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Abrupt Vessel Closure Complicating Coronary Angioplasty: Clinical, Angiographic and Therapeutic Profile

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To assess the clinical, angiographic and procedural correlates of outcome after abrupt vessel closure during coronary angioplasty, results were analyzed of 109 patients (8.3%) who had abrupt vessel closure during 1,319 consecutive coronary angioplasty procedures performed between July 1, 1988 and June 30, 1990. These 109 patients had a mean age of 59 ± 11 years; 63% were male, 57% had had a prior myocardial infarction and 61% had multivessel disease. Coronary angioplasty was performed in the settings of acute myocardial infarction (14%), recent myocardial infarction (36%), unstable angina (34%) and stable ischemia (29%).

Abrupt vessel closure occurred at a median of 27 min (range 0 min to 5 days) from the first balloon inflation. By angiographic criteria, thrombus or coronary dissection was identified in 20% and 28% of cases, respectively; both thrombus and dissection were present in 7% of closures, and 45% were due to indeterminate mechanisms. Successful reversal of abrupt vessel closure, defined as restoration of normal Thrombolysis In Myocardial Infarction (TIMI) grade 3 flow without resultant Q wave myocardial infarction, emergency bypass surgery or death, was achieved in 47 patients (43%). By hierarchal analysis, the incidence of death, emergency coronary bypass surgery, Q wave and non-Q wave myocardial infarction was 8%, 20%, 9% and 11%, respectively.

Univariate analysis using 23 clinical, morphologic and procedural variables demonstrated that successful outcome after abrupt closure was associated with prolonged balloon inflations (>120 s) (odds ratio = 6.87, $p < 0.001$), unstable angina (odds ratio = 2.37, $p = 0.034$) and placement of an intracoronary stent (odds ratio = 5.33, $p = 0.062$). By multivariate analysis, independent correlates of successful outcome were prolonged balloon inflations (odds ratio = 5.11, $p = 0.001$) and intracoronary stenting (odds ratio = 4.37, $p = 0.049$).

Thus, although prolonged balloon inflations and intracoronary stents may improve outcome after abrupt vessel closure, the cumulative risk of morbidity or mortality remains significant and mandates investigation into improved strategies for its prevention and treatment.

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Abrupt vessel closure complicating coronary angioplasty, while infrequent, has important clinical consequences. The National Heart, Lung, and Blood Institute (NHLBI) 1985–86 Percutaneous Transluminal Coronary Angioplasty Registry reported that 20% of all deaths, 40% of myocardial infarctions and 25% of coronary artery bypass operations recorded at 1-year follow-up study occurred in the 6.8% of patients who had periprocedural coronary occlusion (1,2). Although equipment design and operator experience have improved steadily since 1977, the proportion of patients undergoing coronary angioplasty with complex coronary disease or acute ischemic syndromes has also increased (3); the incidence of abrupt closure has thus remained largely unchanged (1,4–7).

Several novel percutaneous techniques have been developed that are directed at reversal of abrupt closure or amelioration of myocardial ischemia caused by vessel occlusion. Although prior data suggest that standard (8,9), perfusion (10,11) and laser balloon angioplasty (12), intracoronary stenting (13), thrombolytic therapy (14,15), catheter reperfusion (16,17) and intraaortic balloon counterpulsation (18) may each be effective in reducing the ischemic consequences of acute coronary occlusion, no study has assessed the ability of these new methods, individually or in combination, to alter the clinical outcome after abrupt closure. Thus, to evaluate the efficacy of current strategies for management of abrupt vessel closure complicating coronary angioplasty, we retrospectively analyzed our experience over a 2-year period with 109 consecutive abrupt vessel closures complicating coronary angioplasty.

Methods

Study patients. From July 1, 1988 until June 30, 1990, 1,342 coronary balloon angioplasty procedures were performed at the University of Michigan Medical Center. Informed written consent was obtained for each procedure,

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and patient confidentiality was maintained during the subsequent retrospective analysis. Cardiac catheterization or morbidity and mortality records for 23 of these patients were unavailable for review. The 1,319 remaining catheterization records were screened for documentation of periprocedural vessel closure or ischemic complications. Additionally, to avoid inadvertent exclusion of patients who experienced in-laboratory abrupt closure without apparent clinical or hemodynamic sequelae, cineangiograms were also reviewed for all patients in whom more than three balloon inflations or balloon inflations lasting >120 s were performed. On the basis of this initial screening, 302 cineangiograms were evaluated for angiographic evidence of abrupt vessel closure. The 109 patients who met criteria for abrupt closure form the basis of this study.

Angioplasty procedure. In general, unless such treatment was contraindicated by a history of allergy or drug intolerance, all patients undergoing coronary angioplasty were pretreated with aspirin, heparin (10,000-U intravenous bolus administered 5 to 10 min before angioplasty, with a 5,000-U bolus repeated hourly throughout the procedure), nitrates and calcium channel antagonists. Coronary angioplasty was performed by methods described in detail elsewhere (19). In the event of intimal or medial disruption or intracoronary thrombus formation at the angioplasty site, final angiography was performed after an observation period of at least 10 min while patients remained in the cardiac catheterization laboratory with the guide wire across the lesion; in these instances, planned dilation of other target stenoses was staged to a later date and patients were usually treated with a heparin infusion for 12 to 48 h. Unless anticoagulation was clinically indicated because of preprocedural myocardial infarction or unstable angina, heparin was discontinued on completion of uncomplicated angioplasty procedures, and patients were monitored for at least 18 h in the cardiology inpatient unit.

Management of abrupt closure. Although operators followed general guidelines based on their interpretation of the video fluoroscopic images, abrupt closure was managed on an individual basis without a uniform protocol. After the angiographic demonstration of abrupt closure, additional heparin and intracoronary or intravenous nitrates were administered. *Hemodynamic instability*, defined by a systolic blood pressure <80 mm Hg unresponsive to crystalloid infusion, was immediately treated with intravenous metaraminol, dopamine or intraaortic balloon counterpulsation. Percutaneous cardiopulmonary bypass was used as adjunctive therapy in the event of cardiovascular collapse. Patients with persistent hemodynamic compromise despite pharmacologic or mechanical support or those with *left main coronary closure* were usually referred for immediate coronary bypass surgery. Otherwise, *coronary dissection* was managed with progressively longer balloon inflations until a satisfactory angiographic result was obtained; the autoperfusion catheter was used in patients who were unable to tolerate prolonged inflations with standard balloons. With a significant residual flap, directional coronary atherectomy was occasionally performed. The Gianturco-Roubin intracoronary stent, available at our institution since March 1990, was implanted in those patients in whom balloon inflations had failed or were thought likely to fail to reestablish a stable vessel lumen.

In the absence of contraindications to thrombolytic therapy, *thrombus* was treated with intracoronary and intravenous administration of urokinase or recombinant tissue-type plasminogen activator (rt-PA), or both agents; unless a substantial intracoronary clot burden was observed, balloon dilation was usually performed if thrombolytic treatment failed to restore adequate vessel patency. When the morphologic etiology of the closure could not be determined or when thrombus appeared superimposed on coronary dissection, repeat balloon dilation, with or without thrombolytic therapy, was generally attempted. Emergency coronary bypass surgery was usually recommended for persistent myocardial ischemia or hemodynamic instability, at which time a "bail-out" translesional perfusion catheter or intraaortic balloon pump was often placed.

Clinical and procedural variables. Clinical and procedural variables were obtained by retrospective review of catheterization laboratory and medical records. Age, gender, prior myocardial infarction (including the administration of thrombolytic agents within the previous 6 weeks) and previous angioplasty attempts were recorded. *Clinical setting* was defined as one or more of the following: acute (≤24 h) or recent (≤6 weeks) myocardial infarction, unstable angina (postinfarction angina, rest angina or crescendo exertional angina), or stable ischemia.

Clinical end points included *successful reversal of abrupt closure*, *procedural success* (defined as a residual target stenosis <50% in diameter), *myocardial infarction*, *emergency coronary artery bypass surgery* or *death*. Additional selected definitions were:

Abrupt vessel closure was considered to be *complete* (Thrombolysis In Myocardial Infarction [TIMI] [20] grade 0 or 1 distal flow) or *partial* (*worsening stenosis with* TIMI grade 2 distal flow) closure of a previously patent (TIMI grade 2 or 3) coronary vessel during or after coronary angioplasty within the period of hospitalization. Abrupt vessel closure of a previously totally occluded artery was defined as closure after establishment of TIMI grade 3 flow during an initially successful dilation.

Successful reversal of abrupt vessel closure was defined by restoration of normal TIMI grade 3 flow beyond the site of closure without death, need for immediate coronary artery bypass surgery or development of resultant Q wave myocardial infarction.

Non-Q wave myocardial infarction was defined as an elevation in peak serum creatine kinase (CK) level to >3 times the upper limit of normal with an MB isoenzyme fraction >3% without the development of pathologic Q waves or a significant R wave in lead V_1.

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Angiographic analysis. Cineangiographic films were reviewed by a single experienced angiographer who had no knowledge of the clinical outcome. Lesion percent diameter stenoses were measured by calipers, and the American College of Cardiology/American Heart Association (ACC/AHA) Task Force stenosis characteristic type (21) as modified by Ellis et al. (22) and TIMI grade coronary flow were qualitatively graded. On the basis of the angiographic appearance of the vessel just before and at the time of abrupt closure, the morphologic cause of closure was defined by one or more of the following:

Coronary dissection was defined by the presence of a curvilinear filling defect parallel to the vessel lumen, contrast medium outside of the vessel lumen persisting after passage of contrast medium, or a spiral-shaped filling defect partially or totally obstructing the coronary artery lumen.

Thrombus was defined by the presence of a discrete or mobile intraluminal filling defect visible at the site of closure.

Indeterminate morphologic cause of closure was present if the vessel's angiographic appearance was of a radiolucent or hazy luminal irregularity or a persistent filling defect that was neither discrete nor mobile, with delayed anterograde flow or total coronary occlusion.

Guide catheter closure was present if the site of occlusion or the proximal extent of dissection was seen to be proximal to the target stenosis and adjacent to the lip of the guide catheter.

Guide wire closure was defined by an occlusion at a site of guide wire manipulation before passage of the angioplasty catheter or at a site distal to the balloon near the terminal end of the guide wire.

When full angiographic views of the closure were not recorded because of the clinical urgency of the abrupt closure (six patients) or when cineangiographic films were not available for review (three patients), morphologic data were not coded and these patients were excluded from the analysis of morphologic variables.

Statistical analysis. Continuous variables were expressed as mean values ± SD. Differences in dichotomous variables were compared using the Fisher chi-square significance test (with the Yates correction when appropriate), and odds ratios for variables significant to the $p < 0.10$ level were calculated. For sequential treatment strategies (inflations of standard duration, prolonged inflations, intracoronary stenting and coronary atherectomy), successful outcome was credited only to the final procedure employed; intermediate treatments producing inadequate angiographic results were coded as failures. Thrombolytic therapy, whose beneficial effects may be delayed after administration, was considered to be a concurrent treatment variable when it was used in combination with other techniques, and was credited with success or failure according to the final procedural outcome. Multivariate analysis by multiple logistic regression was performed to identify variables independently correlated with outcome, using only those variables significant at the $p < 0.10$ level in the univariate analysis. Analyses were performed using SYSTAT (the System for Statistics).

Table 1. Baseline Clinical and Angiographic Characteristics of 109 Patients

Mean age ± SD (yr) (range)	59 ± 11 (21–83)
Male patients (%)	63
Previous PTCA (same site) (%)	13
Prior MI (%)	57
Clinical setting (%)	
Acute MI	14
Recent MI (± unstable angina)	36
Unstable angina	34
Stable ischemia	29
Thrombolytic therapy (≤6 weeks) (%)	18
No. of diseased vessels (>50% DS) (%)	
1	39
2	36
3	25
Vessel dilated (%)	
LAD	35
LCx	24
RCA	37
SVG	4
Modified ACC/AHA lesion score (%)	
A	8
B1	20
B2	44
C	28
Thrombus before dilation (%)	23
Total occlusion dilated (%)	20

ACC/AHA = American College of Cardiology/American Heart Association; DS = diameter stenosis; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; MI = myocardial infarction; PTCA = coronary angioplasty; RCA = right coronary artery; SVG = saphenous vein graft.

Results

Patient characteristics and features of abrupt vessel closure (Tables 1 and 2). During the study period, coronary angioplasty was performed in 1,319 patients, of whom 109 (8.3%) developed abrupt vessel closure (Table 1). Notably, >67% of the 109 patients experienced abrupt vessel closure in the setting of an unstable coronary syndrome (acute or recent myocardial infarction or unstable angina). Nearly 75% of closures occurred on dilation of lesions with relatively unfavorable morphology (modified ACC/AHA score B2 or C). Abrupt closure produced complete interruption (TIMI grade 0 or 1 flow) of distal coronary perfusion in 83% of cases, whereas the initial decrease in blood flow was only partial (TIMI grade 2) in 17% (Table 2). As assessed by strict angiographic criteria, thrombus or dissection was identified as the cause of vessel closure in 20% and 28% of patients, respectively; both mechanisms appeared evident in 7% and the cause of abrupt closure was indeterminate in 45%.

Treatment of abrupt vessel closure. Catheterization laboratory management of abrupt vessel closure is outlined in

Table 2. Clinical and Angiographic Characteristics of Abrupt Vessel Closure in 109 Patients

Complete closure (%)	83
Partial closure (%)	17
Median time of onset (range) (from 1st balloon inflation)	27 min (0 min to 5 days)
Location at closure (%)	
Catheterization laboratory	82
Postprocedure bay area	6
Inpatient unit	12
Mechanism of closure (%)	
Guide catheter	10
Guide wire at target lesion	8
Guide wire distal to target lesion	1
Balloon catheter	81
Morphologic cause of closure (%)	
Thrombus	20
Dissection	28
Thrombus + dissection	7
Indeterminate	45
Hemodynamic instability (%)	32

Figure 1 according to angiographic morphology. For the overall group, repeat balloon dilation was performed in 91 of the 109 patients, with the size of the angioplasty balloon increased by 0.5 mm in 19 patients. Prolonged inflations (>120 s) were carried out in 73 patients, over half of whom had undergone preceding unsuccessful inflations of shorter duration; the Stack autoperfusion balloon catheter was required in 7 patients. Available for clinical use during the last 3 months of the study period, the Gianturco-Roubin intracoronary stent was used in 9 of the 22 closures occurring during that time. Directional atherectomy was performed in an additional 2 patients. Thrombolytic agents were administered to 43 patients after acute coronary occlusion (rt-PA in 7, urokinase in 29, both rt-PA and urokinase in 7). 81% of whom also underwent repeat balloon inflations. Doses ranged from 0.5 to 3 million U of urokinase and 20 to 100 U of rt-PA. A total of 17 patients were referred for coronary artery bypass surgery after unsuccessful attempts at resolution of closure in the catheterization laboratory.

Of the 54 patients with closure of indeterminate morphologic cause, 28 underwent mechanical attempts to reestablish vessel patency (balloon inflations, stenting or directional atherectomy), 4 received thrombolytic agents only and 19 were treated with combined therapy. In contrast, thrombolytic agents were used significantly less frequently in the patients with coronary dissection (only 6 of 35, 3 of whom had angiographically visible thrombus associated with the dissected lesion). Two thirds of patients in the dissection group were treated with a combination of standard or prolonged balloon inflations; intracoronary stenting or atherectomy was used in three patients and one patient, respectively. Intracoronary thrombus leading to abrupt vessel closure was treated with thrombolytic therapy in 70% of cases, although the majority of patients with this morphologic cause of closure also underwent repeat balloon dilations. The intracoronary stent was used in two of these patients, neither of whom had been treated with thrombolysis.

In only 11 patients was no attempt made to perform either repeat dilation or thrombolysis. Of these 11 patients, 8 underwent immediate bypass surgery with left main coronary artery closure (n = 2), shock (n = 3) or failure to pass the guide wire (n = 3); an additional 3 patients were treated medically for closure of the infarct-related vessel during rescue angioplasty for acute myocardial infarction.

Hemodynamic instability occurred in 34 patients (31%) with abrupt vessel closure; instability was successfully reversed in 22. An intraaortic balloon pump was placed in 17 patients: in 11 patients with hemodynamic compromise, in 5 as a bridge to coronary bypass surgery and in 1 elderly patient treated medically for refractory closure. Percutaneous cardiopulmonary bypass was instituted in four patients with hemodynamic collapse, three of whom had remained in unstable condition despite intraaortic balloon counterpulsation. Hemodynamic stability was restored in three of four patients, although only two survived to hospital discharge.

Clinical outcome of abrupt vessel closure (Fig. 2). Abrupt vessel closure was successfully reversed in 47 (43%) of 109 patients, and procedural success, defined by dilation to <50% residual stenosis, was accomplished in 42 patients (38%). Of the 47 patients in whom reversal of abrupt closure was considered successful, postprocedural CK determinations were obtained in 32 (68%); excluding the 6 patients in whom angioplasty was performed in the setting of acute myocardial infarction, 8 patients had elevated (>3 times upper limit of normal) peak CK levels of 1,135 ± 560 IU/liter. Thus, among "successfully" managed patients with abrupt vessel closure whose CK values were measured, the incidence of resultant non-Q wave myocardial infarction was approximately 30%.

Of the 62 patients with an unsuccessful outcome, 9 died (3 after coronary bypass surgery, 5 of refractory cardiogenic shock in the catheterization laboratory and 1 with multiple complications including cerebrovascular hemorrhage 13 days after abrupt vessel closure and intracoronary stenting). Notably, three of four patients with abrupt closure involving the left main coronary artery (during angioplasty of the left anterior descending or circumflex artery) died. Successful coronary bypass surgery was performed in 22 patients, in whom postprocedural CK determinations and electrocardiograms (ECGs) were not routinely obtained. Of the remaining 31 patients with unsuccessful treatment of abrupt vessel closure, 10 had a Q wave myocardial infarction (peak CK 2,148 ± 1,530 IU/liter), 4 developed a non-Q wave myocardial infarction (peak CK 662 ± 193 IU/liter), 8 had undergone rescue angioplasty for ongoing acute myocardial infarction (development of Q waves or CK elevation could not be ascribed to the abrupt closure) and 9 had no clinical sequelae, even though TIMI grade 3 flow could not be restored across the site of closure. Two patients (1.8%) in whom abrupt closure was initially reversed in the catheterization

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laboratory subsequently (at 2.5 and 46 h, respectively) developed reclosure that was unsuccessfully managed; no patient with late reclosure had a successful outcome.

The cumulative risk of morbidity and mortality (death, emergency coronary bypass surgery, Q wave or non-Q wave myocardial infarction) for the entire group of 109 patients with abrupt vessel closure was 48%. This value most likely underestimates the morbidity of closure, because it does not include the 14 patients who developed ECG Q waves or CK elevation in the setting of rescue angioplasty for acute myocardial infarction.

Correlates of successful reversal of abrupt vessel closure. Table 3 summarizes rates of successful resolution of abrupt closure with different treatment modalities for each of the three angiographic morphologic causes of closure. Thrombolytic therapy was unsuccessful in every case in which it was used alone, and concomitant administration of thrombolytic agents at any dose employed did not improve the likelihood of success with other management techniques.

Notably, thrombolytic therapy appeared to be equally ineffective for treating intracoronary thrombus, dissection and closure of indeterminate morphology. Similarly, balloon inflations of standard duration (<120 s) were rarely sufficient to restore an adequate angiographic appearance. Irrespective of the morphologic causes of closure, however, prolonged balloon inflation resulted in successful resolution of closure in nearly 50% of patients in whom it was performed. An intracoronary stent, used in nine patients over a 3-month period, produced an excellent angiographic result in every case, although one patient subsequently died of multiple complications and another had a Q wave myocardial infarction during the 3-h elapsed time between his out-of-laboratory vessel closure and its treatment. Both patients undergoing directional atherectomy had successful resolution of vessel closure.

Clinical, angiographic and procedural correlates of successful outcome after abrupt vessel closure are listed in Table 4. By univariate analysis, prolonged balloon inflations ($p < 0.001$), unstable angina ($p = 0.034$) and intracoronary stenting ($p = 0.062$) were associated with a successful outcome to a significance level of $p < 0.10$, whereas stable ischemia ($p = 0.04$), guide wire-induced closure ($p = 0.061$), and closure during angioplasty of the left anterior descending coronary artery ($p = 0.075$) were associated with unsuccessful outcome. Notably, closure morphology (thrombus or dissection) had no demonstrable correlation with the likelihood of successful outcome or with the effectiveness of the various treatment strategies. With use of multivariate analysis, two factors were found to be independent correlates of successful resolution of closure: prolonged balloon inflations

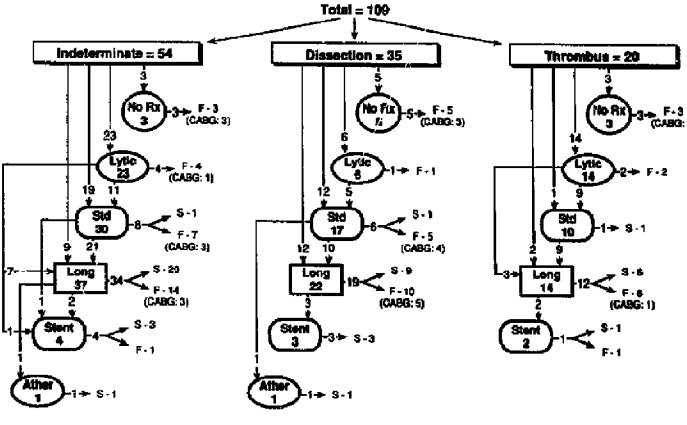

Figure 1. Diagram illustrating the management of abrupt vessel closure in 109 patients. Patients are grouped according to the angiographic morphologic cause of closure as indeterminate (n = 54, including the 6 patients with inadequate visualization of closure and the 3 whose cineangiographic films were unavailable), dissection (n = 35, including the 7 patients with mixed dissection and thrombus morphology) and thrombus (n = 20). Ather = coronary atherectomy; CABG = coronary artery bypass graft surgery; F = failed resolution of closure; Long = repeat balloon inflations of duration ≥120 s; Lytic = intravenous or intracoronary administration of recombinant tissue-type plasminogen activator, urokinase, or both agents; Rx = treatment; S = successful resolution of closure; Std = repeat balloon inflations of duration <120 s.

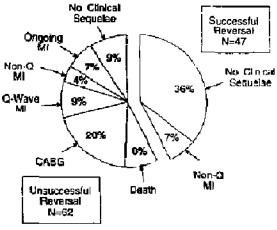

Figure 2. Chart illustrating final hierarchic (death, emergency coronary bypass surgery, Q wave myocardial infarction, non-Q wave myocardial infarction) outcome for all 109 patients after abrupt vessel closure. The shaded pie slices represent unsuccessful outcome among 57% of patients; the white slices successful resolution in 43% of patients. MI = myocardial infarction; ongoing MI = patients undergoing rescue angioplasty for acute myocardial infarction, in whom development of Q waves or serum creatine kinase elevations could not be attributed to abrupt closure; other abbreviations as in Figure 1.

(odds ratio = 5.11, p = 0.001) and intracoronary stenting (odds ratio = 4.37, p = 0.049).

Discussion

In the current series, abrupt vessel closure complicated 109 (8.3%) of 1,319 angioplasty procedures performed over the 2-year period from July 1988 through June 1990. Despite the availability of a wide array of adjunctive treatment methods and support devices, successful resolution of abrupt vessel closure was accomplished with percutaneous techniques in only 43% of patients. Abrupt closure carried a substantial risk of morbidity and mortality; by hierarchal analysis, 8% of patients died, 20% required emergency bypass surgery and at least 9% and 11%, respectively, had a Q wave or non-Q wave myocardial infarction. Moreover, even among "successfully" managed patients, a non-Q wave myocardial infarction occurred in approximately 30%. Multivariate analysis demonstrated that prolonged balloon inflations and intracoronary stenting were independently correlated with successful outcome.

Incidence and clinical consequences of abrupt vessel closure. Depending on the definition used, the reported incidence of abrupt vessel closure after coronary angioplasty has been variable (Table 5). Although the 8.3% incidence rate of abrupt closure in the present series is higher than may be expected from prior work, previous studies have often excluded patients with incomplete reduction in coronary perfusion (6,23), angioplasty for acute myocardial infarction (4,24), in-laboratory closure (25,26) or closure preceding successful angioplasty (6,23). The reported incidence of abrupt closure in this study, therefore, may more accurately reflect the expected frequency of abrupt closure in a more heterogeneous group of patients undergoing coronary angioplasty.

In the present series, nearly 90% of vessel closures occurred while the patient was in the catheterization laboratory or adjacent holding area. Although this frequency of in-laboratory closures may be slightly higher than the 71% to 80% incidence rate reported by other groups (2,23,24), the rigorous criteria for review of cineangiograms in this study may have detected more patients with transient in-laboratory closure than were detected in prior analyses.

Similar to earlier reports (2,23), abrupt vessel closure was successfully reversed in nearly 50% of patients. Although the mortality rate of 8.3% in the current series is higher than *the 2% to 6% mortality rates previously reported (2,4,23,24),* high risk subgroups such as patients with acute myocardial infarction were included in the present analysis. The 20% incidence rate of emergency bypass surgery and 20% incidence rate of periprocedural myocardial infarction in this series compares favorably with rates in prior studies.

Predictive factors of abrupt closure. Several clinical and angiographic features have been associated with a heightened risk of procedural complications during coronary angioplasty. These clinical correlates include unstable angina (27), diabetes mellitus (22), inadequate antiplatelet therapy (28), female gender (4) and extreme age (29). Two large series (4,22) have also demonstrated that angiographic fac-

Table 3. Rates of Successful Resolution of Abrupt Closure With Different Treatment Modalities in 109 Patients

Treatment	Indeterminate n	Indeterminate Success Rate	Dissection n	Dissection Success Rate	Thrombus n	Thrombus Success Rate	Overall Success Rate
Thrombolytic agents only	4	0%	1	0%	2	0%	0%
Other Rx with thrombolytic agents	19	58%	5	40%	12	50%	53%
Other Rx without thrombolytic agents	28	46%	24	38%	3	67%	44%
Inflations of standard duration	30	3%	17	6%	10	10%	5%
Prolonged inflations	37	54%	22	41%	14	43%	48%
Intracoronary stent	4	75%	3	100%	2	50%	78%
Directional atherectomy	1	100%	1	100%			100%

Rx = treatment.

Table 4. Clinical and Procedural Correlates of Successful Reversal of Abrupt Vessel Closure

	n	Univariate p Value	Odds Ratio	Multivariate p Value	Odds (95% CI) Ratio
Clinical					
Mean age (yr)	—	NS	—	—	—
Male gender	69	NS	—	—	—
Acute MI	15	NS	—	—	—
Recent MI	40	NS	—	—	—
Unstable angina	38	0.034	2.37	NS	—
Stable ischemia	32	0.040	0.47	NS	—
Hemodynamic instability	35	NS	—	—	—
Closure in laboratory	97	NS	—	—	—
Angiographic					
Multivessel disease	67	NS	—	—	—
Angioplasty lesion					
LAD	38	0.075	0.47	NS	—
LCx	26	NS	—	—	—
RCA	41	NS	—	—	—
Initial total occlusion	22	NS	—	—	—
Thrombus before dilation	22	NS	—	—	—
Thrombus at closure	25	NS	—	—	—
Dissection at closure	32	NS	—	—	—
Complete (vs. partial) closure	91	NS	—	—	—
Proximal (vs. mid-distal) closure	46	NS	—	—	—
Guide catheter closure	11	NS	—	—	—
Guide wire closure	10	0.061	0.13	NS	—
Procedural					
Thrombolytic agents	43	NS	—	—	—
Standard inflations	60	NS	—	—	—
Prolonged inflations	73	0.001	6.87	0.001	5.11 (1.8–14.9)
Intracoronary stent	9	0.062	5.33	0.049	4.37 (0.8–25.2)
				$p < 0.0001$	

CI = confidence interval; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; MI = myocardial infarction; RCA = right coronary artery.

tors such as lesion length, stenosis bend angulation >45°, excessive proximal vessel tortuosity, intraluminal thrombus, other stenoses in the same vessel, multivessel disease and the modified ACC/AHA Task Force lesion score are associated with increased risk for major complications. Although these and other correlates of procedural complications may serve to stratify groups of patients according to anticipated risk, they generally have a low positive and negative predictive value (30); closure thus remains largely unforeseeable. Further, high risk characteristics are encountered commonly in clinical practice and may not be avoidable in those patients for whom angioplasty remains the preferred option for revascularization.

Profile of vessel closure. Strict criteria were applied to the angiographic identification of the probable cause of closure in the present series. Nearly 45% of morphologic causes of closure were indeterminate, many of which probably represented thrombus superimposed on a coronary dissection. Coronary artery vasospasm, with acute reduction of lumen diameter by up to 30%, has been shown (31) to occur routinely after balloon angioplasty and may be an important initiating event in the development of abrupt vessel closure. The lack of demonstrable angiographic improvement with intracoronary or intravenous nitrates among patients in the current series, however, indicates that vasospasm was likely not the *predominant* mechanism of closure, a finding consistent with those of the NHLBI Percutaneous Transluminal Coronary Angioplasty Registry (3). Somewhat surprisingly, the angiographic morphologic causes of closure did not influence the likelihood of resolution or the suitability of mechanical versus pharmacologic management strategies in this analysis. This finding may reflect the difficulty in differentiating between thrombus and dissection by angiographic appearance alone. Newer imaging modalities such as intracoronary ultrasound or angioscopy may offer greater utility in defining the mechanism of abrupt closure, with possible improved correlation between the morphologic causes of closure and outcome of various therapies.

Hemodynamic instability accompanied 31% of vessel closures in this series, although this complication was not a significant predictor of adverse outcome and was successfully reversed in >60% of cases. This favorable result may be related to the prompt availability of various methods of pharmacologic and hemodynamic support, the latter including the autoperfusion balloon (Stack) and "bailout" cathe-

Table 5. Reported Incidence of Abrupt Closure Complicating Coronary Angioplasty

Study (ref.)	No. of Patients	Incidence of Abrupt Closure
NHLBI (2)	1,801	6.8%
Emory (4)	4,772	4.4%
Beth Israel (23)	1,160	4.7%
Rotterdam (24)	1,423	7.3%
Current study	1,319	8.1%

ters, the intraaortic balloon pump and emergency percutaneous cardiopulmonary bypass.

Unsuccessful resolution of abrupt closure was associated by univariate analysis with angioplasty of the left anterior descending coronary artery. Although the reason for this association has not been defined, it is likely related to the substantial area of myocardium at risk for infarction during closure of this artery; death due to abrupt closure was more frequent in patients with closure after left anterior descending artery angioplasty (death occurred in 16% and 4% of patients with left anterior descending and other vessel closures, respectively, $p = 0.084$).

Treatment of closure. This study extends previous analyses by focusing on the efficacy of current catheterization laboratory strategies in reversing established abrupt vessel closure. With multivariate regression analysis, two procedural variables were associated with a four- to five-fold improvement in the likelihood of successful reversal of abrupt closure: prolonged (>120 s) balloon inflations and intracoronary stenting. Previous studies have suggested that prolonged balloon inflations may improve the immediate result after coronary angioplasty (32) and help reverse failed angioplasty (10). This effect may be due to improved remodeling of atherosclerotic obstruction or "tacking up" of a coronary dissection flap. The association between prolonged inflations and favorable outcome in the present series may, to an extent, reflect the adverse prognosis for patients in whom therapeutic options were limited by inability to recross the zone of occlusion. Nevertheless, prolongation of balloon inflations to >120 s had a salutary effect on the reversal of abrupt closure, whereas such benefit was not evident in patients treated only with inflations of standard duration. Symptomatic and hemodynamic tolerance of long balloon inflations may have reflected less underlying compromise following coronary occlusion and biased toward a favorable outcome independently of the effect of prolonged inflations as such; the retrospective nature of this study did not permit evaluation of this possibility.

The Gianturco-Roubin intracoronary stent (33), a balloon-expandable flexible stainless steel coil currently under investigation for the treatment of abrupt vessel closure, is designed to compress obstructive dissection flaps against the vessel wall and optimize blood flow across the lesion. These stents have been placed for actual or threatened acute closure in 42 patients in one series (34). During the current study, 9 of 22 cases of abrupt closure occurring during the period wherein stents were available were managed with stent placement, with successful resolution in 7 patients (78%). Notably, the two patients with an adverse clinical outcome had a successful angiographic result. Although the sample size was small, the substantial beneficial effect of this technique in the management of abrupt vessel closure warrants validation in larger groups of patients.

Other centers have reported similar improvements in outcome with new modalities for treatment of abrupt closure. Laser balloon angioplasty was used in 85 patients with severe dissection or abrupt vessel closure in a recent series (35), with an 85% rate of prevention of emergency bypass surgery. Directional coronary atherectomy was used for rescue in 30 patients with failed coronary angioplasty, with an overall success rate of 87% (36). In our study, atherectomy was used in only two patients with abrupt closure, both of whom had a successful outcome.

Despite the relatively large number of patients in this series receiving thrombolytic therapy for abrupt vessel closure (43 patients), no beneficial trend was observed at any dose used, irrespective of the angiographic morphologic cause of closure. This finding is supported by those of related studies, in which prophylactic administration of intracoronary urokinase during coronary angioplasty (14) and treatment of abrupt vessel closure with streptokinase (24) did not reduce the incidence of major complications. In view of the associated bleeding risk, particularly if emergency bypass surgery is required, it may be worthwhile to consider possible alternatives before thrombolytic agents are administered for abrupt closure.

Limitations. The current study has several important limitations. First, although the present analysis provides nearly complete in-hospital follow-up for "hard" clinical end points such as death, emergency bypass surgery and angiographic restoration of patency, complete ECG, enzymatic and hemodynamic data were not available for all patients. Notably, because of the limited specificity and sensitivity of new ECG Q waves or elevations in serum CK MB in the postoperative setting (37), an ECG and CK determinations were not routinely obtained in patients after coronary bypass surgery. Moreover, CK levels were not measured in 32% of patients with successful resolution of abrupt vessel closure. Because such patients tended to be those in whom closure responded promptly to treatment (and therefore had a relatively low likelihood of sustaining substantial myocardial necrosis), it is possible that our data overestimate the frequency of non-Q wave infarction after successful therapy. Nevertheless, the failure to obtain postprocedural CK determinations in all patients experiencing in-laboratory vessel closure during the study period constituted suboptimal clinical practice, and current laboratory protocols require CK measurements in patients after even transient coronary vessel closure.

Second, the treatment strategies for abrupt vessel closure

were not controlled and randomized in the present retrospective study, and with small numbers of patients in certain treatment subgroups, the true effect of these strategies may be difficult to assess. Nevertheless, this analysis provides a review of these management techniques when applied in a generally accepted manner based on the angiographic appearance of the vessel at the time of closure. Third, follow-up data after the hospitalization period were not obtained in our series, and issues of long-term outcome such as restenosis and late coronary bypass surgery after abrupt vessel closure could not be addressed. However, other recent studies of abrupt vessel closure have addressed this issue (2,24,38). The NHLBI Registry (2) documented excess rates of death and bypass surgery in patients with abrupt closure after angioplasty, and the incidence of restenosis after successful treatment of abrupt closure was 57% in a recent series from Emory University (38).

Conclusions. Abrupt vessel closure complicating coronary angioplasty remains an ominous event, with a high cumulative risk of morbidity or mortality. Short-term outcome may be improved by the performance of prolonged angioplasty balloon inflations after vessel closure, with an added benefit derived from intracoronary stenting in selected cases. No other treatment strategy studied, including thrombolytic therapy, was associated with improvement in final result. Although this study is subject to the limitations inherent in a retrospective analysis, these data serve to provide a current framework for management of abrupt vessel closure and underscore the need for new strategies for its prevention and treatment.

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