Intracoronary Stents

RAJIV JAUHAR, M.D., CARDIOLOGY FELLOW
DAVID S. BATTLEMAN, M.D., MEDICAL RESIDENT
DORY B. ALTMANN, M.D., ASSISTANT PROFESSOR OF MEDICINE
MANISH PARIKH, M.D., CARDIOLOGY FELLOW
JACK FLYER, M.D., CARDIOLOGY FELLOW
LISA AUTZ, R.N., RESEARCH NURSE
TIMOTHY A. SANBORN, M.D., DIRECTOR
CARDIAC CATHETERIZATION LABORATORY, DEPARTMENT OF CARDIOLOGY
THE NEW YORK HOSPITAL–CORNELL MEDICAL CENTER, NEW YORK, NEW YORK

Interventional cardiology has undergone exciting changes since the first percutaneous transluminal coronary angioplasty was performed by Andreas Gruentzig in 1977.[1] Over the last several years, a variety of techniques have been developed which provide the interventional cardiologist with a range of options to treat coronary stenoses. In addition, the indications for intervention have radically changed since the original work by Gruentzig, who limited treatment to patients with a single focal stenosis in a large vessel. Advances in balloon angioplasty, as well as the development of new techniques such as directional atherectomy, laser angioplasty, rotational atherectomy, extraction atherectomy, and now intracoronary stents have given the interventional cardiologist the ability to treat multivessel disease, increasingly complex lesions, and less stable patients.[2]

LIMITATIONS OF BALLOON ANGIOPLASTY

Despite improvements over the past 15 years in equipment design and operator experience, significant limitations still remain with balloon angioplasty. The risk of abrupt vessel closure, the total or near-total occlusion of a vessel following angioplasty with the loss of antegrade blood flow, is 2% to 7%.[3,4] This complication is associated with significant morbidity and mortality. Myocardial infarction occurs in approximately 40% of these patients and approximately one-third require emergency bypass surgery.[4,5] Emergency bypass surgery after failed angioplasty carries an increased mortality compared with elective surgery.[6,7] Extensive coronary artery dissection secondary to balloon injury is the most frequent cause of abrupt closure.[8] However, it is thought that endothelial damage, thrombus formation, and vasospasm may also contribute to this problem.[2]

Another significant limitation of balloon angioplasty is restenosis at the site of dilatation. After angioplasty, angiographic restenosis, generally defined as the recurrence of a >50% stenosis within 6 months, occurs in approximately 40% of patients with native coronary lesions[9] and a greater percentage of patients with saphenous vein graft lesions.[10] Clinically significant restenosis is less common. For example, in a recent study, the angiographic restenosis rate after balloon angioplasty was 42%. Revascularization of the original lesion because of recurrent ischemia was performed in 15% of patients.[11] Attempts to reduce the restenosis rate with drugs or newer angioplasty techniques have, until recently, been unsuccessful.

The etiology of restenosis is felt to be both smooth muscle cell and extracellular matrix proliferation (intimal hyperplasia) as well as vessel wall remodeling (Fig. 1). Remodeling refers to a dynamic decrease in arterial size that produces luminal narrowing after angioplasty which cannot be accounted for by tissue proliferation. Geometric remodeling may contribute up

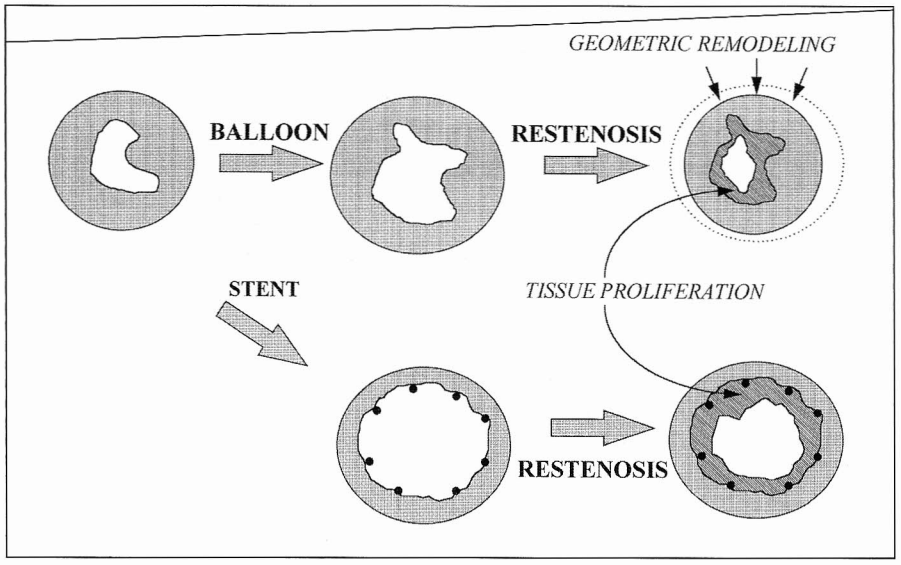

Figure 1. Restenosis. Restenosis resulting in narrowing after balloon angioplasty is a consequence of both tissue proliferation and geometric remodeling of the vessel. Stents serve as a rigid scaffolding to prevent geometric remodeling.

to two-thirds of the late lumen cross-sectional area loss after angioplasty.[12]

Several devices, including directional atherectomy and excimer laser angioplasty, have been developed to address this problem. Directional atherectomy involves the use of a catheter-guided cutting device to remove atherosclerotic plaque. Laser angioplasty uses a focused high energy laser to ablate atheromatous plaques. However, in clinical trials to date, these new devices have been of little benefit in reducing restenosis rates.[13] Randomized trials comparing directional atherectomy with balloon angioplasty have not shown significant differences in follow-up angiographic results or patient outcomes.[14,15]

Thus, the need for an alternative procedure to address these two major limitations of balloon angioplasty, abrupt closure and restenosis, was the rationale for the development of intracoronary stents.

INTRACORONARY STENTS

Intracoronary stents have dramatically changed the way interventional cardiology is practiced. Beginning with the first report by Dotter in 1969, in which it was shown that scaffolding with an open coil spring made long-term patency possible, the field has undergone rapid evolution.[16] In theory, endovascular scaffolding (stenting) would seal dissections, resist vessel remodeling, and ultimately reduce restenosis. Several types of stents have been developed. However, the only devices currently approved by the Food and Drug Administration for intracoronary use are the Palmaz–Schatz and Gianturco–Roubin stents.

The Palmaz–Schatz stent is a stainless steel stent, designed as a continuous tubular mesh. To aid in its flexibility, the stent has two segments joined by a single "articulation" filament.[17] The Gianturco–Roubin stent is a flexible stainless steel stent, composed of monofilamentous stainless-steel loops. This stent, in contrast to the Palmaz–Schatz stent, has an interdigitating coil-like structure.[18,19] Both stents are wrapped around a polyethylene balloon that expands the stent when inflated at the lesion site. The balloon is subsequently deflated and removed, leaving the expanded stent in place. In this report, we describe our results with the Palmaz–Schatz and Gianturco–Roubin stents in the first 213 consecutive patients treated at New York Hospital–Cornell Medical Center with attention to acute (in-hospital) complications. This data will be discussed in the context of other clinical trials.

METHODS

The study population consisted of the first 213 consecutive patients treated with either the Gianturco–Roubin or Palmaz–Schatz stent at New York Hospital–Cornell Medical Center from October 1993 through December 1995. The indications for stenting were either emergency (threatened or abrupt closure) or elective (restenosis prevention). Threatened closure was defined as a suboptimal angiographic result after balloon angioplasty that placed the patient at increased risk for abrupt closure despite normal blood flow. Abrupt closure was defined as the sudden decrease or complete loss of flow beyond the lesion. Elective stenting was considered in patients with objective evidence of ischemia attributable to either a de novo or restenotic lesion. The indications for elective stent placement were (a) stenosis > 70% with documented ischemia, (b) vessel diameter greater than 2.5 mm, and (c) no contraindications to anticoagulation. The patients were followed throughout their hospital stay and then at 1 week, 1 month, 6 months, and 12 months after discharge.

Major in-hospital cardiac complications were defined as death from any cause, Q wave myocardial infarction, or the need for emergency angioplasty or bypass surgery. A vascular complication was said to have occurred when there was a pseudoaneurysm requiring manual compression or surgical repair, arteriovenous fistula, or retroperitoneal hemorrhage.

Stent implantation was performed according to standard clinical practice using the femoral artery approach through a No. 8 French guiding catheter. After fluoroscopic confirmation of optimal position, the device was deployed with balloon inflation to full expansion. Beginning in March 1995, the stented area was further dilated at high pressure in order to achieve maximal stent expansion. For the majority of patients, the expansion of the stent was assessed by angiography alone. However, a small number of patients had stent deployment and expansion confirmed by intravascular ultrasound as well.

Several anticoagulation protocols were employed during this study. The original protocol included aspirin 325 mg po qd, dipyridamole 75 mg po tid, 10% dextran 40, and a bolus injection of heparin (10,000 U) followed by additional injections to maintain an activated clotting time >300 seconds. The arterial sheath was removed either approximately 4 hours after the procedure or the following day, and a heparin infusion was begun 6 hours after hemostasis of the vascular access site. Dextran was stopped once the partial thromboplastin time was >50 seconds on heparin. Warfarin was begun on the day of the procedure and continued until an international normalized ratio of 2.0 - 3.0 was achieved. After discharge from the hospital, warfarin was continued for 1 to 2 months. As a modification of this standard protocol, dextran and persantine were no

longer used after March 1995. Antiplatelet therapy alone (aspirin, usually with ticlopidine 250 mg bid) was applied in patients in whom intravascular ultrasound confirmed optimal deployment and complete stent expansion.

Categorical data are presented as prevalence rates and continuous data as mean ± 1 SD. The Fisher Exact test (two-tail) was used for analysis of discrete variables and Student's t test for continuous variables. A p value less than 0.05 was considered statistically significant.

RESULTS

Between October 1993 and December 1995, intracoronary stents were deployed in 213 patients, with 68% undergoing elective stent placement and 32% emergency stent placement (Table 1). Overall, stents were successfully deployed in 96% of patients. Angiographic examples of intracoronary stenting for abrupt and threatened closure are shown in Figures 2 and 3.

Table 1. Baseline characteristics and outcome in patients undergoing elective or emergency stenting

	OVERALL N=213	ELECTIVE N=145	EMERGENCY N=68
Age (years)	60.5 ± 9.4	60.7 ± 9.4	60.0 ± 10.3
Male (%)	165 (78)	121 (83)	44 (65)*
In-hospital Outcome			
Death (%)	1 (0.5)	1 (0.7)	0 (0)
Q Wave MI (%)	3 (1.4)	2 (1.4)	1 (1.5)
Bypass Surgery (%)	7 (3.3)	3 (2.1)	4 (5.9)
Emergency PTCA (%)	4 (1.9)	4 (2.8)	0 (0)
Any Major Complication (%)	12 (5.6)	7 (4.8)	5 (7.4)
Stent Thrombosis	8 (3.8)	3 (2.1)	5 (7.4)
Vascular Complication (%)	11 (5.2)	6 (4.1)	5 (7.4)

MI = myocardial infarction; PTCA=percutaneous transluminal coronary angioplasty
*p<0.01 compared with elective

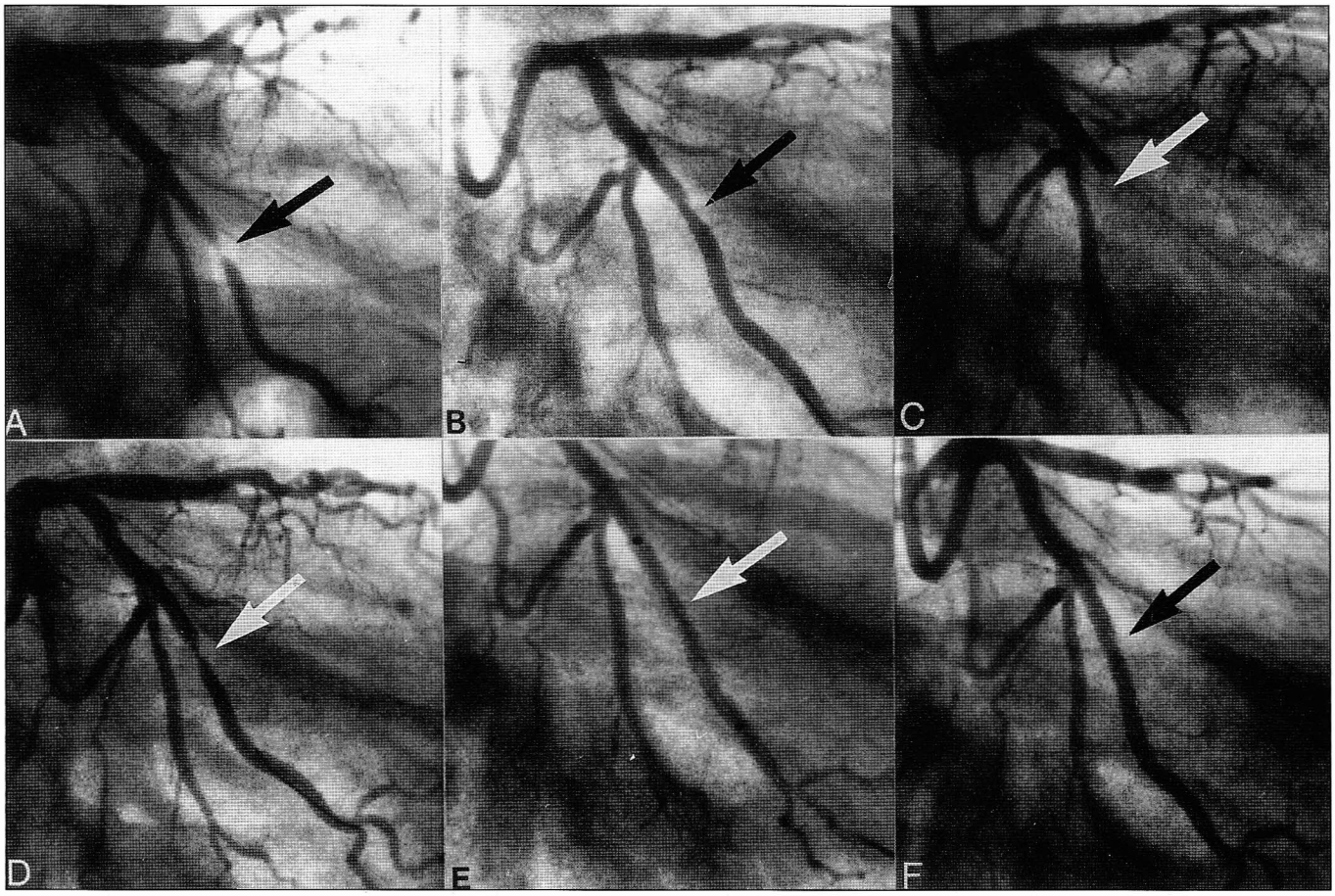

Figure 2. Abrupt closure treated by stenting. (A) Baseline angiogram with 90% stenosis in an obtuse marginal artery (arrow). (B) Result after balloon angioplasty (arrow). (C) Emergency angiogram performed because of chest pain with EKG changes shortly after the procedure. There is acute occlusion of the vessel (arrow). (D) Repeat balloon angioplasty with prolonged balloon inflation still yielded a suboptimal result (arrow). (E) Deployment of a Gianturco–Roubin stent. Stent lies on the balloon between the two black fluoroscopic markers. (F) Final result.

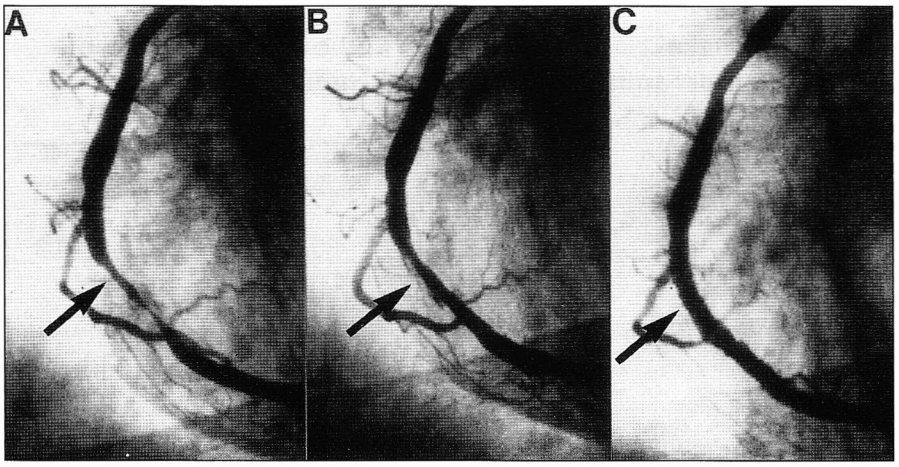

Figure 3. Threatened closure treated with a stent. (A) Baseline angiogram with a stenosis in the right coronary artery (arrow). (B) After balloon angioplasty, there is evidence of a dissection (arrow) which persisted despite prolonged balloon inflation. (C) Final result after placement of an intracoronary stent (arrow).

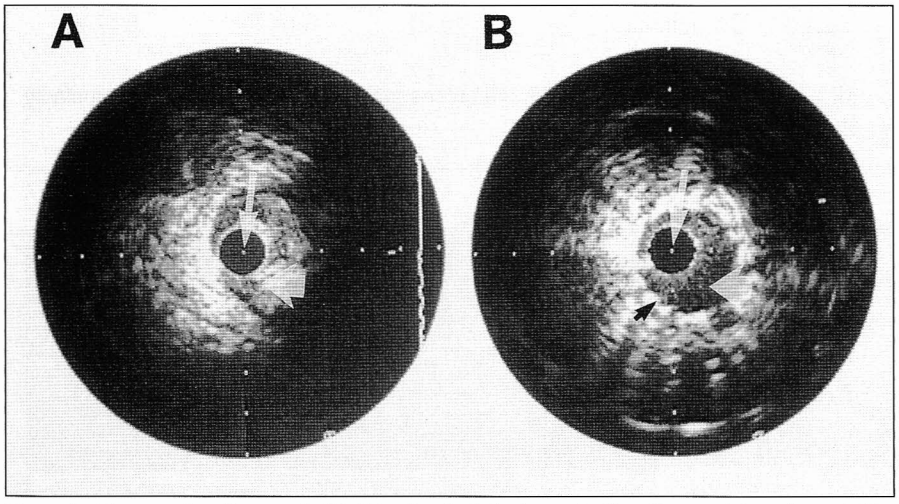

Figure 4. Intravascular ultrasound. (A) Prior to stent deployment, the ultrasound transducer (white arrow) is completely surrounded by atherosclerotic plaque (large white arrow). (B) After deployment of an intracoronary stent, a large lumen is created (large white arrow). Note struts of the stent (small black arrowhead) completely apposed to the vessel wall.

Patient Outcome

In-hospital clinical events for emergency and elective stenting are shown in Table 1. The rate of in-hospital major cardiac events was 4.8% in the elective group and 7.4% in the emergency group (p = NS). In-hospital death occurred in one patient (0.7%) in the elective stent group. That death occurred as a complication of stent thrombosis 4 days after stent implantation. Stent thrombosis occurred in 10 patients overall (4.7%) and in 3.4% and 7.4% of elective and emergency stent recipients, respectively (p = NS). Two patients (1.4%) who had undergone elective stenting developed abrupt stent thrombosis after hospital discharge while all other thromboses occurred in-hospital. Eleven patients developed vascular complications. These consisted of pseudoaneurysms in nine patients and retroperitoneal hemorrhage in two patients. One patient who had a retroperitoneal hemorrhage also developed an arteriovenous fistula. Among the 22 patients treated with antiplatelet therapy alone, there were no major cardiac complications in-hospital or within 14 days of the procedure; however, one patient did develop a pseudoaneurysm.

DISCUSSION

Stent Implantation for Acute or Threatened Closure

Intracoronary stenting has been developed, in part, to treat acute or threatened vessel closure after angioplasty. It provides an endoluminal support structure to seal dissections and maintain lumen patency. The efficacy of intracoronary stenting for acute or threatened closure was established in the Multicenter Registry of Acute and Elective Gianturco–Roubin stent placement.[20,21] In that study, intracoronary stents were shown to be both safe and effective in the treatment of abrupt or threatened closure. Only 3.0% suffered a Q wave myocardial infarction and 4.3% of patients required emergency bypass surgery.[20] These major cardiac complication rates after stent placement compared favorably to previous studies of acute closure after balloon angioplasty in which myocardial infarction occurred in approximately 40% of these patients and about one-third underwent bypass surgery.[4,5] The difference in morbidity may reflect more rapid reestablishment of coronary blood flow compared to bypass surgery.

Stent Implantation for Restenosis

Intracoronary stents have now been shown to reduce the rate of restenosis. Two randomized studies (STRESS and BENESTENT) compared elective intracoronary stents versus balloon angioplasty in patients with focal lesions (<15 mm length) in large native coronary arteries (>3 mm diameter).[11,22] These studies had angiographic stent restenosis rates of 22% and 32%, respectively. This was significantly less than the corresponding angioplasty restenosis rates of 32% and 42% ($p < 0.05$ for both). Intracoronary stenting represents the first clearly established therapy for restenosis and therefore a major advance in interventional cardiology. We are unable to report our restenosis rate because follow-up angiography was not routinely performed and 6-month outcome data is available on only a minority of patients.

Most series to date have dealt with lesions in native vessels. However, saphenous vein graft occlusion after bypass surgery remains a significant clinical problem. Follow-up studies of bypass surgery patients have shown that 15% to 20% of saphenous vein grafts occlude in the first year and 50% to 80% occlude within 10 years.[23,24] Repeat bypass surgery carries an increased morbidity and mortality. The use of balloon angioplasty for saphenous vein graft lesions has been associated with restenosis rates of 40% or higher depending on the location of the lesion.[10,25,26] Unfortunately, newer techniques (other than stents) have not been shown to reduce restenosis. For example, a multicenter randomized trial of balloon angioplasty versus directional atherectomy for patients with saphenous vein bypass graft lesions reported a restenosis rate of 46% for directional atherectomy and 51% for

Table 2. Early and late events in elective and emergency stenting

	EARLY EVENTS (0-14 Days or In-hospital) (%)					LATE EVENTS (6-7 months) (%)			
Reference	Death	Q Wave MI	Bypass	Emergency PTCA	Stent Thrombosis	Death	Q Wave MI	Bypass	PTCA
Elective Stenting									
Serruys[22]	0	1.9	3.1	0.4	3.5	0.8	2.7	5.0	10
Fishman[11]	0	2.9	2.4	0.2	3.4	1.5	1.0	2.4	9.8
Columbo[37]	2.0	2.0	3.0	2.0	3.0	0.0	0.0	4.3	19.5
NYH-Cornell	0.7	1.4	2.1	2.8	3.4	NA	NA	NA	NA
Emergency Stenting									
George[20]	2.2	3.0	7.3	NA	7.7	1.4	0.8	6.9	NA
Robinson[38]	4.9	14.6	19.5	NA	7.3	0	2.4	0	NA
Antoniucci[39]	5.6	2.7	8.3	8.3	NA	0	0	3.0	6.0
Lincoff[40]	3.3	32	4.9	NA	13	1.7	1.7	10.7	10.7
Fishman[11]	NA	NA	NA	NA	21.4	NA	NA	NA	NA
NYH-Cornell	0	0	5.9	0	7.4	NA	NA	NA	NA

MI = myocardial infarction; PTCA = percutaneous transluminal coronary angioplasty.

balloon angioplasty (p = NS).[27] However, in a multicenter prospective nonrandomized study of Palmaz–Schatz stents, restenosis rates of 18% in de novo saphenous vein graft lesions and 38% in restenotic lesions were reported.[28] These rates are substantially lower than those traditionally associated with balloon angioplasty.[10,25,26] More information on stenting of saphenous vein grafts will be obtained from a prospective randomized trial (Saphenous VEin De novo [SAVED]) which is now being conducted.

Limitations of Intracoronary Stents

Intracoronary stents have been shown to treat abrupt or threatened closure and reduce restenosis. However, the widespread use of stents has been tempered by risks of stent thrombosis and bleeding at the vascular access site.

Intracoronary stents are thrombogenic. Despite intense anticoagulation regimens, the subacute thrombosis rate has remained 3% to 5% in elective stenting and 7% to 21% in emergency stenting (Table 2). This event usually occurs within 2 weeks of stent placement and generally presents as an acute myocardial infarction. The current study represents our early experience with stents and also includes more complex lesions than some initial trials. The 2-week thrombosis rate was 3.4% for elective stenting and 7.4% for emergency stenting (p = NS). In addition to the indication for stent placement (elective versus emergency), studies have identified operator inexperience, dissection not covered by stents, and vessel diameter less than 2.5 mm as risk factors for subsequent subacute closure.[19]

The risk of vascular complications is another limitation of intracoronary stenting. Traditionally, a stringent anticoagulation regimen employing both aspirin and warfarin has been advocated to reduce the incidence of subacute stent thrombosis.[29] However, aggressive anticoagulation has been associated with significant vascular complications. The rate of vascular complications at the access site is 5% to 16%.[11,21,22,30] In the BENESTENT trial, major vascular complications, defined as the need for surgical vascular repair or bleeding requiring transfusion, occurred in 13.5% of stented patients and 3.1% of angioplasty patients (p < 0.001).[22] The STRESS trial found roughly twice the number of hemorrhagic and peripheral vascular complications in the patients receiving intracoronary stents.[11] In the present series, the rate of significant vascular complications, defined as pseudoaneurysm requiring compression or repair, arteriovenous fistula, or retroperitoneal hemorrhage, was 5.2%, comparable to that reported in the literature.

Antiplatelet Therapy for Stenting

The technique of stent placement has undergone dramatic evolution over the past 2 years. Among the recent advances in stent deployment have been the advent of intravascular ultrasound and the use of high pressure balloon inflation. Intravascular ultrasound is a catheter-based two-dimensional ultrasound imaging technique that allows visualization of the coronary artery lesion and stent deployment from within the coronary artery. Studies with intravascular ultrasound imaging of intracoronary stents revealed incomplete stent expansion in 50% to 80% of patients with otherwise angiographically acceptable results (Fig. 4).[31,32] Additional balloon inflation at very high

pressure will generally achieve complete expansion. These observations suggest that the thrombogenicity of coronary stents may only in part be due to the procoagulant nature of the stent itself and that incomplete stent dilatation and suboptimal flow characteristics may play a role in subacute thrombosis. In a recent prospective trial enrolling 359 patients, Colombo et al. demonstrated that stent patients could be successfully managed with the antiplatelet agents ticlopidine and aspirin, without anticoagulation if stent deployment was followed by intravascular ultrasound-guided high pressure balloon inflation.[33] In that series, there were only three cases of stent thrombosis (0.9%) within 2 months. Furthermore, the rate of vascular complications was 0.6% at 6-month follow-up. In the present series, of the 22 patients receiving either aspirin and ticlopidine or aspirin alone, there was no subacute thrombosis, and one patient developed a vascular complication. These data suggest that this regimen is safe and effective and that lower subacute thrombosis and vascular complication rates may be achievable using this newer approach. However, the criteria for selecting patients to receive antiplatelet therapy without oral anticoagulation needs to be further delineated.

FUTURE DIRECTIONS

New stent designs, deployment techniques, and pharmacologic protocols are being evaluated to address the problems of subacute thrombosis and restenosis. The development of stents coated with anticoagulants such as heparin, hirudin, or a thrombolytic agent may further reduce the incidence of stent thrombosis.[34] Thromboresistant stents coated with covalently bound heparin will be studied in the forthcoming BENESTENT II clinical trial. Other future directions could involve the coating of intracoronary stents with antiproliferative agents such as growth factors, cytokines, or immune modulators to reduce restenosis. Several investigators have even advocated the use of endovascular radiation therapy to reduce stent restenosis.[35] Intracoronary radiation delivered locally via a beta-particle–emitting stent may prove an effective means to inhibit smooth muscle cell mediated neo-intimal growth after stent implantation.[36] Thus the future of intracoronary stents will be directed toward the development of devices not only for added structural support but also to serve as an effective matrix for local drug delivery and potentially as a vehicle for intracoronary gene therapy. STI

REFERENCES

1. Gruentzig AR, Senning A, Siegenthaler WC. Non-operative dilatation of coronary artery stenosis: percutaneous transluminal coronary angioplasty. New Engl J Med 1979; 301:61-8.
2. Leon MB, Wong SC. Intracoronary stents: a breakthrough technology or just another small step? Circulation 1994;89(3):1323-7.
3. de Feyter PT, de Jaegere PPT, Murphy ES, et al. Abrupt coronary artery occlusion during PTCA. Am Heart J 1992;123:1633-42.
4. Simpfendorfer C, Belardi J, Bellamy G, et al. Frequency, management and follow-up of patients with acute coronary occlusions after percutaneous transluminal coronary angioplasty. Am J Cardiol 1987;59:267-9.
5. Detre KM, Holmes DR, Holubkov R, et al. Incidence and consequence of periprocedural occlusion: The 1985-1986 National Heart, Lung, and Blood Institute PTCA registry. Circulation 1990;82:739-50.
6. Pages VS, Okies JC, Colburn LQ, et al. Percutaneous transluminal cutaneous angioplasty: a growing problem. J Thorac Cardiovasc Surg 1986;92:847.
7. Reul GJ, Cooley DA, Hallman GL, et al. Coronary artery bypass surgery for unsuccessful PTCA. J Thorac Cardiovasc Surg 1984;88:685.
8. Ellis SG, Roubin GS, King SB, et al. Angiographic and clinical predictors of acute closure after native vessel coronary angioplasty. Circulation 1988;77:372-9.
9. Nobuyoshi M, Kimura T, Nogaka H, et al. Restenosis after successful PTCA: serial angiographic followup of 229 patients. J Am Coll Cardiol 1988;12:616-23.
10. de Feyter PT, Van Suyler P, de Jaegere PP, et al. Balloon angioplasty for the treatment of lesions in saphenous vein bypass grafts. J Am Coll Cardiol 1993;21:1539-49.
11. Fischman DL, Leon MB, Baim DS, et al. A randomized comparison of coronary stent placement and balloon angioplasty in the treatment of coronary artery disease. (Stent Restenosis Study Investigation). New Engl J Med 1994;331:496-501.
12. Mintz GS, Kovach JA, Javier SP, et al. Geometric remodeling is the predominant mechanism of late lumen loss after coronary angioplasty. Circulation 1993;88(suppl II):8-654. Abst.
13. Wong SC, Leon MB, Popma TJ. New device angioplasty: the impact on restenosis. Coronary Artery Dis 1993;4:243-53.
14. Topol EJ, Leye F, Pinkerton CA, et al. A comparison of directional atherectomy with coronary angioplasty in patients with coronary artery disease. New Engl J Med 1993;329:221-7.
15. Adelman AG, Cohen EA, Kimball BP, et al. A comparison of directional atherectomy with balloon angioplasty for lesions of the left anterior descending coronary artery. New Engl J Med 1993;329:228-33.
16. Dotter CT. Transluminally placed coil-spring endarterial tube grafts: long term patency in canine popliteal artery. Invest Radiol 1969;4:329-32.
17. Schatz RA. An introduction to intravascular stents. Cardiol Clin 1988;6:357-72.
18. Roubin GS, Pinkerton CA. Gianturco–Roubin stent: development and investigation. In: Sigwart V, Frank GI, eds. Coronary stents. Berlin: Springer-Verlag; 1992. p 79-99.
19. Roubin GS, Cannon AD, Agrawal SK, et al. Intracoronary stenting for acute and threatened closure complicating percutaneous transluminal cutaneous angioplasty. Circulation 1992;85;916-29.
20. George BS, Voorhees WD, Roubin GS, et al. Multicenter investigation of coronary stenting to treat acute or threatened closure after percutaneous transluminal coronary angioplasty: clinical and angiographic outcomes. J Am Coll Cardiol 1993;22:135-43.
21. Sutton JM, Ellis SG, Roubin GS, et al. Major clinical events after coronary stenting: the multicenter registry of acute and elective Gianturco–Roubin stent placement. Circulation 1994;89:1126-37.
22. Serruys PW, de Jaegere P, Kiemeney F, et al. A comparison of balloon-expandable stent implantation with balloon angioplasty in patients with coronary artery disease (BENESTENT). New Engl J Med 1994;331:489-95.
23. Kalan JM, Robert CW. Morphologic findings in saphenous vein used as coronary arterial bypass conduits for longer than 1 year: necropsy analysis of 53 patients, 123 saphenous vein, and 1865 5 mm segment of vein. In: Yacoub M, Pepper JA, eds. Annals of Cardiac Surgery. Current Science 1991:182.
24. Bourassa MG, Fisher LD, Campean L, et al. Long-term fate of bypass grafts. The coronary artery surgery study (CASS) and Montreal Heart Institute Experience. Circulation 1985;72(suppl V):71.
25. Platkow P, Hollman J, Whitlow PL, et al. Percutaneous transluminal cutaneous angioplasty of saphenous vein graft stenosis: long term followup. J Am Coll Cardiol 1989;14:1645.
26. Meesler BJ, Samson M, Suryapranata H, et al. Long term followup after attempted angioplasty of saphenous vein graft: the Thoraxcenter experience. 1981-1988. Eur Heart J 1991; 72: 648-53.
27. Holmes DR, Topol EJ, Califf RM, et al. A multicenter, randomized trial of coronary angioplasty versus directional atherectomy for patients with saphenous vein bypass graft lesions. Circulation 1995;91:1966-74.
28. Friedrick BP, Davis SF, Kuntz RF, et al. Investigational use of the Palmaz–Schatz biliary stent in large saphenous vein grafts. Am J Cardiol 1993;71:439-41.
29. Schatz RA, Baim DS, Leon M, et al. Clinical experience with the Palmaz–Schatz coronary stent. Initial results of a multicenter study. Circulation 1991;83:148-61.
30. Carrozza JP, Kuntz RE, Levine MJ, et al. Angiographic and clinical outcome of intracoronary stenting: immediate and long-term results from a large single center experience. J Am Coll Cardiol 1992;20:328-37.
31. Goldberg SL, Colombo A, Nakamura S, et al. The benefit of intracoronary ultrasound in

the deployment of Palmaz–Schatz stents. J Am Coll Cardiol 1994;24:996-1003.
32. Nakamura S, Colombo A, Gaglione S, et al. Intracoronary ultrasound observations during stent implantation. Circulation 1994;89:2026-34.
33. Colombo A, Hall P, Nakamura S, et al. Intracoronary stenting without anticoagulation accomplished with intravascular ultrasound guidance. Circulation 1995;91:1676-88.
34. Van der Giessen WJ, Van Bauschom HMM, Van Houtan CD, et al. Coronary stenting with polymer coated and uncoated self-expanding endoprosthesis in pigs. Coronary Artery Dis 1992;30:631-40.
35. Waksman R, Robinson LA, Crocker IR, et al. Endovacular low-dose irradiation inhibits neointima formation after coronary artery balloon injury in swine. A possible role for radiation therapy in restenosis prevention. Circulation 1995;91:1533-9.
36. Fischell TA, Kharma BK, Fischell DR, et al. Low-dose, beta-particle emission from "stent" wire results in complete, localized inhibition of smooth muscle cell proliferation. Circulation 1994;90:2956-63.
37. Colombo A, Maiello L, Almagor Y, et al. Coronary stenting: single institution experience with the initial 100 cases using the Palmaz–Schatz stent. Cath Cardiovasc Diag 1992;26:171-176.
38. Robinson NMK, Thomas MR, Jewitt DE, et al. Comparison of clinical outcome after elective and "bailout" coronary stent insertion. J Invas Cardiol 1995;7:156-164.
39. Antoniucci D, Santoro GM, Bolognese L, et al. Bailout Palmaz–Schatz coronary stenting in 39 patients with occlusive dissection complicating conventional angioplasty. Cath Cardiovasc Diag 1995;35:204-9.
40. Lincoff AM, Topol EJ, Chapekis AT, et al. Intracoronary stenting compared with conventional therapy for abrupt vessel closure complicating coronary angioplasty: a matched case-control study. J Am Coll Cardiol 1993; 21: 866-75.