Angiographic and clinical characteristics of patients with unstable angina showing an ECG pattern indicating critical narrowing of the proximal LAD coronary artery

Chris de Zwaan, MD, Frits W. Bär, MD, Johan H. A. Janssen, MD, Emiel C. Cheriex, MD, Willem R. M. Dassen, PhD, Pedro Brugada, MD, Olaf C. K. M. Penn, MD, and Hein J. J. Wellens, MD. *Maastricht, The Netherlands*

In 1982 we reported on a typical ECG pattern pointing to a critical reduction in flow in the left anterior descending coronary artery (LAD) in patients admitted to the hospital because of unstable angina.[1] These patients characteristically had abnormal ST segments and negative T waves in the precordial leads V_2 and V_3 in the absence of pathologic Q waves. Similar observations were made by Haines et al.[2] and Boden et al.[3] The ECG abnormalities were frequently only present during a limited period of time.

Considering the importance of the LAD in perfusing a large area of the left ventricular myocardium, awareness of this ECG pattern is helpful in identifying patients with unstable angina who are at risk for losing a considerable amount of myocardial tissue. In this article we report our findings in a prospective study of 180 consecutive patients with unstable angina who had this ECG pattern.

METHODOLOGY

Patient selection. From July 1, 1980 to December 31, 1985, a total of 204 patients admitted to the hospital because of unstable angina had typical ECG pattern.[1] Thirteen of them had a myocardial infarction just before cardiac catheterization. Data were incomplete in 11 patients. The remaining 180 patients underwent coronary and left ventricular angiography within a mean of 3.3 days after admission. They represent 14% (180 of 1260) of patients hospitalized during that period with a diagnosis of unstable angina.

From the Departments of Cardiology and Cardiothoracic Surgery, the University of Limburg, Academic Hospital Maastricht.

Received for publication Sept. 6, 1988; accepted Nov. 1, 1988.

Correspondence: Chris de Zwaan, MD, Department of Cardiology, Academic Hospital Maastricht, Annadal 1, PB 1918, 6201 BX Maastricht, The Netherlands.

Diagnosis of unstable angina. Unstable angina was defined as angina of recent onset, sudden worsening of preexisting stable angina, or angina after a pain-free period (at least 3 months) after a nonanterior myocardial infarction. Chest pain had to have started within 6 weeks before admission. Severity of complaints was classified according to New York Heart Association (NYHA) functional class III or IV. The last episode of chest pain should have taken place within 5 days before the patient entered the study and could have lasted for a long period of time (more than 20 minutes).

Other inclusion and exclusion criteria. All patients were less than 76 years of age. If pain was present at the time of admission, it had to respond to sublingual or intravenous nitroglycerin. Patients were excluded if they had severe pump failure (NYHA class III of IV), cardiomyopathy, or previous cardiac surgery or coronary angioplasty; also excluded were those with a pacemaker or anemia.

ECG features. In all patients 12-lead ECGs were routinely obtained twice daily. Additional ECGs were obtained during and after new episodes of chest pain. All patients had at the time of entry into the study and outside an episode of chest pain, ST segment and T wave abnormalities in leads V_2 and V_3 (Fig. 1). The abnormalities in leads V_2 and V_3 consisted of an isoelectric or minimally elevated (<1 mm) take-off of the ST segment followed by a concave or straight ST segment and a symmetrically inverted T wave; 121 patients also had these changes in lead V_1. One hundred thirty-six patients also had ST-T segment abnormalities in lead V_4, and sometimes in V_5 or V_6, characterized by a take-off of the ST segment below the isoelectric line followed by a convex ST segment passing into a symmetrically inverted T wave (Fig. 1, *B*).

Excluded were patients with Q waves or QS

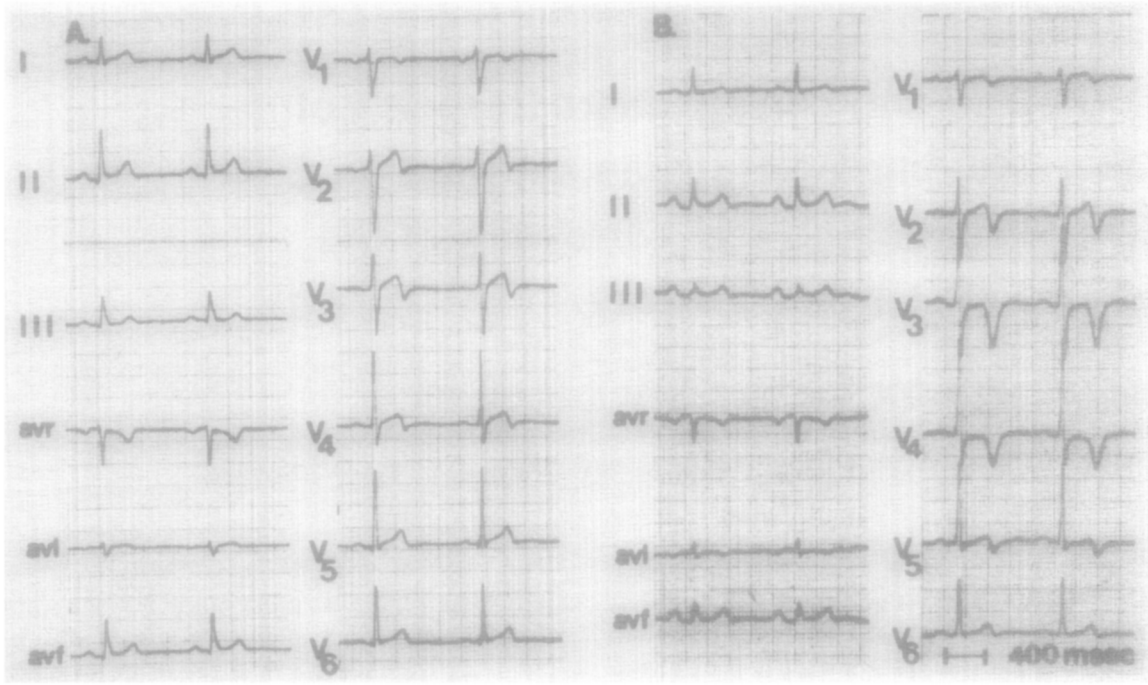

Fig. 1. Examples of ECG patterns found in precordial leads of patients reported. **A**, Pattern found in 44 patients. **B**, Pattern found in 136 patients. (See text for details).

complexes in leads V_2 to V_4, patients with a left or right bundle branch block pattern or left or right ventricular hypertrophy, and patients with a loss of initial R wave voltage in leads V_1 to V_3.

Enzyme measurements. Serial enzyme values for creatine kinase, serum glutamic oxaloacetic transaminase (SGOT), and serum lactic dehydrogenase were measured in all patients every 8 hours for 3 consecutive days after admission and were repeated after a new attack of chest pain during hospitalization. The peak values had to be less than twice the upper limit of the normal values (220, 40, and 400 U/L, respectively) if the patient was to be included in the study.

Cardiac catheterization. The investigation was performed 3 hours to 10 days (mean 3.3 days) after admission, or 12 hours to 14 days (mean 4.6 days) after the last attack of chest pain. Coronary angiography was performed by the Judkins technique with multiple projections. The severity of the stenosis was graded subjectively. In 61 of 147 patients with a nonoccluded ischemia-related artery, grading was also done quantitatively by means of the previously validated and described computer-based cardiovascular angiography analysis system (CAAS) with high-resolution digital subtraction images.[4]

Collateral filling of the LAD was considered to be present if there was visualization of the distal portion of the LAD different from anterograde flow after injection of the contrast medium either into the right or the circumflex coronary artery or by bridging collaterals from the proximal to the distal segment of the LAD. Diminished flow was considered to be present if there was anterogradely no distal filling within three cardiac cycles.

Abnormalities in left ventricular wall motion were assessed in the 30-degree right anterior oblique and the 60-degree left anterior oblique views by means of the modified method of Ingels et al.[5] The left ventricular silhouette was divided into anterobasal, anterolateral, apical, diaphragmatic, posterobasal and anteroseptal, and posterolateral segments, respectively. Systolic left ventricular wall motion was graded as normal, hypokinetic, akinetic, or dyskinetic. Diastolic motion of the anterior wall segments was considered to be abnormal if a biphasic or triphasic relaxation pattern and a prolonged relaxation time were present. The films were examined by at least two experienced angiographers.

Treatment regimen. Medical management consisted of a beta-adrenergic–blocking agent (metoprolol) and a nitrate (isosorbide dinitrate). A calcium channel blocker (nifedipine) was given when indicated. Since 1984 antiplatelet drugs (aspirin, dipyridamole) have been added to this treatment regimen.

Follow-up. Follow-up ended 1.5 years after the last patient was admitted to the study. Incidence of angina pectoris, myocardial infarction, death, coronary artery bypass surgery, and coronary angioplas-

Table I. Clinical characteristics of patients at time of entry into the study

No. of patients	180
Age in years (mean ± SD)	55 ± 9
Men	124
Previous angina pectoris	86
Previous myocardial infarction	20
Severity of angina pectoris at entry	
Class III	49
Class IV	131
Duration of unstable angina pectoris at entry in days	
Mean ± SD	6.6 ± 9.0
Median	3.0
Time interval between last attack of chest pain and admission in days (mean ± SD)	1.3 ± 1.3
Creatine kinase value	
One to two times upper limit of normal	21
Normal	157

Table II. Coronary angiographic characteristics of patients

No. of patients	180
No. of diseased vessels	
1	75
2	66
3	39
Occluded LAD	33
Narrowed LAD	
Diameter narrowing (subjectively [n = 147], in %, mean ± SD)	85 ± 10
Area of stenosis (CAAS [n = 61] in %, mean ± SD)	79 ± 16
Diminished anterograde flow over LAD	25
Calcification of LAD	97
Collaterals to LAD	75

CAAS = cardiovascular angiography analysis system.

ty was recorded after 1 year and at the end of the follow-up period. The mean duration of follow-up was 4.2 years for the survivors.

OBSERVATIONS

Clinical characteristics. As shown in Table I, the mean age of the 180 patients was 55 years. The majority were men. Almost half of the patients had previously had angina pectoris. In 73% of patients the severity of angina was considered as class IV (attacks of angina at rest). In this group of patients the last attack of pain had occurred 1.2 days (mean) before admission and had lasted an average of 39 minutes. The mean duration of unstable angina was 4.9 days. In patients with angina during slight exercise (class III), these values were 1.8 days, 11 minutes, and 11.4 days, respectively.

Findings at coronary angiography. As shown in Table II, all patients had abnormalities in the LAD. The site of occlusion or narrowing was proximal to the first septal perforator in 29% of patients (n = 53) and between the first and second septal branches in 54% (n = 97). The lesion in the LAD was the only abnormality (single-vessel disease) in 75 patients. Sixty-six patients had additional disease in one vessel, and 39 patients had significant narrowing in both remaining coronary arteries. Severe mainstem disease as the "culprit" lesion was not found in these patients. More than half of the patients had calcification in the LAD.

The LAD was completely obstructed in 33 patients. In the remaining 147 patients the degree of narrowing varied from 50% to 99%, with a mean of 85% according to subjective analysis and 79% according to quantitative analysis (CAAS).

Diminished anterograde flow over the stenosis in the LAD was observed in 25 patients: in 18 of 77 vessels with near-total occlusion (90% to 99%), in 5 of 46 arteries with diameter narrowing of 80% to 90%, and in two vessels with diameter narrowing of 50% to 80%. Collateral circulation was present in all 33 totally obstructed arteries, in 35 of 77 vessels with near-total occlusion (90% to 99%), and in 7 of 46 arteries with diameter narrowing of 80% to 90%.

We did not find any significant relation between results of coronary angiography and duration of unstable angina pectoris, although no occluded vessels and a low incidence of collaterals (4 of 14) were found in 14 patients with a duration of unstable angina of only 1 day. Comparison with severity of anginal complaints, class IV as compared to class III, yielded a reduced incidence of collaterals (49 of 131 vs 26 of 49 patients, chi-square test: $p = 0.08$) and a lower incidence of totally occluded arteries (20 of 131 vs 13 of 49 patients, chi-square test: not significant) in class IV angina.

Findings at left ventricular angiography. Systolic left ventricular wall motion abnormalities related to the diseased LAD were present in 137 patients. The abnormal systolic motion was hypo- or akinesis in 130 patients. In the remaining seven patients regional dyskinesis was observed. Many patients had systolic wall motion abnormalities in more than one segment. The anteroseptal region was most commonly involved (n = 97) as compared to the apical (n = 75), anterolateral (n = 66), and anterobasal

Table III. ECG findings in the outpatient clinic

ECG findings	n		
	I	*II*	*III*
ST-T segment normal at first visit (%)	12/26 (46)	48/101 (48)	6/25 (24)
ST-T segment normalized < 6 months (%)	21/26 (81)	95/101 (94)	21/25 (84)
ST-T segment remained abnormal (%)	5/26 (19)	6/101 (6)	4/25 (16)

I = medical therapy (assigned and ultimate treatment); II = early revascularization; III = late revascularization.

(n = 28) segments. The interval between the last attack of chest pain and angiography was an average of 4.1 (±2.5) days for the 137 patients with abnormal systolic wall motion and 6.4 (±3.3) days for the remaining 43 patients with normal systolic left ventricular function (Student's unpaired *t* test, $p < 0.001$).

Abnormal diastolic left ventricular wall motion was observed in 153 patients: 129 of 137 with abnormal systolic wall motion and 24 of 43 with normal systolic wall motion. No serious complications occurred during or after cardiac catheterization in this group of patients with unstable angina. One patient had nonsustained ventricular tachycardia during coronary angiography.

ECG findings. The ECG findings described in the Methodology section were present at the time of admission to the hospital in 108 patients. In 72 patients the ECG abnormalities developed later: within 24 hours after admission in 56 patients, within 2 days in 10, within 3 days in five, and within 5 days in one. During an attack of chest pain at the time of or after admission to the hospital, the described ST-T segment abnormalities in the precordial leads normalized or the patient had ST segment elevation.

When we studied correlations between clinical and angiographic findings in patients with the typical ECG pattern "at the time of" admission or "after" admission, we found that the mean duration of unstable angina in the former group was insignificantly longer (n = 108, mean ± SD 7.6 ± 8.1 vs n = 72, mean ± SD 5.3 ± 7.1 days). Furthermore, a higher incidence of collaterals (52 of 108 vs 23 of 72, chi-square test: $p < 0.05$) was present in patients with ST-T segment changes on admission.

We also correlated the presence or absence of abnormal ST-T segments in the precordial leads during angiography with the presence or absence of abnormal systolic left ventricular wall motion. If the ECG was still abnormal there was a greater chance of abnormal systolic left ventricular wall motion as when the ECG had normalized (131 of 159 vs 6 of 21 patients, chi-square test: $p < 0.001$).

The long-term duration of the ST-T segment abnormalities could be established from visits to the outpatient clinic in 152 of 180 patients (Table III). As shown normalization of the ST-T segments occurred in the majority of patients. In 28 patients no ECGs were reviewed because of death or the development of a myocardial infarction, or because the ECG was not available.

Enzyme measurements. Twenty-one patients had a small increase in enzyme values. In 10 patients only the creatine kinase level was elevated (mean 294 U/L). In 11 patients there was also release of SGOT, which was always less than twice the upper limit of normal (mean 53 U/L).

Of these 21 patients 19 had class IV angina, 17 had angina of recent onset, and eight had total occlusion of the ischemia-related LAD. These incidences are higher in comparison to the total study population. However, the incidence of collaterals at the time of catheterization (9 of 21) was not different.

Clinical course. At the time of admission to the hospital all patients were treated with medical therapy (nitrates, beta-adrenergic–blocking agents, and/or calcium channel blockers). In view of our earlier experience in 26 patients,[1] where we found a high incidence of anterior wall myocardial infarction during hospitalization and large areas at risk at the time of cardiac catheterization, most of our patients had early revascularization. In the present study nine patients had a myocardial infarction 6 days (mean) after admission to the hospital and before scheduled revascularization. The mean peak SGOT value was 320 U/L. During hospitalization two of the patients with infarctions died, both because of pump failure.

As shown in Fig. 2, 115 patients underwent (within a mean of 12 days after hospital admission) bypass surgery (103 patients) or angioplasty (12 patients), whereas 56 patients were treated medically. During early bypass surgery six patients had myocardial infarction. During hospitalization three early revascularized patients died: two of the six infarcted patients because of pump failure and one of the 109 noninfarcted patients because of sepsis. Of the 56 initially medically treated patients, 26

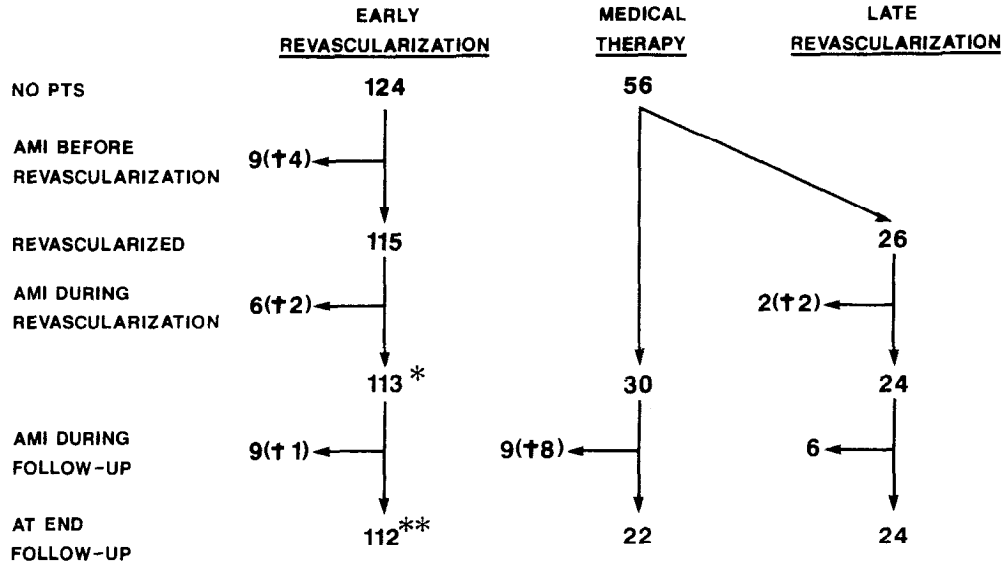

Fig. 2. Flow chart showing modes of treatment and incidence of acute myocardial infarction *(AMI)* and cardiac death during follow-up. *One patient died of sepsis; **five patients died of noncardiac-related causes; numbers in parentheses indicate patients with cardiac death.

remained unstable or became refractory to drug therapy and ultimately underwent a revascularization procedure (a mean of 8 months and 15 days after the initial admission).

Treatment. Twenty-five surgically treated patients received only one bypass graft, 36 received two, 29 received three, 20 received four, 16 received five, two received six, and one patient received seven (mean 2.8). In only 6 of 99 operation reports was a scar of myocardial infarction mentioned as being present. The overall operative mortality rate (early and late revascularization surgery) was 5 of 129 patients; four patients died of pump failure from myocardial injury that developed during surgery (two of these patients also received a valve prosthesis); one patient died of sepsis. During follow-up 98 of 141 revascularized patients became asymptomatic. Five patients required either reoperation (n = 3) or a second angioplasty (n = 2).

Thirty patients did not undergo a revascularization procedure for one or more reasons such as control by medication (n = 21), less than 65% LAD stenosis (n = 4), extensive collateral circulation to the LAD (n = 13), or location of the ischemia-related lesion in the first diagonal branch (n = 2). Other reasons included inoperability (n = 3) and refusal (n = 2). Only 8 of the 30 nonrevascularized patients continued to have no complaints, including all four patients with nonseverely narrowed vessels and 4 of 12 patients with an occluded ischemia-related LAD at the time of the initial angiography. Eight of the 30 patients died (two patients with extensive collateral circulation, two who were considered to be inoperable, and one who refused intervention).

Myocardial infarction and cardiac death. According to Fig. 2, 24 patients had a myocardial infarction during follow-up. For the sake of simplicity, we incorporated in this group of myocardial infarction 6 patients who had sudden death (1 of the early revascularization and 5 of the medical therapy group). In 35 patients who had a proven myocardial infarction we observed the following: (1) According to the baseline angiogram the mean number of diseased coronary arteries was higher compared to the remaining patients in the total study population (2.1 of 35 vs 1.7 of 145 patients, chi-square test: $p < 0.01$). (2) Patients who had the acute myocardial infarction in the hospital (nine before and six during early revascularization) had no occluded ischemia-related LAD (chi-square test: not significant) and less collaterals compared to the noninfarcted patients in the total series (2 of 15 vs 73 of 165 patients, chi-square test: $p < 0.05$). (3) In both the "medically" treated and the revascularization-related groups with myocardial infarction, the infarct was always located in the anterior wall. In the two series of postintervention myocardial infarction this was the case in only 43%.

COMMENTS

In view of the importance of the LAD in supplying blood to the left ventricular myocardium,[6] recognition of patients with unstable angina and severe narrowing of the LAD is of obvious importance. We became aware of an ECG pattern suggestive of a

critical lesion in the proximal LAD when we retrospectively studied the ECG findings in patients admitted with unstable angina. Our study reported in 1982 was also composed of patients studied prospectively. Our impression at that time was that medical treatment of patients with unstable angina showing the described ECG pattern was followed by a high incidence of extensive anterior wall myocardial infarction.[1] This experience and the angiographic finding of extensive segmental myocardial dysfunction prompted us to treat this subgroup of patients with unstable angina—in principle—more aggressively by way of coronary artery bypass grafting or angioplasty.

Location and severity of LAD disease and incidence of collateral circulation. Patients with unstable angina have a higher incidence of left main and proximal left anterior descending coronary artery disease compared to patients with stable angina pectoris.[7] When patients with unstable angina have the typical ECG pattern described, 83% will have their "culprit" lesion proximal to the second septal perforator. In our study 106 patients had a complete (n = 33) or almost totally occluded (n = 73) ischemia-related vessel, which is in agreement with the study of Plotnick et al.[8]

In the stenotic vessels the mean degree of narrowing was 85% with subjective analysis and 79% with quantitative analysis (CAAS). According to McMahon et al.[9] the subjectively graded coronary artery severity is comparable to the quantitatively assessed area of stenosis. The percentage of stenosis determined by videodensitometry correlates well with the percentage of reduction in cross-sectional area as measured by histologic planimetry in postmortem hearts ($r = 0.97$).[10]

Our patients were characterized by absence of the development of Q wave infarction in the anterior wall. It is well known that the presence of collaterals affects the extent and severity of myocardial injury.[8,11,12] Collateral vessels were visualized in this study in all (of 33) totally occluded coronary arteries, in 35 of 77 vessels with near-total occlusion (90% to 99%), and in 7 of 46 segments with 80% to 90% diameter narrowing.

Angiographic findings related to clinical findings. An occluded LAD and collaterals were more often seen in class III angina pectoris than in class IV, although these differences were not statistically significant. The mean duration of the interval between the last attack of chest pain and angiography was longer in the former compared to the latter group (5.9 vs 4.2 days) as was the mean duration of unstable angina at the time of admission (11.4 vs 4.9 days). It is possible that class III unstable angina is the result of a slowly diminishing flow over a gradually increasing vessel diameter narrowing and a moderate flow over collaterals, whereas class IV unstable angina is caused by a "sudden" reduction of flow in a severely stenosed artery and sluggish flow by way of collaterals (if present).

It is of interest that all 15 patients who subsequently had an anterior myocardial infarction had no occluded LAD and few collateral vessels at baseline angiography, whereas none of the 33 patients with an occluded vessel at the time of initial angiography had an acute myocardial injury during hospitalization. Aggressive therapy therefore seems to be more important in unstable patients with proximal LAD stenosis and no collaterals compared to patients with an occluded LAD and collateral circulation.

Left ventricular wall motion. Whereas only 21 patients had a slight increase in enzyme levels, abnormal systolic wall motion was found in 137 patients. These wall motion abnormalities were most likely caused by hibernating or stunned myocardium.[13-16] The fact that in only 6 of 99 operation reports an anterior wall myocardial scar was mentioned to be present is suggestive of that mechanism. Although not quantified the alterations described in diastolic wall motion can be another illustration of stunning.[17] Recent evidence suggests that abnormalities in diastolic function persist beyond the recovery of normal systolic function in patients rendered ischemic for brief periods during percutaneous transluminal coronary angioplasty.[18]

ECG observations. During the phase of terminal T wave negativity in the left precordial leads most patients had transient QT prolongation. These changes were partly produced by incorporation of a negative U wave in the terminal part of the negative T wave. The group with ST-T segment abnormality at the time of admission to the hospital had a history of unstable angina that was 2.3 days longer, whereas the severity of coronary disease was the same compared to the population in which the abnormality developed later. This finding suggests that duration of myocardial ischemia was responsible for development of the ST-T segment changes.

Ninety percent of patients regained a normal ST-T segment over time. The recovery time was longest in patients who continued to have complaints or became refractory to medical therapy, also suggesting persistence of myocardial ischemia. However, more collaterals were visualized at the initial angiography in the group with abnormal ECGs on admission compared to those in whom the changes developed during hospitalization (52 of 108 vs 23 of 72 patients). This suggests that reperfusion by col-

laterals may have accelerated disintegration of injured cells in the former group.[19]

Myocardial infarction and death. During the study period 204 patients initially had unstable angina and a typical ECG pattern. Eleven patients had incomplete data, and 13 patients had an anterior wall myocardial infarction before angiography was performed. Fifteen of 180 patients who entered the study had an extensive anterior wall infarction soon after angiography. Therefore 28 of 204 (13.7%) patients initially seen with the syndrome had extensive myocardial damage during hospitalization. As a consequence of this injury nine patients died shortly after admission (four of the earlier mentioned preangiography infarcted 13 patients and 5 of the 15 postangiography infarcted patients). The incidence of myocardial infarction in this high-risk group was low in comparison to data in the literature.[20, 21] We believe that this low incidence was obtained by our approach, which was to treat these patients aggressively on an urgent basis. The death-acute myocardial infarction ratio was high, indicating the short-term "risk" in this patient population.

Following discharge after the initial hospitalization, at the end of the follow-up period, another 26 patients had acute myocardial infarction (two during late revascularization), and 11 patients died of cardiac-related causes. Most of the cardiac deaths occurred in the nonrevascularized patients (Fig. 2) suggesting better outcome with revascularization. However, in the absence of randomization to medical or revascularization therapy, such a conclusion should be made with caution.

Revascularization. Data have been published showing that disease of the proximal LAD is associated with a high risk[22] and that those patients have a better chance of survival when treated with coronary bypass surgery.[23] It is suggested that ischemia (in our study illustrated by the large areas of jeopardized left ventricular myocardium during angiography) is likely to be responsible for cardiac death and morbidity[24] and that revascularization is far more effective in relieving ischemia than pharmacologic therapy.[24-26]

Coronary bypass surgery was ultimately performed in the present study in 129 patients. Five patients died. If we exclude the three patients with an additional artificial valve the operative mortality rate (1.6%) is as low as that described by Rahimtoola et al.[27] in 1282 patients with unstable angina, namely, 1.8%. The perioperative myocardial infarction rate in our study of unstable angina was 8 of 129 patients (6.2%).

Limitations of the study. The present study does not allow a comparison of efficacy of medical versus revascularization therapy because no randomization procedure was followed in either treatment group. Also the study was done at a time when coronary angioplasty was not performed as often as at the present time and thrombolytic therapy was not used in unstable angina. Now early revascularization by percutaneous transluminal coronary angioplasty plays an important role in this subgroup of patients with unstable angina.[28, 29] However, although a high incidence of thrombosis has recently been shown in unstable angina,[30, 31] the value of thrombolytic therapy in these patients has not yet been established.

Practical implications. Risk stratification and correct treatment of patients admitted because of acute coronary syndromes is one of the most important challenges in modern cardiology. This study stresses the importance of being aware that a distinct ECG pattern, present on admission or developing shortly thereafter, allows recognition of patients with unstable angina and a critical lesion proximal in the LAD as having a poor prognosis. We found that the risk of myocardial infarction was lowest in patients with an occluded LAD and collateral circulation and highest in those with a high degree of stenosis without collaterals. Especially the latter group of patients should be revascularized on an urgent basis. We believe that early catheterization is indicated in these patients to identify those requiring early revascularization.

SUMMARY

One hundred eighty of 1260 patients consecutively admitted to the hospital because of unstable angina pectoris had the typical ST-T segment changes suggestive of a critical stenosis in the proximal LAD. In 108 patients the ECG abnormalities were present at the time of admission. In the remaining 72 patients they developed shortly thereafter. The difference between these two groups was a longer duration of anginal complaints in the former (mean 2.3 days). Results of coronary angiography, performed a mean of 4.6 days after the last attack of chest pain, showed 50% or more narrowing in the proximal LAD in all patients. Thirty-three patients had complete occlusion of the LAD and 75 had collateral circulation to the LAD.

Results of left ventricular angiography showed abnormal systolic left ventricular wall motion in 137 patients and normal systolic motion in the remaining 43 patients. The difference between these two groups was a shorter mean time interval between the last attack of chest pain and angiography in the former group ($p < 0.001$). Twenty-four patients had only abnormal diastolic wall motion.

Twenty-one patients had a small increase in the

creatine kinase level at the time of admission. Fifteen patients (nine before and six during early revascularization) had an anterior wall myocardial infarction in the hospital; these patients had a patent but severely narrowed LAD and a low incidence of collateral circulation to the LAD. One hundred fifteen patients underwent revascularization shortly after admission. Of 56 patients treated medically, 26 eventually underwent revascularization (a mean of 8 months and 15 days after the initial admission).

During the follow-up period (mean 4.2 years) eight of the 30 patients treated medically died of a cardiac cause. There was one cardiac death among the 136 patients discharged after revascularization. During follow-up most of the patients showed normalization of the ECG and left ventricular wall motion abnormalities, suggesting hibernation or stunning of the myocardium as the mechanism of these abnormalities.

We conclude that in risk stratification of patients with unstable angina, the ECG is of great value for recognizing a subset of patients with a proximal LAD lesion having a poor prognosis because of a substantial area of jeopardized myocardium. Early cardiac catheterization is indicated so that appropriate therapy can be selected.

REFERENCES

1. de Zwaan C, Bär FWHM, Wellens HJJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. Am Heart J 1982;103:730-5.
2. Haines DE, Raabe DS, Gundel WD, Wackers FJT. Anatomic and prognostic significance of new T-wave inversion in unstable angina. Am J Cardiol 1983;52:14-18.
3. Boden WE, Bough EW, Benham J, Shulman RS. Unstable angina with episodic ST segment elevation and minimal creatine kinase release culminating in extensive recurrent infarction. J Am Coll Cardiol 1983;2:11-20.
4. Reiber JHC, Serruys PW, Kooyman CJ. Assessment of short-, medium-, and long-term variations in arterial dimensions from computer-assisted quantitation of coronary cineangiogram. Circulation 1985;71:280-8.
5. Ingels Jr NB, Daughters GT, Stinson EB, Alderman EL. Evaluation of methods for quantitating left ventricular segmental wall motion in man using myocardial markers as a standard. Circulation 1980;61:966-72.
6. Edwards WD, Tajk AJ, Seward JB. Standardized nomenclature and anatomic basis for regional tomographic analysis of the heart. Mayo Clin Proc 1981;56:479-97.
7. Plotnick GD, Greene HL, Carliner NH, Becker LC, Fisher ML. Clinical indicators of left main coronary artery disease in unstable angina. Ann Intern Med 1979;91:149-53.
8. Plotnick GD, Fisher ML, Lerner B, Carliner NH, Peters RW, Becker LC. Collateral circulation in patients with unstable angina. Chest 1982;82:719-25.
9. McMahon MM, Brown BG, Cukingnan R, Rolett EL, Bolson E, Frimer M, Dodge HT. Quantitative coronary angiography: measurement of the "critical" stenosis in patients with unstable angina and single vessel disease without collaterals. Circulation 1979;60:106-13.
10. Nichols AB, Gabrieli CFO, Fenoglio JJ, Esser PD. Quantification of relative coronary arterial stenosis by cinevideo densitometric analysis of coronary arteriograms. Circulation 1984;69:512-22.
11. Gorlin R, Fuster V, Ambrose JA. Anatomic-physiologic links between acute coronary syndromes. Circulation 1986;74:6-9.
12. Epstein SE. Influence of stenosis severity on coronary collateral development and importance of collaterals in maintaining left ventricular function during acute coronary occlusion. Am J Cardiol 1988;61:866-8.
13. Kloner RA. Persistent myocardial abnormalities following brief periods of temporary coronary occlusion not associated with necrosis [Abstract]. Circulation 1980;62:III-80.
14. Flameng W, Wouters L, Sergeant P, Lewi P, Borgers M, Thone F, Suy R. Multivariate analysis of angiographic, histologic and electrocardiographic data in patients with coronary heart disease. Circulation 1984;70:7-17.
15. Braunwald E, Rutherford JD. Reversible ischemic left ventricular dysfunction: evidence for the "hibernating myocardium." J Am Coll Cardiol 1986;8:1467-70.
16. Preuss KC, Gross GJ, Brooks HL, Warltier DC. Time course of recovery of "stunned" myocardium following variable periods of ischemia in conscious and anesthetized dogs. Am Heart J 1987;114:696-703.
17. Przyklenk K, Patel B, Kloner RA. Diastolic abnormalities of postischemic "stunned" myocardium. Am J Cardiol 1987; 60:1211-13.
18. Wijns W, Serruys PW, Slager CJ, Grimm J, Krayenbuehl HP, Hugenholtz PG, Hess OM. Effect of coronary occlusion during percutaneous transluminal angioplasty in humans on left ventricular chamber stiffness and regional diastolic pressure-radius relations. J Am Coll Cardiol 1986;7:455-63.
19. Jennings RB, Reimer KA. Factors involved in salvaging ischemic myocardium: effect of reperfusion of arterial blood. Circulation 1983;68:I-25-36.
20. Neill WA, Whorton Jr TP, Fluri-Lundeen J, Cohen JS. Acute coronary insufficiency-coronary occlusion after intermittent ischemia attacks. N Engl J Med 1980;302:1157-62.
21. HINT Research Group. Early treatment of unstable angina in the coronary care unit: a randomised, double-blind, placebo-controlled comparison of recurrent ischaemia in patients treated with nifedipine or metoprolol or both. Br Heart J 1986;56:400-13.
22. Ellis S, Alderman E, Cain K, Fisher L, Sanders W, Bourassa M, and the CASS investigators. Prediction of risk of anterior myocardial infarction by lesion severity and measurement method of stenoses in the left anterior descending coronary distribution: a CASS registry study. J Am Coll Cardiol 1988;11:908-16.
23. Neill WA. Staccato left anterior descending artery occlusion: a recognizable subset of unstable angina. J Lab Clin Med 1985;105:390-6.
24. Rahimtoola SH. Coronary bypass surgery for unstable angina. Circulation 1984;69:842-8.
25. Rahimtoola SH. Coronary bypass surgery for chronic angina—1981: a perspective. Circulation 1982;65:225-41.
26. Mock MB, Fisher LD, Holmes Jr DR, Gersh BJ, Schaff HV, McConney M, Rogers WJ, Kaiser GC, Ryan TJ, Meyers WO, Killip TH III, and participants in the CASS. Comparison of effects of medical and surgical therapy on survival in severe angina pectoris and two-vessel coronary artery disease with and without left ventricular dysfunction: a CASS registry study. Am J Cardiol 1988;61:1198-1203.
27. Rahimtoola SH, Nunley D, Grunkemeier G, Tepley J, Lambert L, Starr A. Ten-year survival after coronary bypass surgery for unstable angina. N Engl J Med 1983;308:676-81.
28. de Feyter PJ, Serruys PW, Suryapranata H, Beatt K, van den Brand M. Coronary angioplasty early after diagnosis of unstable angina. Am Heart J 1987;114:48-54.
29. Leeman DE, McCabe CH, Faxon DP, Lorell BH, Kellett MA, McKay RG, Varricchione T, Baim DS. Use of percutaneous transluminal coronary angioplasty and bypass surgery

despite improved medical therapy for unstable angina pectoris. Am J Cardiol 1988;61:38 G-44 G.
30. Gold HK, Johns JA, Leinbach RC, Yasuda T, Grossbard E, Zusman R, Collen D. A randomized, blinded, placebo-controlled trial of recombinant human tissue-type plasminogen activator in patients with unstable angina pectoris. Circulation 1987;75:1192-9.
31. Gotoh K, Minamino T, Katoh O, Hamano Y, Fukui S, Hori M, Kusuoka H, Mishima M, Inoue M, Kamada T. The role of intracoronary thrombus in unstable angina: angiographic assessment and thrombolytic therapy during ongoing anginal attacks. Circulation 1988;77:526-34.
32. de Zwaan C, Bär FW, Janssen JHA, de Swart HB, Vermeer F, Wellens HJJ. Effects of thrombolytic therapy in unstable angina: clinical and angiographic results. J Am Coll Cardiol 1988;12:301-9.

Pacemakers in children: An update

John D. Kugler, MD, and David A. Danford, MD. *Omaha, Nebr.*

Although the first attempt to pace the heart involved resuscitating a stillborn infant in 1929, pacemaker technology was static until the invention of the transistor 20 years later.[1-4] Permanent pacemakers in children were first reported in the early 1960s.[5-14] Steady advances in lead technology, programmability, and generator miniaturization were achieved through the late 1970s and 1980s.[15-40] Based simply on the increased number of adults who need pacemakers compared to children, pacemaker technology has focused on the older population. However, the pacemaker industry has discovered that advances for the pediatric patient (e.g., smaller generator size) also substantially benefit adults. Conversely, the technological improvements intended for adults benefit children who despite differing pathogenesis may have similar arrhythmia and conduction system disturbances. Pediatric electrophysiologists have aggressively applied the new technology, and this has provided the pediatric cardiologist with an abundance of new pacemaker data.[31-68] The purposes of this report are to review recent pacemaker technological advances, how they apply in infants, children, and adolescents, and to update the indications for permanent pacemaker implantation in children.

INDICATIONS FOR PACEMAKER IMPLANTATION

Pediatric pacemaker technology is developing so rapidly that major advances have occurred since the 1984 report "Guidelines for Permanent Cardiac Pacemaker Implantation"[69] was prepared by the Joint Task Force of the American College of Cardiology and The American Heart Association. The 11 member physician committee (which included a pediatric cardiologist) was to define the role of pacemaker implantation in the management of cardiovascular disease for adults and children. Based on current data and on standard practice, the committee classified indications for pacemaker implantation into three groups (Table I). Class I indications included diagnoses for which general agreement existed among all cardiologists for pacemaker implantation. Class II consisted of conditions for which a divergence of opinion existed whether or not pacemaker implantation was indicated. Class III included diagnoses for which general agreement prevailed that pacemaker implantation was unnecessary. The intent in this update is not to discuss each Task Force recommendation, but to highlight those recommendations that can be viewed differently in light of data available in 1989.[70-76] This involves some disorders that were not included in the 1984 classification that can now be considered appropriate for permanent pacemaker implantation.

Congenital complete atrioventricular (AV) block in asymptomatic patients. In 1987, Dewey et al.[77] reported a prospective ambulatory ECG (Holter) monitoring study in 27 patients with congenital complete AV block. Follow-up for a mean of 8 ± 3 years revealed that 8 of the 13 patients with a mean heart rate of less than 50 beats/min while awake developed cardiac complications of sudden death (three), syncope or presyncope (three), or excessive fatigue (two). These investigators also found that six of the eight symptomatic patients showed instability of the

From the Section of Cardiology, Department of Pediatrics, University of Nebraska Medical Center.

Received for publication Sept. 15, 1988; accepted Nov. 1, 1988.

Reprint requests: John D. Kugler, MD, Dept. of Pediatrics (Cardiology), University of Nebraska Medical Center, 42nd & Dewey Ave., Omaha, NE 68105.