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New Electrocardiographic Criteria for Predicting Either the Right or Left Circumflex Artery as the Culprit Coronary Artery in Inferior Wall Acute Myocardial Infarction

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In the present study, we attempted to predict the culprit artery by assessing the relative ST-segment deviations in different leads during inferior wall acute myocardial infarction (AMI). To overcome the main limitation of most previous studies, we selected a relatively homogeneous group of patients with significant narrowing limited to either the right coronary artery (RCA) or the left circumflex coronary artery (LCX).

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The study population included all patients with a diagnosis of inferior wall AMI who were admitted to our coronary care unit between January 1993 and May 1996, and who subsequently underwent coronary angiography during hospitalization. Diagnosis of inferior wall AMI was based on chest pain lasting >30 minutes, evolving characteristic electrocardiogram abnormalities that included ST-segment elevations of >1 mm in ≥ 2 inferior leads (II, III aVF), and an increase in serum creatine kinase >2 times the upper normal limit. Patients with previous AMI, coronary artery bypass grafting, or electrocardiographic evidence of left ventricular hypertrophy or bundle branch block were excluded.

All standard admission 12-lead electrocardiograms were evaluated by 2 investigators blinded to the angiographic findings. ST-segment deviation from the isoelectric line was measured manually to the nearest 0.5 mm in every lead at 0.08 seconds after the J point. A ST-segment deviation of >1 mm was considered significant. The magnitude of ST-segment elevation in

TABLE I Correlation Between ST-Segment Deviation and the Infarcted Coronary Artery

Group	RCA (n = 66)	LCX (n = 17)	p Value
Lead I ST ≤ 1 mm	36 (54%)	5 (29%)	0.1
Lead aVL ST ≤ 1 mm	62 (94%)	5 (29%)	<0.001
Lead V ₅ -V ₆ ST ≥ 1 mm	16 (24%)	6 (35%)	0.37
Criteria A	53 (80%)	1 (6%)	<0.001
Criteria B	58 (88%)	1 (6%)	<0.001
Criteria A + B positive	46 (70%)	0	<0.001
Criteria A + B negative	0	15 (88%)	<0.001

Criteria A = higher ST-segment elevation in lead III than in lead II. Criteria B = greater ST-segment depression in lead aVL than in lead I.

leads II and III was compared, as was the ST-segment depression in leads aVL and I.

Coronary cineangiography films were reviewed by 2 investigators who were blinded to the electrocardiographic findings. The infarct-related artery was determined by the following criteria: coronary artery disease resulting in total or subtotal occlusion of 1 artery supplying the area of asynergy as seen on left ventriculography, arteriographic features suggestive of acute thrombus, or a ruptured plaque in 1 artery. Patients with significant stenosis in both the RCA and LCX artery were excluded.

Patients were divided into 2 groups according to the culprit artery. The chi-square test was used to compare electrocardiographic differences between groups. Where there were a few patients in a category, Fisher's exact test was performed. All tests of significance were 2-tailed and p values of <0.05 were considered statistically significant.

The study population consisted of 83 patients (68 men and 15 women), aged 57 ± 12 years (Table I).

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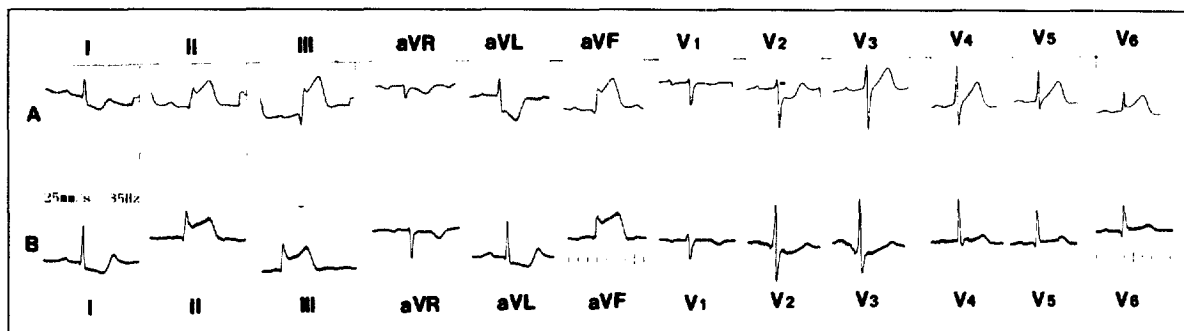


FIGURE 1. Electrocardiogram of patient with RCA-related infarction (A) and LCX artery-related infarction (B).

TABLE II Sensitivity, Specificity, Positive Predictive Value (PPV), and Negative Predictive Value (NPV) for the Different Criteria

	Sensitivity	Specificity	PPV	NPV
ST ↓ lead I	55%	71%	88%	29%
ST ↓ aVL	94%	71%	94%	75%
ST lead V ₅ -V ₆ ≥ 1 mm	35%	76%	27%	82%
Criteria A	88%	94%	98%	67%
Criteria B	80%	94%	98%	55%
Both A + B positive	70%	100%	100%	46%
Both A + B negative	0%	12%	0%	100%
Both A + B negative for LCX	88%	100%	100%	97%

Criteria A = higher ST-segment elevation in lead III than in lead II. Criteria B = greater ST-segment depression in lead aVL than in lead I.

Involvement of the left anterior descending artery was found in 20 patients in the RCA group and in 7 in the LCX group ($p = 0.12$). Table I lists the electrocardiographic findings in the 2 groups. Significant ST depression in lead aVL was more common in the RCA group ($p < 0.001$), with a sensitivity and specificity of 94% and 71%, respectively. Higher ST-segment elevation in lead III than in lead II (Figure 1A) was found in 80% of the RCA group and in 6% of the LCX group ($p < 0.001$); deeper ST-segment depression in lead aVL than in lead I (Figure 1B) was found in 88% in the RCA group and in 6% of the LCX group ($p < 0.001$). Both criteria were positive in 70% of the patients in the RCA group and in none of the LCX group ($p < 0.001$), whereas both were negative in 88% of the LCX patients and none of the RCA patients ($p < 0.001$). No significant differences were found for significant ST-segment elevation in leads V₅ to V₆ (35% of the LCX group and 24% of the RCA group, $p = 0.37$), or for frequency of ST-segment depression in the anterior precordial leads.

The sensitivity, specificity, positive predictive values, and negative predictive values of the various electrocardiographic criteria are shown in Table II. ST-segment depression in lead aVL had a high sensitivity for predicting RCA obstruction (94%), but a low specificity (71%). When the ST-segment deviation in leads I and aVL was taken into consideration (criteria B), the sensitivity decreased to

88%, but the specificity increased to 94%. The positive predictive values and the negative predictive values were 98% and 67%, respectively. Moreover, when both criteria were positive, there was no LCX obstruction; when both were negative, there was no RCA obstruction. These results did not change with the variation of the obstruction site along the coronary artery (proximal vs distal).

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We found that in patients with inferior wall AMI, a higher ST-segment elevation in lead III than in lead II and/or a greater ST-segment depression in lead aVL than in lead I is a sensitive and specific marker for RCA (and not LCX) obstruction. If both criteria are negative, the positive predictive values for LCX obstruction is 100%. We found these criteria to be more sensitive, more specific, and less affected by the site of the obstruction in the coronary artery than the traditional electrocardiographic criteria. The presence or absence of ST-segment elevation in precordial leads V₅ to V₆ or ST-segment depression in leads V₁ to V₄ was not of discriminatory value.

Occlusion of the LCX artery is difficult to diagnose using the standard 12-lead electrocardiogram.^{1,2} Because the posterolateral and inferoapical segments of the left ventricle are supplied by the LCX, their involvement in LCX occlusion is expected. Radionuclide studies show that thallium defects in the posterolateral segments are relatively specific for LCX AMI, whereas defects in the posterobasal segment are seen with equal frequency in RCA AMI.^{1,3} Indeed, the lateral limb leads are highly significant in inferior wall AMI.^{1,4} The aVL lead faces the high-lateral segment of the left ventricular artery, and is the only lead truly reciprocal to the inferior wall.⁵ Birnbaum et al⁴ recently reported that ST segment depression in lead aVL is a sensitive early electrocardiographic sign of inferior wall AMI and Berry et al² showed that ST-segment depression in leads I and aVL was observed only during RCA occlusion. Huey et al¹ reported that ST-segment depression in lead I is significantly less common with LCX-related AMI than with RCA-related AMI. These studies did not compare the relative magnitude of ST-segment depression in the 2 leads, as was performed in this study. In the case

of RCA infarction, the ST-segment depression in leads aVL and I represents reciprocal changes that are more prominent in the true reciprocal lead, aVL; in LCX AMI, the high posterolateral and apical segments are ischemic, therefore the ST-segment depressions in these leads are canceled out and an even ST-segment elevation can appear.

Standard lead III is oriented to the right inferior segment, whereas lead II is oriented principally to the left inferior segment and also tends to be oriented to the inferior region of the left lateral or superior wall of the left ventricle.⁵ Consequently, lead III is more influenced by RCA-related AMI, whereas lead II is more influenced by LCX-related AMI. By comparing the magnitude of deviations of the ST segment in different leads (aVL to I and II to III), we were able to overcome the limitation found by Hasdai et al⁶ who could not differentiate RCA occlusion from distal LCX occlusion.

In summary, this study indicates that it is possible to predict the culprit artery in inferior wall AMI using the readily obtainable measures on the admission electrocardiogram. A higher ST-segment elevation in lead III than in lead II and a

deeper ST-segment depression in lead aVL than in lead I are sensitive and specific markers for RCA-related AMI. As a result of the present study we are currently planning to examine these observations prospectively in patients with inferior wall AMI in order to test the predictive values in patients who may have multivessel disease.

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Treatment of Hyperlipidemia by Specialists Versus Generalists as Secondary Prevention of Coronary Artery Disease

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It is estimated that <40% of patients with coronary artery disease and hyperlipidemia requiring drug therapy actually receive it. It is not known whether the untreated individuals are followed primarily by general practitioners or cardiologists. This study attempted to determine which specialty more closely follows established guidelines for the pharmacologic treatment of hyperlipidemia for secondary prevention of coronary artery disease.

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The design of the study was a retrospective cohort. We identified all patients hospitalized at the Durham Veterans Administration (VA) Medical Center from January 1990 to January 1994 with myocardial infarction as defined by (1) creatine kinase-MB >9 mg/dl and (2) creatine kinase-MB index >3% of total creatine kinase. Patients were identified through the laboratory computer database. All VA clinic visits were identified for 2 years following hospital discharge. Only those patients

who had ≥ 2 visits over a 2-year period in either general medicine or cardiology clinics were included. The laboratory database was searched for cholesterol levels and complete lipid profiles. All prescriptions, including archived ones, were reviewed for a cholesterol-lowering agent.

Main outcomes were (1) proportion of patients in each clinic who were screened for elevated cholesterol and lipid profile in the clinic or during hospitalizations and (2) proportion of patients who received a cholesterol-lowering agent stratified by low-density lipoprotein (LDL) cholesterol level.

Demographic characteristics, co-morbidity as assessed by Charlson's index, and cholesterol/lipid profile measurements and treatment were compared between clinics. For continuous variables, differences were assessed with a 2-sample *t* test using a 2-tailed *p* <0.05 level. For categorical variables, differences were evaluated using chi-square tests; *p* values <0.05 were regarded as significant. Because of baseline differences in age, age-adjusted odds ratios were calculated for patients receiving pharmacologic treatment. Data were analyzed using SAS (SAS Institute, Cary, North Carolina).

There were 735 patients hospitalized at the Durham VA Medical Center from 1990 to 1994 who met the criteria for acute myocardial infarction. Of these, 140

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