Intracoronary Stenting for Acute and Threatened Closure Complicating Percutaneous Transluminal Coronary Angioplasty

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Background. Acute closure remains a significant limitation of percutaneous transluminal coronary angioplasty (PTCA) and underlies the majority of ischemic complications. This study details the clinical and angiographic characteristics of a series of patients receiving an intracoronary stent device to manage acute and threatened closure and presents the early clinical results.

Methods and Results. From October 1989 through June 1991, 115 patients undergoing PTCA received intracoronary stents to treat acute or threatened closure in 119 vessels. Sixty-three percent had multivessel coronary disease, 33 (29%) had undergone prior coronary artery bypass grafting (CABG), and 52 (45%) had previous PTCA. Using the American College of Cardiology/American Heart Association (ACC/AHA) classification, 15% of lesions were class A, 55% were class B, and 30% were class C. Eight patients were referred with severe coronary dissection and unstable angina after PTCA at other institutions. Acute closure was defined as occlusion of the vessel with TIMI (Thrombolysis in Myocardial Infarction) 0 or 1 flow immediately before stent placement. Threatened closure required two or more of the following criteria: 1) a residual stenosis greater than 50%, 2) TIMI grade 2 flow, 3) angiographic dissection comprising extraluminal dye extravasation and/or a length of greater than 15 mm, 4) evidence of clinical ischemia (either typical angina or ECG changes). Twelve vessels (10%) met the criteria for acute closure, and 87 vessels (73%) satisfied the criteria for threatened closure. Twenty vessels (17%) failed to meet two criteria. Stenting produced optimal angiographic results in 111 vessels (93%), with mean diameter stenosis (±1 SD) reduced from 83±12% before to 18±29% after stenting. Overall, in-hospital mortality was 1.7% and CABG was required in 4.2%; Q wave myocardial infarction (MI) occurred in 7% and non–Q wave MI in 9%. Stent thrombosis occurred in nine patients (7.6%). For the 108 patients who presented to the catheterization laboratory without evolving MI, Q wave MI occurred in 4% and non–Q wave MI occurred in 7%. Angiographic follow-up has been performed in 81 eligible patients (76%), and 34 patients (41%) had a lesion of ≥50%.

Conclusions. This stent may be a useful adjunct to balloon dilatation in acute or threatened closure. Randomized studies comparing this stent with alternative technologies are required. (Circulation 1992;85:916–927)

KEY WORDS • coronary stenting • acute closure • coronary dissection

Suboptimal luminal results are responsible for the majority of the early complications of percutaneous transluminal coronary angioplasty (PTCA). Primarily because of arterial dissection and recoil of atherosclerotic plaque, acute or threatened closure underlies the need for emergent coronary artery bypass graft surgery (CABG) and the occurrence of myocardial infarction (MI) and death. Previous studies have reported an incidence of 2–11% for these events.1–10 The National Heart, Lung, and Blood Institute (NHLBI) PTCA registry report showed the combined incidence of death, MI, and emergent coronary artery surgery for elective PTCA in the presence of one-, two-, and three-vessel disease as 5%, 8%, and 10%, respectively.2 A recent report on multivessel PTCA reported an 8.6% incidence of the same events.4 Using a modified American College of Cardiology/American Heart Association (ACC/AHA) classification of morphological criteria, this group found that the incidence of ischemic complications ranged from 2% in type A lesions up to 21% in type C lesions.

Although pre-PTCA lesion morphology may predict the probability of an ischemic event, angiographic appearance after dilation is the most important determinant of a complication. A number of studies have shown that a residual stenosis5–7 and/or the presence of significant dissection4–3,11 are predictive of a major complication. Urgent or emergency CABG has been used12–17 to manage suboptimal PTCA results but has significant limitations. Apart from important logistical and eco-
nomic problems associated with CABG for failed PTCA, more than 50% of patients suffer significant myocardial damage, one third suffer Q wave myocardial infarction, and mortality ranges from 1.4% up to 26% in reported studies.\textsuperscript{13–15,18–22}

Intracoronary stents (intra-arterial prosthetic scaffolding devices) have recently been developed to reduce the problems of acute closure complicating PTCA.\textsuperscript{17,23–26} Use of the intracoronary stent for acute or threatened closure should serve to control dissection and residual stenosis, the morphological predictors of a major ischemic event. Because the use of these intracoronary prostheses is not benign and because some patients with suboptimal PTCA results do well in the long term, one difficulty has been in selecting patients who may benefit from the implantation of these devices. The purpose of this study is to present the experience of a single center in stenting for acute or threatened closure and to describe definitions of acute and threatened closure, which may aid in the investigation of this and other devices used to treat suboptimal results from PTCA.

**Methods**

**Population**

From October 1989 through June 1991, 1,338 patients underwent PTCA at the University of Alabama Hospital. One hundred seventy-nine of these procedures included placement of a balloon-expandable intracoronary stent device (Cook, Inc., Bloomington, Ind.). The device was used under Food and Drug Administration investigational device exemption status as part of a multicenter study. Sixty-four patients had stenting procedures electively for recurrent restenosis and 115 for acute or threatened closure. These 115 patients who underwent 118 separate stenting procedures form the basis of this report. All patients gave written informed consent for this investigational device before the procedure. The study was approved by the Institutional Review Board of the University of Alabama at Birmingham.

**Percutaneous Transluminal Coronary Angioplasty Procedure**

PTCA was done using standard techniques.\textsuperscript{27} All patients received aspirin, calcium channel blockers, either diltiazem or nifedipine the night before, and most received dipyridamole. The pre-PTCA medical regimen evolved during the period of the study. Early in the study period, enteric-coated aspirin was administered, dipyridamole was omitted, and frequently, antiplatelet agents were not given on the morning of the procedure. As the importance of antiplatelet therapy became apparent, soluble 325 mg aspirin was given (at least two doses, including one on the morning of the procedure) and pretreatment with dipyridamole was more strictly applied to all patients undergoing PTCA. In addition, patients thought to have lesions at high risk for acute closure were pretreated with Dextran 40, 10% solution, at 50–75 ml/hr for at least 2 hours before commencing the procedure. Heparin (10,000 units) was given intraruminally at the start of the procedure and supplemented as needed, aiming for an activated clotting time (ACT) (Hemotech Co., Inc., high-range assay) level of $\geq$300 seconds. Preliminary angiography of the coronary artery to be dilated was performed in two near-orthogonal views. A balloon catheter was chosen so that its inflated diameter was equal in size to the normal lumen of artery adjacent to the lesion to be dilated. The balloon catheter system was inserted, and patients received 100–200 $\mu$g selective intracoronary nitroglycerin. The balloon was then positioned across the lesion and inflated. When initial inflations failed to produce a satisfactory result, patients generally received prolonged inflations with the same balloon or with a balloon $\frac{1}{2}$ mm larger before proceeding to stenting. A perfusion balloon system was used when considered necessary. This was at the discretion of the primary operators and, if appropriate, the patient proceeded directly to the stent insertion. When a decision was made to proceed with stenting, the patient received an intravenous bolus dose of 100–200 ml Dextran 40, 10% solution, after premedication with 250 mg hydrocortisone acetate. Dextran was then infused at 100 ml/hr for 2 hours and continued at 50 ml/hr until the sheaths were removed and a therapeutic infusion of heparin was established (partial thromboplastin time [PTT], 50–80 seconds). The infusion was begun in the catheterization laboratory immediately after the bolus and was continued on return to the ward.

**Stenting Procedure**

The stenting device used in this study was designed specifically for the control of dissection and acute closure complicating PTCA. Stenting was performed by three primary operators (G.S.R., L.S.D., W.A.B.). One of the operators had significant experience at another institution and assisted with most of the initial cases performed by the other two operators. The stent prototype used is made of 0.006-in. surgical stainless steel suture wire wrapped in a cylindrical shape with bends adopting an inverted “U” every 360° and comes in 12- or 20-mm lengths. The stent is supplied by the manufacturer tightly wrapped around a standard polyvinylchloride compliant balloon catheter. The balloon length varies, with the length of the stent 20 mm for the shorter model and 30 mm for the longer model. Stent sizes range from 2 to 4 mm in diameter. The expanded diameter of the stent is determined in part by the size of the balloon and the inflation pressure used at the time of deployment. As the stents undergo a 15–20% diameter recoil after release, they are mounted on balloons with a nominal diameter 0.5 mm larger than the nominated stent size. Accordingly, oversizing of the stent is associated with marked oversizing of the balloon. In the early part of the study, the importance of this in producing distal dissection was not appreciated. Later in the study, more conservative sizing of the stent balloon combination was used and may have contributed to the improvement in results. The stent does not shorten with expansion. The deflated balloon stent catheter is relatively flexible and is positioned in the vessel by standard over-the-guide wire technique, with some modifications. The considerable profile of the balloon/stent catheter combination requires the use of large-lumen 8F guiding catheters when 2.0-mm, 2.5-mm, and 3.0-mm stents are used and large-lumen 9F guiding catheters when 3.5-mm and 4.0-mm stents are used. In addition, guiding catheters chosen must be able to provide optimum backup support. Usually well-fitting
right and left Judkins catheters were used, but in some cases involving tortuous right coronary, left circumflex, and left anterior descending arteries, left Amplatz shapes were required. Before a stent could be placed at the site of arterial dissection, it was necessary on occasion to dilate any proximal lesions or segment of plaque that obstructed tracking of the stent. Finally, the use of extra-support 0.018-in. coronary guide wires (Advanced Cardiovascular Systems, Inc., Santa Clara, Calif.) markedly enhanced the trackability of these devices. In this manner, the balloon stent assembly was carefully advanced to the stenosis and the balloon was inflated in the usual fashion. After ensuring balloon deflation, the balloon catheter was withdrawn, leaving the expanded stent in situ. Angiography was then performed and, if necessary, further inflations were done inside the stent to produce an optimal luminal result. If it was not possible to track the stent to the desired site, it was removed by withdrawing the stent to the tip of the guiding catheter and removing the balloon/stent catheter and guiding catheter as one through the arterial sheath. At the end of the procedure, catheters were removed, the sheaths were left in situ, and the patient was taken to the coronary care unit. Some patients had intracoronary urokinase infusions at 80,000 units/hr for 12–18 hours to treat residual thrombus in the stented vessel. In general, the sheaths were removed at 4–6 hours once the ACT fell to 150 seconds. Pressure was held for 1 hour, and the heparin infusion was then restarted. Dextran was continued until the PTCA was in the therapeutic range. After PTCA, the patients received an ECG immediately and daily for 2 days. Creatinine kinase (CK) estimations (including isoenzymes) were done immediately and every 8 hours for 24 hours. The patients continued on aspirin, dipyridamole, and a calcium channel blocker and were anticogulated with coumadin.

The mobilization and ambulation protocol proved critical in determining the incidence of femoral artery complications. In the early part of the study, patients were mobilized according to routine post-PTCA protocols. Approximately halfway through the study period, the mobilization protocol was adjusted as follows and appeared to have markedly reduced the incidence of groin complications. For the first 24 hours after sheath removal, the patients lay flat in bed. Following this, they were allowed to sit up in bed for the next 24 hours. Finally, on the third postprocedure day, the patients were able to sit out of bed. After 24–48 hours, the patients were transferred to the cardiac ward, where heparin was continued until the prothrombin time was greater than 17 seconds on two blood specimens 12 hours apart. On day 4, they were able to mobilize freely in their rooms and were discharged once stable anticoagulation with coumadin was established. After discharge, the patients continued persantine and coumadin for 2 months, with prothrombin times maintained between 17 and 20 seconds. Aspirin was continued indefinitely.

Data Collection

Indications for stenting and angiographic and clinical data from the procedure were recorded prospectively on standard forms by a research nurse in collaboration with a physician. Clinical and laboratory details, including the presence of angina, ECG changes, and procedural complications, were routinely collected throughout the admission and entered into a data file for each patient. Relevant information not available at the time of review was collected retrospectively from hospital charts.

All angioplasty and, where available, diagnostic cine-angiograms were reviewed. Information recorded included the severity of the coronary artery disease and the ACC/AHA classification of the lesion in the stented vessel. After initial balloon dilatation, the residual stenosis, TIMI (Thrombolysis in Myocardial Infarction) grade flow, and the presence or absence and morphology of dissection were noted. This information was again recorded after supplementary balloon inflations in the immediate present angiograms. Finally, the appearance of the vessel after stenting was recorded. The angiogram immediately before stenting was used to classify the patients according to our definition of acute or threatened closure.

In the early period of the study, both 2-month and 6-month angiographic follow-up was recommended. Later, only 6-month angiographic follow-up was advised. Patients were admitted for angiography earlier, depending on clinical circumstances. Angiographic restenosis was defined as a ≥50% diameter narrowing at the stent site at any angiographic follow-up study.

Definitions and Measurements

For the purpose of this study, acute closure was defined as occlusion of a vessel with TIMI grade 1 or 0 flow, based on the appearance of the vessel immediately before stent insertion. Thus, if patients with acute ischemia who were returned to the catheterization laboratory had partial resolution of the occlusion with repeat balloon dilatation, they were not included in the acute closure group in this study. Threatened closure was defined as the presence of two or more of the following four criteria: 1) a residual stenosis ≥50%, 2) TIMI grade 2 flow, 3) a significant dissection, or 4) evidence of clinical ischemia (either typical angina or ECG changes).

Dissection was considered present angiographically if there was a luminal filling defect suggestive of an intimal flap or marked irregularity of the vessel wall after PTCA. A significant dissection was defined as the presence of extravascular dye or a dissection with a length of more than 15 mm.11 The remaining lesions with intimal disruption were defined as simple dissections.

Successful angioplasty after stenting was defined as a 50% residual stenosis and freedom from a major ischemic complication (death, MI, and CABG).

The diameters of the stenoses before PTCA and immediately before and after stenting were measured using digital calipers. Measurements were taken from the angiographic view that showed the lesion most clearly (free of foreshortening and side branches) and the most severe narrowing. A frame was selected that showed the clearest borders of the presumed flow channel.

Myocardial infarction was defined as the development of new pathological Q waves (≥40 msec) and/or elevation of CK more than twice the upper limit of normal with an elevated CK-MB fraction.
TABLE 1. Distribution and Classification of Lesions in Coronary Arteries Undergoing Percutaneous Transluminal Coronary Angioplasty

<table>
<thead>
<tr>
<th>Vessel (n=119)</th>
<th>AHA/ACC lesion classification</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
</tr>
<tr>
<td>LAD</td>
<td>8</td>
</tr>
<tr>
<td>RCA</td>
<td>4</td>
</tr>
<tr>
<td>LCx</td>
<td>2</td>
</tr>
<tr>
<td>LMCA</td>
<td>0</td>
</tr>
<tr>
<td>SVG</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
</tr>
</tbody>
</table>

AHA, American Heart Association; ACC, American College of Cardiology; LAD, left anterior descending artery; RCA, right coronary artery; LCx, left circumflex artery; LMCA, left main coronary artery; SVG, saphenous vein graft.

Pseudoaneurysms and atrioventricular fistulas were defined using color flow Doppler ultrasonography performed on clinical suspicion.

Results

The group comprised 74 men and 41 women with a mean age of 58±10 years (mean±1 SD; range, 35–80 years). Thirty-three patients (29%) had prior CABG and 52 (45%) had a prior PTCA, 48 (42%) to the same site. The most common indication for PTCA was angina pectoris, which was present in 81% of the group. Of these, 57 (48%) were in Canadian Cardiovascular Society class IV. Eight patients were referred to our hospital with unstable angina and coronary dissection complicating previous PTCA. Eleven patients developed acute closure on the floor after previous PTCA in our hospital and were returned emergently to the catheterization laboratory (six with evolving MI and five with unstable angina). Another three patients had the index PTCA for acute MI. The remaining patients were referred with significant lesions found at coronary angiography after recent non-Q wave MI. The MI occurred on an average of 3 weeks previously with a range of 1–12 weeks, and each of the patients was considered to have further myocardium in jeopardy.

Coronary Anatomy and Lesion Morphology

Patients with one-vessel, two-vessel, and three-vessel disease made up 45%, 28%, and 27% of the patient group, respectively. The mean ejection fraction (available in 103 patients) was 51±9% (range, 25–70%). Five patients had an ejection fraction <30% and 22 <50%. Table 1 shows the distribution of coronary artery lesions dilated. Applying the AHA/ACC classification to those lesions, 28 (15%) were class A, 65 (55%) were class B, and 36 (30%) were class C. In 17 patients, the vessel dilated was totally occluded.

These 115 patients had PTCA stent procedures in 119 coronary arteries, three patients having another vessel stented at a second procedure and one patient having two vessels stented at one procedure. The left anterior descending artery was stented in 43 patients (36%) and in the mid or distal segments in 23 of 43 (53%). The right coronary artery was stented in 40 patients (34%), with 55% in the mid and 8% in the distal vessel. The left circumflex arteries or the marginal branches were stented in 21 patients (18%). The left main stem was stented in three patients with prior CABG and protected left coronary branches. Saphenous vein grafts were stented in 12 patients (10%).

Indications for Stenting

Of the 119 vessels stented, 12 (10%) had acute closure with TIMI grade 0–1 flow at the time of stenting (Figure 1). All 12 had clinical signs of myocardial ischemia. The remaining 107 vessels had stents placed for impending or threatened closure. This was done to improve the acute clinical status of the patient and/or the immediate angiographic appearance of the PTCA site.

Eighty-three of the patients had prolonged inflations (≥120 seconds), and in 14 of these patients, a perfusion balloon system was used in an attempt to maintain vessel patency and control dissection.

Table 2 and Figure 2 show the remaining 107 vessels classified according to residual percentage diameter stenosis, clinical manifestations of myocardial ischemia, significant dissection, and TIMI grade flow. Ninety-nine of these 107 patients (93%) had a >50% residual diameter stenosis and 55 patients (51%) had >70% stenosis. Seventy-three of the 107 patients (68%) had clinical manifestations of myocardial ischemia, 55 patients (50%) had significant dissections (Figure 3), and 17 patients (16%) had TIMI grade 2 flow. Another 35 had angiographic evidence of a simple dissection (Figure 4). Forty-two patients (39%) met at least three of the above criteria and 87 patients (81%) at least two of the above criteria fulfilling our definition of threatened closure. Of the remaining 20 patients, eight had more than 70% residual stenosis, five had more than 50% residual stenosis, and another three had a significant dissection.

Stenting Success

One hundred fifteen patients underwent 118 separate stent angioplasty procedures in which 132 stents were successfully deployed in 119 arteries. Of these, 27 were the shorter 12-mm model. In all 118 procedures, at least one stent was successfully deployed; 11 patients received two stents and one patient received three stents in a badly dissected right coronary artery. Of the stents deployed, eight (6%) were 2.0 mm, 63 (48%) were 2.5 mm, 39 (29%) were 3.0 mm, 13 (10%) were 3.5 mm, and nine (7%) were 4.0 mm. In 111 arteries, the final result showed no significant residual stenosis and TIMI grade 3 flow. The mean diameter stenosis was 83±12% before PTCA, 66±16% after PTCA, and 18±29% after stenting.

In eight patients, the stent failed to produce optimal results (Table 3). In three patients, severe dissection of >50 mm was not fully covered by one or more stents, although each had subsequently uncomplicated hospitalization. In three patients, there was failure to control dissection distally with subsequent vessel occlusion. All three suffered non-Q wave MI. Of the two remaining patients, one had residual dissection at the initial procedure and was referred for surgery after failed PTCA for acute closure of the vessel. The final patient had a stent placed distal to the ostial stenosis in a protected left main coronary artery. She developed refractory pulmonary edema later and died.

On five occasions, there was failure to deploy a stent. On two occasions, stents had been successfully deployed in the right coronary artery proximally and there was failure...
to deploy another stent though the proximal stent. This was not associated with adverse outcome. In three patients, it was not possible to track a stent into the lesion. This was the result of diffusely diseased, calcified, and/or tortuous proximal vessels. In each case, nondeployed stents were successfully removed from the patient.

The overall technical success rate for stent deployment was 132 of 137 attempts (96%). Overall procedural success with satisfactory arterial lumen after stenting was 111 of 119 procedures (93%).

**Complications**

There were two deaths in the series (see Table 4 and Figure 5). The first occurred in a 70-year-old man who underwent PTCA to a proximal left anterior descending lesion complicated by acute closure. Two hours later, he developed severe pain and cardiogenic shock. He died 1 week later of multiorgan system failure. At autopsy, the stent was patent but he had sustained a large posterolateral infarction due to an undilated stenosis in the left circumflex artery.

<table>
<thead>
<tr>
<th>Table 2. Patients With Threatened Acute Closure Grouped According to TIMI Flow, Percent of Residual Stenosis, Ischemia, and Significant Dissection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TIMI grade 2 flow</strong></td>
</tr>
<tr>
<td><strong>Residual Stenosis</strong></td>
</tr>
<tr>
<td>Angina pectoris and ECG changes</td>
</tr>
<tr>
<td>Angina pectoris only</td>
</tr>
<tr>
<td>ECG changes only</td>
</tr>
<tr>
<td>No clinical ischemia</td>
</tr>
<tr>
<td>Total patients</td>
</tr>
</tbody>
</table>

TIMI, Thrombolysis in Myocardial Infarction; ECG, electrocardiogram.

*Evidence of extraluminal contrast and/or length of dissection ≥15 mm.
†Failed to meet two or more criteria for threatened closure.
The second death was in an 80-year-old woman with refractory unstable angina and heart failure after coronary artery grafting 6 months previously (ejection fraction was 30%). This patient underwent PTCA to a left main lesion protected by a graft to the circumflex; the vessel closed acutely and was successfully stented. Six hours later, she developed refractory pulmonary edema and expired. At autopsy, the stent was patent but had been deployed just inside the aortic wall with a residual 90% stenosis present at the ostium.

Nineteen patients (16%) had MI. There were eight Q wave MIs (7%) and 11 non-Q wave MIs (9%). In the 108 patients not entering the catheterization laboratory with evolving MI (Figure 5), there were 12 MIs (11%), of which four were Q wave (4%). Three of the Q wave MIs were the result of secondary subacute thrombosis during the early period of the study, and one was the result of distal embolization during PTCA of a vein graft. In the eight patients with non-Q wave MI, the mean peak CK level was 1,846±1,311 IU (range, 632–4,930 IU). Four of these events occurred in patients with unsuccessful stenting resulting from distal dissection not covered by the stent and one in a patient with subacute stent thrombosis.

In the 10 patients who entered the catheterization laboratory with acute evolving MI (Figure 6), four had a Q wave MI, three had non-Q wave MI, and three suffered no significant myocardial damage.

Five patients (4.2%) underwent CABG. One patient had a stent thrombosis and failed repeat angioplasty. Three patients had residual dissection after stenting and/or continuing angina. The final patient had a dissection and perforation with Excimer laser therapy that was initially controlled by stenting but required CABG after developing cardiac tamponade.

Nine patients (7.6%) suffered subacute stent thrombosis (Table 5) between days 1 and 5 after stent deployment. The nine patients with stent occlusion received 11 stents: six received single 2.5-mm stents, one received a single 3.0-mm stent, and one patient received tandem 3.0-mm stents. Another patient received a proximal right coronary artery 3.0-mm stent and a distal 2.5-mm stent. In seven of the patients, the site of vessel occlusion was within the 2.5-mm stent. Seven of the nine patients were stented in the first group of 40 patients who received a stent for acute or threatened closure. In all nine patients, an attempt was made to reopen the stented vessel with PTCA. In eight patients this was possible, although three had Q wave and two had non-Q wave infarctions. Three patients suffered no sequelae, although one required another stent to control a refractory lesion. The patient whose vessel could not be opened with PTCA required emer-
FIGURE 4. Angiograms. Panel A: Before percutaneous transluminal coronary angioplasty (PTCA); left anterior descending artery stenosis. Panel B: Right anterior oblique projection; vessel after a series of PTCA balloon catheter inflations with production of a localized dissection (arrow). Panel C: Left anterior oblique projection; this was classified as a simple dissection and did not meet our criteria for threatened closure. Panel D: After deployment of a 3.0-mm stent (arrow). Stent covers septal and diagonal branches that remain patent.

TABLE 3. Characteristics of Patients With Poor Angiographic Outcome After Stenting

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex</th>
<th>Vessel/lesion %</th>
<th>Class</th>
<th>Closure status</th>
<th>Stent size (mm)</th>
<th>Result after stent</th>
<th>Major complication (MI/death/CABG)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52/F</td>
<td>RCA/60% proximal</td>
<td>C</td>
<td>TC</td>
<td>3.0</td>
<td>Severe spiral dissection persists, TF 3</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>47/M</td>
<td>RCA 100%/mid</td>
<td>C</td>
<td>TC</td>
<td>2.5</td>
<td>Spiral dissection proximal, TF 3</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>60/M</td>
<td>RCA 70%/mid</td>
<td>C</td>
<td>TC</td>
<td>3.5</td>
<td>Persistent distal dissection, TF 3</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>68/M</td>
<td>RCA/mid 90%</td>
<td>C</td>
<td>TC</td>
<td>3.0</td>
<td>Spiral dissection, distal SVG to PDA patent, PLSA occlusion</td>
<td>NQMI</td>
</tr>
<tr>
<td>5</td>
<td>64/M</td>
<td>LAD/70% proximal</td>
<td>B</td>
<td>TC</td>
<td>2.5</td>
<td>Distal dissection, vessel occlusion</td>
<td>NQMI</td>
</tr>
<tr>
<td>6</td>
<td>39/M</td>
<td>LCx/ostia 70%</td>
<td>B</td>
<td>TC</td>
<td>2.5</td>
<td>Distal dissection, side branch occlusion</td>
<td>NQMI</td>
</tr>
<tr>
<td>7</td>
<td>57/M</td>
<td>RCA/distal 90%</td>
<td>C</td>
<td>AC</td>
<td>2.5</td>
<td>*Severe dissection, failed repeat PTCA</td>
<td>NQMI/CABG</td>
</tr>
<tr>
<td>8</td>
<td>80/F</td>
<td>LMCA</td>
<td>B</td>
<td>TAC</td>
<td>3.5</td>
<td>Stent failed to cover ostial lesion†</td>
<td>Pulmonary edema/ death</td>
</tr>
</tbody>
</table>

MI, myocardial infarction; CABG, coronary artery bypass grafting; F, female; RCA, right coronary artery; TC, threatened closure; TF, TIMI (Thrombolysis in Myocardial Infarction) flow grade; M, male; SVG, saphenous vein graft; PDA, posterior descending artery; PLSA, posterolateral surface artery; NQMI, non-Q wave myocardial infarction; LAD, left anterior descending artery; LCx, left circumflex artery; AC, acute closure; PTCA, percutaneous transluminal coronary angioplasty; LMCA, left main coronary artery.

*This patient presented with acute closure after prior PTCA.
†This patient had good initial angiographic result. Autopsy showed recoil of left main coronary artery ostia.
TABLE 4. Major Complications

<table>
<thead>
<tr>
<th>Adverse event</th>
<th>Patients (n=118)</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>2</td>
<td>1.7</td>
</tr>
<tr>
<td>Q wave MI*†</td>
<td>8</td>
<td>6.7</td>
</tr>
<tr>
<td>Non-Q wave MI†</td>
<td>11</td>
<td>9.3</td>
</tr>
<tr>
<td>CABG</td>
<td>5</td>
<td>4.2</td>
</tr>
<tr>
<td>Major hemorrhage</td>
<td>12</td>
<td>10.2</td>
</tr>
<tr>
<td>Femoral vascular repair</td>
<td>5</td>
<td>4.2</td>
</tr>
</tbody>
</table>

MI, myocardial infarction; CABG, coronary artery bypass surgery.
*Includes one patient undergoing CABG.
†Includes patients presenting with acute MI.

Emergency CABG. There was no stent thrombosis event in the last 35 patients.

An additional patient developed pericardial tamponade and was successfully treated with pericardiocentesis and cessation of anticoagulation that had included an intracoronary urokinase infusion. The patient had Dressler’s syndrome, which presumably contributed to the pericardial effusion.

Twelve patients (10%) had major bleeding events. There were four gastrointestinal bleeds, two retroperitoneal hemorrhages, and six local groin hematomas.

Another 13 patients required a transfusion of one or more units.

Five patients (6%) required vascular surgical repairs, four for expanding pseudoaneurysms, and one for failure to control femoral arterial bleeding. Four other patients developed local femoral artery complications: three pseudoaneurysms and one arteriovenous fistula, none of which required surgical intervention.

**Early Postdischarge Outcome**

Follow-up was available in all 107 patients who survived without CABG 6±3 months (range, 1–16 months) after stenting. No patient suffered MI, and no patient had a clinical event caused by acute thrombosis of the stented vessel. Two patients had totally occluded vessels at angiographic follow-up. Eight patients (7%) have required CABG since hospital discharge with bypass grafting of the stented vessel. One of these patients had no restenosis of the stented vessel but had developed progression of a left main lesion. There have been two late deaths, one from progressive heart failure without MI and one from a cerebrovascular event after cessation of coumadin.

Eighty-one (76%) of the 107 eligible patients (no death or in-hospital CABG) have had follow-up angiography of 82 stented vessels. Fifty-seven patients had angiography before 3 months (mean, 2±1 month; range, 1–3 months). The mean stenosis at this time was 39±27% (range, 0–98%). Nineteen patients (33%) had a greater than 50% diameter stenosis. Seventeen were treated with repeat PTCA and two underwent CABG. Angioplasty reduced the lesions from 71±10% to 26±10%. Five patients with 2-month follow-up and no
restenosis have refused 6-month angiography. All remained symptom free.

Another 56 patients had follow-up angiography of 57 stented arteries 6±1 month (range, 3–9 months) after stent deployment, with a mean artery stenosis of 35±29% (range, 0–100%). This group had either no 2-month angiography or no restenosis at that time. Fifteen (26%) of these patients had restenosis, with 13 undergoing PTCA. Angioplasty reduced the lesion from 78±15% before PTCA to 28±73% after PTCA. One patient had failed PTCA when a guide wire could not be passed across a total occlusion. One patient underwent CABG and another patient was treated medically. Overall, restenosis occurred in 35 of 82 (41%) restudied arteries.

### Discussion

Treatment of acute closure remains a problem in clinical practice. The incidence of this complication varies from 2% to 11% in 1–3,5,10,29,30 and is increased in patients with multivessel disease.2,30–32 In this study, the use of the stent appeared to decrease the need for emergency CABG and may have decreased the overall incidence of ischemic complications. Alternative technical approaches such as very prolonged inflations and oversized balloons were used in most but not all of the patients. Therefore, this series cannot be used to judge the absolute role of stents in managing dissection and acute closure. Treatment of the vessels was not delayed until absolute vessel obstruction supervened, and it remains uncertain what proportion could have been managed with anticoagulation alone. Prospective randomized studies comparing various technologies and strategies will be needed to further define these issues.

On the basis of lesion and vessel morphology and clinical presentation, it is possible to predict which patients are at greatest risk of acute closure,1–7,9,29,33,34 but the nature of balloon dilation makes such assessment imprecise. Furthermore, the ischemic consequences of a residual lesion or dissected segment after PTCA varies markedly, depending on the pathophysiological circumstances (i.e., amount of myocardium in jeopardy, presence of collaterals, and extent of disease and baseline left ventricular dysfunction).35 Although angiographically evident dissection or intimal tearing has been reported in 30–40% of all vessels having PTCA,3 only approximately one third will develop an ischemic complication.27,36

For any device or study examining the treatment of acute closure, the most important question is when to intervene in the process. The purpose of this study was not only to describe the early experience with this stent prototype but to characterize the spectrum of patients who might potentially benefit from such an intervention. The definition of threatened closure in this study used the presence of two or more adverse angiographic or clinical criteria. Such a definition should have clinical use and be sufficiently sensitive to enable the recognition and management of problems before patients leave the angioplasty suite. Because coronary stenting is not without some risk and additional costs, any definition of threatened closure must also have a level of specificity that avoids a large percentage of unnecessary stenting procedures. The proposed criteria appear to meet these needs, but prospective evaluation of outcome comparing alternative nonsurgical strategies is needed.

The post-PTCA angiographic appearance of the dilated artery is most predictive of an acute ischemic event. Intimal dissection and residual percent diameter stenosis have been shown repeatedly to increase the probability of a major complication3–9,29,37 and formed the basis of our definition of threatened closure. The presence of intimal dissection after PTCA is associated with a sixfold risk of an acute closure or an ischemic event.3,4 The absolute length and the presence of extraluminal contrast are the most important characteristics of a dissection and predict ischemic events with odds ratios of 15.6 and 4.2, respectively.7 In the present study, we used these two features to define the presence of a significant dissection. Such a dissection was present in 53 patients (50%) of the study population. Of these patients, 34 (64%) had evidence of myocardial ischemia. Angiographic dissection, however, is not the sole cause of acute closure. Recoil of the dilated artery with a residual significant stenosis is important in some pa-
tients. Of the 66 patients in our group without significant dissection, 35 (53%) had angiographic evidence of some intimal disruption, 30 (45%) had a ≥70% residual stenosis, and 38 (56%) had evidence of clinical ischemia. The residual percent diameter stenosis is probably the most compelling angiographic feature because it constitutes failure of dilation and is a major impetus for surgical intervention after PTCA. The 10% incidence of acute closure (100% occlusion) in this series reflects the strict angiographic definition used. For example, seven patients with acute ischemia who were returned to the laboratory for emergency repeat PTCA and who only had partial success from repeat dilatation were classified angiographically as threatened closure based on the appearance of the vessel immediately before stenting. Because luminal borders after PTCA are sometimes difficult to assess, contrast flow was used as a physiological marker of residual obstruction. Seventeen patients (14%) had TIMI grade 2 flow, and this was associated with a significant dissection or ≥50% stenosis in each patient. Sixteen of the 17 patients had evidence of clinical ischemia. Thus, in this series, TIMI flow added little to the characterization of the patients at risk. Overall, ongoing ischemia or its recurrence in the early period after PTCA occurred in 73 of 107 (68%) patients with threatened closure.

One important question to be resolved is the timing of stent placement in the management of suboptimal PTCA results. Whereas additional dilatation with higher inflation pressures or large balloons may solve the problem in some patients, it may also extend the dissection in others. In the present series, extensive dissection was more difficult to control with stenting. In asymptomatic patients with TIMI grade 3 flow, despite a poor angiographic appearance, a trial of anticoagulation may be attempted. Although some patients in this category do well, in this series and others, 10 acute closure after leaving the catheterization laboratory was associated with significant MI. In addition, once the coronary guide wire has been removed, it is sometimes difficult to recross the dissected vessel segment should this become necessary. The NHLBI registry showed that even transient occlusion complicating PTCA is associated with increased mortality during the first 18 months of follow-up.

In this series, acute or threatened closure resulting in stent placement occurred in 118 of 1,338 procedures (8.8%). This incidence of closure or threatened closure is relatively high; however, as a tertiary referral center for PTCA, many of the patients are referred with complex lesions, specifically for consideration of stenting should dissection eventuate. Eighty-five percent of the lesions stented were the more complex class B or C lesions, which have been reported to have an incidence of acute complications up to 21%. Furthermore, some patients with severe dissection were referred to our institution specifically for insertion of a stent as alternative management to coronary bypass surgery.

Historically, the management of acute or threatened closure has involved referral for CABG. In-hospital CABG was used in 27% of the PTCA patients in the first NHLBI registry. The management of acute closure has changed as experience has shown that repeat dilation can produce satisfactory results. In many patients, prolonged inflations and the use of perfusion balloon catheters have proved useful as methods of controlling acute closure or dissection and avoiding referral for urgent CABG. In one series with an 8% incidence of acute closure, 45% of patients had the vessel reopened with repeat PTCA. Overall, mortality was 7.5%; 22% had CABG, 10% had Q wave MI, and 20% suffered a non-Q wave MI. The repeat PTCA patients had a 30% incidence of non-Q wave MI.

Coronary artery bypass grafting currently remains the standard therapy for acute or threatened closure after PTCA, despite the significant operative mortality and the high incidence of MI. The most recent NHLBI PTCA registry report demonstrated a 6.8% incidence of emergency CABG. Only 20% had coronary occlusion, with another 31% having coronary dissection defined as extending distally or remote from the lesion; another 21% had prolonged angina or coronary spasm as the indication for surgery. Other studies reporting events that lead to CABG after PTCA noted dissection in 35–65% and artery occlusion in 38–44%. The incidence of Q wave MI in reported surgical series ranges from 21% to nearly 70%. The operative mortality for emergency coronary surgery varies from 1.4% to 26%. The incidence of postoperative hemorrhage, sternal wound infections, and other postoperative complications are also higher in patients having emergency surgery.

The initial in-hospital results in this series of patients stented for acute or threatened closure appear to suggest stenting as an acceptable alternative management for these patients. The 1.7% mortality is similar to that reported for elective bypass surgery. Although 7% of our group had Q wave MI, including those patients with evolving MI before PTCA, this appears favorable compared with surgical series. In the patients entering the laboratory without evolving MI, the incidence of Q wave MI was 4%, and all were due to early problems with stent thrombosis. Most importantly, CABG was used in only 4% of the series. At our institution, the incidence of surgical referral after PTCA has fallen from 3.4% of 378 cases in the year up to October 1989 to 1.1% of the last 1,338 cases done over a similar time period. We believe that use of the stent has been largely responsible for this reduction in CABG after PTCA. Overall, the incidence of MI in this series appears to be less than previously reported when similar patients are treated with urgent repeat PTCA alone.

The 10% incidence of major bleeding events does include the five patients who required vascular surgical repair of their femoral access vessels. This represents a significant limitation of the stenting procedure and clearly reflects the aggressive anticoagulation needed to maintain initial stent patency. The occurrence of bleeding complications has been reduced from 20% in the first third of the series to 6% in the last third, reflecting the growing expertise in managing these patients, particularly in limiting ambulation for 48 hours after the procedure.

The occurrence of late stent occlusion occurring between days 1 and 5 after the procedure (Table 5) is a further limitation that resulted in a significant number
of ischemic complications. One factor that may have played a role in these occlusions is the stent size. A 2.5-mm-diameter stent was used in 47% of our patients but was associated with 77% of the late occlusions. Seven of the nine events occurred in the first 40 patients. The decrease in the incidence of stent thrombosis with time related to both experience gained in managing dissections and better control of anticoagulation and antiplatelet therapy. All stent thrombosis events occurred within 20-mm devices. There has been no stent thrombosis in the 27 patients who have received the shorter 12-mm model.

Although the overall insertion success rate was 97%, eight patients had a suboptimal angiographic result. In each of these patients, the placement of a stent failed to fully control dissection within the vessel. Thus, although the stent frequently produced good control of dissection, it is not entirely fail-safe, and alternative therapies (i.e., CABG) may be needed.

Other groups22–25 have reported the use of stent devices to treat acute closure. Sigwart et al23 published a small series of 11 patients in 1988. They reported no deaths, two non-Q wave MIs, one patient requiring a transfusion, and one late stent thrombosis at 3 months. A preliminary report of stenting for suboptimal PTCA results was provided by Schatz et al24; they reported on 93 patients who received the Palmaz-Schatz stent for suboptimal PTCA results. There were no deaths in the group; there was a 3.2% incidence of infarction, and 2.3% underwent CABG and 5.3% had late stent closure. This preliminary report suggests a retrospective identification subgroup of a larger study of patients who were carefully selected for elective use of the stent as a primary adjunct to balloon dilation. In contrast, the patients in the present series were stented on an emergency basis, having more severe residual narrowing and more extensive dissection.

Although in-hospital results appeared acceptable, repeat revascularization, PTCA, or CABG was required in 38 patients. The majority (30 patients) were successfully managed with repeat PTCA. Preliminary results suggest that the incidence of restenosis is similar to that seen in acute closure managed by dilatation alone and can be managed by repeat conventional dilatation.46 Recent data from our laboratory have demonstrated that the incidence of restenosis is significantly lower in patients with stents 3.0 mm or greater in diameter (25%) and in patients with a stent-to-artery ratio of ≥1 (26%). Additional dilation of the previously stented site is technically straightforward, and immediate and late results are encouraging. One report has suggested that patients should be sent to CABG after being stabilized with the use of intracoronary stents.47 The short-term follow-up results in our series indicate that such an approach is unnecessary, at least with the stent used in the present study. The large majority of patients avoided Q wave MI and CABG. Whether this will impact favorably on late outcome remains to be determined.

Conclusions

In this series, stenting for acute or threatened closure appeared to limit the need for emergency CABG and to probably reduce the incidence of MI. Although much additional work is required to define the role of these and similar devices, these data suggest that stenting represents a useful adjunct to balloon dilatation. A multicenter study is in progress to examine the broader application of this device in the community, and prospective randomized trials comparing stents with alternative methods are planned.

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