

CORONARY HEART DISEASE

Electrocardiographic and Coronary Arteriographic Correlations During Acute Myocardial Infarction

HEINER BLANKE, MD, MARC COHEN, MD, GERHARD U. SCHLUETER, MD,
KARL R. KARSCH, MD, and K. PETER RENTROP, MD

One hundred fifty-two patients underwent cardiac catheterization and coronary arteriography within 6.3 ± 6.0 hours from the onset of acute myocardial infarction (AMI). All had standard 12-lead electrocardiograms recorded within 1 hour of cardiac catheterization. The electrocardiographic abnormalities present were correlated with the infarct-related artery as determined by coronary arteriography. ST-segment elevation was the most common finding in patients with the left anterior descending (LAD) or right coronary artery as the infarct-related artery. ST-segment depression was the most common abnormality in patients with the left circumflex (LC), artery as the infarct-related artery. A classic pattern of anteroapical AMI was seen in 93% of all patients with the LAD as the infarct-related artery. A classic pattern of inferior AMI was seen in 53% of patients with right or LC narrowing taken as 1 group. The pattern of true posterior and isolated lateral wall AMI in the absence of classic changes

in the inferior leads was highly specific and predictive of LC narrowing. In contrast, the pattern of an inferior wall AMI, in the absence of true posterior or lateral wall changes, was highly specific and predictive of right coronary artery narrowing. Fifty-six percent of patients with LC artery as the infarct-related artery presented with non-classic electrocardiographic abnormalities. The electrocardiographic patterns in patients with subtotal occlusions were similar to those of patients with total occlusions. Thus, the electrocardiogram obtained in the first few hours of AMI is reliable in localizing the LAD as the infarct-related artery. Certain patterns are specific but not sensitive in localizing the right coronary artery as opposed to the LC artery as the infarct-related artery. Presentation with signs and symptoms of AMI and a nonclassic electrocardiogram is suggestive of LC narrowing.

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Previous studies have established the usefulness of the standard 12-lead electrocardiogram in localizing the

From the Division of Cardiology, Department of Medicine, Mount Sinai School of Medicine, and Mount Sinai Hospital, New York, New York, and the Department of Medicine, University of Goettingen, Goettingen, West Germany. This study was supported in part by the SFB 89, Goettingen, West Germany, of the Deutsche Forschungsgemeinschaft. Manuscript received February 10, 1984; revised manuscript received April 18, 1984, accepted April 23, 1984.

Address for reprints: Marc Cohen, MD, Division of Cardiology, Mount Sinai Medical Center, 1 Gustave L. Levy Place, New York, New York 10029.

anatomic location of an acute myocardial infarction (AMI).¹⁻¹¹ These studies were based on postmortem findings, correlating the pathologic site of AMI with the electrocardiogram. Other investigators, using left ventriculography, correlated ventricular wall motion abnormalities with the electrocardiogram.^{12,13} More recent investigations have attempted to correlate abnormalities seen on serial electrocardiograms with the site of coronary artery stenosis in patient status after AMI.¹²⁻¹⁶ The coronary arteriograms in these later studies were all obtained at least several weeks after

AMI. This study of 152 patients correlates the electrocardiographic abnormalities seen during the first hours of AMI with the coronary arteriographic data obtained within several hours after hospital admission. The usefulness of the "admission" electrocardiogram in predicting the AMI-related artery is assessed.

Methods

Patients: Since January 1977, patients who presented to the emergency room with complaints of acute chest pain suggestive of myocardial ischemia were screened for possible immediate cardiac catheterization and coronary arteriography. Inpatients seen in emergency cardiac consultation for acute chest pain were screened in a similar fashion. Patients with valvular or congenital heart disease, incurable chronic diseases or contraindication to possible fibrinolytic therapy were excluded from immediate catheterization. No patients were excluded because of hypotension or cardiogenic shock. Patients with more than 30 minutes of persistent ischemic chest pain suggestive of AMI underwent immediate cardiac catheterization. Six patients who underwent immediate catheterization were excluded from further study. Five patients had unstable angina and 1 had normal coronary arteries. In 152 patients, the diagnosis of AMI was established retrospectively based on creatine kinase levels and creatine kinase-MB elevation to at least twice the upper limit of normal. The characteristics of these 152 patients who form the basis of this study are: The mean age of the study group was 57 ± 10 years. The male/female distribution was 132/20. Eleven patients with an electrocardiographic pattern of a previous transmural AMI, based on documentation in an old hospital chart, were entered into this study because the more recent electrocardiographic changes were in a different distribution than the earlier infarction. The mean time from onset of symptoms to angiography was 6.3 ± 6.0 hours. The infarct-related artery was the left anterior descending artery (LAD) in 82 patients, the right coronary artery in 39 and the left circumflex artery (LC) in 25. One, 2- and 3-vessel coronary disease was present in 64, 43 and 45 patients, respectively. Left main disease occurred in 8 patients. The initial creatine kinase at the time of admission was 169 ± 226 mU/ml.

Electrocardiogram: All patients had a standard 12-lead electrocardiogram recorded within 1 hour of cardiac catheterization. If more than 1 electrocardiogram was recorded, the 1 taken just before angiography was analyzed. All electrocardiograms were recorded on either 3- or 6-channel recorders using a paper speed of 50 mm/s and including 1 mV/cm standardization. Q waves of 0.04 second duration or 0.03 second in duration in association with a Q/R ratio $\geq 1:3$ were considered abnormal if they occurred in leads I, II, III, aVL, aVF and V_5 - V_6 . In leads V_1 - V_4 , any Q waves were considered abnormal unless they occurred in the setting of counter-clockwise rotation or in the presence of left-axis deviation greater than 30° .¹⁷ In lead V_1 , R > S, and R > 5 mm was considered pathologic. Poor R-wave progression was defined as an R-wave of 1.5 mm or less in lead V_2 or 3 mm or less in lead V_3 .¹⁸ ST-segment elevation to 1 mm or more above the baseline (as defined by the preceding TP segment) and occurring 0.02 second after the end of the QRS complex in any lead was considered abnormal. Analysis of Q waves and ST-segment elevation was done in all patients with right bundle branch block, left anterior hemiblock and intraventricular conduction abnormality less than 0.11 second in duration. In patients with right bundle branch block, analysis of ST-segment elevation was not done in leads V_1 - V_3 . Q-wave, ST-segment and T-wave analysis were not performed in the 4 patients who presented with left bundle branch block. ST-segment depression of 1

mm or more and occurring 0.08 second after the J point in any lead was considered significant. T-wave inversion occurring in leads I, II, aVL, aVF and V_2 - V_6 was considered to be abnormal. T-wave inversion in lead III was considered abnormal only if R was greater than S. Left ventricular hypertrophy was considered present if the sum of $SV_1 + RV_5$ was 3.5 cm or greater. ST-segment depression and T-wave analysis was not performed in patients taking digoxin (3 patients) or in patients with left ventricular hypertrophy. Other electrocardiographic abnormalities were diagnosed using standard criteria.

The electrocardiographic diagnosis of anterior AMI was made if abnormal Q waves or abnormal ST-segment elevation (as previously defined) occurred in at least 2 of the following leads: I and V_1 - V_4 . No attempt was made to distinguish anterior from anteroapical or from anterolateral infarction. Acute inferior myocardial infarction was diagnosed if abnormal Q waves or ST-segment elevation occurred in at least 2 of the following leads: II, III or aVF. Isolated acute lateral wall myocardial infarction was considered present when abnormal Q waves or ST-segment elevations occurred in at least 2 of the following leads: aVL or V_4 - V_6 . Lateral wall infarction in the setting of an extensive anterolateral wall infarction was excluded from the analysis of patients with isolated lateral infarction. True posterior wall infarction was diagnosed if, in lead V_1 , R was greater than S and R was greater than 5 mm in the presence of an upright T wave. Patients with any of these electrocardiographic changes were considered to have *classic* electrocardiographic abnormalities.

A number of patients presented with ST-segment depression, T-wave inversions, Q waves or ST-segment elevation that did not meet the aforementioned criteria. These patients were considered to have *non-classic* electrocardiographic abnormalities.

Cardiac catheterization: The technique of angiography during the AMI has been described in a previous publication.¹⁹ The degree of luminal narrowing was assessed by the method of Gensini.²⁰ Wall motion abnormalities were described according to the method of Herman et al²¹ with use of biplane left ventriculography.

Determination of the infarct-related artery: The infarct-related artery was determined by the following angiographic criteria: Coronary artery disease resulting in 90% or greater stenosis in only 1 artery occurred in 115 patients. In 37 patients there was more than 90% stenosis in 2 or 3 arteries. The following criteria were applied to these 37 patients: (1) In 10 of these patients, arteriographic features suggested acute thrombus in 1 of the arteries. The arteriographic findings of persistent staining of intraluminal material with contrast material or intraluminal filling defects completely surrounded by contrast were considered suggestive of thrombus.^{22,23} (2) In 10 patients, the wall motion abnormalities seen during left ventriculography combined with determination of the dominance of the coronary circulation allowed determination of the infarct artery. (3) In 11 patients, a previous infarction was documented by an old electrocardiogram that demonstrated a Q-wave infarction pattern. In 6 of these patients the electrocardiogram demonstrated an old inferior wall infarction. Left ventriculography, however, revealed wall motion abnormalities in the anterior as well as the inferior segments, suggesting that the acute infarct-related artery was the LAD. In 5 of the 11 patients, the old electrocardiogram indicated a previous anterior infarction. Left ventriculography, however, revealed wall motion abnormalities in the inferior as well as in the anterior segments. The acute infarct-related artery was presumed to be the right or LC artery, depending on the severity of lesions seen in these 2 arteries. (4) In 6 patients, the infarct-related artery could not be determined. Five of the 6 had more than 90% stenosis in 3 coronary arteries, and 1 of the

TABLE I Correlation Between Infarct Artery and Electrocardiographic Changes in Individual Leads*

Infarct Vessel	1	aVL	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆	II	III	aVF	V _{1R}
Q Waves												
LAD (n = 79) [†]	0	5	33	39	31	7	3	0	3 (7)	10 (8)	7 (9)	0
Total occl (n = 57)	0	4	26	29	23	5	3	0	1 (7)	7 (8)	5 (9)	
Subtotal (n = 22)	0	1	7	10	8	2	0	0	2	3	2	
Right (n = 39) [‡]	0	0	3	2	4	2	2	2	5 (10)	17 (13)	8 (15)	6
Total occl (n = 29)	0	0	3	2	4	2	2	2	4 (8)	14 (9)	6 (11)	5
Subtotal (n = 10)	0	0	0	0	0	0	0	0	1 (2)	3 (4)	2 (4)	1
LC (n = 25) [§]	1	1	0	1	1	0	1	1	1 (1)	6 (2)	2 (4)	6
Total occl (n = 18)	0	0	0	1	1	0	1	1	1 (1)	4 (2)	2 (3)	6
Subtotal (n = 7)	1	1	0	0	0	0	0	0	0	2	0 (1)	0
ST Elevation												
LAD (n = 79)	17 (5)	25 (7)	53 (5)	68 (2)	60 (7)	44 (4)	24 (6)	9 (5)	3 (2)	6 (1)	2 (3)	
Total occl (n = 57)	16 (4)	21 (6)	30 (3)	50 (2)	43 (6)	33 (2)	21 (2)	8 (2)	3 (1)	4	2 (1)	
Subtotal (n = 22)	1 (1)	4 (1)	13 (2)	18	17 (1)	11 (2)	3 (4)	1 (3)	0 (1)	2 (1)	0 (2)	
Right (n = 39)	0	1 (1)	2	6	6 (1)	3 (1)	1 (2)	3 (3)	21 (2)	23 (3)	22 (2)	
Total occl (n = 29)	0	1 (1)	1	3	4	3 (1)	1 (2)	3 (2)	18 (1)	17 (3)	16 (2)	
Subtotal (n = 10)	0	0	0	0	2 (1)	0	0 (1)	0 (1)	3 (1)	6	6	
LC (n = 25)	1 (2)	2 (1)	0	0	1	2 (2)	8 (1)	9 (1)	7 (1)	7 (1)	5 (3)	
Total occl (n = 18)	1	1 (1)	0	0	1	2 (1)	6 (1)	7 (1)	5 (1)	5 (1)	3 (1)	
Subtotal (n = 7)	0 (2)	1	0	0	0	0 (1)	2	2	2	2	2 (2)	
ST Depression												
LAD (n = 78)	8	5	1	3	7	8	16	11	22	24	21	
Total occl (n = 56)	4	2	1	3	5	5	11	5	14	18	16	
Subtotal (n = 22)	4	3	0	0	2	3	5	6	8	6	5	
Right (n = 36)	16	20	7	12	14	15	15	7	2	1	1	
Total occl (n = 27)	12	14	7	11	13	13	13	4	0	1	1	
Subtotal (n = 9)	4	6	0	1	1	2	2	3	2	0	0	
LC (n = 25)	4	4	7	12	11	11	8	5	4	2	1	
Total occl (n = 18)	3	4	6	9	8	7	5	2	4	2	1	
Subtotal (n = 7)	1	0	1	3	3	4	3	3	2	1	0	
T-Wave Abnormalities												
LAD (n = 78)	12	17	—	18	17	21	19	10	4	16	10	
Total occl (n = 56)	9	14	—	14	13	14	14	7	2	13	7	
Subtotal (n = 22)	3	3	—	4	4	7	5	3	2	3	3	
Right (n = 36)	8	15	—	3	2	4	8	10	10	12	11	
Total occl (n = 27)	6	12	—	2	2	4	7	7	7	9	7	
Subtotal (n = 9)	2	3	—	1	0	0	1	3	3	3	4	
LC (n = 25)	3	6	—	4	4	5	4	4	4	5	5	
Total (n = 18)	3	5	—	2	2	4	4	4	2	3	4	
Subtotal (n = 7)	0	1	—	2	2	1	0	0	1	2	1	

* Numbers in parentheses refer to additional patients with Q waves or ST elevation that do not meet criteria.

[†] Four patients with acute total occlusion of the left anterior descending coronary artery had a previous inferior wall infarction. Two patients with subtotal occlusion of the left anterior descending artery had a previous inferior infarction.[‡] Four patients with an acute total occlusion of the right coronary artery had an old anterior infarction.[§] One patient with an acute total circumflex occlusion had an old anterior infarction.

LAD = left anterior descending artery; LC = left circumflex artery; occl = occlusion.

TABLE II Electrocardiographic Patterns of Myocardial Infarction in Relation to Infarct Artery*

LAD (n = 82)	Total Occl (n = 59)	Subtotal Occl (n = 23)
Isolated anterior MI	53 (90%)	19 (83%)
Anterior + inferior	2	0
Left bundle branch block	2	1
Nonclassic	2	3
Normal	0	0
Right (n = 39)	Total Occl (n = 29)	Subtotal Occl (n = 10)
Isolated inferior	16 (55%)	6 (60%)
Inferoposterior	1	0
Inferolateral	1	0
Inferoposterolateral	1	0
Posterior isolated	2	0
Inferior + anterior	1	1
Lateral isolated	0	0
Nonclassic	6 (21%)	3 (30%)
Anterior	1	0
Normal	0	0
LC (n = 25)	Total Occl (n = 18)	Subtotal Occl (n = 7)
Inferior isolated	2	1
Inferoposterolateral	2	0
Inferolateral	1	1
Posterior isolated	2	0
Postero lateral	2	0
Nonclassic	8 (44%)	4 (57%)
Normal	1	1

* Electrocardiographic patterns present from previous infarction not included in this analysis.

Abbreviations as in Table I.

6 had 2 total occlusions that were both demonstrated to be acute occlusions at autopsy. Wall motion abnormalities were present in the areas subtended by the infarct-related artery in all 146 patients in whom the infarct artery was determined.

Statistics: Continuous variables are presented in the tables as the mean value \pm standard deviation. Sensitivity, specificity and predictive value are calculated in the standard fashion.

Results

Electrocardiographic changes in individual leads (Table I): The incidence in the different leads of abnormal Q waves, ST-segment elevation, ST-segment depression and T-wave inversions are listed in relation to the infarct-related artery in Table I. ST-segment elevation was the most common finding for all 3 infarct-related arteries. In patients in whom the LAD was the infarct-related artery, ST-segment elevation occurred most commonly in lead V₂ (83%). Patients with the right coronary artery as the infarct-related artery had ST-segment elevation most commonly in lead III (59%). Patients with LC disease had ST-segment elevation most often in lead V₆ (36%). Regardless of the infarct-related artery, ST-segment elevation of less than 1 mm was seen infrequently. In patients in whom the LAD was the infarct-related artery, abnormal Q waves occurred most often in lead V₂ (48%). Patients with the right coronary artery as the infarct-related artery had abnormal Q waves most often in lead III (44%). Q waves that did not meet the criteria previously described occurred in 38% of patients in lead aVF. Patients with LC

disease had Q waves most often in lead III (24%). Q waves that did not meet the criteria specified in the methods section occurred in 16% of these patients.

ST-segment depression occurred most often in lead III in patients with LAD disease (29%). ST-segment depression occurred most often in lead V₄ in patients with right coronary artery disease (39%). Forty-eight percent of patients with LC disease had ST-segment depression in lead V₂. T-wave abnormalities occurred infrequently with respect to all 3 infarct-related arteries. The incidence of ST-segment elevation or Q waves in patients with subtotal occlusions was comparable to that of patients with total occlusions of the infarct-related artery.

Electrocardiographic patterns of myocardial infarction in relation to the infarct vessel (Table II): Eighty-one percent of the entire group presented with classic electrocardiographic changes in AMI. Nineteen percent of patients presented with nondiagnostic electrocardiograms, that is, nonpathologic Q waves, or nonspecific ST-T wave changes. Two patients presented with a normal electrocardiogram. Ninety-three percent of patients in whom the infarct artery was the LAD presented with classic patterns of anterior myocardial infarction. Only 5 of the 82 patients with the LAD as the infarct artery presented with a nonclassic pattern: 2 patients with total occlusion and 3 patients with subtotal occlusion.

Patients with right coronary artery and LC as the infarct artery presented with variable classic patterns; isolated inferior, inferoposterior, inferolateral, inferoposterolateral, isolated true posterior, posterolateral and isolated lateral infarctions. Twenty-three percent of patients with the right coronary artery as the infarct artery and 48% of patients with the LC as the infarct artery presented with nonclassic electrocardiographic abnormalities.

The electrocardiographic patterns of infarction with respect to total vs subtotal occlusion of the infarct artery is presented in Table II. Patients with subtotal occlusion of the LAD or right coronary artery presented with classic electrocardiographic patterns of AMI with comparable frequency as those with total occlusion. Fifty percent of patients with total occlusion of the LC presented with classic patterns of AMI, compared with 29% (2 of 7 patients) with subtotal occlusion. Of the 2 patients with LC narrowing who presented with AMI and a normal electrocardiogram, 1 had a total occlusion and 1 a subtotal occlusion.

Non-classic electrocardiographic patterns (Table III): The specific electrocardiographic abnormalities in patients who did not present with classic electrocardiographic patterns are listed in Table III. Two patients in whom the LAD was the infarct artery presented with detectable ST-segment elevation which was, however, less than 1 mm high. ST-segment depression in association with T-wave inversions was the most frequent finding in patients who had the right coronary artery as the infarct artery, but who did not have a classic electrocardiographic pattern of infarction. Although several of these patients had Q waves in the inferior leads, these Q waves did not fulfill criteria. The most common ob-

TABLE III Correlation Between Infarct Artery and Electrocardiographic Abnormalities in Patients with Non-Classic Electrocardiographic Changes

Infarct Artery	Pt. ID No.	Q Waves*	ST Elevation*	ST Depression	T-Wave Abnormality	Poor R-Wave Progression	R-Wave Decay	Normal
LAD total	014		V ₂ , V ₃	V ₄ -V ₆				
occl	002			V ₂ -V ₆				
LAD subtotal	36			aVL, V ₂ -V ₅	V ₂ -V ₃			
	52		1, aVL, V ₄ -V ₆		V ₂ -V ₄			
	139				V ₂ -V ₄			
Right total	18	2, F		2, 3, F				
occl	69	2, F		2, 3, F, V ₅ , V ₆ , aVL	3, F, V ₅ , V ₆			
	70	2, 3, F		2, 3, F, V ₅ , V ₆	2, 3, F, V ₅ , V ₆			
	99			V ₄ , V ₅ , V ₆	2, F, V ₅ , V ₆ , L			
	134				2, 3, F			
Right subtotal	152		V ₂ -V ₄		3, F			
	29	2, 3, F				F		
	94					2, 3, F		
LC total	90		V ₄ -V ₆					
occl	21		2, 3, V ₅ , V ₆		V ₄ -V ₆			
	34						V ₄ -V ₆	
	56			2, V ₁ -V ₆				
	61			3, F, V ₂ -V ₆				
	87			1, 2, V ₂ -V ₆	V ₄ -V ₆			
	92							
LC subtotal	114	V ₄ -V ₆			V ₄ -V ₆			In all leads
	005			aVL, V ₄ -V ₆				
	008			V ₄ -V ₆				
	022				Hyperacute V ₄ -V ₆			
	116							In all leads

* Q waves and ST elevation in these patients do not meet criteria. Abbreviations as in Table I.

servations in patients with LC as the infarct artery who did not manifest classic infarction patterns on the electrocardiogram were ST-segment depression in the lateral precordial leads and T-wave abnormalities.

Sensitivity, specificity and predictive value of the electrocardiographic pattern of myocardial infarction (Tables IV and V): The correlation between the electrocardiographic pattern of anterior, inferior, isolated lateral and true posterior AMI, and the infarct-related artery is shown in Tables IV and V. The sensitivity, specificity and predictive value of the diagnosis of anterior AMI in predicting the LAD as being the infarct-related artery was 90, 95 and 96%, respectively. In 3 patients who presented with a pattern of anterior AMI, the LAD was not the infarct artery. These patients had acute occlusions of the right coronary artery, but presented with ST-segment elevation in the anterior precordial leads in addition to having classic electrocardiographic changes in the inferior leads. All 3 patients had 80 to 90% diameter reduction in the proximal LAD and severe right coronary narrowing.

The sensitivity, specificity and predictive value of the pattern of inferior wall AMI predicting either the right coronary artery or the LC artery as the infarct artery was 53, 98 and 94%, respectively. Two patients in whom the infarct artery was the LAD presented with classic electrocardiographic changes in the inferior leads in addition to changes in the anterior leads. Both of these patients had a large LAD that extended to and around the ventricular apex. The pattern of a true posterior or isolated lateral AMI was highly specific and predictive of the right coronary artery or LC as the infarct-related

artery. No patient in whom the LAD was the infarct vessel presented with changes in the posterior or lateral distribution. The pattern of true posterior and lateral wall AMI in the absence of classic changes in the inferior leads was highly specific and predictive of the LC as opposed to the right coronary artery as the infarct artery (Table V). In contrast, the pattern of an inferior wall AMI by electrocardiogram in the absence of posterior or lateral changes was highly specific and predictive of right coronary artery narrowing.

Discussion

This study attempts to relate the electrocardiographic location of Q waves, ST-segment elevation, ST-segment depression and T-wave changes, to the infarct-related artery during AMI. Several previous studies have correlated serial electrocardiographic changes with the site of significant coronary artery obstructions. All of these angiographic studies, however, were performed after healing of AMI. The recent study of Fuchs et al,¹⁶ for example, related the electrocardiographic location of Q waves, ST-segment elevation and depression, and T-wave changes seen in serial electrocardiograms during the hospital phase of AMI to the site of coronary artery disease seen during coronary arteriography in the chronic phase.

In this study, more than 80% of our patients presented with classic electrocardiographic patterns of AMI. ST-segment elevation of 1 mm or greater was the most sensitive marker. The addition of patients with less than 1 mm of elevation would not have significantly improved the sensitivity of this marker. Abnormal Q waves occurred less often than ST-segment elevation,

TABLE IV Correlation Between Infarct Artery and Electrocardiographic Pattern of Myocardial Infarction*

Infarct Artery		Anterior Infarction	
LAD (n = 82)	No	No	Yes
	Yes	61 8	3 74
		Sensitivity 90 %; specificity 95 %; predictive value 96 %	
		Inferior Infarction†	
Right/LC (n = 64)	No	No	Yes
	Yes	80 30	2 34
		Sensitivity 53 %; specificity 98 %; predictive value 94 %	
		Posterior or Lateral Myocardial Infarction‡	
Right/LC (n = 64)	No	No	Yes
	Yes	82 51	0 13
		Sensitivity 20 %; Specificity 100 %; predictive value 100 %	

* Electrocardiographic patterns present from previous infarction not included in this analysis.

† Inferior infarction, with or without additional posterior or lateral changes.

‡ Includes patients who may or may not have additional inferior wall changes.

Abbreviations as in Table I.

TABLE V Correlation Between Infarct Artery and Electrocardiographic Pattern of Myocardial Infarction: Right Coronary Versus Left Circumflex Artery

Infarct Artery		Posterior or Lateral Infarction Without Inferior ECG Changes	
LC (n = 25)	No	119	2
	Yes	19	6
		Sensitivity 24 %; specificity 98 %; predictive value 75 %	
		Inferior AMI Without Posterior or Lateral Changes	
Right (n = 39)	No	104	3
	Yes	17	22
		Sensitivity 56 %; specificity 97 %; predictive value 88 %	

AMI = acute myocardial infarction; LC = left circumflex artery.

probably because of the relatively early timing of the electrocardiograms. The addition of patients with nonpathologic Q waves, which occurred in a significant number of patients with right coronary or LC narrowing would not have improved the sensitivity of the electrocardiogram as a marker, because in most patients abnormal ST-segment elevation was already present. Almost one-fifth of our study group presented with nonclassic electrocardiograms. ST depression and T-wave abnormalities occurred as the solitary markers for AMI in many patients in whom the right coronary artery or LC was the infarct-related artery.

In patients with classic ST-segment elevation or Q waves, "reciprocal," ST depression, i.e., changes in leads at a distance from the infarct zone, is common regardless of the infarct-related artery.

The initial electrocardiogram obtained during AMI is reliable in localizing the LAD as the infarct artery. In addition, classic electrocardiographic patterns are seen in most patients with disease of the right or LC when these 2 subsets are evaluated as 1 group. Our findings suggest that the ability to distinguish the right coronary as opposed to the LC artery as the infarct-related artery, is significantly enhanced if patterns of isolated inferior or isolated true posterior or lateral infarction are seen. These markers, however, are not very sensitive.

As described by Sullivan⁹ and Ward¹¹ and their co-workers, infarction of the lateral wall of the heart caused by disease in the LC rarely presents with classic electrocardiographic changes. Our findings are in keeping with these investigations. More than 50% of our patients in whom the LC was the infarct-related artery did not present with an electrocardiographic pattern diagnostic of transmural AMI. In fact, 2 of these patients presented with normal electrocardiograms, suggesting that infarctions secondary to LC disease may be electrocardiographically silent. By relying only on classic ST-segment elevation or Q waves as a marker of acute infarction, almost half of the patients with LC disease and almost one-fourth of the patients with right coronary artery disease would not be diagnosed as having AMI and possibly be excluded from clinical trials. Recently published data from the Multicenter Investigation on the Limitation of Infarct Size indicate that 21% of the patients with documented AMI did not have abnormal Q waves or ST-segment elevation.²⁴

More than one-fourth of our patients presented with subtotal occlusion of the infarct-related artery. The incidence of classic electrocardiographic changes were comparable whether a patient had a total vs a subtotal occlusion. This applied for all 3 infarct-related arteries.

Limitations of the study: Our study patients were selected from a larger group of patients with AMI, and therefore this study may be subject to some selection bias. No log of all patients screened but not eventually catheterized was kept. In addition, patients with unstable angina but with transient electrocardiographic patterns suggesting acute ischemia were not included in this study. This study did not restrict itself to patients with 1-artery disease. While most patients had 1 clearly identifiable infarct artery, several patients had severe disease in 2 or more arteries. Using generally accepted arteriographic, ventriculographic and previous electrocardiographic criteria, we made a determination of the infarct artery in most patients with multivessel disease. Lastly, the mean time between onset of symptoms to initial electrocardiogram was 6 hours. A shorter or longer interval may have affected our observations regarding electrocardiographic changes.

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