of patient selection is clearly evident when PTCA is attempted in the presence of intracoronary thrombus. In one series,[1] dilatation was complicated by occlusion in 73% of patients compared to only 8% when thrombus was absent; the latter was a result of dissection.

We derived a simple risk factor score from reports of the American College of Cardiology and the American Heart Association Task Force and major university hospitals.[2,3] Our results confirm the validity of these guidelines. (Possibly, the risk score should have been weighted, so that thrombus was allocated at least 2 points.) Risk factors were identified by retrospective analysis of very large numbers of cases with a low rate of complications. Allowing for the technical expertise and experience of these operators, one could also assume that these risk factors were absent in most patients who did well, and the clinical material was therefore selected. With awareness of these lesion-specific risk factors, we believe we can now refer patients who will have fewer complications. Indeed, careful selection of low risk patients could probably blunt the so-called learning curve for the new PTCA operator. The results of PTCA would be more meaningful if, in future studies, risk factors were clearly defined prospectively for each patient. Because we acted as referring physicians, our data may have relevance to the results of PTCA in the community. Although the results are less than satisfactory, they may be similar to those obtained outside major centers that choose to report their results.

In summary, careful assessment of angiographic lesion-specific risk factors can identify a group of patients at high risk for intimal dissection and coronary artery occlusion. Because of the excellent prognosis of patients with 1-vessel disease treated medically,[4] a high risk factor score may militate against PTCA and suggest further medical treatment or coronary artery bypass surgery.

Addendum: Since submission of this manuscript, an additional 66 patients had 79 arteries dilated (66 one- and 13 two-vessel) at institution C between May 1988 and May 1989. The primary success rate was 94%. Acute closure with nonfatal myocardial infarctions complicated 4 cases (6%). The lesion risk factor score was 1.75 ± 0.48 for acute occlusion and 0.44 ± 0.8 (p <0.001) for primary success. These findings confirm the favorable effect of patient selection.

1. Mabin TA, Holmes DR, Smith HC, Vlietstra RE, Bove AA, Reeder GS, Chesebro JH, Bresnahan JF, Orszulak TA. Intracoronary thrombus: role in coronary occlusion complicating percutaneous transluminal coronary angioplasty. JACC 1985;5:198–202.
2. Ryan TJ, Faxon DP, Gunnar RM, Kennedy JW, King SB III, Loop FD, Peterson KL, Reeves TJ, Williams DO, Winters WL Jr, Fisch C, DeSanctis RW, Dodge HT, Weinberg SL. Guidelines for percutaneous transluminal coronary angioplasty. JACC 1988;12:529–545.
3. 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;72:1044–1052.
4. Danchin N, Brengard A, Ethevenot G, Briancon S, Cuilliere M, Aliot E, Pernot C, Gilgenkrantz JM, Mathieu P, Cherrier F. Ten year follow up of patients with single vessel coronary artery disease that was suitable for percutaneous transluminal coronary angioplasty. Br Heart J 1988;59:275–279.

Restenosis After Excellent Angiographic Angioplasty Result for Chronic Total Coronary Artery Occlusion— Implications for Newer Percutaneous Revascularization Devices

Stephen G. Ellis, MD, Richard E. Shaw, PhD, Spencer B. King, III, MD, Richard K. Myler, MD, and Eric J. Topol, MD

The incidence of restenosis after coronary angioplasty for treatment of chronic total coronary occlusion is unacceptably high.[1] The pathophysiology of restenosis after coronary angioplasty may be conceptually divided into an exuberant myointimal proliferation,[2] and a residual partial obstruction that serves as a platform for atheroma regrowth and may potentiate that process by augmenting blood flow turbulence and platelet deposition. The techniques of atherectomy or laser ablation[3] may lessen the likelihood of restenosis by minimizing the residual stenosis, although currently each may require supplemental balloon angioplasty to achieve this result. However, the effect of these techniques on later myointimal proliferation in human beings is largely unknown. The

concept that intracoronary stenting[4] may reduce restenosis is based largely on the supposition that, by forcing and maintaining the obstructive atheroma out of the normal arterial lumen, turbulence and hence platelet deposition would be reduced[5] and a large amount of myointimal proliferation would be required to recreate an obstruction of physiologic consequence.

To assess the likelihood of restenosis after an excellent angiographic result with routine coronary angioplasty for chronic total occlusion, and thereby to provide inferential data on the likelihood that newer interventional techniques will decrease restenosis in this setting, we reviewed all patients with successful coronary angioplasty (final diameter stenosis <50% by electronic calipers or a validated computer-edged detection system) for chronic total occlusion (>3 days' duration) and angiographic follow-up from Emory University Hospital, the San Francisco Heart Institute and the University of Michigan Hospital before December 31, 1986. There were 257 stenoses that met these criteria, representing 53% of all chronic total occlusions successfully dilated

TABLE I Angiographic Documentation of Restenosis			
	Within 3 Months*	Within 6 Months*	Within 12 Months*
Group 1 (n = 37)			
Chronic total occlusion, final stenosis ≤10% (%)	11	30	52
Group 2 (n = 69)			
Chronic total occlusion, final stenosis 11–20% (%)	14	39	59
Group 3 (n = 151)			
Chronic total occlusion, final stenosis >20% (%)	24	45	73†
Group 4 (n = 56)			
Subtotal occlusion, final stenosis 0–49% (%)	7	26	39‡

* By lifetable analysis; † p <0.001 group 3 versus groups 1 and 2 by log rank analysis; ‡ p <0.01 vs groups 1 through 3 by log rank analysis.

during this time period. Patient age was 54 ± 10 years (mean ± standard deviation), 79% were men, the duration of occlusion was 11 ± 13 weeks and the diameter stenosis after angioplasty was 24 ± 12%. During follow-up, 82% of patients received aspirin, 27% received dipyridamole and 63% received warfarin. Stenoses were prospectively divided by final diameter stenosis. Group 1 comprised 37 patients with final stenosis 0 to 10%, group 2 included 69 patients with final stenosis 11 to 20% and group 3 had 151 patients with final stenosis >20%. In addition, 56 stenoses with initial diameter stenosis 50 to 95% provided a "control" to inferentially assess the effect of incomplete angiographic follow-up in these patients (group 4). Groups 1 through 3 did not differ (Pearson chi-square >0.05) with regard to duration of

total occlusion or distribution of arteries dilated, factors known to influence the risk of restenosis in this setting.[1]

Restenosis (diameter stenosis ≥50% from multiple views) was angiographically documented as listed in Table I. Thus, although the exact rates of restenosis cannot be determined without 100% angiographic follow-up, these data suggest that, although final diameter stenosis is an important predictor of risk of restenosis after angioplasty for chronic total occlusion, even chronic total occlusions with excellent angiographic results after angioplasty are at high risk for restenosis. These findings may have important implications regarding the likelihood of restenosis after successful recanalization by newer ablative techniques or intracoronary stenting in this setting. Pharmacologic, immunologic or mechanical methods of altering the neointimal blood interface and thereby preventing myointimal hyperplasia may be required to markedly reduce the risk of restenosis after any mechanical intervention to treat chronic total coronary artery obstruction.

1. Ellis SG, Shaw RE, Gershony G, Thomas R, Roubin GS, Douglas JS, Topol EJ, Stertzer SH, Myler RK, King SB. Risk factors, time course, and treatment effect for restenosis after successful percutaneous transluminal coronary angioplasty of chronic total occlusion. Am J Cardiol 1989;63:897–901.
2. Austin GE, Ratliff NB, Homan J, Tabei S, Phillips DG. Intimal proliferation of smooth muscle cells as an explanation of recurrent coronary artery stenosis after percutaneous transluminal coronary angioplasty. JACC 1985;6:369–375.
3. Topol EJ. Emergency strategies for failed percutaneous transluminal coronary angioplasty. Am J Cardiol 1989;63:249–251.
4. Ellis SG, Topol EJ. Intracoronary stents—will they fulfill their promise as an adjunct to coronary angioplasty? JACC;13:1425–1430.
5. Jorgenson L, Packham MA, Rowsell HC, Mustard JF. Exposition of formed elements of blood from the intima and signs of intimal injury in the aorta of a rabbit, pig, and man. Lab Invest 1972;27:341–350.

Assessment by Doppler Color Flow Mapping of Ventricular Septal Defect After Acute Myocardial Infarction

Gerald Maurer, MD, Lawrence S.C. Czer, MD, Prediman K. Shah, MD, and Aurelio Chaux, MD

Rupture of the ventricular septum is an uncommon but life-threatening complication of acute myocardial infarction (AMI). It occurs in <1% of infarcts. The noninvasive evaluation of such defects by 2-dimensional echocardiography has been reported.[1] These defects, however, can be difficult to image, particularly after anterior AMI,[2,3] when they are usually located in the apical region. Doppler color flow mapping allows visualization of the shunt flow abnormality and may thus aid in the preoperative assessment of such defects. Color flow mapping has been used intraoperatively to evaluate valve repair[4] and closure of congenital shunt lesions.[5] It may also be a useful tool in assessing the adequacy of repair of

ventricular septal rupture after AMI, which continues to carry a high mortality rate despite refinement of surgical technique. We report the use of Doppler color flow mapping for the bedside assessment of ventricular septal defects complicating AMI, and its intraoperative application to assess the effectiveness of surgical repair.

We studied 6 consecutive patients who presented to our coronary care unit with clinical manifestations suggestive of acute ventricular septal rupture after anterior (n = 4) or inferior wall (n = 2) AMI. The relevant clinical features are listed in Table I. The diagnosis of ventricular septal rupture was confirmed in all patients by oximetry, as well as in 4 of the 6 patients at the time of surgery. Two patients died before surgery and autopsies were not performed.

Echocardiographic imaging was performed from the parasternal, apical and subcostal windows. In addition to the standard planes, intermediate views were obtained by angling the transducer to visualize the defect, as well as the shunt flow, optimally.