the Diagnosis of Myocardial Infarction

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A definite diagnosis of anterior myocardial infarction is often difficult to make in patients when a pattern of poor R wave progression in the precordial leads is present on the electrocardiogram. The purpose of this study was to determine whether a mathematical model could be devised to identify patients with anterior infarction among 102 consecutive patients with poor R wave progression. Each patient underwent exercise testing with thallium scanning. The diagnosis of anterior infarction was established in 20 (20%) of the 102 patients by the presence of fixed thallium-201 perfusion defects in the anterior wall or septum, or both. With the use of a multivariate stepwise discriminant analysis of clinical and electrocardiographic variables, five variables (sex, ST-T changes, S wave amplitude in leads V_2 and V_3 and the sum of the R wave amplitude in leads V_3 and V_4) that were statistically significant by univariate analysis were selected by the model to identify patients with anterior infarction (sensitivity 85%, specificity 71%). The discriminant model was subsequently applied prospectively to an additional 21 patients with poor R wave progression and provided a sensitivity of 85% and a specificity of 88%.

Thus, anterior infarction (fixed thallium-201 defects in the anteroseptal segments) was present in 20% of patients with poor R wave progression in the precordial leads; and a mathematical model can be used to identify a subset of patients with anterior infarction in a group of patients with poor R wave progression.

Electrocardiographic criteria of poor R wave progression in the precordial leads have been associated with various cardiac diseases (1–4). The definition and clinical implication of this entity are controversial (5). The electrocardiographic pattern of poor R wave progression frequently is described by terms such as "possible," "probable," "consistent with," or "cannot rule out" anterior myocardial infarction. Despite prior studies (6–8) in which findings from vectorcardiography, contrast ventriculography or autopsy were used to define the presence or absence of myocardial infarction in patients with poor R wave progression, the incidence of the latter and its predictive value have not been established.

Most electrocardiograms (such as those in the heart station) are interpreted without direct access to the patient or technician to ascertain proper electrode position. False positive and negative poor R wave progression may be associated with change in position of superior and inferior leads, respectively. Therefore, at least two electrocardiograms should be recorded on different occasions with attention paid to electrode position to ensure that poor R wave progression is not artifactual. If poor R wave progression is recognized, it would be important to interpret it in terms of the probability that this electrocardiographic abnormality is due to anteroseptal myocardial scar.

Thallium-201 myocardial scintigraphy is useful in evaluating patients with coronary artery disease; a fixed perfusion defect is specific and sensitive for the diagnosis of myocardial infarction (9). Quantitation of the amount of infarcted myocardium by thallium scintigraphy correlates well with pathologic findings (10). The purposes of this study were to determine 1) the incidence of poor R wave progression in the precordial leads; 2) the correlation between the size of infarction in two groups of patients with anterior infarction, one with poor R wave progression and the other with Q waves in the precordial leads; and 3) whether a mathematical model can be devised to differentiate patients

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with poor R wave progression and anterior infarction from the remaining patients with poor R wave progression but no infarction.

Methods

Study patients. We reviewed the rest 12 lead electrocardiograms of 1,250 consecutive patients who subsequently underwent exercise thallium-201 scintigraphy. All electrocardiograms were performed with the patient in the supine position. Each patient had two electrocardiograms, one before and one after the exercise. All electrocardiograms were performed by one individual who has had extensive experience in recording electrocardiograms and is aware of the importance of proper electrode placement; therefore, interobserver variability in electrode placement was not a problem. Finally, the exercise studies were supervised and electrode placement was automatically checked if poor R wave progression was noted on the electrocardiogram.

One hundred two patients had electrocardiographic criteria of poor R wave progression or reverse R wave progression in the precordial leads. These patients were between the ages of 34 and 77 years (mean age ± standard deviation 52 ± 9). There were 50 men and 52 women.

Electrocardiography. All patients had chest pain and were being evaluated for suspected angina pectoris. Poor R wave progression was considered present when the R wave amplitude was 3 mm or less in precordial lead V_3 (the electrocardiogram was standardized so that a 10 mm deflection was equal to 1 mV) and when the R wave amplitude in lead V_2 was equal to or smaller than the R wave amplitude in V_3. A reverse R wave progression was defined as the presence of decreasing R wave amplitude such that the R wave in lead V_4 was smaller than the R wave in V_3, or the R wave in V_3 was smaller than the R wave in V_2, or the R wave in V_2 was smaller than the R wave in V_1 (or any combination of these), provided the R wave amplitude in V_3 was 3 mm or less (5–8). In this study, as in other studies (5–8), poor and reverse R wave progression are both referred to as poor R wave progression.

Patients with wide QRS complexes (QRS duration 110 ms or greater), left anterior fascicular block, Wolff-Parkinson-White syndrome, left ventricular hypertrophy or electrocardiographic criteria of myocardial infarction at other lead sites were excluded. Similarly, patients with a QS pattern in precordial lead V_2 or V_3, or both, and those with low QRS voltages were excluded. The electrocardiograms were interpreted without knowledge of the results of thallium scintigraphy. Abnormalities of repolarization in leads V_2 and V_3 were recorded.

The amplitude of the R and the S waves was measured and averaged over three cycles in leads I, V_1, V_2, V_3 and V_4, because these leads were analyzed for detecting anterior myocardial infarction in patients with poor R wave progression (Table 1). The R/S ratio in several leads and the summation of the R wave in multiple leads were also measured.

Control patients. During the same time period, 21 patients with prior transmural anterior infarction (defined as abnormal Q waves 0.04 second or more in duration in the precordial leads) underwent stress thallium-201 scintigraphy. The results of scintigraphy in these patients were analyzed separately and were compared with those of patients with fixed perfusion defects, but with poor R wave progression.

Thallium-201 imaging. Each patient underwent multistage treadmill exercise testing in the fasting state in accordance with the Bruce protocol. The exercise was continued until the patient complained of angina with or without ST segment depression, excessive fatigue, leg weakness or shortness of breath, or when we observed hypotension, frequent premature ventricular beats or ventricular tachycardia. At peak exercise, 2 mCi of thallium-201 was injected intravenously and the patient was asked to continue exercising for 30 to 60 seconds. Ten minutes after the termination of the exercise, images were obtained in three projections. The techniques for exercise testing and imaging, and the interobserver and intraobserver variability in interpreting the results, have been previously described in detail (11,12). Redistribution images were obtained 4 hours after injection.

The nature of the defects (fixed vs. transient) and their locations were determined. Persistence of the defect in the delayed images because of absent or partial redistribution was considered to indicate the presence of residual myocardial scar. If these defects involved the anterolateral segment (in the anterior projection), the septum (in the shallow left anterior oblique projection) or the anterior wall (in a steep left anterior oblique projection), they were considered to represent anterior scar. The size of the defects in the delayed images was quantitatively analyzed by the perimetric method (12,13). The size of the defect was determined by measuring the perimeter of the defect with a computerized Hewlett-Packard 982A digitizer and expressing it as a percent of the total left ventricular perimeter in each projection, the region of the valve being excluded. This method is similar to that used by Feild et al. (14) in assessing the extent of akinetic-dyskinetic segments by means of left ventriculography. The average abnormal perimeter was determined from the three projections (Fig. 1).

Statistical analysis. Univariate and multivariate statistical analyses were performed. The presence or absence of anterior scar as determined by thallium imaging was considered to be the dependent variable and all other variables were considered the independent variables. Statistical significance was determined using chi-square analysis with Yates' correction and Fisher's exact test. Stepwise discriminant analysis was performed to derive a multivariate function to classify patients with poor R wave progression into

Table 1. Clinical and Electrocardiographic Variables in Patients With (Group I) or Without (Group II) Thallium Scintigraphic Evidence of Anterior Wall Myocardial Infarction

	Group I (n = 20)		Group II (n = 82)		
	No.(%)	Mean ± Standard Deviation	No.(%)	Mean ± Standard Deviation	p Value
Age (yr)		52 ± 8		52 ± 9	NS
Sex					
Male	15(75)		35(43)		NS
Female	5(25)		47(57)		NS
PRWP	16(80)		77(94)		NS
RRWP	4(20)		5(6)		NS
R_1 (mm)		5.5 ± 3.5		5.3 ± 3.1	NS
S_1 (mm)		0.70 ± 0 84		0.47 ± 0.77	NS
RV_1 (mm)		0.65 ± 0.71		0.62 ± 0.61	NS
SV_1 (mm)		9 ± 3.0		8.5 ± 3.1	NS
RV_2 (mm)		1.2 ± 0.89		1.4 ± 1.3	NS
SV_2 (mm)		12.4 ± 4.9		10.6 ± 3 9	0.02
RV_3 (mm)		1 6 ± 0.8		2.0 ± 0.8	NS
SV_3 (mm)		11 0 ± 5.3		10.0 ± 4.4	NS
RV_4 (mm)		7.7 ± 5 3		9.8 ± 5.0	NS
SV_4 (mm)		4 7 ± 3.5		5.1 ± 3.5	NS
ST-T changes					
In either V_2 or V_3	3(14)		4(5)		NS
In V_2 + V_3	5(23)		2(3)		NS
R/S ratio in V_1		0 08 ± 0.08		0.09 ± 0.10	NS
R/S ratio in V_2		0.11 ± 0.10		0.17 ± 0.20	NS
R/S ratio in V_3		0.20 ± 0.14		0 25 ± 0 15	NS
R/S ratio in V_4		2.3 ± 2 7		4.2 ± 8 4	NS
RV_3 + RV_4 (mm)		9 3 ± 5 5		11 8 ± 5.2	NS
RV_1 + RV_2 + RV_3 + RV_4 (mm)		11 2 + 6 3		13.8 ± 5.5	NS
RV_2 + RV_3 (mm)		2 8 ± 1.4		3 4 ± 1.8	NS
RV_2 + RV_3 + RV_4 (mm)		10 5 ± 5.9		13.2 ± 5 4	NS
RV_3 < 1.5 mm	11(55)		28(34)		NS

n = number of patients, p = probability; PRWP = poor R wave progression, RRWP = reverse R wave progression

outcome categories (anterior infarction or no infarction). Independent variables were considered candidates for forward entry into the function and backward elimination from the function if their association with the dependent variable and their contribution to the discriminant function were significant (F = 1.5). A discriminant function was calculated from the derivation sample (15).

Definition of statistical terms. From the electrocardiograms and the thallium imaging results, the following terms are defined:

True positive (TP): The presence of anterior infarction by the discriminant score (a score greater than zero) and anterior scar by thallium imaging.

True negative (TN): Absence of anterior infarction according to the discriminant score and absence of anterior scar by thallium imaging.

False positive (FP): Presence of anterior infarction by the discriminant score but no anterior scar by thallium imaging.

False negative (FN): Absence of anterior infarction by discriminant score but presence of anterior scar by thallium imaging.

$$Sensitivity = \frac{TP}{TP + FN} \times 100,$$

$$Specificity = \frac{TN}{TN + FP} \times 100,$$

$$Positive\ predictive\ value = \frac{TP}{TP + FP} \times 100,$$

$$Negative\ predictive\ value = \frac{TN}{TN + FN} \times 100,$$

$$Predictive\ accuracy = \frac{TP + TN}{TP + TN + FP + FN} \times 100,$$

$$Predictive\ error = \frac{FN}{TN + FN,}$$

$$Relative\ risk = \frac{\text{Positive predictive value}}{\text{Predictive error.}}$$

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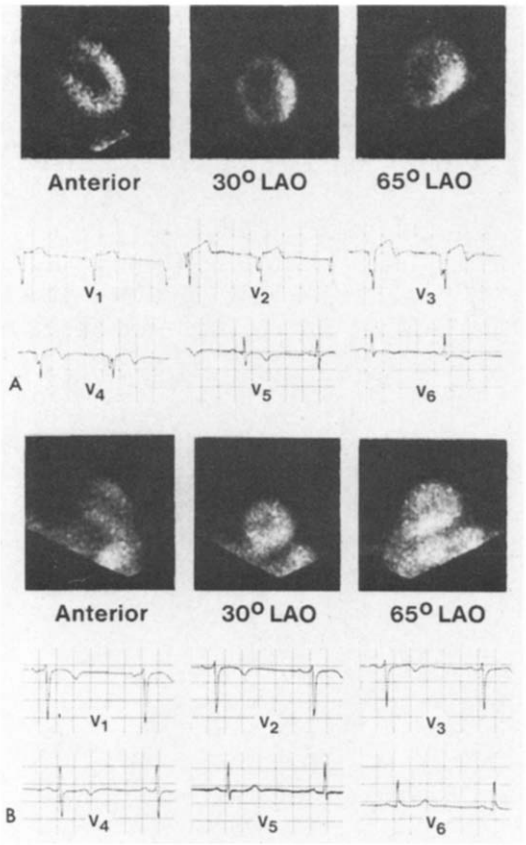


Figure 1. Electrocardiographic precordial leads and thallium-201 images in a patient with Q wave infarction **(A)** in a patient with poor R wave progression **(B).** The anteroseptal perfusion defects are more extensive in the patient with Q wave infarction. LAO = left anterior oblique view.

Results

Clinical features. Of the 102 patients with poor R wave progression in the precordial leads, 20 (20%) had anterior scar by thallium-201 scintigraphy (Group I) and the remaining 82 patients (80%) did not (Group II). There was no significant difference in age between the two groups (52 ± 8 years in Group I vs. 52 ± 9 years in Group II; probability [p]: not significant [NS]). Men constituted 75% of the patients in Group I (15 of 20 patients) and 43% in Group II (35 of 82 patients; p:NS).

Discriminant analysis. The results of the clinical and electrocardiographic independent variables in the two groups are listed in Table 1. Of the 24 independent variables, only 5 were included in the final model of discriminant analysis. The coefficients and associated F values are listed in Table 2. The performance of the model on the original data set from which it was derived is shown in Figure 2.

The results, based on five variables, are derived according to the following equation:

Equation 1: $Y = 0.95\ (\text{sex}) + 1.38\ (\text{ST-T changes}) + 0.17\ (SV_2) - 0.12\ (SV_3) - 0.07\ (RV_3 + RV_4) - 0.54.$

Table 2. Predictive Function of Five Variables Used in the Model

Variable	Coefficient	F Value
ST-T ($V_2 + V_3$)	1.38	19.8
Male sex	0.95	4.4
SV_2*	0.17	2.09
SV_3*	−0.12	3.83
$RV_3 + RV_4$*	−0.07	3.10

*The absolute amplitude of the remaining three variables was used. If the sex was male, a score of 1 was given; if female, a score of 0 was given. In the presence of ST-T changes in leads V_2 or V_3, a score of 1 was given; in their absence a score of 0 was given.

A score of 1 is given for male sex and 0 for the female sex. For ST-T wave changes, a score of 2 is given for ST depression or T wave inversion in both leads V_2 and V_3; a score of 1 for ST depression or T wave inversion in either V_2 or V_3; and a score of 0 for normal ST segment and T wave in V_2 and V_3. SV_2 is S wave amplitude (in mm) in lead V_2; SV_3 is S wave amplitude in lead V_3; and $RV_3 + RV_4$ is the sum of R wave amplitude in leads V_3 and V_4.

Using a binary cutoff at the discriminant score of zero, values above this level correctly classified 17 of 20 patients in Group I (anterior infarction by thallium scintigraphy) (sensitivity 85%) and 58 of the 82 patients in Group II (no anterior infarction) (specificity 71%) (Table 3).

Sixteen (16%) of the 102 patients had a history of previous documented myocardial infarction. There were seven patients (35%) in Group I and nine (11%) in Group II. When the discriminant model was reconstructed to include the presence or absence of history of previous infarction, seven variables were selected to predict the presence of infarction using the following equation:

Equation 2: $Y = 0.80\ (\text{sex}) + 0.36\ (S_1) + 0.18\ (SV_2) - 0.38\ (RV_3) - 0.14\ (SV_3) + 1.2\ (\text{ST-T changes}) + 1.2\ (\text{history of infarction}) - 0.8.$

A score of 1 is given for a history of previous myocardial infarction and a score of 0 for absence of previous infarction. Other scores are as in equation 1. Using this model, 16 of the 20 patients in Group I and 65 of the 82 patients in Group II were correctly identified (sensitivity 80% and specificity 79%) (Table 3).

Prospective study. The discriminant function was subsequently applied prospectively to an additional 21 patients with poor R wave progression (8 with and 13 without anterior infarction by thallium scintigraphy) and provided a sensitivity of 85%, a specificity of 88% and a predictive accuracy of 86%.

Q wave anterior infarction. All 21 patients with anterior infarction in association with abnormal precordial Q waves had abnormal thallium-201 scintigrams consistent with anterior scar. The mean abnormal perimeter was larger in patients with Q waves than in patients with anterior in-

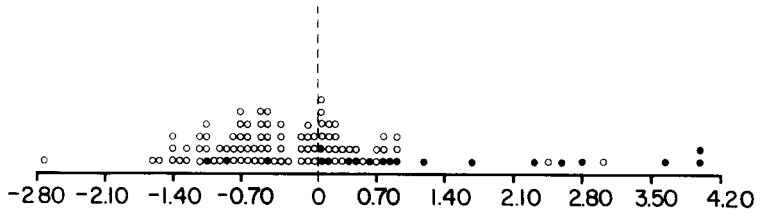


Figure 2. The result of discriminant analysis of five variable models to predict the presence or absence of anterior infarction in patients with poor R wave progression. The **closed circles** represent patients with infarction and the **open circles** represent patients without infarction. The **dotted line** represents the binary cutoff point.

farction and poor R wave progression (Group I) (42 ± 12 vs. 28 ± 13%, $p < 0.001$) (Fig. 3).

Discussion

Poor R wave progression. Our study was undertaken to determine whether patients with anterior myocardial infarction can be identified within a larger group of patients with poor R wave progression in the precordial leads. Loss of anterior forces of depolarization because of anterior infarction has long been established clinically and experimentally to produce poor R wave progression (5–8); however, poor R wave progression is also recognized to be an unreliable criterion in the diagnosis of anterior infarction because it may result from many other sources (1–4). Only recently (5–8) have there been attempts to differentiate patients with anterior infarction from other patients with poor R wave progression.

The definition of poor R wave progression in this study was identical to that used by Zema and Kligfield (5–8). They used an algorithm to identify patients with infarction; we used a multivariate stepwise discriminant analysis, a statistical technique that sequentially selects variables in the order in which they contribute information to differentiate patients with infarction from those without infarction. Abnormality of repolarization in leads V_2 and V_3 was found to be an independent variable that best separated the patients into infarction and noninfarction groups. This finding is in agreement with the results of Zema and Kligfield (5–8).

Other variables identifying anterior infarction. After accounting for the discriminant power provided by the variable, we examined the remaining variables to determine which provided additional independent discriminant power for separation of patients. This process continued sequentially until there were no additional variables that significantly differentiated between groups with infarction (fixed defects) and without infarction (no fixed defects). In this process four additional variables: sex, the amplitude of the S wave in leads V_3 and V_4 and the amplitude of the R wave in V_3 and V_4, entered into the analytic model. If the discriminant score was positive (> 0), 17 0f the 20 patients with anterior infarction were correctly identified (sensitivity 85%) and 58 of the 82 patients without anterior infarction were also correctly identified (specificity 71%) (Fig. 2). The importance of the ST-T wave abnormalities in the precordial leads as markers for anterior infarction has been noted by other investigators (5–8).

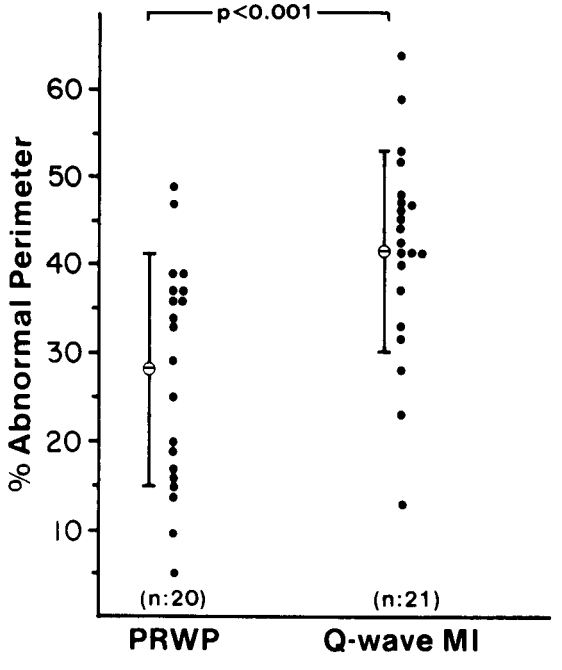
Similarly, the amplitude of the R wave in leads V_3 and V_4 and the S wave in V_2 and V_3 may be independent markers for anterior infarction. The importance of sex in the discriminant model emphasizes the importance of Bayes' theorem in interpreting test results according to the prevalence

Table 3. Results of Analysis in the Study Group

	Five Variable Model	Seven Variable Model*
Sensitivity (%)	85	80
Specificity (%)	71	79
Positive predictive value (%)	41	48
Negative predictive value (%)	95	94
Predictive accuracy (%)	74	79
Predictive error (%)	5	6
Relative risk	8.2	8

*The seven variable model was derived when prior history of myocardial infarction was included among the variables. The five variable model was derived when history of myocardial infarction was not included.

Figure 3. The abnormal perimeter is a measure of the size of infarction in patients with a Q wave infarction and in patients with poor R wave progression (PRWP). Values are mean ± standard deviation.



of the disease in the group being tested; coronary artery disease is more frequent in men than in women.

The additional information obtained from the presence or absence of prior myocardial infarction did not significantly improve the sensitivity or specificity (Table 3). This is probably because patients with a history of prior infarction may have sustained infarction in areas other than the anterior wall.

Previous studies on clinical diagnosis of previous anterior infarction. The results of several recent studies, which attempted to identify a subset of patients with anterior infarction within a group of patients with poor R wave progression, are listed in Table 4 (6–8,16). The results of these studies were obscure because of the small number of patients (range 16 to 56), the variable time between electrocardiographic results and documentation of anterior infarction (48 hours to 3 months) and the inclusion of patients with left ventricular hypertrophy or left axis deviation among the patients with poor R wave progression. The criteria for determining the presence of anterior infarction in patients with left ventricular hypertrophy, using multivariate analysis, remain to be determined.

The diagnosis of anterior infarction in patients with poor R wave progression is difficult. It is known that anterior wall asynergy, as determined by contrast ventriculography, may be due to ischemia rather than scar (17). The findings on vectorcardiography are certainly not specific for anterior infarction (18). Although autopsy results are considered the standard for the diagnosis of anterior infarction, the time lag between the electrocardiographic changes and death (up to 3 months) in these studies and the lack of quantitative assessment of the degree of fibrosis detract from the value of the results.

Our standard for anterior infarction was the presence of fixed thallium-201 perfusion defects. Fixed thallium-201 perfusion defects have been shown to be specific and sensitive markers of myocardial fibrosis due to coronary artery disease (9), although, on occasion, diseases other than coronary artery disease may produce perfusion defects (19).

Overall, approximately 20% of prior myocardial infarcts cannot be recognized on thallium-201 scans either at rest or during exercise (20). These are, in particular, small and nontransmural myocardial infarcts. It is entirely conceivable that among the patients with poor R wave progression and normal thallium scans, a previous infarction occurred that can only be detected by histologic examination. Therefore, our results may relate more precisely to the presence or absence of perfusion defects rather than to myocardial infarction.

Infarct size. Quantitation of infarct size revealed that the infarct in patients with poor R wave progression is smaller than in patients with anterior infarction and precordial Q waves (Fig. 3). This finding is consistent with autopsy and angiographic studies (16,21) that showed that in patients with poor R wave progression, the infarct is small and often nontransmural and that left ventricular function is less severely impaired than in patients with Q wave infarction.

Limitations of the study. Several points regarding our results should be considered: First, our subjects were consecutive patients undergoing stress testing for suspected coronary artery disease. The results may be different, therefore, in entirely asymptomatic patients, because a low incidence of coronary artery disease is expectable in this group. However, it is important to note that even in our selected group an anterior scar was present in only 20% of the patients.

Second, an anterior scar was defined by thallium imaging. The potential problems with this and other methods (contrast angiography, autopsy and vectorcardiography) have been discussed. All eight patients in Group I with a fixed defect who had catheterization studies had significant disease of the left anterior descending artery and seven of the eight had asynergy of the left ventricular anterior wall. The

Table 4. Summary of Reported Data Regarding Anterior Infarction in Patients With Poor R Wave Progression

First Author	No. of Patients	Definition of Anterior Scar	Method of Analysis	Results (%)				
				SN	SP	+PV	−PV	PA
Zema (6)	55*	Vectorcardiography	Algorithm	90	72	64	93	79
Zema (7)	40†	Contrast angiography	Algorithm	85	56	48	88	65
Zema (8)	33‡	Autopsy	Algorithm	85	75	69	88	79
Hart (16)	16§	Thallium scintigraphy	Lower interspace ECG recording	71	78	71	78	75

*Patients with left ventricular hypertrophy or left anterior hemiblock were included. †Patients with left ventricular hypertrophy or left anterior hemiblock were included; there was a time lag up to 1 month between electrocardiography and angiography ‡Patients with left ventricular hypertrophy, left anterior hemiblock and right ventricular hypertrophy were included; there was a time lag up to 3 months between electrocardiography and autopsy. §Only patients with chronic obstructive lung disease were examined.

ECG = electrocardiographic; PA = predictive accuracy; −PV = negative predictive value; +PV = positive predictive value; SN = sensitivity; SP = specificity.

last patient had a septal defect only, and the motion of the septum was not evaluated by contrast angiography. It should be noted that many of our patients had ischemic defects in the distribution of one or more vessels and fixed defects involving the inferior or posterior walls of the left ventricle, but these abnormalities were thought not to produce poor R wave progression and, therefore, were not analyzed in detail.

Third, the redistribution images were used to diagnose and quantitate fixed defects rather than rest studies. The pharmacokinetics of thallium-201 imaging indicate that this is an acceptable method because reperfusion is nearly complete 4 hours after injection of thallium-201 (22).

The use of the discriminant model when applied to an individual patient with poor R wave progression should be viewed as a relative risk factor with regard to the probability of anterior infarction. The relative risk of anterior infarction in patients with a discriminant score of zero or higher was eight times that of patients with poor R wave progression with a lower score.

Conclusions. In our study, fixed perfusion defects involving the anteroseptal segments were seen in 20% of patients with poor R wave progression. These patients can be identified by multivariate stepwise discriminant analysis with a sensitivity of 85%, specificity of 71% and an overall predictive accuracy of 74%. Prospective validation of the model provided a sensitivity of 85%, a specificity of 88% and predictive accuracy of 86%. If the results are confirmed, this model can be of considerable aid to physicians who are involved in interpreting routine electrocardiograms.

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References

1. Pruitt RD, Barnes AR, Essex HE. Electrocardiographic changes associated with lesions in deeper layers of mycardium: experimental study. Am J Med Sci 1945,210.100–13
2. Prinzmetal M Shaw CM Jr, Maxwell MH, et al Studies on mechanisms of ventricular activity. Depolarization complex in pure subendocardial infarction Am J Med 1954;16·469–89
3. Cook RW, Edward JE, Pruitt RO. Electrocardiographic changes in acute subendocardial infarction Circulation 1958;18·603–22.
4. Berger M, Schack JA Significance of poor R-wave progression Adv Cardiol 1977;19:218–9
5. Zema MJ, Kligfield P. Electrocardiographic poor R-wave progression. Arch Intern Med 1982;142:1145–8
6. Zema MJ, Kligfield P. Electrocardiographic poor R-wave progression. I Correlation with the Frank vectorcardiogram J Electrocardiol 1979;12:3–10.
7. Zema MJ, Kligfield P. Electrocardiographic poor R-wave progression. II. Correlation with angiography. J Electrocardiol 1979;12:11–5.
8. Zema MJ, Collins M, Alonso DR, Kligfield P. Electrocardiographic poor R-wave progression: correlation with postmortem findings. Chest 1981,79:195–200.
9. Wackers FJT, Sokole EB, Samson G, et al Value and limitations of thallium-201 scintigraphy in the acute phase of myocardial infarction. N Engl J Med 1976,295.1–5.
10. Wackers FJT, Becker AE, Samson G, et al. Location and size of acute transmural myocardial infarction estimate from thallium-201 scintiscans. a clinicopathological study. Circulation 1977;56:72–8.
11. Iskandrian AS, Segal BL Value of exercise thallium-201 imaging in patients with diagnostic and nondiagnostic exercise electrocardiograms Am J Cardiol 1981;48:233–8
12. Iskandrian AS, Lichtenberg R, Segal BL, et al. Assessment of jeopardized myocardium in patients with one-vessel disease. Circulation 1982,65.242–7.
13. Iskandrian AS, Hakki AH, Segal BL, Kane SA. Assessment of jeopardized myocardium in patients with multivessel disease. Am Heart J (in press).
14. Feild BJ, Russell RO Jr, Dowling JT, Rackley CE. Regional left ventricular performance in the year following myocardial infarction. Circulation 1972;46:679–89.
15. Dixon WJ, Brown MB, Engelman L, Frane JW, Jennrich RI (eds). Biomedical Computer Programs, P-series (BMDP-79) (Program was developed at the Health Science Computing Facility, UCLA, and sponsored by NIH Special Research Resources Grant RR-3) Berkeley, California: University of California Press, 1979:399–417.
16. Hart GJ, Barret PA, Barnaby PF, Clark EH, Lyons NR, Burke JJ. Diagnosis of old anterior myocardial infarction in emphysema with poor R-wave progression in anterior chest leads. Br Heart J 1981;45·522–6
17. Bodenheimer MM, Banka VS, Helfant RH Q waves and ventricular asynergy: predictive value and hemodynamic significance of anatomic localization Am J Cardiol 1975;35:615–8.
18. Murata K, Matsushita S, Kurihara H, Seki M. Some pitfalls of vectorcardiographic diagnosis of myocardial infarction with particular respect to emphysema Circulation 1967;35:172–7.
19. Dunn RF, Uren RF, Sadick N, et al Comparison of thallium-201 scanning in idiopathic dilated cardiomyopathy and severe coronary artery disease. Circulation 1982;66:804–11
20. Uthorralt N, Parodi O, Severi S, Davies G, Maseri A. Thallium-201 scintigraphy for diagnosis of old myocardial infarction: comparison with electrocardiographic, ventriculographic and coronary arteriographic findings Br Heart J 1980;43.527–34.
21. Savage RM, Wagner GS, Ideker RE, Pololsky SA, Hackel DB. Correlation of postmortem anatomic findings with electrocardiographic changes in patients with myocardial infarction. Circulation 1977;55·279–85.
22. Watson DD, Campbell NP, Read EK, Gibson RS, Teates CD, Beller GA Spatial and temporal quantitation of plane thallium myocardial images. J Nucl Med 1981;22:277–84