11. Huang SK, Bharati S, Lev M, Marcus FI. Electrophysiologic and histologic observations of chronic atrioventricular block induced by closed-chest catheter desiccation with radiofrequency energy. PACE (In press).
12. Scheinman MM, Davis JC. Cathether ablation for treatment of tachyarrhythmias: Present role and potential promise. Circulation 1986;73:10.
13. Bardy GH, Reichenbach D, Greene HL, Thomas R, Breazeale DG, Ivey TD. Unipolar vs bipolar catheter shocks at the coronary sinus orifice (abstr). Circulation 1985;72(suppl III):III-390.
14. Hoyt RH, Huang SK, Marcus FI, Odell RC. Factors influencing trans-catheter radiofrequency ablation of the myocardium. J Appl Cardiol 1986;1:469.

Coronary angioplasty early after diagnosis of unstable angina

Coronary angioplasty (PTCA) was performed early after diagnosis of unstable angina in 71 patients who responded favorably with initial pharmacologic treatment and who also had persistent exertional angina. The patients selected for PTCA had predominantly single-vessel disease and a normal or slightly abnormal left ventricular function. PTCA was successful in 87% (62/71) of the patients and unsuccessful in 13% (9/71). There were no deaths related to PTCA. The incidence of myocardial infarction during the procedure was 10% (seven of the 71 patients). Urgent bypass surgery was necessary in 11% (eight of 71 patients) of the patients. All patients were followed up for 12 months. There was one late death and one late nonfatal myocardial infarction. During 12 months of follow-up there was recurrence of angina pectoris in 25% of the patients (14/62). The restenosis rate was 25% (13/52) in the patients with an initial successful PTCA who underwent repeat angiography. Improved cardiac functional status after sustained successful PTCA was demonstrated by the normal exercise capacity on bicycle exercise testing and the absence of ischemia on thallium 201 scintigraphy studies in 70% of the patients. At the 1-year follow-up visit after attempted coronary angioplasty in all 71 patients, the total incidence of deaths was 1.5% (one patient), myocardial infarction 11% (eight patients), and the need for revascularization 25% (emergency surgery eight patients, late surgery three patients, and repeat PTCA seven patients); 91% (64 of 70 patients) were symptom free. It is concluded that PTCA in selected patients with unstable angina initially stabilized with medical treatment is an effective treatment with an acceptable complication rate and an excellent 1-year prognosis. (Am Heart J 1987;114:48.)

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Unstable angina pectoris, a syndrome intermediate in severity between stable angina and acute myocardial infarction, is well recognized.[1,2] Although there is no universally accepted definition, unstable angina is often defined as angina occurring at rest and accompanied by reversible ECG changes. Its most striking features are the clinical instability[1,2] and rapid progression in extent and severity of coronary atherosclerosis.[3,4] The relative roles of initial medical and surgical therapy in unstable angina are well defined.[2,5-7] It is now accepted practice to begin treatment with medical therapy and to proceed to coronary artery bypass grafting or coronary angioplasty only in those patients refractory to medical treatment.[2,5,8-10] However, the use of angioplasty early after diagnosis in patients with unstable angina who have an initially favorable response to pharmacologic therapy is controversial. The merits of angioplasty in this setting are unclear because in this group of patients with unstable angina a medical program that includes aspirin antiplatelet therapy is associated with a low risk of subsequent nonfatal infarction or cardiac death.[11,12] The combined incidence of death and nonfatal myocardial infarction during a 12-week study period was 5%[11] and during a follow-up of up to 2 years (mean 18

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Received for publication Sept. 25, 1986; accepted Jan. 12, 1987.

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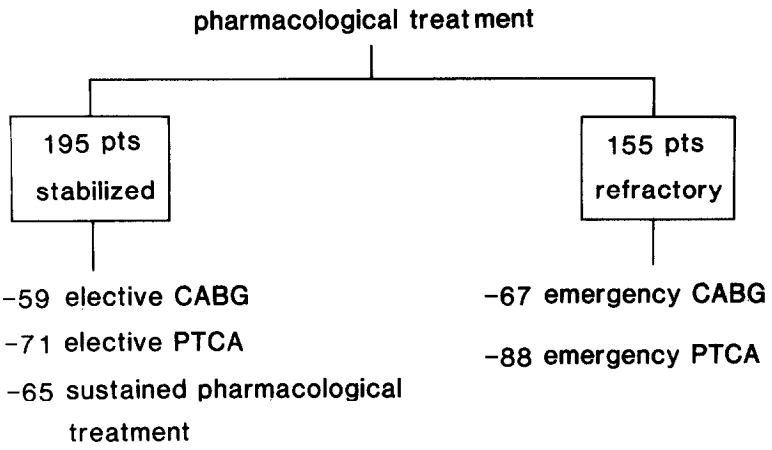

Fig. 1. Outcome of patients with unstable angina managed according to established protocol (Table I). *CABG* = Coronary artery bypass grafting; *PTCA* = coronary angioplasty.

months) the incidence was 8.6%.[12] However, the crossover rate to surgery was about 30%.[12] The present study reports our experience with coronary angioplasty early after diagnosis of unstable angina, initially stabilized with pharmacologic treatment. Angioplasty was performed before discharge from the hospital. The patients were followed up for 1 year after coronary angioplasty.

METHODS

During the period between January 1983 and June 1985, 2185 patients were admitted to our coronary care unit. Unstable angina pectoris was defined as chest pain at rest lasting for at least 15 minutes, associated with documented ST segment and T wave changes and no subsequent signs of necrosis as evidenced by a rise in cardiac enzymes less than twice normal and no Q wave development. Three hundred fifty patients had unstable angina and they were managed according to an established protocol (Table I).

Following the protocol (Fig. 1), a consecutive series of 71 patients satisfied all of the following criteria: (1) diagnosis of unstable angina pectoris, (2) stabilization in the acute phase with pharmacologic therapy but persisting exertional angina, (3) potentially suitable candidates for coronary bypass surgery, and (4) angiographically demonstrated coronary lesions suitable for coronary angioplasty. These seventy-one patients composed the study group. The angiograms were made in multiple views including oblique, cranial, and caudal projections. Patients were considered to have one-, two-, or three-vessel disease if they had >50% luminal narrowing in one, two, or three major coronary arteries (left anterior descending, right coronary, and circumflex arteries). The global left ventricular ejection fraction was calculated from the 30-degree right anterior oblique projection as the ratio between end-systolic and end-diastolic volume.

The baseline clinical and angiographic characteristics are presented in Tables II and III. The documented ST segment and T wave changes during pain at rest are shown in Table IV. Coronary angioplasty was performed with preformed guiding catheters, steerable dilating balloon catheters, and a pneumatic inflation device. A No. 7 French pacing electrode catheter (Zucker, USCI, Billerica, Mass.) was positioned in the right ventricle. On beginning the procedure, heparin, 100 mg, and acetylsalicylic acid, 250 mg, were administered intravenously and low molecular-weight dextran was infused slowly. ECG and blood pressure were monitored continuously. To prevent coronary spasm, nifedipine or isosorbide was given.[13] Initial balloon inflation pressure was 2.0 atmospheres, with subsequent inflations ranging to 12 atmospheres. Inflation was maintained according to ECG changes, degree of blood pressure drop, or induced pain and was in no instance longer than 60 seconds. The number of balloon inflations varied depending on the appearance of the lesion as seen during immediate postdilatation angiogra-

Table I. Management of patients with unstable angina pectoris

Bed rest and sedation (coronary care unit)
Treatment of precipitating factors (hypertension, anemia, and tachycardia)
Stepwise intensification of pharmacologic regimen with individual tailoring to achieve a heart rate <60 bpm and a systolic blood pressure <110 mm Hg; medication included a combination of nitrates, β-blockers, and Ca antagonists
Anticoagulants (heparin) or antiplatelet (aspirin) treatment
Coronary angiography in all patients
Emergency coronary angioplasty or emergency surgery in refractory unstable angina
Elective coronary angioplasty or surgery in patients with stabilized unstable angina if there are persisting exertional angina detected either clinically or by exercise testing and suitable lesion(s) for angioplasty or surgery

Table II. Clinical characteristics of 71 patients with UAP initially "stabilized"

Men (%)	82
Mean age (range) (yr)	57 (36-74)
Prior MI (%)	24
Prior CABG (%)	3
History of UAP (%)	—
Recent onset of AP at rest (within 3 months)	49 —
Worsening of preexisting AP	45
post MI (within 4 weeks)	6

UAP = unstable angina pectoris; MI = myocardial infarction; CABG = coronary artery bypass grafting; AP = angina pectoris.

Table III. Angiographic characteristics of 71 patients with UAP initially "stabilized"

One-vessel disease (%)	68
Two-vessel disease (%)	25
Three-vessel disease (%)	6
Left main stem disease (%)	1
Ejection fraction (% of patients)	
Mean (±SD)	0.59 ± 10
>0.50	79
0.30-0.50	21
<0.30	0

UAP = unstable angina pectoris.

Table IV. Documented ST-T changes during chest pain at rest in patients with stabilized angina pectoris

	mV	%
Transient ST elevation	≥0.1	13
Transient ST depression	≥0.1	13
Persisting negative T wave	≥0.1	35
Transient minor ST-T changes:		39
ST depression or elevation	<0.1	
Pseudonormalization of T wave		
T wave changes	<0.1	

Table V. Clinical follow-up after attempted coronary angioplasty in 71 patients with unstable angina, stabilized after pharmacologic therapy

	12-Mo follow-up				
	Death	*Non-fatal MI*	*CABG*	*Re-PTCA*	*Asympto-matic*
PTCA successful (62 patients)	1	1	3	7	57
PTCA unsuccessful (9 patients)	0	7	8*	0	7

PTCA = percutaneous transluminal coronary angioplasty; MI = myocardial infarction; CABG = coronary artery bypass grafting.
*Includes six patients who developed MI.

phy and on the residual transstenotic pressure gradient. Coronary angioplasty was considered successful after a reduction of the severity of the obstruction to <50% luminal-diameter narrowing and abolition of acute ischemic symptoms and no progression to myocardial infarction or death. A decrease of the transstenotic gradient index to <0.30[14] (mean proximal pressure minus mean distal pressure divided by mean aortic pressure) was used as a guide, but not as a sole criterion of success. In patients with multivessel disease only the ischemia related vessel was dilated.[15]

After the procedure all patients were monitored for 24 hours in the coronary care unit where ECGs and enzyme levels were measured. They were discharged after 2 to 3 days. They were treated with nifedipine, 40 to 60 mg daily, and acetylsalicylic acid, 500 mg daily, during a period of 6 months. All procedures were carried out with cardiac surgical team on standby. A perioperative myocardial infarction was diagnosed if new pathologic Q waves developed. Clinical follow-up information was obtained by either personal interview or information obtained from the referring physician. Patients were evaluated for the occurrence of myocardial infarction and recurrence of angina pectoris. The majority of the patients underwent ECG exercise testing with thallium 201 scintigraphy and repeat angiography. Patients performed symptom-limited exercise on the bicycle ergometer with stepwise increments of 10 W/min. The three orthogonal leads XYZ of the Frank lead system were recorded. An ischemic response was defined as at least a 0.1 mV ST segment depression, 0.08 second after the J point. The maximum work load achieved was expressed as a percentage of the normal work load predicted for age, sex, and body length. Thallium exertional scintigraphic imaging was performed in the anterior and left anterior oblique 45- and 65-degree views, immediately after injection of 1.5 mCi thallium 201 at peak stress. The postexercise images were obtained 4 hours later. Images were obtained with a Searle Phogamma V camera (Searle Radiographics, Des Plaines, Ill.) and processed with computer interface as described previously.[16] Defects with redistribution were considered to represent exercise-induced ischemia. Persistent defects without redistribution were considered to represent scars.

Repeat angiograms were obtained in multiple views (including hemiaxial views for the left coronary artery) and interpreted visually without knowledge of the patient's clinical status. The estimated percent of obstruction was derived from the angiographic view showing the greatest reduction in diameter for the vessel in question. Restenosis was defined as an increase of the luminal diameter stenosis of the dilated lesion >50%.

Results are presented as mean ± SD. Comparisons were made with the χ^2 test.

RESULTS

The coronary angioplasty procedure was successful in 87% (62) of the 71 patients. The success rate for the left anterior descending artery was 89% (40

Table VI. Exercise ECG testing and thallium scintigraphy after successful PTCA in 44 symptom-free patients

Extent of CAD	*No. of patients*	*Exercise capacity (% achieved of predicted value)*	*ST-segment depression ≥1 mm (%)*	*Thallium-reversible perfusion defect (%)*
One-vessel disease	31	99.5 ± 16	4/31 (13)	5/29 (17)
Multivessel disease	13	96 ± 16	5/13 (38)*	7/13 (53)†
Total	44	98.5 ± 16	9/44 (20)	12/42 (30)

CAD = Coronary artery disease.
*Not significant; †$p < 0.02$ (one vessel vs multivessel).

of 45), for the right coronary artery 72% (eight of 11), for the left circumflex artery 92% (12 of 13), for the left main stem 100% (one of one), and for the bypass 100% (one of one). The inflation pressure per procedure was a mean of 9 ± 1.9 atmospheres (range 6 to 12). The number of inflations per procedure was a mean of 3.8 ± 1.3; the average inflation duration was a mean 48 ± 12 seconds. The transstenotic pressure gradient measured was of adequate quality in 55 of the 62 patients who underwent a successful angioplasty. The gradient dropped from 0.63 ± 0.17 before angioplasty to 0.17 ± 0.10 after angioplasty. The procedure was unsuccessful in 13% (9/71) of the patients. In three patients the procedure was complicated by total occlusion of the vessel while attempting to cross the lesion, two had dissection with poor runoff, in three patients the dilatation was incomplete resulting in chest pain and ST-segment elevation shortly after the procedure, and in one patient the guide wire, embedded in a side branch, broke and had to be removed by the surgeon. Eight of these patients underwent immediate coronary artery bypass grafting, but six patients sustained a myocardial infarction. Another patient without surgery developed a myocardial infarction. Thus the procedure-related incidence of myocardial infarction was 10% (7/71 patients). There were no deaths related to the procedure.

All patients were followed up for 12 months after successful coronary angioplasty (Table V). There was one late death, and one patient progressed to late myocardial infarction. Fourteen patients had recurrence of angina pectoris, the majority within 6 months after coronary angioplasty. Two of these demonstrated progression of disease in a nondilated segment. Seven patients underwent a successful repeat angioplasty procedure. The patients who underwent coronary artery bypass grafting after unsuccessful coronary angioplasty were followed up for 12 months; one had recurrence of angina pectoris, adequately controlled with medical therapy. Thus at 1-year follow-up after attempted coronary angioplasty (inclusive emergency and late elective surgery and reangioplasty) the total incidence of death was 1.5% (1/71) and of myocardial infarction 11% (8/71); 91% (64/70) were symptom free. A total of 11 patients (16%) underwent surgery (emergency surgery and late elective surgery).

The exercise test and thallium scintigraphy data are presented for 44 patients with successful coronary angioplasties. They were symptom free at the time of the test (Table VI). Stress tests were performed 2.1 ± 1.6 months after angioplasty. Although symptom free, 20% of the patients showed an exercise-induced ischemic ST depression and 30% a reversible perfusion defect. ST-segment depression or a reversible perfusion defect was more frequent in patients with multivessel disease and in whom only the ischemia-related vessel was dilated than in those with single-vessel disease. The difference in the perfusion defects was statistically significant.

Repeat angiograms were available in 84% of the patients (52/62). In the 10 patients who refused repeat angiography, one had recurrent angina controlled by medical treatment and 9 were symptom free. The angiograms were performed 3.4 ± 3.2 months after coronary angioplasty. Restenosis occurred in 13 patients (25%), of whom one was symptom free and 12 had recurrent angina pectoris. All patients without restenosis were symptom free.

DISCUSSION

By definition, unstable angina is an unstable state leading to either improvement or culmination into myocardial infarction or death.[1,2] Treatment is directed toward relief of ischemic symptoms and prevention of progression to myocardial infarction or death.[2,5-7] The exact pathophysiologic mechanism is unknown. Increased coronary artery tone, frank coronary artery spasm, plaque fissuring with platelet aggregation and formation of a mural thrombus, or even an occlusive thrombus may play a role.[8,17-21] However, in nearly all patients one can find a high-grade coronary atherosclerotic lesion.[22,23] It is

most likely that dynamic and fixed coronary stenoses act in combination.[8] Dynamic stenosis at a fixed obstruction causes a critical intermittent reduction in flow that leads to reversible ischemia. Standard treatment consists of restriction of activity and administration of β-blockers and nitrates. Recently the addition of Ca antagonists, a vasodilator designed to increase oxygen supply and decrease O_2 demand, has been shown to be effective also.[8-10, 24] Modern pharmacologic treatment seems to control pain but not death and morbidity.[25] However, administration of aspirin has been shown to have a beneficial effect on the incidence of cardiac death and myocardial infarction.[10, 11] Nevertheless, patients remain at risk for progression to myocardial infarction or cardiac death, and the need for coronary bypass surgery is not reduced.[8-10, 24, 25] It was recently shown that unstable angina is often associated with increased progression of coronary artery disease.[3, 4] The coronary artery obstruction is at risk of becoming a permanent occlusion leading to myocardial infarction or death. Therefore coronary angioplasty, designed to reduce the severity of the obstruction,[26] seems a logical step in an attempt to improve morbidity and mortality rates. It has been shown that coronary angioplasty, combined with pharmacologic therapy, can be performed safely and successfully in selected patients with refractory unstable angina.[27-29] In our study the success rate of angioplasty early after the diagnosis of unstable angina with a favorable response to pharmacologic treatment was 87%, which is comparable to the success rates currently reported in patients with stable angina.[30, 31] No procedure-related death occurred in our study. However, the incidence of myocardial infarction during the procedure was 10%, definitely higher than that reported in stable angina.[30, 31]

The high frequency of myocardial infarction during the procedure is probably related to the high frequency of "complicated" lesions in patients with unstable angina.[32] Intracoronary instrumentation may more readily induce total occlusion resulting in myocardial infarction. After a successful procedure, during a follow-up of 12 months the disease had further progressed to late death in 1.0% and late nonfatal myocardial infarction in 1.0% of the patients. Thus at 12 months the total incidence of mortality was 1.5% and of myocardial infarction 11%. These results are comparable to those reported by the National Heart, Lung and Blood Institute Percutaneous Transluminal Coronary Angioplasty and Coronary Artery Surgery Study Registry.[33] The latter study does not contain detailed information about persistence of angina after diagnosis of unstable angina; however, the patient population described seemed comparable to ours. In their study the incidence of in-hospital mortality was 0.9%, the 18-month mortality 2.6%, and the 18-month combined death/myocardial infarction rate 10.8%. In both studies the majority of the events occurred during initial hospitalization and were related to the angioplasty procedure, which in these patients carries an increased risk. It is to be noted that the patients in our study and those in the National Heart, Lung and Blood Institute Percutaneous Transluminal Coronary Angioplasty and Coronary Artery Surgery Registry study do not represent a "normal" population with unstable angina. These patients are a selected group and consist of patients with predominantly one-vessel disease and preserved left ventricular function. Furthermore, in our study we included, in addition to patients with transient ST segment and T wave changes, patients with persistent T wave inversion and chest pain at rest, a subgroup known to have a poor prognosis.[34, 35]

The results of the ECG exercise testing and thallium scintigraphy performed approximately 2 months after the procedure indicate good functional recovery after successful coronary angioplasty, although about 30% of the symptom-free patients had either ST-segment depression or a reversible perfusion defect. This objective evidence of ischemia in otherwise symptom-free patients is partly the result of untreated disease in patients with multivessel disease and only dilatation of the ischemia-related vessel and partly the result of early stenosis. It has been shown that thallium scintigraphy performed within 2 months after coronary angioplasty can predict a later stenosis.[36] At this stage the severity of this early stenosis is limited and does not result in symptoms but may induce homogeneous myocardial perfusion, which can be detected by thallium scintigraphy.

After a successful procedure the prognosis with respect to death and late progression to myocardial infarction is excellent. The recurrence rate of angina pectoris during the 12-month follow-up after successful angioplasty was 23% (14/62). This recurrence rate is in agreement with the rates reported in patients with stable angina.[31] Repeat angiography showed restenosis in 25% of the patients with an initially successful angioplasty. This restenosis rate is comparable to the restenosis rate in patients with stable angina.[37] The majority of these patients underwent a successful repeat angioplasty or surgery. At 1-year follow-up after attempted coronary

angioplasty of all patients (inclusive emergency surgery, elective surgery, and repeat angioplasty), 92% were symptom free. Thus, with acceptable risk, coronary angioplasty appears to improve coronary flow, with long-lasting symptomatic improvement in the majority of patients. However, for coronary angioplasty to be accepted as an alternative form of therapy, it should effectively relieve angina with mortality and morbidity rates at least equal to those of other therapies for this syndrome. Currently, in the setting of severe coronary artery disease or continued disabling angina, coronary artery bypass grafting has been the treatment of choice. In a recent observational study, the acute and long-term results of coronary angioplasty and coronary artery bypass grafting in patients with unstable angina and one-vessel disease were compared.[33] The two patient groups with similar baseline characteristics were recruited, one from the National Heart, Lung and Blood Institute Percutaneous Transluminal Coronary Angioplasty and Coronary Artery Surgery Study Registry and one from the Coronary Artery Surgery Study Registry. Comparison of the two groups showed that coronary angioplasty compared favorably with surgery. Our study does not compare the results of coronary angioplasty with modern pharmacologic therapy or surgery in patients with unstable angina but supports the suggestion that coronary angioplasty is an effective alternate form of therapy in these patients. However, the results were obtained in a highly selected group of patients with unstable angina. They had predominantly single-vessel disease and a normal or slightly abnormal left ventricular function. The results of coronary angioplasty in patients with unstable angina and predominantly multivessel disease and severely compromised left ventricular function remain unanswered.

We are indebted to Gusta Koster and Anja van Huuksloot for assistance in the preparation of the manuscript.

REFERENCES

1. Cairns JA, Fantus IG, Klassen GA. Unstable angina pectoris. Am Heart J 1976;92:373.
2. Scanlon PJ. The intermediate coronary syndrome. Prog Cardiovasc Dis 1981;23:351.
3. Neill WA, Wharton TP, Fluri-Lundeen J, Cohen JS. Acute coronary insufficiency—coronary occlusion after intermittent ischemic attacks. N Engl J Med 1980;302:1157.
4. Moise A, Theroux P, Taeymans Y, Descoings B, Lesperance J, Waters DD, Pelletier GB, Bourassa MG. Unstable angina and progression of coronary atherosclerosis. N Engl J Med 1983;309:685.
5. Plotnick GD. Approach to the management of unstable angina. Am Heart J 1979;98:243.
6. Russell RO, Morashi RE, Kouchoukos N, et al. Unstable angina pectoris: national cooperative study group to compare surgical and medical therapy. Am J Cardiol 1978;42:839.
7. Michels R, Hugenholtz PG, Haalebos M, van den Brand M, Serruys PW, Balakumaran K. Management of unstable angina pectoris. In: Adelman AG, Goldman BS, eds. Unstable angina—recognition and management. Littleton, MA: PSG Publishing Co Inc, 1981:143.
8. Hugenholtz PG, Michels HR, Serruys PW, Brower RW. Nifedipine in the treatment of unstable angina, coronary spasm and myocardial ischemia. Am J Cardiol 1981;47:163.
9. Moses JW, Wertheimer JH, Bodenheimer MM, Banka VS, Feldman M, Helfort RH. Efficacy of nifedipine in rest angina refractory to propranolol and nitrates in patients with obstructive coronary artery disease. Ann Intern Med 1981; 94:425.
10. Gerstenblith G, Ouyang B, Achuff SC, Bulkley BH, Becker LC, Mellits ED, et al. Nifedipine in unstable angina: a double-blind, randomized trial. N Engl J Med 1982;306:885.
11. Lewis HD, Davis JW, Archibald DG, et al. Protective effects of aspirin against acute myocardial infarction and death in men with unstable angina. N Engl J Med 1983;309:396.
12. Cairns JA, Gent M, Singer J, et al. Aspirin, sulfinpyrazone or both, in unstable angina: results of a Canadian multicenter trial. N Engl J Med 1985;313:1369.
13. Serruys PW, van den Brand M, Brower RW, Hugenholtz PG. Regional cardioplegia and cardioprotection during transluminal angioplasty, which role for nifedipine? Eur Heart J 1983;4(suppl):115.
14. Wijns W, Serruys PW, Reiber JHC, van den Brand M, Simoons ML, Kooyman C, Balakumaran K, Hugenholtz PG. Quantitative angiography of the left anterior descending coronary artery: correlation with pressure gradient and exercise thallium scintigraphy. Circulation 1985;71: 273.
15. Feyter de PJ, Serruys PW, Arnold A, Simoons L, Wijns W, Geuskens R, Soward A, van den Brand M, Hugenholtz PG. Coronary angioplasty of the unstable angina related vessel in patients with multivessel disease. Eur Heart J 1986;7:460.
16. Reiber JHC, Lie SP, Simoons ML, Wijns W, Gerbrands JJ. Computer quantification of location, extent and type of thallium 201 myocardial perfusion abnormalities. IEEE Comp Cardiol 1982; p 123.
17. Epstein SE, Talbot TL. Dynamic coronary tone in precipitation, exacerbation and relief of angina pectoris. Am J Cardiol 1981;48:797.
18. Maseri A, L'Abbate A, Baroldi G, Chierchia S, Marzilli M, Ballestra AM, Severu S, Parodi O, Biagini A, Distante A, Pesola A. Coronary vasospasm as a possible cause of myocardial infarction: a conclusion derived from the study of "preinfarction" angina. N Engl J Med 1978;299:1271.
19. Mandelkorn JB, Wolf NM, Singh S, Schechter JA, Kersh RI, Rodgers DM, Workman MB, Bentivolglio LG, La Porte SM, Meister SG. Intracoronary thrombus in nontransmural myocardial infarction and unstable angina pectoris. Am J Cardiol 1983;52:1.
20. Davies MJ, Thomas DC. Plaque fissuring—the cause of acute myocardial infarction, sudden ischemic death and crescendo angina. Br Heart J 1985;53:363.
21. Falk E. Unstable angina with fatal outcome: dynamic coronary thrombosis leading to infarction and or sudden death. Circulation 1985;71:699.
22. Mc Mahon NM, Brown BG, Calungnan R, Rolett EL, Bolson E, Frimer M, Dodge HT. Quantitative coronary angiography: measurements of the "critical" stenosis in patients with unstable angina and single-vessel disease without collaterals. Circulation 1979;60:106.
23. Rafflenbeul W, Smith LR, Rogers WJ, Mantle JA, Rackley CE, Russel RO. Quantitative coronary arteriography: coronary anatomy of patients with unstable angina pectoris reexamined one year after optimal medical therapy. Am J Cardiol 1979;43:699.
24. Blaustein AS, Heller GV, Kolman BS. Adjunctive nifedipine therapy in high-risk, medically refractory, unstable angina pectoris. Am J Cardiol 1983;52:950.

25. Rahimtoola SH. Coronary bypass surgery for unstable angina. Circulation 1984;69:842.
26. Gruntzig AR, Senning A, Siegenthaler WE. Nonoperative dilatation of coronary artery stenosis: percutaneous transluminal coronary angioplasty. N Engl J Med 1979;301:61.
27. Williams DO, Riley RS, Singh AK, Gewirtz H, Most AS. Evaluation of the role of coronary angioplasty in patients with unstable angina pectoris. Am Heart J 1981;102:1.
28. Meyer J, Schmitz H, Kiesslich T, Erbel R, Krebs W, Schulz W, Bardos P, Minale C, Messmer BJ, Effert S. Percutaneous transluminal coronary angioplasty in patients with stable and unstable angina pectoris: analysis of early and late results. Am Heart J 1983;106:973.
29. de Feyter PJ, Serruys PW, van den Brand M, et al. Emergency coronary angioplasty in refractory unstable angina. N Engl J Med 1985;313:342.
30. Bredlau CE, Roubin GS, Leimgruber PP, Douglas JS, King SB, Gruentzig AR. In-hospital morbidity and mortality in patients undergoing elective coronary angioplasty. Circulation 1985;72:1042.
31. Block PC. Percutaneous transluminal coronary angioplasty: role in the treatment of coronary artery disease. Circulation 1985;72(suppl):V-161.
32. Ambrose JA, Winters SL, Stern A, Eng A, Teichholtz LE, Gorlin R, Fuster A. Angiographic morphology and the pathogenesis of unstable angina pectoris. J Am Coll Cardiol 1983; 5:609.
33. Faxon DP, Detre KM, McGabe CH, Fisher L, Holmes DR, Cowley J, Bourassa MG, van Raden M, Ryan TJ. Role of percutaneous coronary angioplasty in the treatment of unstable angina: report from the National Heart, Lung and Blood Institute Percutaneous Transluminal Coronary Angioplasty and Coronary Artery Surgery Study Registries. Am J Cardiol 1983;53:131C.
34. 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.
35. Haines DE, Raabe DS, Gundel WD, Wackers FJ. Anatomic and prognostic significance of new T-wave inversion in unstable angina. Am J Cardiol 1983;52:14.
36. Wijns W, Serruys PW, Reiber JHC, Feyter de PJ, van den Brand M, Simoons ML, Hugenholtz PG. Early detection of restenosis after successful percutaneous transluminal coronary angioplasty by exercise-redistribution thallium scintigraphy. Am J Cardiol 1985;55:357.
37. Leimgruber PP, Roubin GS, Hollman J, et al. Restenosis after successful coronary angioplasty in patients with single-vessel disease. Circulation 1986;73:710.

Metabolic support during coronary reperfusion

The limitation of infarct size by thrombolysis could potentially be improved by an early metabolic intervention. We therefore evaluated the effects of a 48-hour infusion of glucose-insulin-potassium (GIK) in patients with anterior infarctions. Seventeen patients were randomized to receive intravenous GIK (n = 10) or placebo (n = 7). All patients additionally received streptokinase. Changes in left ventricular function were assessed by comparing the global ejection fractions and the regional infarct area ejection fractions of the first ventriculogram with the 10-day second ventriculogram. There was a significantly greater improvement in the global ejection fraction of patients receiving GIK (↑0.07 ± 0.04) than in those randomized to placebo (↓0.08 ± 0.04) ($p < 0.02$). There was also a much greater improvement in the area ejection fractions of the group receiving GIK vs the group receiving placebo in the anterolateral (↑0.24 ± 0.07 vs ↓0.02 ± 0.04 [$p < 0.02$]) and diaphragmatic (↑0.08 ± 0.08 vs ↓0.17 ± 0.05 [$p < 0.005$]) segments. Thus in patients with anterior infarctions receiving streptokinase, GIK improves ventricular function and reduces the size of the segmental wall motion abnormality. (Am Heart J 1987;114:54.)

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The potential beneficial effects of thrombolytic therapy have been under intense investigation. Left ventricular global and regional function has not consistently demonstrated improvement in multiple randomized trials.[1] Therefore, reperfusion cannot simply be equated with myocardial salvage. This is probably secondary to the multiple subcellular derangements that result in adenosine triphosphate depletion, a rapid influx of calcium, and the loss of integrity of cellular membranes secondary to free

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Received for publication Nov. 21, 1986; accepted Jan. 2, 1987.

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