Unstable angina: ST segment depression with positive versus negative T wave deflections—Clinical course, ECG evolution, and angiographic correlation

Thirty-two consecutive patients who initially had horizontal or downward-sloping ST segment depression confined to the precordial leads were studied. Patients were divided into two groups: group A included 21 patients with horizontal or downward-sloping ST depression with peaked positive T waves, and group B comprised 11 patients with peaked negative T waves and downward or horizontal ST depression. The incidence of acute myocardial infarction (AMI) was similar (group A 38.1% vs group B 36.4%; $p > 0.05$). In-hospital mortality was much more significant in group B ($p = 0.03$). Coronary arteriography was performed in 31 patients. Of the 10 patients in group B who were catheterized, seven (70%) had left main occlusion. Of the 21 patients in group A, none had a significant left main lesion ($p = 0.001$), although eight (38.1%) had single-vessel disease ($p = 0.05$). Thus the ECG pattern of horizontal or downward-sloping ST depression passing into a peaked negative T wave identifies a subgroup of high-risk patients in whom the prognosis is poor once AMI occurs. Early catheterization is recommended when this ischemic pattern is apparent on the ECG. (Am Heart J 1988;116:933.)

Samuel Sclarovsky, MD, Eldad Rechavia, MD, Boris Strasberg, MD, Alex Sagie, MD, Roni Bassevich, MD, Jairo Kusniec, MD, Aviv Mager, MD, and Jacob Agmon, MD.
Petah Tikvah and Tel Aviv, Israel

The ECG finding of ST segment deviation is accepted as the most reliable marker of myocardial ischemia. In most instances ST segment elevation and tall upright T waves reflect transient total occlusion of a major epicardial coronary artery.[1,2] Downward displacement of the ST segment may represent either a reduction of flow or an increase in myocardial oxygen demand. Presumably these patients have a reduction in coronary flow as a result of diffuse narrowing of a major coronary artery or subtotal spastic occlusion superimposed on a preexisting atherosclerotic lesion.[3-5] The recording of ST segment depression coupled with coronary spasm is much more frequent in patients with severe coronary artery disease and a good collateral circulation.[6,7] Less certain, however, is the significance of ST segment depression associated with an increased oxygen demand or coronary spasm in patients with angiographically normal coronary anatomy.[8-10] It is also important to differentiate between subendocardial ischemia and a "benign" electrical phenomenon of a "concealed" transmural ischemia affecting the inferior reciprocal territory.[11]

The broad spectrum of clinical and angiographic observations related to ischemic ST depression may be explained by the different patterns of ST segment displacement and the wide variations in heart rate, systolic pressure, and contractility among patients included in this category in previous studies. There is clearly a need to determine subgroups of patients having anatomically or functionally severe coronary obstruction based on standard ECG interpretation.

In the suitable clinical context, the appearance of tall upright T waves represents a transmural ischemia.[12,13] Unlike the presence of positive T waves, there are essentially no natural history data on the evolution and characterization of early negative T wave deflections associated with ST segment depression recorded during an active attack of ischemia. In the present study patients with horizontal or

From the Israel and Ione Massada Center for Heart Diseases, Beilinson Medical Center, and the Tel Aviv University Sackler School of Medicine.

Received for publication Jan. 20, 1988; revision accepted June 10, 1988.

Reprint requests: S. Sclarovsky, MD, Massada Center for Heart Diseases, Beilinson Medical Center, Petah Tikva 49100, Israel.

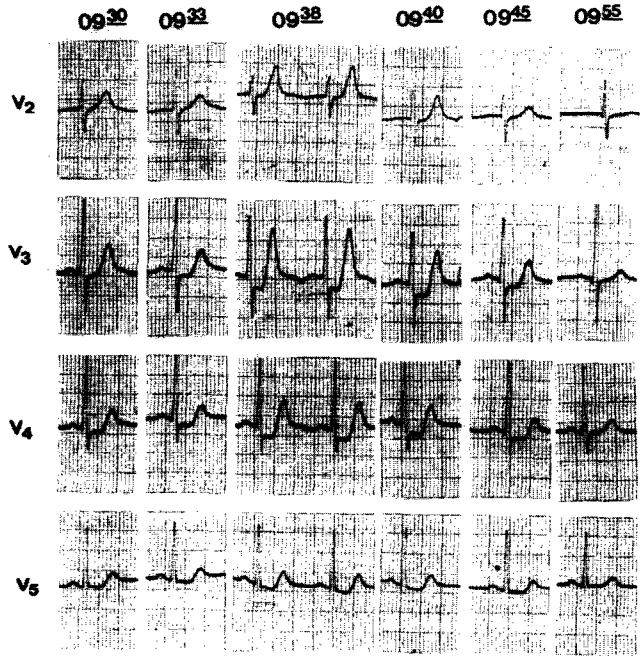

Fig. 1. Representative ECG of evolving anterior wall ischemia (group A). Maximal ischemic changes of horizontal ST segment depression and peaked positive T waves are seen in V_3.

downward-sloping ST segment depression during episodes of chest pain were divided into two groups based on the presence of peaked positive or negative T wave deflections. The purpose of this study was to describe the clinical course, sequential evolutionary ECG changes, and angiographic findings in this group of patients.

METHODS

Between December, 1984, and June, 1987, a total of 35 consecutive patients without previous myocardial infarction or coronary artery bypass surgery were admitted to the coronary care unit with acute myocardial ischemia consisting of chest pain and downsloping or horizontal ST segment depression (>2 mm) confined to the precordial leads. Excluded were patients with ST segment shifts in leads remote from the ischemic anterior wall and those with ECG evidence of tachycardia (>90 beats/min), previous myocardial infarction, or intraventricular block. Treatment was initiated with intravenous isosorbide dinitrate and heparin except in those for whom anticoagulants were contraindicated. None of the patients included in this study received thrombolytic therapy, since the presence of ST segment depression is generally accepted as an exclusion criterion for thrombolysis.[14] Lidocaine was given intravenously as indicated clinically. Patients were followed-up with serial ECGs and determinations of cardiac serum enzymes for at least 4 days. Patients without an enzymatic criterion of infarction (twice the upper limit of normal creatine phosphokinase) were considered to have acute myocardial ischemia. The diagnosis criteria for an evolving Q wave infarction was based on the ECG appearance of new pathologic Q waves of 30 msec or longer appearing two or more contiguous precordial leads and a typical curve of serum cardiac enzymes. In the absence of Q waves, enzymatic criteria associated with ST-T wave changes, with or without a decrease in R wave amplitude, were required for a diagnosis of non–Q wave infarction.

At the time of admission a QRS complex with no baseline drift was identified, and a horizontal baseline connecting consecutive TP segments was shown. ST segment displacement and T wave deflection were referred to this baseline. ST segment depression was measured at 80 msec after the J point. The T wave was measured as the maximal deflection (positive or negative) more than 120 msec after the J point. The location of maximal horizontal or downward sloping lasting at least 80 msec from the J point was determined in one of the precordial leads. This lead was then repeatedly observed for subsequent evolving ST-T wave changes. Patients with junctional ST segment depression (depressed J point) were not included. Patients were then divided into two groups: Group A included 21 patients (13 men and eight women; mean age 61 ± 6.1 years) with horizontal or downward-sloping ST segment depression with peaked positive T waves (Fig. 1). Group B included 11 patients (seven men and four women; mean age 66 ± 8.8 years) with horizontal or downward-sloping ST segment depression with peaked negative T waves (Fig. 2). From the initial study population, three patients were excluded from further analysis because of undetermined peaked T wave deflection.

Coronary arteriography was performed in multiple projections in all patients (mean 8 ± 4 days after admission) except one who had an acute infarction complicated by ventricular septal defect and cardiogenic shock before catheterization. The angiograms were reviewed by two independent observers. Stenosis of 70% or more luminal diameter reduction of the right coronary, left anterior descending, or circumflex artery and stenosis of 50% or more of the left main artery were considered significant. Stenosis of 70% or more of a large diagonal, marginal, or posterior descending artery was also considered significant.

Statistics. Statistical analysis was performed by means of Fischer's exact probability test. Student's t and chi-square tests were used for analysis of age and sex, respectively. p Values <0.05 were considered statistically significant and data are expressed as mean ± standard deviation.

RESULTS

Clinical characteristics (Tables I and II). There were no statistically significant differences between groups A and B with regard to age, sex, and risk factors (Table III). Of the eight patients in group A

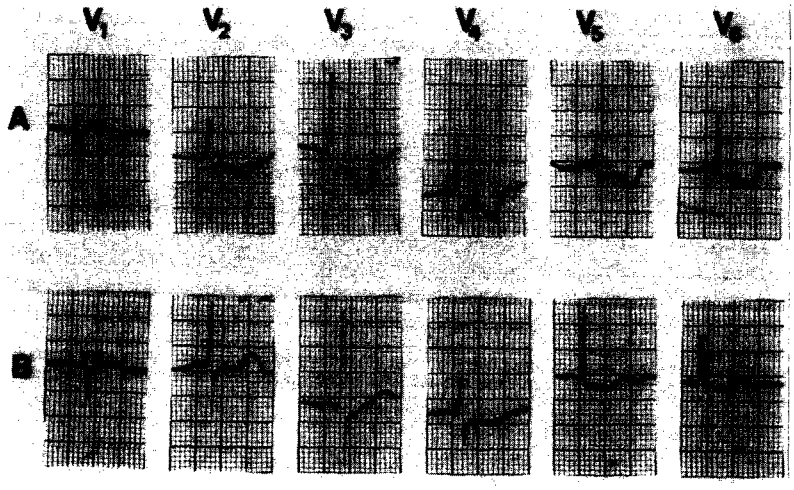

Fig. 2. Example of ischemic ST segment depression with negative T wave deflection (group B). *A*, During severe chest pain. *B*, Six hours later, showing a lesser degree of ST segment displacement and positivity of T waves.

(38.1%) who had myocardial infarction, none died during hospitalization. In group B four patients (36.4%) had acute myocardial infarction, and three of them died. (In-hospital mortality rate 27.3% [$p = 0.03$], statistically significant vs group A.) Death was not related to hazards of cardiac catheterization. Aortocoronary bypass surgery was performed in 14 patients, six (28.6%) from group A and eight from group B (72.7%; $p = 0.001$, statistically significant). Six patients from group A underwent coronary angioplasty (not statistically significant vs group B). Most of them had recurrent ischemic episodes.

ECG findings. As shown in Fig. 1, all 21 patients in group A had a typical ECG pattern consisting of a depressed ST segment passing into a peaked symmetrically positive T wave in the absence of tachycardia or reciprocal ST changes. Of the 21 patients who had the reported ST-T segment abnormalities, transition to other configuration was noted on serial ECG tracings (Table IV). The most common pattern was symmetric inversion of T waves with isoelectric ST segment (Fig. 3), which was seen in eight patients. Three of them had non–Q wave infarction, and the remaining five had myocardial ischemia. In seven more patients the ECG became normal after combined treatment with heparin and isosorbide dinitrate (Fig. 1). In the three patients in whom a Q wave infarction developed, a gradual upright shifting of the ST segment followed by a Q wave appearance and T wave inversion was noted. The three remaining patients had persistent ST segment depression with positive T waves, decreased R wave

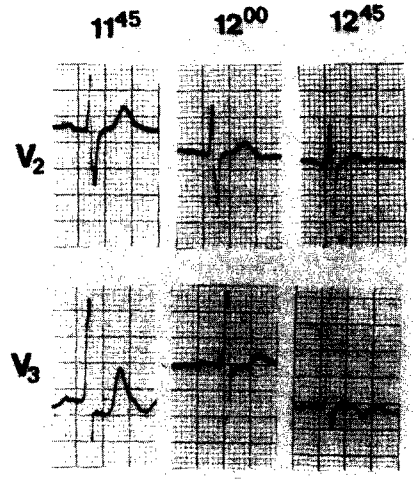

Fig. 3. Regression of ischemia initiated with pattern of downward displacement of ST segment (maximal in V_3), passing into tall upright T wave.

amplitude with normalization of ST-T, and upright shifting followed by normalization of the ST segment and inversion of T waves without the appearance of Q waves.

All 11 patients included in group B met our criterion of a depressed ST segment passing into a peaked negative T wave (Fig. 2). Evolving ECG changes are listed in Table V. Normalization of the ECG was the dominant evolving manifestation in this group of patients.

Angiographic findings. Table VI summarizes and correlates the angiographic findings of both groups. Coronary artery disease in group A ranged from

Table I. Group A: Clinical data, ECG evolution, and angiographic findings of patients first seen with ischemic ST segment depression (downward or horizontal pattern) passing into peaked upright T waves

Patient No.	*Age (yr)*	*Sex*	*Risk factors*	*Location of maximal ST depression*	*Subsequent evolving ECGs*	*Clinical diagnosis*	*Complications*	*Coronary angiographic data*	*Outcome*
1	59	F	Hypertension	V_5	Return of ST to isoelectric line, T wave inversion	Non–Q wave AMI	—	100% LCx	Medical therapy, discharged
2	62	M	Hypertension	V_3	Return of ST to isoelectric line, T wave inversion	Myocardial ischemia	—	90% 1st diagonal	Medical therapy, discharged
3	56	F	—	V_4	Normalization of ECG	Myocardial ischemia	Reischemia	95% LAD	PTCA
4	62	F	Hypertension	V_3	Normalization of ECG	Myocardial ischemia	Reischemia	95% LAD 80% LCx	PTCA (LAD)
5	64	M	Smoking	V_5	Return of ST to isoelectric line, T wave inversion	Non–Q wave AMI	—	95% 1st marginal	Medical therapy, discharged
6	54	F	Smoking	V_4	Persistent ST↓ with positive T wave	Myocardial ischemia	—	95% proximal LAD, 80% distal LAD 80% LCx	PTCA × 2 (proximal LAD and LCx)
7	65	F	Hypertension, smoking	V_3	Normalization of ST and T wave, ↓R wave amplitude	Non–Q wave AMI	VT, VF	90% LAD 99% LCx 100% RCA	CABG
8	71	M	DM	V_3	Normalization of ECG	Myocardial ischemia	Reischemia	90% LAD 80% RCA	PTCA (LAD)
9	69	M	Hypertension	V_4	Return of ST to isoelectric line, T wave inversion	Myocardial ischemia	—	70% LAD 85% LCx	Medical therapy discharged
10	56	M	—	V_3	ST↑ proceeding to Q and negative T waves	Q wave AMI	—	100% LAD	Medical Therapy, discharged
11	54	F	Hypertension	V_4	Normalization of ECG	Myocardial ischemia	—	NCA	Discharged

AF = Atrial fibrillation; AMI = acute myocardial infarction; CABG = coronary artery bypass grafting; ↓ = decrease; DM = diabetes mellitus; LAD = left anterior descending; LCx = left circumflex; LV = left ventricular; NCA = normal coronary arteries; PTCA = percutaneous transluminal coronary angioplasty; RCA = right coronary artery; RPD = right posterior descending; ST↓ = ST segment depression; ST↑ = ST segment elevation; VF = ventricular fibrillation; VT = ventricular tachycardia.

normal coronary anatomy to triple-vessel disease. One patient had angiographically normal coronary arteries (4.8%), eight had single-vessel disease (38.1%, statistically significant vs group B), eight had double-vessel disease (38.1%, statistically significant vs group B), and the remaining four had triple-vessel disease (19%). None of the patients in group A had angiographic evidence of significant left main occlusion.

Of the 10 patients in group B, one had double-vessel disease (10%) and two had triple-vessel disease (20%). Seven additional patients had significant left main coronary artery disease ($p = 0.001$, statistically significant vs group A). None of the patients had single-vessel disease or normal coronary arteries.

DISCUSSION

ST segment depression during experimental coronary occlusion has been ascribed to functional col-

Table I. Cont'd

Patient No.	*Age (yr)*	*Sex*	*Risk factors*	*Location of maximal ST depression*	*Subsequent evolving ECGs*	*Clinical diagnosis*	*Complications*	*Coronary angiographic data*	*Outcome*
12	54	M	Smoking	V_4	Return of ST to isoelectric line, T wave inversion	Myocardial ischemia	Reischemia	90% LAD	PTCA
13	73	M	Hypertension	V_3	Normalization of ECG	Myocardial ischemia	—	70% LAD 80% LCx	Medical therapy discharged
14	70	F	Hypertension	V_4	ST↑, normalization of ST, final inversion of T wave	Non–Q wave AMI	Reischemia	70% LAD 80% LCX 80% RCA	Medical therapy discharged
15	64	M	—	V_4	Normalization of ECG	Myocardial ischemia	—	90% LAD 90% LCx	CABG
16	59	M	—	V_4	ST↑ proceeding to Q and negative T waves	Q wave AMI	AF, mobile pedunculated LV thrombus	99% LAD	CABG, thrombectomy
17	61	M	Hypertension, DM	V_5	ST proceeding to Q and negative T waves	Q wave AMI	LV aneurysm, reinfarction	99% LAD 80% LCx 95% RCA 80% 1st diagonal	Medical therapy discharged
18	61	M	Hypertension	V_5	Return of ST to isoelectric line, T wave inversion	Myocardial ischemia	—	95% LAD 90% LCx	CABG
19	53	M	—	V_5	Return of ST to isoelectric line, T wave inversion	Non–Q wave AMI	AF, VT	95% LAD 80% RPD 99% 1st marginal	CABG
20	55	M	Smoking	V_4	Return of ST to isoelectric line, T wave inversion	Myocardial ischemia	Reischemia	90% LAD	PTCA
21	64	F	Hypertension, smoking	V_3	Normalization of ECG	Myocardial ischemia	—	90% LAD 95% LCx	CABG

lateral circulation to the ischemic area.[15] In humans, where coronary anatomy and dynamics of flow are more complicated than traditional experimental studies suggest, even the clinical course cannot be predicted. Findings of subendocardial ischemia may correspond to the earliest stage of acute non–Q wave infarction. It may either evolve into transmural ischemia (ST elevation) and subsequent Q wave infarction or resolve itself spontaneously.[16, 17] The difficulty in interpreting these changes may stem largely from the heterogeneous group of patients who were first seen with ST segment depression during chest pain. With a fixed coronary stenosis, ischemia affecting the subendocardial layers is directly proportional to increases in heart rate.[18] On the other hand, ST depression may be caused by vasospasm or an increase in oxygen demand in the presence of normal coronary arteries.[9] It may also represent the reciprocal electrical phenomenon of a "concealed" transmural ischemia taking place in the high segment of the posterior wall.[11]

Downward-sloping or horizontal ST segment depression during exercise testing accurately identifies patients with suspected coronary artery disease. The potential clinical value of these findings encountered in the setting of impending infarction and in the absence of tachycardia has not yet been defined. As far as we are aware, previous studies

Table II. Group B: Clinical data, ECG evolution, and angiographic findings of patients first seen with ischemic ST segment depression (downward or horizontal pattern) passing into negative T wave deflection

Patient No.	*Age (yr)*	*Sex*	*Risk factors*	*Location of maximal ST depression*	*Subsequent evolving ECGs*	*Clinical diagnosis*	*Complications*	*Coronary angiographic data*	*Outcome*
1	69	M	Hypertension	V_3	Normalization of ECG	Myocardial ischemia	—	90% LM 80% LAD	CABG
2	73	F	Hypertension, DM	V_3	Return of ST to isoelectric line, persistent negative T waves	non–Q wave AMI	VI, reischemia	99% LAD 100% LCx 90% RCA	CABG
3	64	F	Hypertension	V_4	Persistent ST ↓ and positive waves	Myocardial ischemia	—	90% LM 70% LCx 100% RCA 80% 1st diagonal	CABG
4	44	M	Hypertension, smoking hyperlipidemia	V_4	ST↑, positivity of T waves, appearance of Q waves	Q wave AMI	Acute VSD, cardiogenic shock	—	Died
5	66	M	Hypertension	V_4	Normalization of ECG	Myocardial ischemia	—	80% LM 80% LAD 100% LCx 80% RCA	CABG
6	70	M	—	V_4	Normalization of ECG	Myocardial ischemia	—	75% LM 80% LAD 90% LCx	CABG
7	63	M	Hypertension, DM, smoking	V_3	Normalization of ECG	Myocardial ischemia	Reischemia	90% LM 99% LAD 90% RCA 90% RPD	CABG
8	66	F	Hypertension, DM	V_4	Return of ST to isoelectric line, persistent negative T waves	Myocardial ischemia	Reischemia	99% LAD 100% RCA	CABG
9	65	M	Smoking	V_4	Normalization of ECG	Myocardial ischemia	—	99% LAD 90% LCx 80% RCA 60% LM	CABG
10	80	F	Hypertension	V_4	Persistent ST ↓ and negative T waves, ↓R wave amplitude	Non–Q wave AMI	Reinfarction, cardiogenic shock	90% LM	Died
11	64	M	DM, smoking	V_3	Persistent ST ↓ and negative T waves, ↓R wave amplitude	Non–Q wave AMI	Reinfarction, cardiogenic shock	99% LAD 90% LCx 80% RPD	Died

AMI = Acute myocardial infarction; CABG = coronary artery bypass grafting; ↓ = decrease; DM = diabetes mellitus; LAD = left anterior descending; LCx = left circumflex; LM = left main; RCA = right coronary artery; RPD = right posterior descending; ST↓ = ST segment depression; ST↑ = ST segment elevation; VSD = ventricular septal defect; VT = ventricular tachycardia.

Table III. Clinical correlates

	Group A (21 patients)	*Group B (11 patients)*	*p Value*
Age (yr)			
Mean	61 ± 6.1	66 ± 8/8	NS
Range	53-73	44-80	
Sex			
Male	13 (61.9%)	7 (63.6%)	NS
Female	8 (38.1%)	4 (36.4%)	
Risk factors			
Hypertension	11 (52.4%)	8 (72.7%)	NS
Diabetes mellitus	2 (9.5%)	4 (36.4%)	NS
Smoking	6 (28.6%)	4 (36.4%)	NS
Hyperlipidemia	—	1 (9.1%)	NS
No. of risk factors per patient	0.9	1.5	NS
Acute event			
Myocardial ischemia	13 (61.9%)	7 (63.6%)	NS
Non-Q wave infarction	5 (23.8%)	3 (27.3%)	NS
Q wave infarction	3 (14.3%)	1 (9.1%)	NS
Total no. of patients with infarct	8 (38.1%)	4 (36.4%)	NS
Outcome			
Death	—	3 (27.3%)	Significant ($p = 0.03$)
Reischemia and reinfarction	7 (33.3%)	5 (45.5%)	NS

Table IV. ECG data: Group A (21 patients)

Evolving ECGs	*No. of patients*
Symmetric inversion of T waves with isoelectric ST segment	8 (38.1%)
Normalization of ECG	7 (33.3%)
ST↑, Q wave appearance, T wave inversion	3 (14.3%)
Persistent ST↓ with positive T waves	1 (4.8%)
Decreased R wave amplitude	1 (4.8%)
ST↑ followed by normalization of ST and T wave inversion	1 (4.8%)

Table V. ECG data: Group B (11 patients)

Evolving ECGs	*No. of patients*
Normalization of ECG	5 (45.4%)
Return of ST to isoelectric line with persistent negative T waves	2 (18.2%)
Persistent ST↓ with positivity of T waves	1 (9.1%)
ST↑, positivity of T wave, Q wave appearance	1 (9.1%)
Persistent ST↓ with negative T waves	1 (9.1%)
Persistent ST↓, negative T waves, R wave amplitude	1 (9.1%)

were not uniform with respect to patient populations and ECG criteria and did not analyze separately the clinical course of patients who were first seen with downward-sloping or horizontal ST segment depression passing into tall peaked T waves versus negative T wave deflection. The potential effect of such an admixture of patients may contribute to the misconception of this clinical entity and the lack of distinct prognostic implications.

We studied a homogeneous group of patients at one point in the natural course of their coronary artery disease. Our findings indicate that the ECG characteristics of ST segment displacement, as well as the direction of T wave deflection, may contribute to risk stratification of patients with unstable angina. It is also obvious that ischemic ST depression associated with peaked negative T waves should be cause for suspicion of multivessel coronary artery disease or left main occlusion.

In the serial ECG follow-up during progression and regression of ischemia, peaking of T waves may represent the only ECG marker of transmural ischemia. It may originate from an initially negative T wave (the so-called pseudonormalization phenomenon), and it occurs along with or before the appearance of ST segment elevation. In group A, as long as the ischemic event endures, T waves heighten and the downward displacement of the ST segment becomes more prominent (Fig. 4). Deviation of the ST segment and the T wave in opposite directions is an unusual ECG presentation during acute ischemia or impending infarction. This ischemic pattern has neither been debated in the literature nor correlated with angiographic findings. Although T wave abnormalities are less specific than ST changes,[19] we may assume that the significance of tall upright T waves associated with ST depression is identical to those related to T waves recorded in the absence of ST segment deviation. In keeping with this hypothesis are the evolutionary ECG changes observed in group A. Of the 21 patients included in this group, three had progressive changes consisting of upward movement of the ST segment. The direction of the ST shift was constant, since ST elevation was preceded

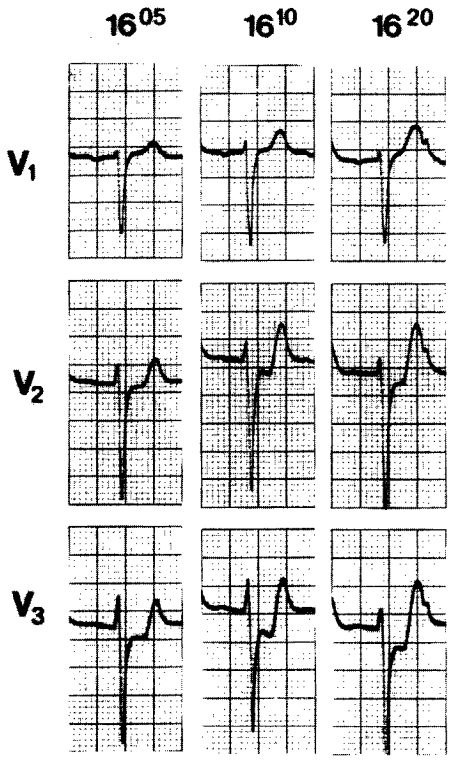

Fig. 4. Progressive changes of ischemia with same pattern as in Fig. 3. Note that T waves gradually heighten, whereas ST segment displacement becomes more prominent.

by depression but not vice versa. Regardless of the pathophysiologic sequence of coronary events, it is not inconceivable that subtotal spastic occlusion culminating in total occlusion accounted for these sequential ECG changes.

The significance of early negative T wave deflections associated with a pattern of ischemic injury (ST depression) has not yet been specified. In group B negative T wave deflections deepen with the length of the ischemic period. These changes may as well initiate mostly from positive or flattened T waves. Even though T wave morphology may be affected by the adjacent ST segment displacement, in our opinion those T wave changes are not directly related to abnormalities of ventricular repolarization. As shown in Fig. 1, T waves are clearly separated from the displaced ST segment, including the proximal limbs and not only the peak of the T waves.

Coronary vasospasm has been documented angiographically during episodes of angina, accompanied by ST segment depression and positivity of T waves.[16] These findings could also be interpreted in light of perfusion imaging procedures.[12] Deficits in

Table VI. Angiographic correlates

	Group A (21 patients)	*Group B (10 patients)*	*p Value*
Normal coronary arteries	1 (4.8%)	—	NS
Single-vessel disease	8 (38.1%)	—	Significant ($p = 0.05$)
Two-vessel disease	8 (38.1%)	1 (10%)	Significant ($p = 0.02$
Three-vessel disease	4 (19%)	2 (20%)	NS
Left main disease	—	7 (70%)	Significant ($p = 0.001$)
Left main and three-vessel disease	4 (19%)	9 (90%)	Significant p = 0.001)
PTCA	6 (28.6%)	—	NS
CABG	6 (28.6%)	8 (72.7%)	Significant ($p = 0.001$)

thallium uptake were caused by acute reduction of regional perfusion rather than by an increase in flow to viable nonischemic muscle. Good correlation was found between the perfusion defect and the site of "normalization" of negative T waves rather than the location of ST segment depression.

Patients in group A had a high incidence of single-vessel disease. In contrast most of the patients in group B had multivessel disease with left main involvement. Those patients undoubtedly may benefit from early catheterization and revascularization. In view of the excessive morbidity and mortality linked with catheterization of unstable patients with left main occlusive lesions,[20] recognition of this ischemic pattern might be beneficial, taking *a priori* into account the possibility of left main coronary artery disease.

Ogawa et al.[21] classified patients with non–Q wave infarction into three groups based on the ECG changes seen in the acute phase. Patients with ST segment depression proceeding to non–Q wave infarction had a higher incidence of multivessel disease and mortality than patients with initial ST segment elevation or T wave changes. In the present study in-hospital mortality was much more significant in patients with an initial pattern of downward ST segment displacement with negative T wave deflections during chest pain. The relatively late deaths of two patients who survived their initial coronary event of non–Q wave infarction (Table III) were the result of reinfarction and subsequent hemodynamic deterioration. This observation lends support to results of previous studies demonstrating

that nontransmural infarcts are frequently followed by reinfarction or sudden death.[22, 23] Therefore our clinical attention should be confined to patients with non–Q wave infarction and especially to those initially seen with an ischemic pattern of ST segment depression. Its propensity toward successive ischemic events strongly supports an early diagnostic evaluation, even in patients who are apparently well after an uncomplicated infarction.

Clinical implications. We believe that patients who are first seen with horizontal or downsloping ischemic ST segment depression, and especially those with negative T wave deflections, should be regarded as an extremely high-risk subset of patients. Aggressive management, including thrombolytic therapy, should be instituted to prevent the evolution of infarction.

We thank Judith Sasdi and Gill Sher for assistance in the preparation of the manuscript.

REFERENCES

1. De Wood MA, Spores JA, Notske R. Prevalence of total coronary occlusion during the early hours of transmural myocardial infarction. N Engl J Med 1980;303:897.
2. Yasue H, Omote S, Takizawa A, et al. Comparison of coronary arteriographic findings during angina pectoris associated with ST elevation or depression. Am J Cardiol 1981;47:539.
3. Yasue H, Omote S, Takizawa A, Nagao M, Tanaka S. Exertional angina pectoris caused by coronary arterial spasm: effects of various drugs. Am J Cardiol 1979;43:647.
4. De Servi S, Specchia G, Ardissino D, et al. Angiographic demonstration of different pathogenetic mechanisms in patients with segment depression. Am J Cardiol 1980; 45:1285.
5. Shea MJ, Deanfield JE, Wilson R, Delansheere C, Jones T, Selwyn AP. Transient ischemia in angina pectoris. Am J Cardiol 1985;56:34E.
6. Maseri A. Pathogenetic mechanisms of angina pectoris: expanding views. Br Heart J 1980;43:648.
7. Maseri A, Chierchia S, L'Abbate A. Pathogenetic mechanisms underlying the clinical events associated with atherosclerotic heart disease. Circulation 1980;62(suppl):3.
8. Vincent GM, Abildskov JA, Burgess MJ. Mechanisms of ischemic ST segment displacement. Circulation 1977; 55:559.
9. Legrand V, Hodgson JM, Bates ER, et al. Abnormal coronary flow reserve and abnormal radionuclide exercise test results in patients with normal coronary angiograms. J Am Coll Cardiol 1985;6:1245.
10. Feldman RL. Coronary thrombosis, coronary spasm and coronary atherosclerosis and speculation on the link between unstable angina and acute myocardial infarction. Am J Cardiol 1987;59:1187.
11. Sclarovsky S, Topaz O, Rechavia E, Strasberg B, Agmon J. ST segment depression in V_2-V_3 as the presenting electrocardiographic feature of posterolateral infarction. Am Heart J 1987;113:1085.
12. Parodi O, Uthurralt N, Severi S, et al. Transient reduction of regional myocardial perfusion during angina at rest with ST segment depression or normalization of negative T waves. Circulation 1981;63:1238.
13. Lewin RF, Sclarovsky S, Rosenberg B, et al. Positivization of T wave with or without ST segment elevation in patients with unstable angina. Coronary angiographic findings and in-hospital prognosis. Eur Heart J 1987;8:31.
14. The ISAM Study Group. A prospective trial of intravenous streptokinase in acute myocardial infarction. N Engl J Med 1986;314:1465.
15. Ekmekci A, Toyoshima H, Kwoczynski JK, Nagaya T, Prinzmetal M. Clinical and experimental difference between ischemia with ST elevation and ischemia with ST depression. Am J Cardiol 1961;7:412.
16. Maseri A, Severi S, De Nes M, et al. "Variant" angina: one aspect of a continuous spectrum of vasospastic myocardial ischemia. Am J Cardiol 1978;42:1019.
17. Maseri A, Chierchia S. Coronary artery spasm: demonstration, definition, diagnosis and consequences. Prog Cardiovasc Dis 1982;25:169.
18. Gobel FL, Nordstrom LA, Nelson RR, Jorgensen CR, Wang Y. The rate-pressure product as an index of myocardial oxygen consumption during exercise in patients with angina pectoris. Circulation 1978;57:549.
19. Papapietro SE, Niess GS, Paine TD, et al. Transient electrocardiographic changes in patients with unstable angina: relation to coronary arterial anatomy. Am J Cardiol 1980; 46:28.
20. Gordon PR, Abrams C, Gash AK, Carabello BA. Pericatheterization risk factors in left main coronary artery stenosis. Am J Cardiol 1987;59:1080.
21. Ogawa H, Hiramori K, Haze K, et al. Classification of non-Q wave myocardial infarction according to electrocardiographic changes. Br Heart J 1985;54:473.
22. De Wood MA, Stifter WF, Simpson CS, et al. Coronary arteriographic findings soon after non-Q wave myocardial infarction. N Engl J Med 1986;315:417.
23. Cannom DS, Levy W, Cohen LS. The short and long term prognosis of patients with transmural and non-transmural myocardial infarction. Am J Cardiol 1976;61:452.