MEDICAL GRAND ROUNDS

THE UNIVERSITY OF TEXAS HEALTH SCIENCE CENTER

DALLAS, TEXAS

JUNE 21, 1984

PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

L. David Hillis, M.D.

OUTLINE

R.D. is a 36 year old man who was admitted to Parkland Hospital on 2/26/84 with an acute inferolateral myocardial infarction. Subsequently, he had numerous episodes of post-infarction chest pain, which was finally controlled with verapamil, 480 mg/day, and transderm nitroglycerin, 10 mg/day. Because of his post-infarction pain, he was referred for cardiac catheterization 10 days after infarction.

Physical examination revealed a thin, healthy-appearing man in no distress. BP was 110/70, pulse 64/minute and regular. An S4 gallop was audible, but there was no S3 or murmurs. The remainder of his examination was unremarkable. His chest xray and routine blood work were within normal limits, and his ECG revealed evidence of an evolving inferolateral infarction.

At cardiac catheterization, the patient had a cardiac output of 4.7 liters/minute; his intracardiac pressures were normal; and his LV ejection fraction was 0.69. Selective coronary arteriography revealed a 95% stenosis of the proximal portion of the first large circumflex marginal coronary artery. The coronary arteries were otherwise widely patent.

The patient continued to have chest pain at rest and with minimal exertion, and, therefore, he was returned to the catheterization laboratory 4 days later for attempted percutaneous transluminal coronary angioplasty. Angioplasty was successful in reducing the patient's circumflex marginal stenosis from a 95% luminal diameter narrowing to approximately a 30% narrowing. Two days later, an exercise tolerance test was performed and was negative, and the patient was discharged on aspirin, dipyridamole, and verapamil. He has continued to be angina-free.

In 1964, Dotter and Judkins (1) introduced the technique of transluminal angioplasty for the treatment of atherosclerotic obstruction of the femoral artery. Subsequently, although their technique was largely ignored in the United States, it was used extensively by angiographers in Europe. In the late 1970s, Gruentzig and his associates (2) modified the dilatation catheter to allow its use in patients with severe stenoses of the renal arteries (3) and the coronary arteries (2). Over the past 5 years, percutaneous transluminal coronary angioplasty (abbreviated PTCA) has been widely used throughout the world in individuals with atherosclerotic coronary artery disease, so that presently it is performed on a routine basis in hundreds of hospitals by an ever-growing number of angiographers. My purpose today is to provide you with a broad overview of PTCA and to attempt to familiarize you-- the practicing internist or the non-cardiologic medical subspecialist-- with what you can realistically expect when one of your patients undergoes this procedure.

I. PATIENT SELECTION

The ideal candidate for PTCA should fulfill several criteria. (a)He or she should be a reasonable candidate for coronary artery bypass surgery. Thus, the patient should have continuing angina despite adequate medical therapy, and he or she should not have an associated disease (such as severe pulmonary disease or a bleeding diathesis) which renders intrathoracic surgery prohibitive. (b)The patient should demonstrate objective evidence of reversible myocardial ischemia (by exercise tolerance testing with or without radionuclide imaging). (c)The patient should have arteriographic evidence of single vessel (or, at most, double vessel) coronary artery disease, and the stenosis should be proximal, discrete, subtotal, relatively concentric (as opposed to eccentric), and noncalcified. Finally, (d)the patient should not have a hemodynamically significant stenosis of the left main coronary artery (4).

A number of studies have attempted to assess the approximate percentage of patients undergoing coronary arteriography who satisfy all the above criteria. Although Gruentzig et al (2) estimated that the procedure would be applicable to 10-15% of patients having coronary artery bypass surgery, subsequent retrospective analyses have demonstrated that the percentage of patients who fulfill all the above criteria ranges from 2.1% to 8.0%, with an average of only 3.5% (Table 1).

TABLE 1

% OF PATIENTS WHO ARE IDEAL CANDIDATES FOR PTCA

AUTHORS	# PTS CATHETERIZED	# PTS IDEAL FOR PTCA	%
Hamby et al (5)	500	40	8.0
Hlatky et al (6)	5126	110	2.1
Holmes et al (7)	21,478	796	3.7
TOTALS	27,104	946	3.5

In fact, in our own experience here at Parkland, patients who fulfill all the criteria and are, therefore, ideal candidates comprise only 2.4% of those who are found at catheterization to have coronary artery disease.

Hamby et al (5) suggested that certain clinical characteriestics may help identify individuals with angina who are especially likely to be good candidates for PTCA. In their review of 500 consecutively studied patients with arteriographic evidence of coronary artery disease, the 40 who met all criteria for PTCA noted angina for an average of 2.0

years, whereas the 460 with coronary artery disease who were not good candidates for angioplasty had angina for 4.1 years. There was a clear relationship between the duration of angina and the likelihood that the patient would have a coronary stenosis suitable for PTCA (Figure 1), a finding also noted by Holmes et al (7). Of the 631 patients reported from the voluntary National Heart, Lung,

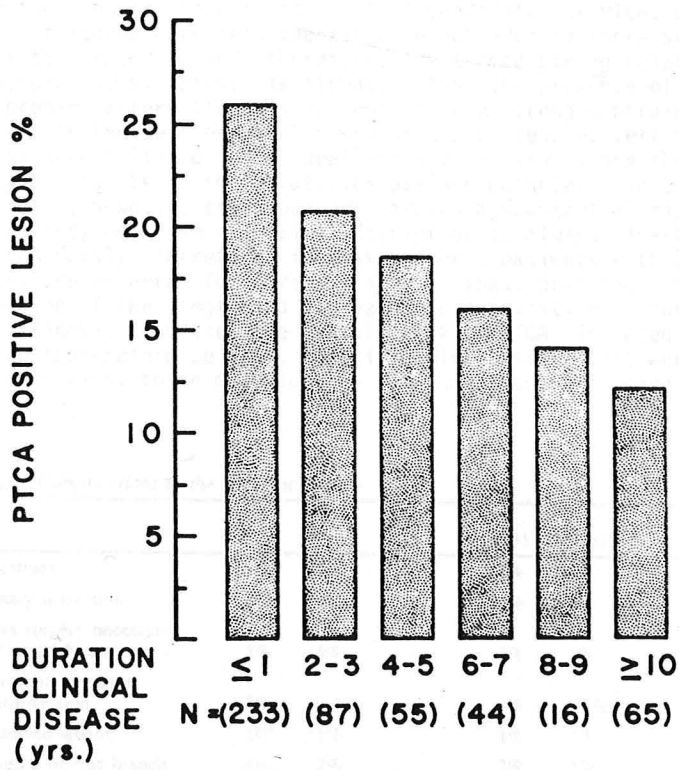


Figure 1 The frequency of stenoses ideal for PTCA, expressed as a % of patients catheterized, in relation to the duration of clinical disease (in years). Note that the suitability for PTCA gradually declines as the duration of symptoms increases (taken from reference # 5).

and Blood Institute PTCA Registry, 46% had noted angina for less than 6 months before PTCA, and another 23% had noted it for less than 1 year (4). Furthermore, Hamby et al (5) noted that only 15% of good PTCA candidates had sustained a previous myocardial infarction, whereas 43% of those not suitable for angioplasty had suffered prior infarction.

In addition to the clinical and arteriographic criteria already listed that render the patient an ideal candidate for PTCA, several anatomic features have been identified which greatly increase the risk of the procedure and, therefore, are termed strong relative contraindications. First, as already noted, the presence of a left main coronary artery stenosis presents a very strong contraindication to PTCA. In the developmental phase of the procedure, left main stenoses were believed to be ideal for angioplasty, since they were so readily accessible to the dilatation balloon catheter. In these individuals, however, the procedure carries a substantial risk, since catheter-induced spasm or intimal disruption is always disastrous and often fatal. Therefore, in most centers, patients with left main stenoses are referred for coronary artery bypass grafting. Second, a combination of the length and degree of eccentricity of a coronary artery stenosis increases the overall risk of PTCA, as noted in Table 2 (taken from reference # 8). Specifically, lesions that were noted by arteriography to be both long (> 5 mm) and eccentric were associated

TABLE 2 *Complications in the Different Groups*

	L (153)	S (265)	E (155)	C (338)	L+E (51)	S+C (177)
Complications	**16%**	**14%**	**21%**	**14%**	**24%**	* **12%**
Coronary dissection	5%	7%	9%	6%	8%	6%
Bypass surgery necessary within 24 hours	5%	5%	8%	4%	10%	3%
Arrhythmia requiring cardioversion	2%	0.5%	1%	0.5%	2%	—
Unresolved spasm	1%	1%	1%	2%	2%	1%
Occlusion of side branch	4%	2%	2%	2%	6%	2%
Myocardial infarct by ECG	2%	1%	0.5%	2%	2%	2%
Blood loss requiring transfusion	0.5%	0.5%	0.5%	0.5%	—	—
Other complications	5%	3%	4%	4%	6%	4%
Deaths	—	—	—	—	—	—

Numbers in parentheses represent number of patients.
*$p < 0.05$.
Abbreviations: L = long; S = short; E = eccentric; C = concentric.

with complications in 24% of patients. In contrast, stenoses that were short (< 4 mm) and concentric were associated with complications in only 12% (Table 2). Third, patients in whom a branch of an involved coronary artery originates from within the atherosclerotic plaque are at risk of iatrogenic occlusion of the branch during attempted angioplasty of the main vessel, whereas branches whose origins are adjacent to the atherosclerotic plaque (but not involved in it) are at considerably lower risk of iatrogenic occlusion. In a review of their experience with side branches, Meier et al (9) noted that 17 of 122 (14%) side branches of the "Group I" type were iatrogenically occluded during angioplasty of the main lesion; in contrast, only 3 of 243 (1%) side branches of the "Group II" type were occluded by PTCA ($p < 0.001$, Figure 2).

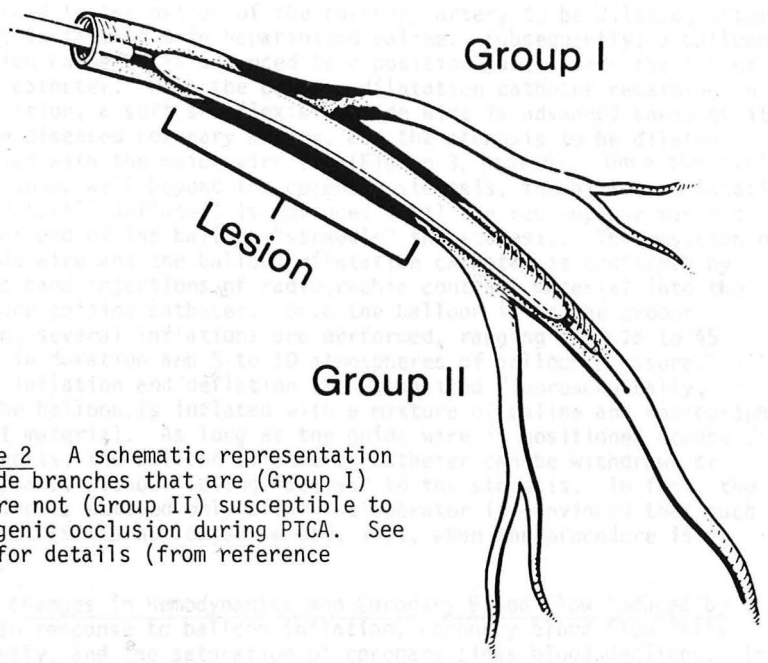


Figure 2 A schematic representation of side branches that are (Group I) and are not (Group II) susceptible to iatrogenic occlusion during PTCA. See text for details (from reference # 9).

In short, PTCA as it is practiced today appears to be a reasonable alternative to coronary artery bypass surgery in a relatively small percentage of patients. Although it could, no doubt, be attempted in many individuals who do not meet the listed criteria of an ideal candidate, such indiscriminant use of the procedure would probably be associated, first, with its performance in some patients in whom it is not warranted and, second, with a substantial risk of failure or complications.

II. PERFORMANCE OF THE PROCEDURE

A. Technical Issues In the patient in whom PTCA is attempted, a large-bore guiding catheter (9.2 French, 3.15 mm outer diameter) is advanced to the ostium of the coronary artery to be dilated, after which it is flushed with heparinized saline. Subsequently, a balloon dilatation catheter is advanced to a position just inside the tip of the guiding catheter. With the balloon dilatation catheter remaining in this position, a soft and flexible guide wire is advanced ahead of it into the diseased coronary artery, and the stenosis to be dilated is crossed with the guide wire (10)(Figure 3, page 9). Once the guide wire is positioned well beyond the coronary stenosis, the balloon dilatation catheter (still deflated) is advanced until the radioopaque markers at either end of the balloon "straddle" the stenosis. The position of the guide wire and the balloon dilatation catheter is confirmed by periodic hand injections of radiographic contrast material into the large-bore guiding catheter. Once the balloon is in the proper position, several inflations are performed, ranging from 15 to 45 seconds in duration and 5 to 10 atmospheres of balloon pressure. Balloon inflation and deflation are visualized fluoroscopically, since the balloon is inflated with a mixture of saline and radiographic contrast material. As long as the guide wire is positioned across the stenosis, the balloon dilatation catheter can be withdrawn or even replaced without losing "access" to the stenosis. In fact, the guide wire is removed only after the operator is convinced that such ready "access" is no longer needed, i.e., when the procedure is completed.

B. Changes in Hemodynamics and Coronary Blood Flow Induced by PTCA In response to balloon inflation, coronary blood flow falls transiently, and the saturation of coronary sinus blood declines. In the 30 seconds after balloon deflation, a reactive hyperemic response is noted, in that coronary blood flow increases, and the saturation of coronary sinus blood rises (Figure 4, page 10)(11). During the 15-45 seconds of balloon inflation, systemic arterial pressure and left

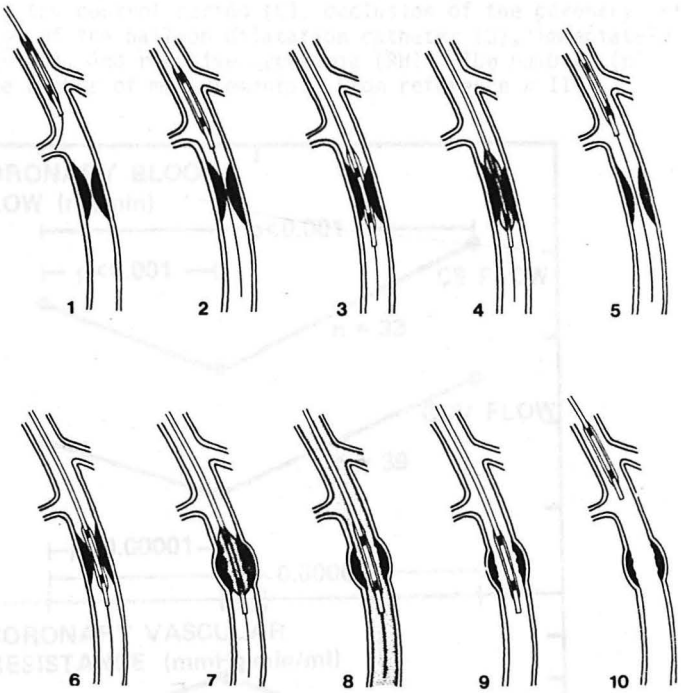
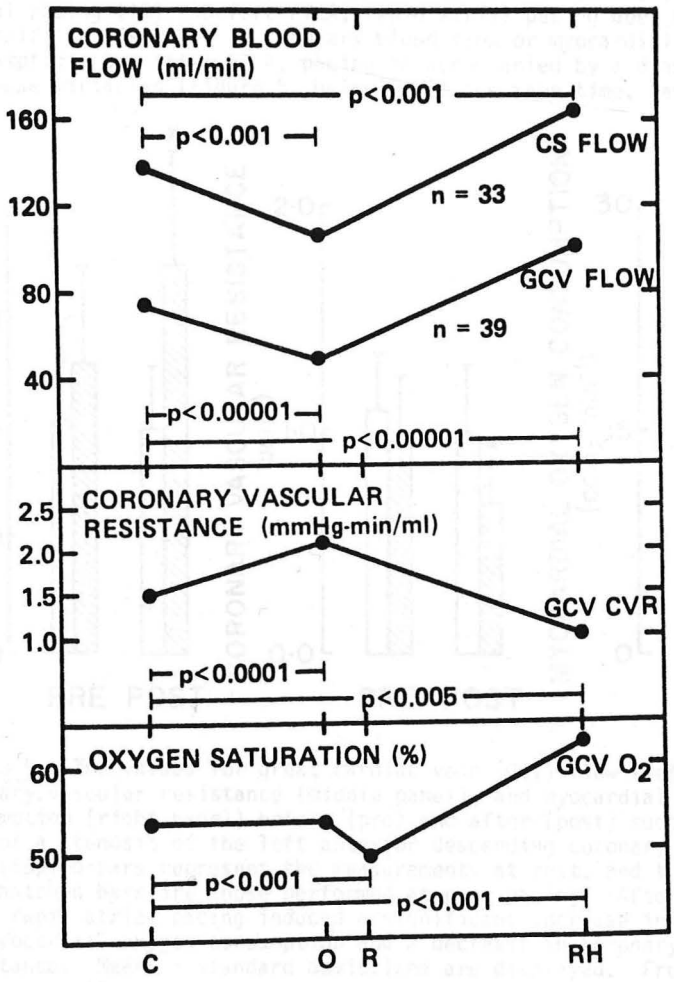


Figure 3 A schematic diagram of the dilatation process. (1)A curved guide wire is used to direct the balloon catheter past the most proximal side branch, but it is not suitable for crossing the stenosis. (2)With the balloon dilatation catheter positioned beyond the origin of the first branch vessel, the curved guide wire is removed, straightened, and advanced across the stenosis. (3)The deflated balloon is advanced over the guide wire so that it "straddles" the stenosis. (4)An initial balloon inflation is performed. (5)If evidence of myocardial ischemia develops, the balloon catheter can be withdrawn, permitting reperfusion of the distal vessel. (6)The balloon dilatation catheter is advanced once again across the stenosis, and (7)repeated balloon inflation is successful in dilating it. (8)An injection of contrast material into the guiding catheter demonstrates that the dilatation is successful. (9)The guide wire is removed so that a pressure gradient across the residual stenosis can be measured, after which (10)the balloon dilatation catheter is removed, and the procedure is terminated (from reference # 10).

Figure 4 The changes in total coronary sinus (CS) flow and great cardiac vein (GCV) flow (top panel), coronary vascular resistance (middle panel), and great cardiac vein oxygen (O_2) saturation (bottom panel) during the control period (C), occlusion of the coronary artery with inflation of the balloon dilatation catheter (O), immediately after release (R), and reactive hyperemia (RH). The numbers (n) represent the number of measurements. From reference # 11.



ventricular dP/dt decline modestly (mean systemic arterial pressure, 96 ± 10 mmHg before PTCA and 90 ± 11 mmHg during PTCA; left ventricular dP/dt, 1200 ± 130 mmHg/sec before PTCA and 1037 ± 103 mmHg/sec during PTCA), reflecting the immediate effect of regional left ventricular ischemia on systolic left ventricular performance. Similarly, successful PTCA alters the response of coronary blood flow to an increase in myocardial oxygen demands, such as those induced by rapid atrial pacing (12). Before PTCA, rapid atrial pacing does not induce a significant increase in coronary blood flow or myocardial oxygen consumption, but after PTCA, pacing is accompanied by a dramatic rise in these variables (Figure 5, below). At the same time, rapid atrial

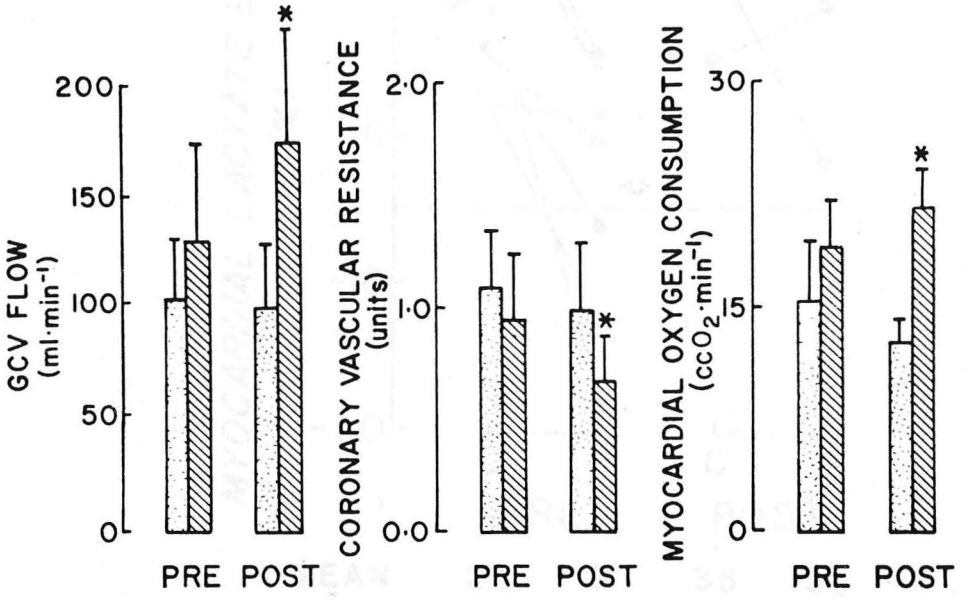


Figure 5 The values for great cardiac vein (GCV) flow (left panel), coronary vascular resistance (middle panel), and myocardial oxygen consumption (right panel) before (pre) and after (post) successful PTCA of a stenosis of the left anterior descending coronary artery. The stippled bars represent the measurements at rest, and the crosshatched bars are those performed at peak pacing. After successful PTCA, rapid atrial pacing induced a significant increase in GCV flow and myocardial oxygen consumption and a decrease in coronary vascular resistance. Means ± standard deviations are displayed. From reference # 12.

pacing in patients before PTCA causes myocardial lactate production in some individuals and a substantial fall in lactate extraction in others; following PTCA, myocardial lactate extraction is not influenced by pacing (Figure 6, below).

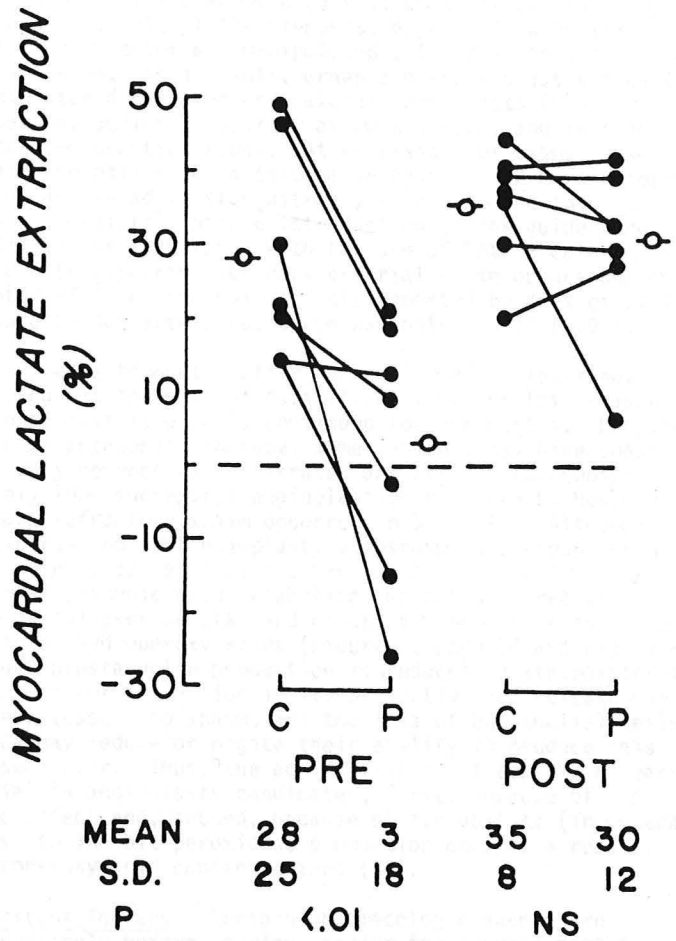


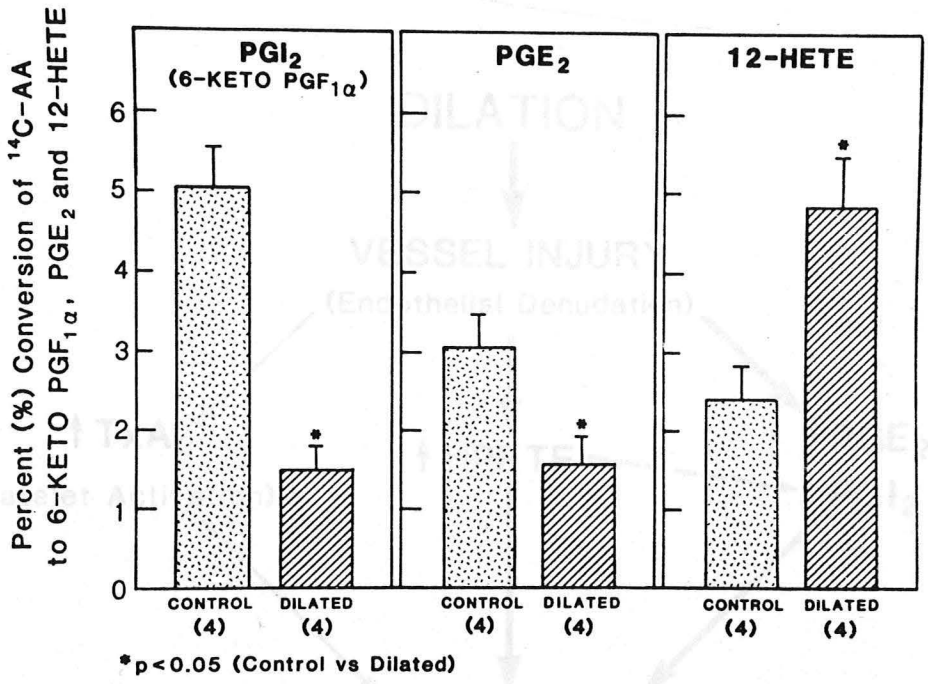
Figure 6 Myocardial lactate extraction, expressed as a %, before (pre) and after (post) successful PTCA both at rest (labelled C) and at peak pacing (labelled P). Prior to PTCA, pacing induced lactate production in 3 patients and a fall in lactate extraction in 3 others. After PTCA, pacing caused lactate production in none and a substantial fall in extraction in only 1. From reference # 12.

C. PTCA-Associated Vasospasm The early experience with PTCA in Europe and in this country demonstrated that coronary arterial spasm often occurred at the site of the stenosis, especially when the balloon dilatation catheter was manipulated into position and repetitively inflated. As a result, Gruentzig and his associates (2) routinely administered nitrates and calcium antagonists (usually nifedipine) before, during, and after attempted PTCA, and this has become the accepted practice today. At Parkland, for example, we routinely begin the patient on a calcium antagonist 1-2 days before attempted PTCA, and we administer nitroglycerin into the involved coronary artery immediately before introduction of the guide wire and balloon dilatation catheter. With the use of both a calcium antagonist and nitroglycerin, coronary arterial spasm occurs uncommonly during attempted PTCA: of the 631 patients reported by Kent et al (4) in whom angioplasty was attempted, spasm was noted in 12 (1.9%).

During the weeks to months after successful PTCA, spasm may occasionally occur at the site of dilatation, and, for this reason, the calcium antagonist is usually continued for 3-6 months. Despite continued calcium antagonist therapy, some patients may have spasm that is relatively refractory to nitrates and calcium antagonists. Of approximately 1000 successful angioplasties reported by Hollman et al (13), such refractory spasm occurred in 5 (0.5%). Although the mechanism of intra- and postangioplasty vasospasm is unknown, Cragg et al (14) have demonstrated that the procedure causes a local derangement of arachidonic acid metabolism, which is characterized by a decrease in vasodilator prostaglandins and a concomitant increase in vasoconstrictor hydroperoxy acids (Figure 7, page 14 and Figure 8, page 15). Since prostacyclin production is reduced in atherosclerotic arteries, even a minor diminution in its production and release may predispose these vessels to spasm, and the loss of endothelial cells induced by PTCA may reduce or negate their ability to produce this endogenous vasodilator. Thus, the administration of aspirin appears to be beneficial in angioplasty candidates, first, because of its antithrombotic effect and, second, because of its ability (in moderate or large doses) to inhibit peroxidase production and, as a result, to reduce hydroperoxy acid concentrations (15).

D. Concomitant Therapy Certain pharmacologic agents are administered routinely before, during, and/or for several months after PTCA. In most centers, aspirin (75-650 mg/day) and dipyridamole (25-100 mg 3 times daily) are initiated 1-2 days before PTCA and are continued for 6 months after the procedure (4). As noted, these agents are given, first, for their antithrombotic effect (16) and, second, for their possible role in preventing an imbalance between vasoconstrictor and vasodilator prostaglandins, with resultant spasm at the site of dilatation. A calcium antagonist is usually begun 1-2 days before attempted PTCA and is also continued for 3-6 months afterward.

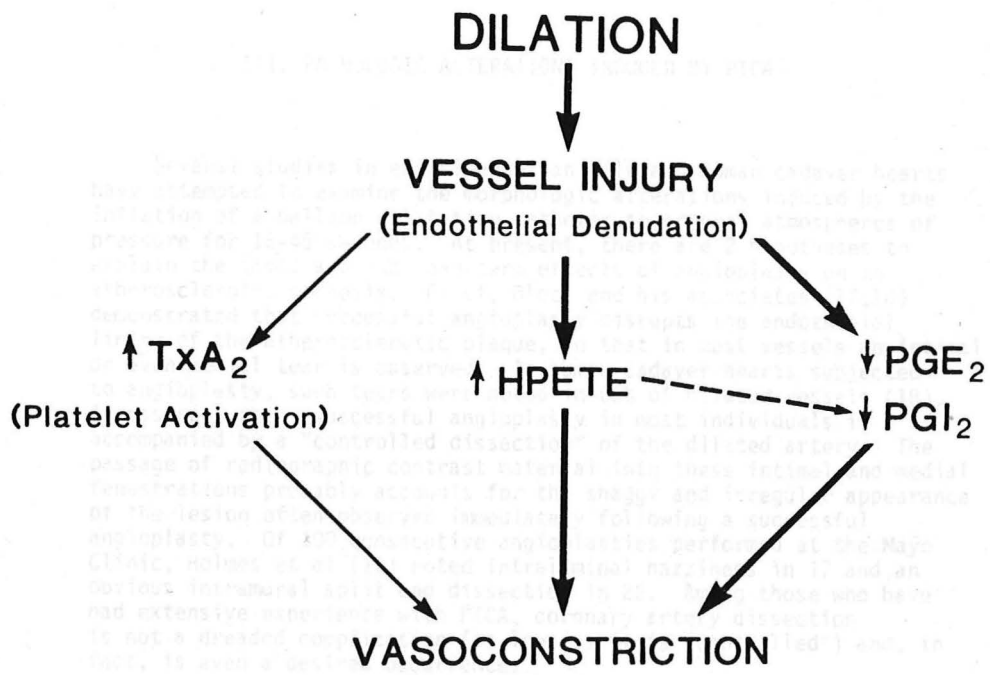
Figure 7 Vessel wall conversion of ^{14}C-arachidonic acid (AA) to 6 keto-$PGF_{1\alpha}$ (the stable metabolite of prostacyclin [PGI_2]), PGE_2, and 12-hydroxy-eicosatetraenoic acid (HETE) in the carotid artery of dogs. In comparison to the contralateral (control) carotid artery (stippled bars), the artery subjected to angioplasty (dilated)(crosshatched bars) demonstrated a lesser concentration of vasodilatory prostaglandins (PGI_2 and PGE_2) and a greater concentration of vasoconstrictor substances (12-HETE). From reference # 14.



Although nifedipine has been most widely employed, verapamil and diltiazem have also been used, and there are no data to suggest that one is better than another. The calcium antagonists are believed to be beneficial because of their antispasmodic effect and because of their possible inhibitory influence on the progression of atherosclerosis at the site of dilatation.

During angioplasty, nitroglycerin is administered sublingually and, if necessary, directly into the diseased coronary artery. When

Figure 8 A schematic representation of how PTCA may induce vasospasm. Dilatation causes endothelial denudation, leading to platelet activation, diminished vasodilatation, and vasoconstriction. From reference # 14.



the large-bore guiding catheter is introduced at the beginning of the procedure, the patient is given 7000-10,000 units of heparin, and supplemental boluses of 2000-3000 units are administered every 45-60 minutes to insure a continued effect. At the end of the procedure, protamine sulfate is not administered; rather, the effect of heparin is simply allowed to diminish gradually.

After the procedure, the patient is continued on aspirin (75-650 mg/day), dipyridamole (25-100 mg 3 times daily), and a calcium antagonist. Although coumadin was used frequently during the "early days" of angioplasty, it is rarely employed today. Three to 6 months after PTCA, the calcium antagonist may be discontinued, and at 6 months, aspirin and dipyridamole may be stopped.

III. PATHOLOGIC ALTERATIONS INDUCED BY PTCA

Several studies in experimental animals and human cadaver hearts have attempted to examine the morphologic alterations induced by the inflation of a balloon dilatation catheter to several atmospheres of pressure for 15-45 seconds. At present, there are 2 hypotheses to explain the immediate and long-term effects of angioplasty on an atherosclerotic stenosis. First, Block and his associates (17,18) demonstrated that successful angioplasty disrupts the endothelial lining of the atherosclerotic plaque, so that in most vessels an intimal or even medial tear is observed. In human cadaver hearts subjected to angioplasty, such tears were noted in 65% of dilated vessels (18). In essence, then, successful angioplasty in most individuals is accompanied by a "controlled dissection" of the dilated artery. The passage of radiographic contrast material into these intimal and medial fenestrations probably accounts for the shaggy and irregular appearance of the lesion often observed immediately following a successful angioplasty. Of 100 consecutive angioplasties performed at the Mayo Clinic, Holmes et al (19) noted intraluminal hazziness in 17 and an obvious intramural split and dissection in 22. Among those who have had extensive experience with PTCA, coronary artery dissection is not a dreaded complication (as long as it is "controlled") and, in fact, is even a desired occurrence.

Second, other investigators have not observed a high incidence of intimal or medial tears following angioplasty, but rather they have noted endothelial desquamation in association with medial and adventitial stretching (20). In essence, angioplasty in most of these instances induced stretching of the vessel with localized aneurysm formation (21). Holmes et al (19) noted smooth-walled

dilatation (without evidence of intimal fissuring or splitting) in 41 of their 100 patients.

In short, it appears that successful angioplasty may be associated with one of two morphologic alterations. In about half, inflation of the balloon dilatation catheter causes a tear in the intima and even the media, leading to a "controlled" dissection. In the other half, balloon inflation simply stretches the media and adventitia and, therefore, causes a localized coronary artery aneurysm. In most individuals, such aneurysms are small, but in an occasional patient they may become quite large (22).

IV. SPECIAL CLINICAL SUBSETS FOR WHICH PTCA MAY BE USED

Although PTCA has been used predominantly in patients with stable angina of effort who meet most, if not all, of the criteria described in Section I (Patient Selection), it has more recently been employed by some investigators in individuals with other manifestations of ischemic heart disease.

A. Unstable Angina Pectoris Preliminary reports suggest that PTCA can be performed safely and effectively in patients with unstable angina whose coronary anatomy is suitable. Meyer et al (23) attempted to perform PTCA in 50 patients with unstable angina and compared the results to those obtained in 50 individuals with stable angina. PTCA was deemed successful in 66% of the stable and 74% of the unstable patients (NS). One patient (2%) in each group required emergency coronary artery bypass surgery. Restenosis within 6 months of dilatation occurred in 18% of the stable patients and in 24% of those with unstable angina (NS). Similarly, McCallister et al (24) reported good overall results of PTCA in patients with unstable angina. Of 123 patients with unstable angina in whom PTCA was attempted, 1 (0.8%) died, and 5 (4.1%) sustained a myocardial infarction. In 30 (24%), PTCA was unsuccessful, and the patient was referred for bypass grafting. During the same period, 251 patients with unstable angina underwent coronary artery bypass grafting, of whom 1 (0.4%) died and 19 (7.6%) sustained an infarction. Finally, Alcan and his associates (25) performed emergent PTCA in 14 patients in whom intraaortic balloon counterpulsation was used simultaneously for control of unstable angina. Of these, 13 eventually were discharged from the hospital. In short, it appears that PTCA can be performed in the patient with unstable angina if (a)coronary anatomy is suitable and (b)the operator has acquired

a substantial angioplasty experience in patients with stable angina.

B. Variant Angina Pectoris Since PTCA apparently induces coronary arterial spasm in some patients, the presence of known variant angina has served as a contraindication to the procedure, first, because continued vasospasm after angioplasty was believed to be likely and, second, because such vasospasm might increase the chance of accelerated atherogenesis at the site of dilatation. In 11 patients with known variant angina in whom PTCA was attempted (26), initial successful dilatation was accomplished in 10, but continued episodes of coronary vasospasm subsequently occurred in 9, and restenosis was noted in 7. Thus, although PTCA is technically feasible in patients with variant angina pectoris, symptoms due to coronary arterial spasm often persist. Furthermore, the occurrence of restenosis appears to be especially likely during the months following the procedure.

C. Acute Myocardial Infarction (With and Without Thrombolytic Therapy) Several case reports have described the administration of intracoronary streptokinase followed immediately (27,28) or within 1-2 days (29) by PTCA. In addition, von Essen and associates (30) have reported a patient in whom a totally occluded left main coronary artery was unaffected by intracoronary streptokinase but was successfully opened with angioplasty. More recently, Hartzler et al (31) have demonstrated that PTCA may be performed with or without concomitant thrombolytic therapy in selected patients with acute myocardial infarction. These investigators emergently catheterized 41 patients with acute infarction an average of 3.3 hours after the onset of chest pain. The 29 with a totally occluded infarct-related coronary artery were given streptokinase. Thrombolysis occurred in 17, at which time PTCA was performed. Of the other 12 individuals with subtotal coronary artery occlusions, 11 underwent angioplasty without prior thrombolytic therapy. Thus, of the 41 patients, 28 had angioplasty in the setting of acute infarction, of which 24 (86%) had prompt relief of pain and a stable clinical course, and only 3 (13%) sustained a reocclusion. Thus, angioplasty in close temporal proximity to successful thrombolysis may reduce the risk of late reocclusion.

D. Total Coronary Artery Occlusion PTCA has been attempted in a relatively small number of patients with totally occluded coronary arteries not in the setting of acute myocardial infarction. In 13 individuals with coronary artery occlusion in whom angioplasty was attempted by Dervan et al (32), primary success was achieved in 7 (54%), and there were no complications. An identical rate of success was reported from the Mayo Clinic by Holmes et al (33): of 24 patients in whom angioplasty of a totally occluded artery was attempted, success occurred in 13 (54%). In the 19 individuals whose total occlusion was believed to be < 12 weeks in duration, angioplasty was successful in 13 (68%); in contradistinction, in the 5 patients whose occlusion

was estimated to be > 12 weeks old, PTCA was not successful (Figure 9, below). Thus, although the chance of primary success is lower in patients with total occlusion than in those with subtotal occlusion, angioplasty can be performed safely in these individuals. The chance of success, however, appears to be very low in patients whose occlusions are old (> 12 weeks).

E. Saphenous Vein Graft Stenoses In centers with extensive angioplasty experience, the procedure appears to be safe and effective in individuals whose saphenous vein grafts have become narrowed. Douglas et al (34) attempted PTCA in 62 saphenous grafts. Of the 34 patients in whom the stenosis was located at the anastomosis of the graft and coronary artery, PTCA was initially successful in 32 (94%), 4

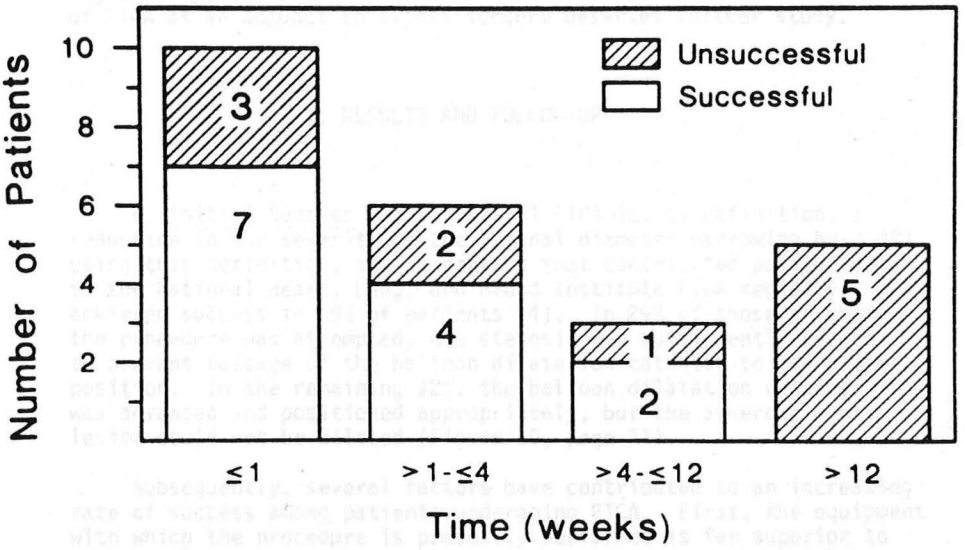


Figure 9 The number of patients with a totally occluded coronary artery in whom PTCA was successful (open bars) or unsuccessful (crosshatched bars) in relation to the duration of the total occlusion (in weeks). Note that angioplasty was often successful in those whose total occlusion occurred recently (< 12 weeks), whereas it was unsuccessful in all 5 patients whose occlusion occurred in the remote past (> 12 weeks). From reference # 33.

of whom later developed restenosis. Of the 23 patients in whom the stenosis was located in the body of the saphenous vein graft (i.e., did not involve an anastomosis), PTCA was successful in 22 (96%), but restenosis developed in 9. Finally, of the 5 individuals whose stenoses were located at the anastomosis of the saphenous vein graft and the aorta, PTCA was successful in 4, 1 of whom developed restenosis. In short, saphenous vein graft stenoses appear to be amenable to angioplasty in most individuals.

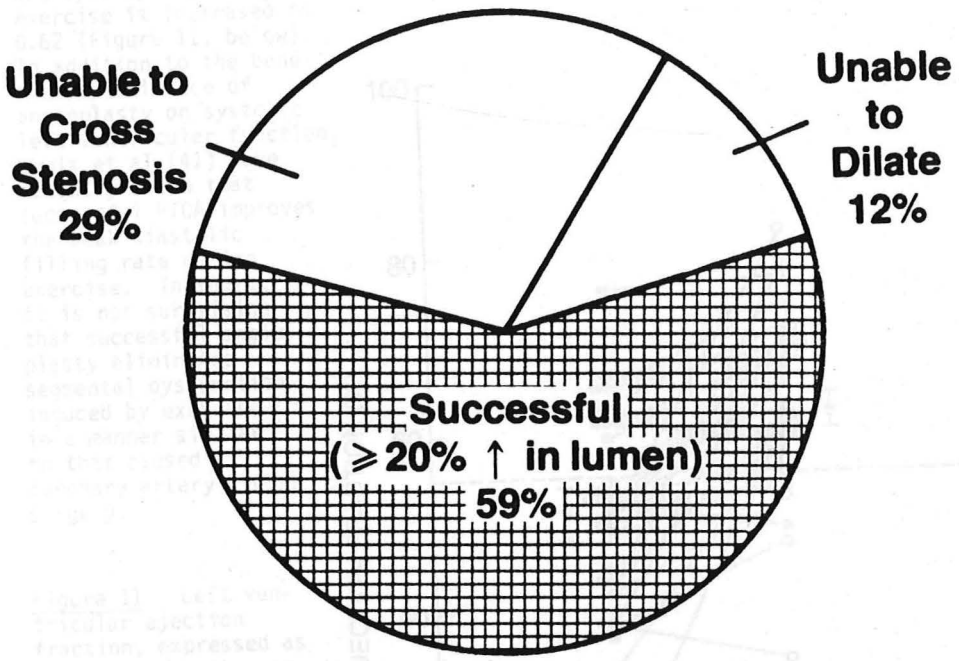
F. Intraoperative Applications Jones and King (35) have reported that intraoperative angioplasty may be beneficial in the treatment of coronary artery stenoses (a)that are inaccessible to normal grafting procedures (i.e., a large septal perforator) and (b)that occur in sequence and, therefore, render bypass grafting technically difficult. Of 14 patients having intraoperative angioplasty, the procedure was deemed successful in 10. The use of PTCA as an adjunct to bypass surgery deserves further study.

V. RESULTS AND FOLLOW-UP

A. Initial Success A successful PTCA is, by definition, a reduction in the severity of the luminal diameter narrowing by ≥ 20%. Using this definition, the 34 centers that contributed patient data to the National Heart, Lung, and Blood Institute PTCA registry achieved success in 59% of patients (4). In 29% of those in whom the procedure was attempted, the stenosis was sufficiently severe to prevent passage of the balloon dilatation catheter to its proper position. In the remaining 12%, the balloon dilatation catheter was advanced and positioned appropriately, but the atherosclerotic lesion could not be dilated (Figure 10, page 21).

Subsequently, several factors have contributed to an increasing rate of success among patients undergoing PTCA. First, the equipment with which the procedure is presently performed is far superior to that utilized in the late 1970s and even early 1980s. Second, physicians have gained considerable experience in better selecting patients in whom angioplasty will probably be successful. Third, in every institution in which angioplasty is performed, a "learning curve" for new operators exists, so that the rate of primary success increases gradually until the physician has done 90-100 procedures (36). It is not surprising, therefore, that Gruentzig's recent results demonstrate that 90% of stenoses are successfully crossed and 86% are successfully dilated (36).

Figure 10 A schematic representation of the fate of the 651 patients in whom PTCA was attempted in the 34 centers contributing patient data to the National Heart, Lung, and Blood Institute PTCA registry. From reference # 4.



B. Exercise Capability, Myocardial Perfusion, and Left Ventricular Function Successful PTCA is as effective as coronary artery bypass surgery in increasing the patient's work capacity. In 160 patients successfully dilated by Meier et al (37), work capacity increased from 74 ± 42 [mean ± SD] watts before angioplasty to 122 ± 47 watts afterward. A similar increase was noted in 16 patients in whom angioplasty was unsuccessful and coronary artery bypass grafting was performed electively (65 ± 37 watts before surgery, 119 ± 41 watts afterward) as well as in 12 individuals in whom failed PTCA required emergent surgery (73 ± 34 watts before, 120 ± 41 watts after).

Using thallium-201 scintigraphy or equilibrium gated blood pool imaging at rest and during exercise, several studies have demonstrated

that successful PTCA eliminates radionuclide evidence of exercise-induced hypoperfusion (38) and left ventricular segmental and global dysfunction (39,40). Of 38 patients having successful PTCA at the National Institutes of Health (40), left ventricular ejection fraction before PTCA averaged 0.55 at rest and 0.51 at peak exercise. Following angioplasty, ejection fraction at rest was unchanged, but during exercise it increased to 0.62 (Figure 11, below). In addition to the beneficial influence of angioplasty on systolic left ventricular function, Lewis et al (41) have recently shown that successful PTCA improves the peak diastolic filling rate during exercise. In short, it is not surprising that successful angioplasty eliminates segmental dysfunction induced by exercise in a manner similar to that caused by coronary artery bypass surgery.

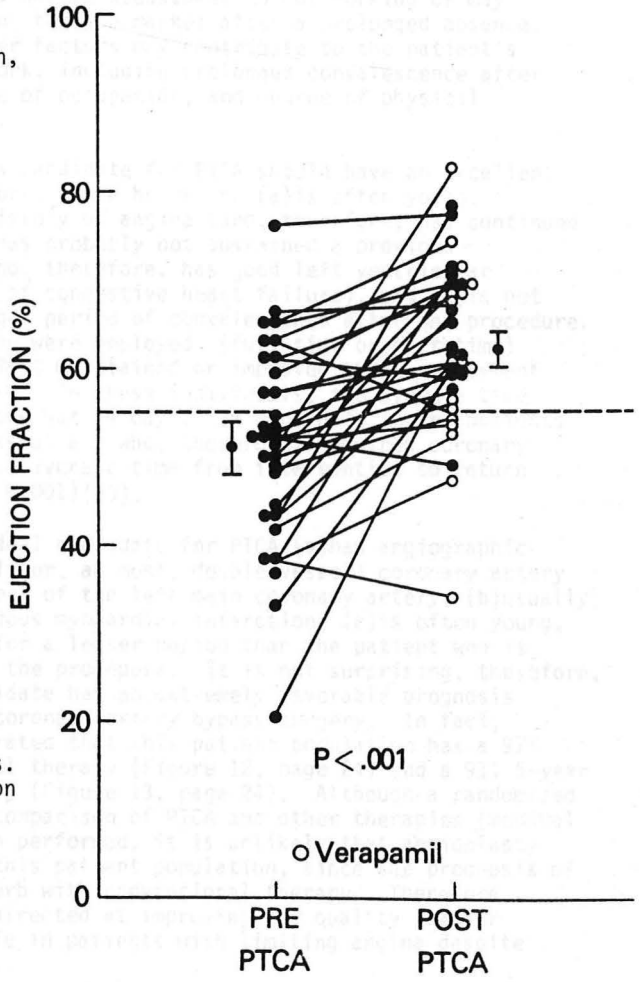


Figure 11 Left ventricular ejection fraction, expressed as a %, during peak exercise before (pre) and after (post) PTCA. Each line represents the data from 1 patient, and the means ± SEM are displayed on either side of each set of lines. Exercise ejection fraction increased significantly after PTCA. From reference # 40.

C. Employability Previous studies (42) have demonstrated that the best predictor of work status 1-2 years after coronary artery bypass surgery is the patient's work status during the several months before surgery. Thus, the patient who has continued to work until coronary bypass grafting is likely to return to work after surgery, and, conversely, the patient who has not worked during the months before surgery is unlikely to do so. It has been postulated that patients who have been on disability or prolonged sick leave before bypass surgery may become accustomed to not working or may have difficulty reentering the job market after a prolonged absence. In addition, several other factors may contribute to the patient's inability to return to work, including prolonged convalescence after bypass surgery, age, type of occupation, and degree of physical disability after surgery.

The patient who is a candidate for PTCA should have an excellent chance of returning to work, since he or she (a)is often young, (b)usually has a short history of angina (and, therefore, has continued to work until PTCA), (c)has probably not sustained a previous myocardial infarction (and, therefore, has good left ventricular function and no evidence of congestive heart failure), and (d)is not confronted with a prolonged period of convalescence after the procedure. Indeed, among patients who were employed (full-time or part-time) before successful PTCA, 98½% maintained or improved their employment status after the procedure. In these individuals, the average time from PTCA to return to work was 14 days. In contrast, in the patients in whom PTCA was unsuccessful and who, therefore, required coronary artery bypass surgery, the average time from intervention to return to work was 60 days ($p < 0.001$)(43).

D. Survival The ideal candidate for PTCA (a)has angiographic evidence of single vessel (or, at most, double vessel) coronary artery disease without involvement of the left main coronary artery, (b)usually has not sustained a previous myocardial infarction, (c)is often young, and (d)has noted angina for a lesser period than the patient who is not a good candidate for the procedure. It is not surprising, therefore, that the ideal PTCA candidate has an extremely favorable prognosis with medical therapy or coronary artery bypass surgery. In fact, Hlatky et al (6) demonstrated that this patient population has a 97% 5-year survival on medical therapy (Figure 12, page 24) and a 91% 5-year survival following surgery (Figure 13, page 24). Although a randomized and properly controlled comparison of PTCA and other therapies (medical or surgical) has not been performed, it is unlikely that angioplasty can improve survival in this patient population, since the prognosis of these individuals is superb with conventional therapy. Therefore, the procedure should be directed at improving the quality (rather than the quantity) of life in patients with limiting angina despite adequate medical therapy.

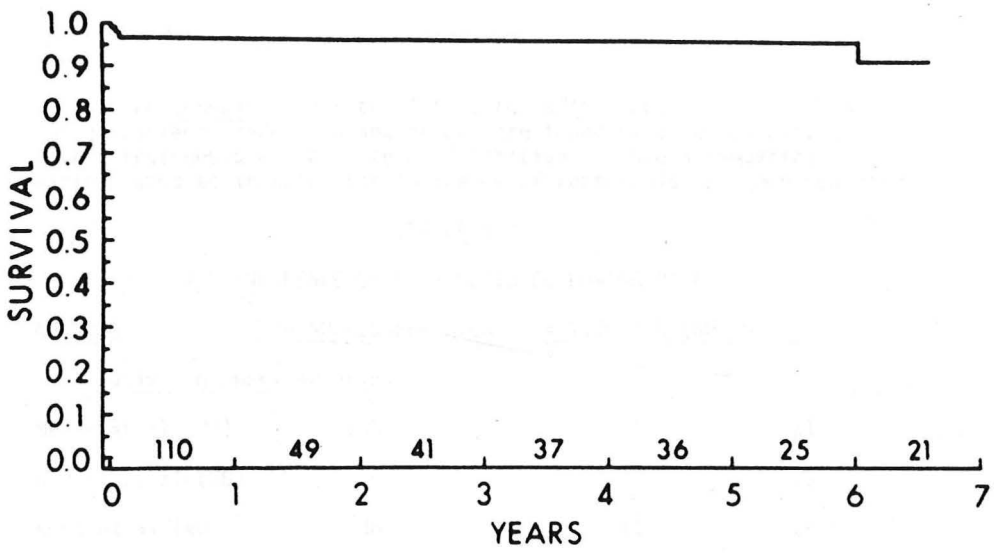


Figure 12 Cumulative survival among good PTCA candidates treated with medical therapy. The numbers above the horizontal axis denote the number of patients under observation during the interval. Survival on medical therapy was excellent. From reference # 6.

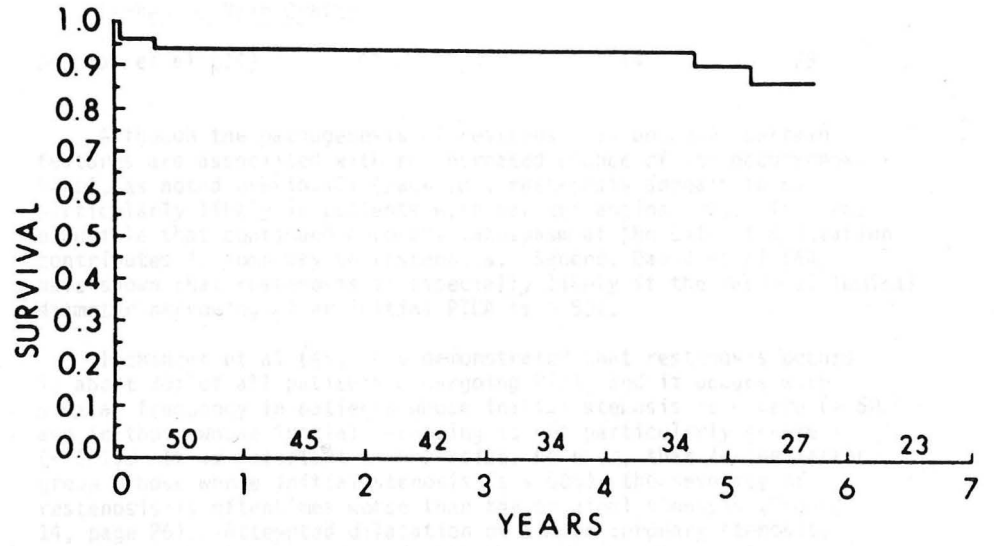


Figure 13 Cumulative survival among good PTCA candidates treated with coronary artery bypass surgery. The numbers above the horizontal axis denote the number of patients under observation during the interval. Survival following bypass surgery was excellent. From reference # 6.

E. Restenosis Over the 4-6 months after successful PTCA, about 25% of patients redevelop angina and are found by arteriography to have a restenosis at the site of dilatation. Table 3 summarizes the various studies in which the incidence of restenosis has been reported.

TABLE 3

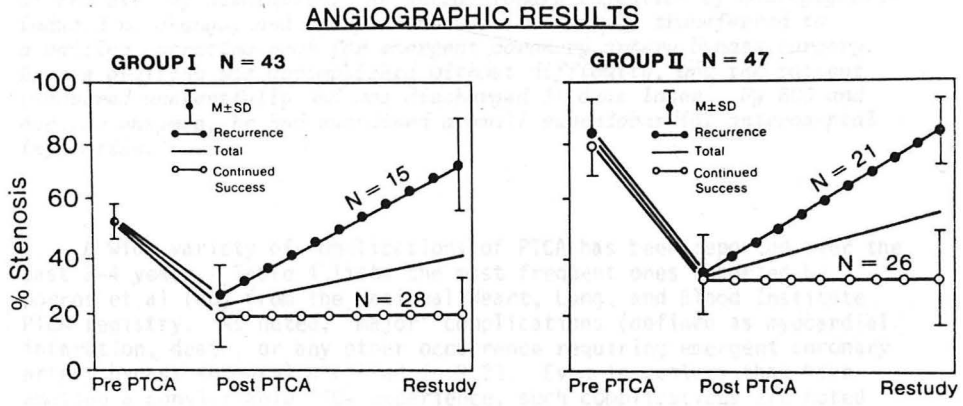
THE INCIDENCE OF RESTENOSIS FOLLOWING PTCA

AUTHORS	# WITH SUCCESSFUL PTCA	# WITH RESTENOSIS	%
Native Coronary Arteries			
Meyer et al (23)	100	21	21
Hirzel et al (38)	16	3	19
Kent et al (40)	38	11	29
David et al (44)	180	53	29
Ischinger et al (45)	130	41	32
TOTAL	464	129	28
Saphenous Vein Grafts			
Douglas et al (34)	62	14	23

Although the pathogenesis of restenosis is unknown, certain features are associated with an increased chance of its occurrence. First, as noted previously (page 18), restenosis appears to be particularly likely in patients with variant angina (26). It seems plausible that continued coronary vasospasm at the site of dilatation contributes in some way to restenosis. Second, David et al (44) have shown that restenosis is especially likely if the residual luminal diameter narrowing after initial PTCA is > 50%.

Ischinger et al (45) have demonstrated that restenosis occurs in about 30% of all patients undergoing PTCA, and it occurs with similar frequency in patients whose initial stenosis is severe (> 60%) and in those whose initial narrowing is not particularly severe (< 60%). It is important to emphasize, however, that in the latter group (those whose initial stenosis is < 60%), the severity of restenosis is oftentimes worse than the original stenosis (Figure 14, page 26). Attempted dilatation of a mild coronary stenosis, therefore, should be performed with the understanding that a

Figure 14 The % stenosis before (pre PTCA), immediately after (post PTCA), and at remote restudy in 2 groups of patients: Group I = those whose initial stenosis is < 60%, and Group II = those whose initial stenosis is > 60%. Note that the patients who develop restenosis in Group I have a more severe narrowing than that noted initially. From reference # 45.



recurrence of the stenosis may be more severe than the original narrowing.

VI. RISKS AND COMPLICATIONS

S.D. is a 44 year old man who was hospitalized at PMH on 2/13/84 with an acute anteroseptal infarction. On arrival in the Emergency Room, he was given intravenous streptokinase (750,000 units over 20 minutes), and within 1 hour his chest pain disappeared, and his widespread ST segment elevation resolved without the concomitant appearance of Q waves. He was treated with heparin by continuous intravenous infusion for the next 10 days, at which time catheterization was performed, revealing a cardiac output of 4.8 liters/minute, normal pressures and resistances, and a left ventricular ejection fraction of 0.71. By selective coronary arteriography, the patient was noted to have a 95% luminal diameter narrowing of the mid-left anterior descending coronary artery; the rest of his coronary arteries were normal.

Six days later, the patient was returned to the Catheterization Laboratory for attempted PTCA. The left anterior descending stenosis was traversed with an angioplasty guide wire and dilatation balloon catheter, after which 4 inflations (each lasting 15-30 seconds) were performed. Within 2 minutes of the final balloon inflation, the patient complained of retrosternal chest pain; his ECG revealed obvious ST segment elevation anteriorly; and repeat coronary arteriography showed that the left anterior descending coronary artery was totally occluded at the site of dilatation. An intracoronary injection of nitroglycerin induced no change, and the patient was immediately transferred to a waiting operating room for emergent coronary artery bypass surgery. Bypass grafting was accomplished without difficulty, and the patient recovered uneventfully and was discharged 11 days later. By ECG and cardiac enzymes, he had sustained a small subendocardial anteroseptal infarction.

A wide variety of complications of PTCA has been reported over the past 3-4 years. Table 4 lists the most frequent ones reported by Dorros et al (46) from the National Heart, Lung, and Blood Institute PTCA registry. As noted, "major" complications (defined as myocardial infarction, death, or any other occurrence requiring emergent coronary artery bypass surgery) occurred in 9.2%. Even in centers that have amassed a considerable PTCA experience, such complications are noted in about 8-10% of patients: in Gruentzig's review of his first 624 patients (36), none died, 20 (3.2%) sustained a myocardial infarction, and 33 (5.3%) required emergent bypass surgery.

TABLE 4

COMPLICATIONS OF PTCA [1500 PROCEDURES AT 73 CENTERS]

	n	%
Number of patients having a complication	314	21
Most frequent complications:		
a. Prolonged angina	121	8.1
b. Myocardial infarction	72	4.8
c. Coronary artery occlusion	70	4.7
d. Emergent bypass surgery required	102	6.8
e. In-hospital death	16	1.1
f. Major complication (myocardial infarction, death, or emergency bypass surgery)	138	9.2

Several points deserve emphasis in assessing the incidence and severity of complications related to PTCA. First, although Meyer et al (23)

and McCallister et al (24) reported that PTCA can be performed safely in patients with unstable angina pectoris, Dorros et al (46) noted a higher incidence of nonfatal complications in these patients than in those with a stable anginal pattern. Second, as the data in Table 5 demonstrate, the incidence of complications gradually diminishes as the physician performing the procedure gains experience. Interestingly, however, the incidence of complications appears to *increase* transiently

TABLE 5. *Complications by Patient Sequence*

Patient sequence no.	No. of pts	All complications		Major complications*	
		n	%	n	%
1–10	471	126	26.75	62	13.16
11–20	230	50	21.74	19	8.26
21–30	172	32	18.60	18	10.47
31–40	129	24	18.60	10	7.75
41–50	97	16	16.50	8	8.25
51–60	77	15	19.48	7	9.09
61–70	56	14	25.00	8	14.29
71+	268	37	13.81	17	6.34
Total	1500				

*Death, myocardial infarction, or emergency surgery.

This table shows the rate of all complications and the rate of major complications (death, myocardial infarction, or emergency surgery) by the sequence of performance.[9] The first 10 cases of all investigators were combined, as were their second 10 cases, etc. These groupings were then related to the complications.

at a time when the physician has done 50-70 procedures, after which it once again falls. Possibly many physicians, after performing 50 or so angioplasties, become overly confident of their abilities and-- for a short time-- attempt the procedure in less ideal candidates, a group of individuals in whom, for technical reasons, complications occur more frequently. Third, it should be emphasized that emergent coronary artery bypass grafting is required in 5-7% of patients. For this reason, it is our approach-- and that of most other centers-- to request that the cardiac surgeons evaluate the patient before PTCA,

obtain his or her permission for bypass surgery, and be immediately available as the procedure is being performed in the event that emergent surgery is required.

VII. CONCLUSIONS

PTCA appears to be an effective alternative to coronary artery bypass surgery in patients whose coronary artery anatomy is suitable--that is, an individual with single (or, at most, double) vessel coronary artery disease whose stenoses are proximal, discrete, subtotal, concentric, and noncalcified. Since emergent coronary artery bypass surgery is required in 5-7% of patients even when PTCA is attempted by an experienced physician, the patient should be an acceptable candidate for surgery from both a cardiac and noncardiac standpoint. Unfortunately, ideal PTCA candidates are a distinct minority among the thousands of patients with coronary artery disease. If the procedure is reserved for ideal (or nearly ideal) candidates, the rate of success should approach 75-80%, and the incidence of major complications should be below 10%. Although the procedure appears to be effective in alleviating angina, it is unlikely that it will exert a beneficial effect on survival when compared to either medical therapy or coronary artery bypass surgery.

REFERENCES

1. Dotter CT, Judkins MP. Transluminal treatment of arteriosclerotic obstruction: Description of a new technic and a preliminary report of its application. Circulation 1964; 30:654-70.

2. Gruentzig AR, Senning A, Siegenthaler WE. Nonoperative dilatation of coronary artery stenosis. Percutaneous transluminal coronary angioplasty. N Engl J Med 1979; 301:61-8.

3. Gruentzig AR, Kuhlmann U, Vetter W, Lutolf U, Meier B, Siegenthaler W. Treatment of renovascular hypertension with percutaneous transluminal dilatation of a renal artery stenosis. Lancet 1978; 1:801-2.

4. Kent KM, Bentivoglio LG, Block PC, Cowley MJ, Dorros G, Gosselin AJ, Gruentzig AR, Myler RK, Simpson J, Stertzer SH, Williams DO, Fisher L, Gillespie MJ, Detre K, Kelsey S, Mullin SM, Mock MB. Percutaneous transluminal coronary angioplasty: Report from the registry of the National Heart, Lung, and Blood Institute. Am J Cardiol 1982; 49:2011-20.

5. Hamby RI, Katz S. Percutaneous transluminal coronary angioplasty: Its potential impact on surgery for coronary artery disease. Am J Cardiol 1980; 45:1161-6.

6. Hlatky MA, Califf RM, Kong Y, Harrell FE Jr, Rosati RA. Natural history of patients with single vessel disease suitable for percutaneous transluminal coronary angioplasty. Am J Cardiol 1983; 52:225-9.

7. Holmes DR Jr, Vlietstra RE, Fisher LD, Smith HC, Mock MB, Faxon DP, Gosselin AJ, Ryan TJ, Judkins MP, Pettinger M. Follow-up of patients from the Coronary Artery Surgery Study (CASS) potentially suitable for percutaneous transluminal coronary angioplasty. Am Heart J 1983; 106:981-8.

8. Meier B, Gruentzig AR, Hollman J, Ischinger T, Bradford JM. Does length or eccentricity of coronary stenoses influence the outcome of transluminal dilatation? Circulation 1983; 67:497-9.

9. Meier B, Gruentzig AR, King SB III, Douglas JS Jr, Hollman J, Ischinger T, Aueron F, Galan K. Risk of side branch occlusion during coronary angioplasty. Am J Cardiol 1984; 53:10-14.

10. Simpson JB, Baim DS, Robert EW, Harrison DC. A new catheter system for coronary angioplasty. Am J Cardiol 1982; 49:1216-22.

11. Rothman MT, Baim DS, Simpson JB, Harrison DC. Coronary hemodynamics during percutaneous transluminal coronary angioplasty. Am J Cardiol 1982; 49:1615-22.

12. Williams DO, Riley RS, Singh AK, Most AS. Coronary circulatory dynamics before and after successful coronary angioplasty. J.A.C.C. 1983; 1:1268-72.

13. Hollman J, Austin GE, Gruentzig AR, Douglas JS Jr, King SB III. Coronary artery spasm at the site of angioplasty in the first 2 months after successful percutaneous transluminal coronary angioplasty. J.A.C.C. 1983; 2:1039-45.

14. Cragg A, Einzig S, Castaneda-Zuniga W, Amplatz K, White JG, Rao GHR. Vessel wall arachidonate metabolism after angioplasty: Possible mediators of postangioplasty vasospasm. Am J Cardiol 1983; 51:1441-5.

15. Siegel MI, McConnell RT, Cuatrecasas P. Aspirin-like drugs interfere with arachidonate metabolism by inhibition of the 12-hydroperoxy-5,8,10,14-eicosatetraenoic acid peroxidase activity of the lipoxygenase pathway. Proc Natl Acad Sci 1979; 76:3774-8.

16. Steele PM, Chesebro JH, Stanson AW, Holmes DR, Badimon L, Fuster V. Balloon angioplasty: Effect of platelet-inhibitor drugs on platelet-thrombus deposition in a pig model. J.A.C.C. 1984; 3:506 (abstract).

17. Block PC, Baughman KL, Pasternak RC, Fallon JT. Transluminal angioplasty: Correlation of morphologic and angiographic findings in an experimental model. Circulation 1980; 61:778-85.

18. Baughman KL, Pasternak RC, Fallon JT, Block PC. Transluminal coronary angioplasty of postmortem human hearts. Am J Cardiol 1981; 48:1044-7.

19. Holmes DR Jr, Vlietstra RE, Mock MB, Reeder GS, Smith HC, Bove AA, Bresnahan JF, Piehler JM, Schaff HV, Orszulak TA. Angiographic changes produced by percutaneous transluminal coronary angioplasty. Am J Cardiol 1983; 51:676-83.

20. Lee G, Ikeda RM, Joye JA, Bogren HG, DeMaria AN, Mason DT. Evaluation of transluminal angioplasty of chronic coronary artery stenosis. Value and limitations assessed in fresh human cadaver hearts. Circulation 1980; 61:77-83.

21. Sanborn TA, Faxon DP, Haudenschild C, Gottsman SB, Ryan TJ. The mechanism of transluminal angioplasty: Evidence for formation of aneurysms in experimental atherosclerosis. Circulation 1983; 68:1136-40.

22. Hill JA, Margolis JR, Feldman RL, Conti CR, Pepine CJ. Coronary arterial aneurysm formation after balloon angioplasty. Am J Cardiol 1983; 52:261-4.

23. Meyer J, Schmitz HJ, 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; 106:973-80.

24. McCallister BD, Hartzler GO, Reed WA, Johnson TW. Coronary angioplasty for unstable angina: A comparison with coronary artery bypass surgery. J.A.C.C. 1984; 3:470 (abstract).

25. Alcan KE, Stertzer SH, Wallsh E, DePasquale NP, Bruno MS. The role of intraaortic balloon counterpulsation in patients undergoing percutaneous transluminal coronary angioplasty. Am Heart J 1983; 105:527-30.

26. David PR, Waters DD, Scholl JM, Crepeau J, Szlachcic J, Lesperance J, Hudon G, Bourassa MG. Percutaneous transluminal coronary angioplasty in patients with variant angina. Circulation 1982; 66:695-702.

27. Goldberg S, Urban PL, Greenspon A, Lebenthal M, Walinsky P, Maroko P. Combination therapy for evolving myocardial infarction: Intracoronary thrombolysis and percutaneous transluminal angioplasty. Am J Med 1982; 72:994-7.

28. Meltzer RS, van den Brand M, Serruys PW, Fioretti P, Hugenholtz PG. Sequential intracoronary streptokinase and transluminal angioplasty in unstable angina with evolving myocardial infarction. Am Heart J 1982; 104:1109-11.

29. Meyer J, Merx W, Dorr R, Lambertz H, Bethge C, Effert S. Successful treatment of acute myocardial infarction shock by combined percutaneous transluminal coronary recanalization (PTCR) and percutaneous transluminal coronary angioplasty (PTCA). Am Heart J 1982; 103:132-4.

30. von Essen R, Lambertz H, Schmidt W, Rustige J, Uebis R, Effert S. Successful recanalization of a left main coronary artery occlusion. Am J Cardiol 1984; 53:356-7.

31. Hartzler GO, Rutherford BD, McConahay DR, Johnson WL Jr, McCallister BD, Gura GM Jr, Conn RC, Crockett JE. Percutaneous transluminal coronary angioplasty with and without thrombolytic therapy for treatment of acute myocardial infarction. Am Heart J 1983; 106:965-73.

32. Dervan JP, Baim DS, Cherniles J, Grossman W. Transluminal angioplasty of occluded coronary arteries: Use of a movable guide wire system. Circulation 1983; 68:776-84.

33. Holmes DR Jr, Vlietstra RE, Reeder GS, Bresnahan JF, Smith HC, Bove AA, Schaff HV. Angioplasty in total coronary artery occlusion. J.A.C.C. 1984; 3:845-9.

34. Douglas JS Jr, Gruentzig AR, King SB III, Hollman J, Ischinger T, Meier B, Craver JM, Jones EL, Waller JL, Bone DK, Guyton R. Percutaneous transluminal coronary angioplasty in patients with prior coronary bypass surgery. J.A.C.C. 1983; 2:745-54.

35. Jones EL, King SB III. Intraoperative angioplasty in the treatment of coronary artery disease. J.A.C.C. 1983; 1:970-1.

36. Gruentzig AR. Results from coronary angioplasty and implications for the future. Am Heart J 1982; 103:779-83.

37. Meier B, Gruentzig AR, Siegenthaler WE, Schlumpf M. Long-term exercise performance after percutaneous transluminal coronary angioplasty and coronary artery bypass grafting. Circulation 1983; 68:796-802.

38. Hirzel HO, Naesch K, Gruentzig AR, Luetolf UM. Short and long-term changes in myocardial perfusion after percutaneous transluminal coronary angioplasty assessed by thallium-201 exercise scintigraphy. Circulation 1981; 63:1001-7.

39. Sigwart U, Grbic M, Essinger A, Bischof-Delaloye A, Sadeghi H, Rivier JL. Improvement of left ventricular function after percutaneous transluminal coronary angioplasty. Am J Cardiol 1982; 49:651-7.

40. Kent KM, Bonow RO, Rosing DR, Ewels CJ, Lipson LC, McIntosh CL, Bacharach S, Green M, Epstein SE. Improved myocardial function during exercise after successful percutaneous transluminal coronary angioplasty. N Engl J Med 1982; 306:441-6.

41. Lewis JF, Tortoledo FA, Lewis JM, Poliner LR, Raizner AE, Verani MS. Improved diastolic and systolic left ventricular function during exercise after transluminal coronary angioplasty. J.A.C.C. 1984; 3:530 (abstract).

42. Hammermeister KE, DeRouen TA, English MT, Dodge HT. Effect of surgical versus medical therapy on return to work in patients with coronary artery disease. Am J Cardiol 1979; 44:105-11.

43. Holmes DR Jr, Vlietstra RE, Mock MB, Smith HC, Dorros G, Cowley MJ, Kent KM, Hammes LN, Janke L, Elveback LR, Vetrovec GW. Employment and recreation patterns in patients treated by percutaneous transluminal coronary angioplasty. A multicenter study. Am J Cardiol 1983; 52:710-3.

44. David PR, Renkin J, Moise A, Dangoisse V, Val PG, Bourassa MG. Can patient selection and optimization of technique reduce the rate of restenosis after percutaneous transluminal coronary angioplasty? J.A.C.C. 1984; 3:470 (abstract).

45. Ischinger T, Gruentzig AR, Hollman J, King S III, Douglas J, Meier B, Bradford J, Tankersley R. Should coronary arteries with less than 60% diameter stenosis be treated by angioplasty? Circulation 1983; 68:148-54.

46. Dorros G, Cowley MJ, Simpson J, Bentivoglio LG, Block PC, Bourassa M, Detre K, Gosselin AJ, Gruentzig AR, Kelsey SF, Kent KM, Mock MB, Mullin SM, Myler RK, Passamani ER, Stertzer SH, Williams DO. Percutaneous transluminal coronary angioplasty: Report of complications from the National Heart, Lung, and Blood Institute PTCA registry. Circulation 1983; 67:723-30.