

Percutaneous transluminal coronary angioplasty

Richard K. Myler, M.D.

San Francisco, California

It has been a half century since Forssman in Germany initiated the era of invasive diagnostic cardiology. Following his courageous lead, further definition of the possibilities of cardiac catheterization occurred in the United States in the early 1940s. The cardiac catheter as a diagnostic tool achieved a dramatic new plateau two decades ago in Cleveland when Sones et al¹ realized the potential for investigating coronary anatomy, normal and abnormal, and a new era in investigational cardiology began.

The cardiovascular catheter has also been an equally interesting therapeutic device. It has been used to open patent foramen ovale in transposition, close atrial septal defects and patent ductus arteriosus, interrupt inferior vena caval return in patients with recurrent pulmonary embolic disease, and treat heart block (in its many forms) with a variety of brilliant pacemaker catheter devices.

A particularly imaginative therapeutic catheter application was introduced by Dotter and Judkins² in 1964 to improve peripheral blood flow in arteries with diffuse and discrete arteriosclerotic stenosis. This system utilized a coaxial catheter system and the procedure was termed transluminal angioplasty. Their pioneering efforts were developed further in Europe by many investigators, in particular,

Zeitler³ in Nuremberg, Grüntzig^{4,5} in Switzerland who modified the Dotter technique by employing a single catheter system with a distensible (balloon) tip, which achieved great success in treating peripheral arteriosclerotic lesions (initial patency, 83%; 3-year patency, 73%).

In 1976 Grüntzig et al⁶⁻⁸ further modified the peripheral angioplasty system to perform coronary angioplasty initially in dogs and subsequently in human cadaver studies. Then in San Francisco and Zurich, Gruentzig et al⁹ first performed intraoperative coronary angioplasty to examine the technique in dilating living human atherosclerotic plaques and determining whether distal debris would be produced. (It was never noted in these studies.)

Then following a period of probing human coronary arteries in the catheterization laboratories in Zurich and San Francisco measuring pressure gradients across lesions and performing subselective coronary arteriography (without complications). On September 16, 1977, in Zurich, Grüntzig¹⁰ performed the first human percutaneous transluminal coronary angioplasty. This technique utilized a guiding catheter, through which a dilation catheter was passed. The dilation catheter then entered the stenotic arterial segment, traversed the lesion under pressure and angiographic control and was inflated with a pump-controlled pressure of 4 to 6 atmospheres of pressure.

Presently, in 19 centers in Europe and the United States, more than 250 attempts have been made to perform percutaneous transluminal coronary angiography. In the four major centers (Zurich, San Francisco, New York, and Frankfurt) more than 200 cases have been submitted to percutaneous transluminal coronary angiography with a suc-

cess rate of 65% measured by anatomic improvement of coronary luminal stenosis of 85% (mean) to 31% (mean) and significant reduction in mean pressure gradients measured across the lesions following successful "dilation". Corroborative evidence for clinical improvement following percutaneous transluminal coronary angiography was demonstrated by exercise studies (treadmill testing or bicycle ergometry) and thallium and wall motion cardiac scintigraphy.¹⁰⁻¹⁵

Although restenosis developed in about 10% of cases, a larger number actually showed improvement in angiographically defined "percent diameter stenosis" in later studies compared to initial postpercutaneous transluminal coronary angiography arteriographic definition. The explanation for this improvement is unclear at present, but perhaps is related to healing of the "controlled injury" (caused by the balloon inflation, which leads to circumferential compression of the plaque) and leads to improved pressure and flow characteristics of blood passing through the now enlarged coronary lumen.¹⁶

Complications have occurred. In less than 6% of the series, there has been abrupt reclosure of the dilated coronary segment, either due to spasm or localized dissection, and has required emergency coronary bypass surgery. Only one patient in the entire series died as a direct result of the procedure, and death was probably caused by dissection of the left main coronary artery by the guiding catheter. Five other patients in the entire series died days to months following percutaneous transluminal coronary angiography, and apparently in many instances death was unrelated to the procedure.

The experience in San Francisco is illustrated in *Table 1*. Since the initial

Table 1. St. Mary's Hospital and Medical Center; percutaneous transluminal coronary angioplasty (March 1, 1978 to August 1979)

Vessels attempted					
LCA	LAD	RCA	LCx	SVG	Total
4	42	16	2	0	64
Patients, 63			Age range, 26-73		
Men, 49; women, 14			Single vessel, 40		
Postoperative coronary bypass grafting, 7			Multiple vessel, 23		

LCA = left coronary artery, LAD = left anterior descending, RCA = right coronary artery, LCx = left circumflex, SVG = saphenous vein graft.

case was performed in San Francisco on March 1, 1978, we have attempted percutaneous transluminal coronary angiography on 63 patients, with an age range of 26 to 73 years (mean, 52 years), 49 men and 14 women, with angina pectoris refractory (relatively) to medical therapy with abnormal treadmill test and/or thallium cardiac scintigraphy. These patients were considered by their medical and surgical cardiac physicians to be candidates for coronary artery bypass grafting. In this series of 63 patients, 40 had single-vessel disease and 23 had evidence of significant disease in more than one vessel. Seven patients had had coronary artery bypass grafting with closure of at least one graft or progressive sclerosis in the active circulation. Of the 63 patients, there were 64 lesions "attempted". Thirty-eight had the dilation catheter successfully pass the lesion and dilate the lumen showing at least a 20% improvement in percent luminal diameter. Of these 38 patients, two required emergency bypass surgery, one because of localized spasm at the site of the lesion and the other because of spasm noted in another part of the coronary arterial system (this patient had organic and coronary spastic disease).

Two patients died in the 17 months

since our series began. One patient (the one with both organic and spastic coronary disease) died with *Pseudomonas* septicemia secondary to saphenous vein donor site infection 9 weeks postoperatively (and postpercutaneous transluminal coronary angiography). A small subendocardial infarction developed in the other patient 2 hours following successful percutaneous transluminal coronary angiography (she had undergone coronary artery bypass grafting 6 years earlier, which subsequently occluded). Two days later, although stable clinically, she underwent elective double coronary artery bypass grafting and arrested in the operating room; she died 2 days postoperatively. Her postmortem examination showed a 5-mm dissection in the region of the stenosis, but the arterial lumen was still patent. Of the 25 patients in whom the lesion could not be passed with the dilation catheter either because the stenosis was too severe or the stenotic arterial branch could not be entered with the dilation catheter, 18 have already undergone elective coronary artery bypass grafting (usually in their own "home" centers) and seven are awaiting elective coronary artery bypass grafting. In the restudy period, three patients have shown signs of significant restenosis, (one, a 73-year-old patient in whom coronary artery bypass grafting had failed and two others who showed progression not only in the dilated segments, but also in other areas of the coronary arterial system). Twelve other patients restudied at 6 to 12 months postpercutaneous transluminal coronary angiography have either shown continued patency or actual improvement in luminal diameter compared to the immediate postpercutaneous transluminal coronary angiography arteriograms.

At present and after our experience of

nearly 2 years, there are certain clear indications for percutaneous transluminal coronary angiography (Table 2). We would not recommend percutaneous transluminal coronary angiography for patients with left main coronary artery disease (unless they had post-coronary artery bypass grafting with at least one patent distal left coronary artery graft), because three of the five late deaths in the entire series occurred in patients with left main coronary artery disease (perhaps due to coronary spasm). We agree that coronary artery bypass grafting is a better form of therapy for these patients in terms of annual attrition.

It should be emphasized that this has been a medical-surgical collaborative study from its inception and surgical standby is mandatory.

Still many questions posed 2 years ago remain, although some can be at least in part answered now:

1. Does percutaneous transluminal coronary angiography work? Apparently yes.
2. What is the success rate? Presently about 65%, although improvement in technique, technology, and patient selection should increase this percentage in the future.
3. What are the risks and do they outweigh the potential benefits? In a single-vessel disease at the present time the risks are minimal and the

potential benefits considerable.

4. What types of patients and how many are candidates for percutaneous transluminal coronary angiography? This is a more difficult question. At present, patients with single-vessel disease who are candidates for coronary artery bypass grafting and for whom the procedure has been recommended may be candidates for percutaneous transluminal coronary angiography. How many patients in a population would meet these criteria depends upon many factors including the age of the coronary population under investigation, aggressiveness or conservativeness of the local medical and surgical cardiac teams. We estimate that in our centers this percentage varies between 3% and 15%.
5. How does percutaneous transluminal coronary angiography compare to medical and surgical therapy? This will be discussed more fully later, but briefly, percutaneous transluminal coronary angiography is comparable in single-vessel disease, but a higher mortality was noted in the left main artery disease in terms of annual attrition (vs. coronary artery bypass grafting).
6. Although it seems apparent why dilated arterial segments might develop restenosis, we noted angiographic improvement in many instances, and this observation has been corroborated in several centers and in both coronary and peripheral arteries treated by transluminal angioplasty. The cause of this improvement remains the single most provocative question at the present time. There has been considerable interest in pathologic investigation of the effects of percutaneous transluminal coronary angiography in human ca-

Table 2. Indications for percutaneous transluminal coronary angioplasty

Single-vessel disease with lesions
Proximal
Concentric
Discrete
Subtotal
Noncalcific
Must be candidates for coronary artery bypass grafting, surgical standby
Avoid coronary spasm

daver hearts,^{8, 17-21} and an experimental model.²²

Finally, it should be stated that this has been a collaborative effort from its beginning, with scientists and technicians from independent laboratories in Europe and the United States working together, sharing information, and helping each other. Now we are joined by the resources of the National Institutes of Health²³ to develop a registry to gather and tabulate information about patients who have been submitted to percutaneous transluminal coronary angiography so that the procedure can continue in a prospective, controlled fashion.

References

1. Sones FM Jr, Shirey EK, Proudfit WL, et al: Cine-coronary arteriography. *Circulation* **20**: 773-774, 1959.
2. Dotter CT, Judkins MP: Transluminal treatment of arteriosclerotic obstruction: description of a new technic and a preliminary report of its application. *Circulation* **30**: 654-670, 1964.
3. Zeitler E, Grüntzig A, Schoop W, eds. *Percutaneous Vascular Recanalization*. New York, Springer-Verlag, 1978.
4. Grüntzig AR: Die perkutane Rekanalisation chronischer arterieller Verschlüsse mit einem doppelumigen Dilatations-katheter (Dotter-Prinzip). *Fortschr Röntgenstr* **124**: 80-86, 1976.
5. Grüntzig AR: Die perkutane transluminale Rekanalisation chronischer Arterienverschlüsse mit einer neuen Dilatations-technik. Baden-Baden, G. Witzstrock Verlag, 1977.
6. Grüntzig AR: Perkutane Dilatation von Koronarstenosen-Beschreibung eines neuen Kathetersystems. *Klin Wochenschr* **54**: 543-545, 1976.
7. Grüntzig AR, Turina MI, Schneider JA: Experimental percutaneous dilatation of coronary artery stenosis. (Abstr) *Circulation* **54** Suppl II: 81, 1976.
8. Grüntzig A, Schneider HJ: Die perkutane Dilatation chronischer Koronarstenosen—Experiment und Morphologie. *Schweiz Med Wochenschr* **107**: 1588, 1977.
9. Gruentzig AR, Myler RK, Hanna ES et al.; Coronary transluminal angioplasty. (Abstr) *Circulation* **55** and **56**: (Suppl III):III-84, 1977.
10. Grüntzig A: Transluminal dilatation of coronary-artery stenosis. *Lancet* **1**: 263, 1978.
11. Grüntzig A, Myler R, Stertz S, et al: Coronary percutaneous transluminal angioplasty: preliminary results. (Abstr) *Circulation* **57** and **58** (Suppl II):II-56, 1978.
12. Hirzel HO, Gruentzig A, Nuesch K, et al: Thallium-201 imaging for the evaluation of myocardial perfusion after percutaneous transluminal angioplasty of coronary artery stenosis. (Abstr) *Circulation* **58** (Suppl II):II-180, 1978.
13. Stertz SH, Myler RK, Bruno MS, et al: Transluminal coronary artery dilatation. *Pract. Cardiol* **5**: 25-32, 1979.
14. Grüntzig AR, Senning A, Siegenthaler WE: Nonoperative dilatation of coronary-artery stenosis; percutaneous transluminal coronary angioplasty. *N Engl J Med* **301**: 61-68, 1979.
15. Grüntzig AR, Myler RK, Stertz S: Percutaneous transluminal coronary angioplasty (PTCA); present state of the art. *Circulation* In press.
16. Robbins SL, Bentov I: The kinetics of viscous flow in a model vessel; effects of stenoses of varying size, shape, and length. *Lab Invest* **16**: 864-874, 1967.
17. Baughman KL, Pasternak RC, Fallon JT, et al: Coronary transluminal angioplasty in autopsied human hearts. (Abstr) *Circulation* **57-58** (Suppl II):II-80, 1978.
18. Pasternak RC, Baughman KL, Fallon JT, et al: Scanning electron microscopy following coronary transluminal angioplasty with balloon dilatation catheter. (Abstr) *Circulation* **57-58** (Suppl II): 242, 1978.
19. Freudenberg H, Wefing H, Lichtlen PR: Risks of transluminal coronary angioplasty: a post-mortem study. (Abstr) *Circulation* **57** and **58** (Suppl II):II-80, 1978.
20. Simpson JB, Robert EW, Billingham, ME, et al: Coronary transluminal angioplasty in human cadaver hearts. (Abstr) *Circulation* **57** and **58** (Suppl II):II-80, 1978.
21. Lee G, Ikeda RM, Joye JA, et al: Evaluation of transluminal angioplasty of chronic coronary artery stenosis; value and limitations assessed in fresh human cadaver hearts. *Circulation* **61**: 77-83, 1980.
22. Block PC, Baughman KL, Pasternak RC, et al: Transluminal angioplasty; correlation of morphologic and angiographic findings in an experimental model. *Circulation*, **61**: 778-785, 1980.
23. Levy RI, Jesse MJ, Mock MB: Position on percutaneous transluminal coronary angioplasty (PTCA). *Circulation* **59**: 613, 1979.