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COOPERATIVE STUDIES

In-Hospital Cardiac Mortality After Acute Closure After Coronary Angioplasty: Analysis of Risk Factors From 8,207 Procedures

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Cardiac death consequent to acute vessel closure after coronary angioplasty occurred in 13 of 294 closures from 8,207 consecutive procedures performed at two centers since 1981 (0.16% cardiac mortality rate). To determine the predictors of cardiac death after acute coronary closure, 50 clinical, angiographic and procedural variables were analyzed by an observer unaware of the clinical outcome for each of the 13 patients who died and also 100 patients randomly chosen, in whom vessel closure after angioplasty did not result in death during hospitalization. Univariate analysis found female gender ($p < 0.0001$), collateral channels from the vessel dilated ($p < 0.0001$), use of balloon counterpulsation ($p < 0.0002$), pre- and postprocedural hypotension ($p = 0.0003$ and $p = 0.003$, respectively), jeopardy score ≥2.5 ($p = 0.003$), left ventricular hypertrophy ($p = 0.013$), hypertension ($p = 0.02$), diabetes ($p = 0.02$) and multivessel disease ($p = 0.03$) to be predictive of death. Multivariate analysis found collateral vessels, female gender and multivessel disease to be independent predictors of death.

Thus, cardiac death after elective coronary angioplasty is very rare in experienced centers and occurs most often in women with a large amount of potentially ischemic myocardium. Hypotension often precedes the fatal closure event. Close attention to the amount of potentially ischemic myocardium and to the fluid volume status of these patients would seem to be especially warranted.

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Death after coronary angioplasty for stable or unstable angina pectoris is an extremely rare event in experienced centers, being reported in 0.1% of procedures in the largest recent review of angioplasty-related complications (1). In the 1985 National Heart, Lung, and Blood Institute Percutaneous Transluminal Coronary Angioplasty Registry (2), the mortality rate was 0.3% in patients with single vessel disease and 0.7% in those with multivessel disease. As indications for coronary angioplasty expand and more difficult anatomy is approached, the likelihood of fatal complications is increased (1,3).

To better define situations in which the risk of cardiac death is increased, we reviewed the combined experience of two high volume angioplasty centers. Since 1980, acute coronary closure has resulted in 13 cardiac deaths (Table 1) after 8,207 elective angioplasty procedures at the Emory University Hospital and the San Francisco Heart Institute (0.16% of procedures). To determine the risk factors for death after closure, these cases were reviewed in detail and compared with 100 cases of acute closure (4) that did not result in a fatal outcome.

Methods

Patient population. From July 4, 1980 (Emory University Hospital) and September 1, 1983 (San Francisco Heart Institute) until July 1, 1986, 8,306 percutaneous transluminal coronary angioplasty procedures were performed at the two institutions. Ninety-nine procedures were performed in the setting of an acute myocardial infarction (typical ischemic chest pain ≥30 min with associated electrocardiographic [ECG] changes). These procedures, performed in a relatively high risk setting (3), were excluded from this analysis. The remaining 8,207 procedures form the basis for this

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Table 1. Characteristics of 13 Patients With Cardiac Death After Coronary Angioplasty

Patient No.	Date	Age (yr) & Sex	Vessel Dil	Others >70%	Prior MI	LVH	Jeop Sc	Coll	Cause of Death
1	1/83	36F	Prox LCx	Prox LAD 100% Prox RCA 60%	Anterior	No	5.0	Yes	Left main dissection. Died secondary to cardiogenic shock 5 h after emergency CABG.
2	1/84	64M	Prox LAD Prox LCx Mid-RCA	None	No	Yes	5.5	No	RCA could not be dilated. LAD, LCx successfully dilated, but LAD closed in lab. Died secondary to cardiogenic shock 2 days after emergency CABG.
3	1/85	65F	Mid-LAD	Non-dom RCA 90%; OM 100%	Apical	Yes	2.5	No	Closed in lab. IABP placed. To CPB in 133 min, but died secondary to cardiogenic shock 2 days after CABG.
4	7/84	69F	Mid-LAD	PDA 70%	Apical	No	2.5	No	Hypotension of uncertain cause after angiography. Otherwise uncomplicated PTCA. Died suddenly the following night of apparent occlusion-related arrhythmia.
5	9/84	50F	Mid-RCA	Distal Cx 100%	No	No	2.0	Yes	Acute RCA closure in lab. Emergency CABG to RCA. Postop course complicated by right ventricular and inferior MI requiring IABP and pressors, then anuria and death on sixth postop day.
6	1/85	46F	Prox LAD	Distal PDA 100%	Anterior	No	2.5	Yes	Chest pain and anterior ST segments 30 min after return to floor, resolving with NTG/nifedipine. LIMA to LAD, but died of cardiogenic shock the next day.
7	2/85	67F	Prox LAD	Prox RCA 100%	Inferior	No	4.0	Yes	PTCA to RCA unsuccessful on previous day. LAD closed abruptly in lab and despite CABG with CPB in 40 min, patient died of cardiogenic shock 7 h after surgery.
8	9/85	72F	Prox LAD	Prox RCA 70%	No	Yes	4.0	No	Hypotension after NTG, nifedipine and angiography. LAD closed abruptly and patient could not be resuscitated for CABG.
9	10/85	68F	Prox LAD Mid-LCx	Mid-RCA 70%	Septal	Yes	4.0	No	LAD dilated first with hazy, but otherwise good result. LCx dissected during PTCA and with hypotension both vessels closed. CPR required before CABG. Died 28 days later of cardiac and multisystem failure.
10	3/86	65F	Prox LAD	None	No	No	3.0	No	Hypotension after NTG and angiography. LAD closed abruptly in lab with cardiac arrest. CPR caused cardiac contusion and tamponade. Patient died of cardiogenic shock.
11	3/86	70F	Prox RCA	Prox LCx 100%	Lateral	Yes	3.0	Yes	RCA closed abruptly 3 h after apparently successful PTCA. Brought back to lab in hypotensive condition and died secondary to shock despite the attempted PTCA.
12	5/86	55F	Prox LCx PDA	None	Lateral	No	3.0	Yes	Both LCx and PDA closed just after leaving lab. Repeat PTCA to PDA was successful, but LCx could not be crossed. Acute mitral regurgitation led to hypotension. Taken to OR with CPR and IABP, but died 6 h later of cardiogenic shock.
13	6/86	64F	OM graft	Prox RCA 100%	Inferior & apical	Yes	3.0	Yes	Graft dilated successfully, but closed with resultant pulmonary edema next day. Died after CABG because of cardiac and multisystem failure.

CABG = coronary artery bypass graft surgery; Coll = collateral vessels; CPB = cardiopulmonary bypass; CPR = cardiopulmonary resuscitation; F = female; IABP = intraaortic balloon pump; Jeop sc = jeopardy score; lab = cardiac catheterization laboratory; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LIMA = left internal mammary artery; LVH = left ventricular hypertrophy; M = male; MI = myocardial infarction; Non-dom = nondominant; NTG = nitroglycerin; OM = obtuse marginal artery; OR = operating room; PDA = posterior descending artery; Post-op = postoperative; Prox = proximal; PTCA = percutaneous transluminal coronary angioplasty; RCA = right coronary artery; Vessel dil = vessel dilated.

analysis. Of these, 294 (3.6%) resulted in acute coronary closure, defined as procedural-related ischemia resulting in emergency coronary artery bypass graft surgery, myocardial infarction, emergency repeat angioplasty or return to the cardiac catheterization laboratory, occurring at any time during hospitalization. Patients in whom ischemia resulted solely from closure of a side branch (5) were excluded from this definition. The predictors of acute closure itself during coronary angioplasty have recently been described (4).

One hundred patients from the group of patients with

acute closure who did not experience a fatal outcome during hospitalization were randomly selected for comparison with those who had a cardiac death. Thirteen patients with acute closure had a cardiac death (death in which congestive heart failure, myocardial ischemia or cardiac arrhythmias were directly contributory), and their records were compared with the group of patients that did not experience a fatal outcome. Six patients died under circumstances that were related to angioplasty, but in which cardiac complications were not believed to be directly causative (intracerebral hemorrhage in three, sepsis in one, anaphylaxis secondary to protamine in one and pulmonary embolus in one), and they were excluded from this analysis.

Coronary angioplasty procedure. The technique of angioplasty used here has been described elsewhere (6). Complications of the acute closure syndrome were managed on an individual basis. In general, however, if the occlusion occurred in the cardiac catheterization laboratory (55% of procedures) and the patient did not have resultant hypotension or an extensive dissection, an attempt was made to redilate the area of closure. If this failed, or if the patient's condition was unstable, an intraaortic balloon pump was inserted and the patient was transported for emergency surgery. More recently, translesional perfusion or "bailout" catheters (7) have been utilized if ongoing ischemia was present. If the coronary closure occurred after dismissal from the catheterization laboratory (45% of procedures), the patient was generally returned for an attempt at angioplasty if the initial dilation had produced a good result; the patient was sent immediately to the operating room if the initial dilation had resulted in a large intimal dissection or if other technical factors associated with the initial procedure suggested that successful repeat redilation would be unlikely.

Data collection and entry. Demographic and clinical information, angiographic and angioplasty measurements and postangioplasty data were recorded on standard forms by a physician prospectively during hospitalization. Specific laboratory data including electrocardiographic (ECG) results and creatine kinase serum levels and postangioplasty complication information, were recovered by a physician at the time of hospital discharge. This information was audited for completeness and entered into a computerized data bank. Further angiographic information was obtained by retrospective review of the patient's chart or cineangiograms, or both, by a reviewer unaware of clinical outcome. Coronary artery diameter was measured using a previously validated electronic digital caliper system (8) and expressed as the average of several apparently normal sites (left anterior descending coronary artery, proximal and mid; left circumflex, coronary artery proximal and mid; right coronary artery, proximal and mid and distal before the posterior descending branch) (9). Diameter stenosis was measured using digital calipers and expressed as the mean of two orthogonal views. Hemodynamic data consequent to the acute closure were obtained from the patient chart, catheterization laboratory log or pressure recordings. The clinical, angiographic, procedural and hemodynamic data collected for risk factor analysis are outlined in Table 2.

Table 2. Variables Analyzed as Potential Risk Factors for Cardiac Death After Coronary Angioplasty

Clinical	
Age	Time of day PTCA performed
Gender	Prior myocardial infarction
Hypertension	Prior bypass surgery
Diabetes mellitus	Pre-PTCA medications (nitrates, calcium channel and beta-blocking agents)
Hypercholesterolemia	
Height	
Weight	
Body surface area	ECG evidence of LVH
Unstable angina	
Angiographic	
Multivessel disease	Total jeopardy score
LV ejection fraction	Collateral vessels from artery dilated
Vessel dilated	
Pre-PTCA % stenosis	Coronary diameter (LAD, LCx, RCA, contralateral vessels)
Infarct score	
Closure score	
Procedural	
Year of PTCA	BP on arrival in catheterization laboratory
Brachial/femoral approach	
Multivessel dilation	
Pre-PTCA translesional gradient	Nitroglycerin pre-PTCA
Post-PTCA percent stenosis	Nifedipine pre-PTCA
Post-PTCA translesional gradient	Blood pressure after angiography
Dissection	
Location of acute closure (laboratory, corridor, floor)	Blood pressure after acute closure
Emergency PTCA attempted	Heart rate on arrival in laboratory
Balloon counterpulsation	
"Bailout" catheter	Heart rate after closure
Intraortic balloon counterpulsation	
Time on cardiopulmonary bypass	
Calculated	
Contractile reserve function	

BP = blood pressure; ECG = electrocardiogram; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; LV = left ventricular; LVH = left ventricular hypertrophy; PTCA = percutaneous transluminal coronary angioplasty; RCA = right coronary artery.

Definition of terms. Multivessel disease was defined as a ≥50% diameter stenosis in two or more major epicardial arteries or a large branch thereof. The "infarction score" was defined as a measure of the amount of dysfunctional left ventricular myocardium caused by previous myocardial infarction based on the jeopardy score of Califf et al. (10). In this scoring system the coronary tree is divided into six segments of nearly equal myocardial perfusion (11). A score

of 0.5 was given for each area of myocardium corresponding to a coronary segment that was hypokinetic, and a score of 1.0 was given to each area that was akinetic or dyskinetic. The total infarction score was the sum of scores for each of the six segments.

The "closure score" and total "jeopardy score" were defined as measures of potentially ischemic myocardium based on the same jeopardy score (10). The closure score was the sum of scores for the six areas if the vessel dilated were to close (assuming the myocardium perfused by that vessel would become akinetic), and the total jeopardy score was the sum of scores that would result if the myocardium subserved by the vessel dilated and all stenoses ≥70% were to become akinetic.

An index of contractile reserve function was developed as a measure of the contractile reserve of the nonjeopardized myocardium after acute coronary closure. This was defined as systolic blood pressure after acute closure divided by 6 minus the total jeopardy score.

Jeopardized collateral vessels were defined as collateral vessels emanating from the artery dilated beyond the site of dilation.

Statistical analysis. The chi-square significance test was used to assess differences in categorical variables, and the unpaired Student's *t* test was used to assess differences in continuous variables. Univariate and multiple stepwise logistic regression analyses were performed to determine the clinical, angiographic, procedural and hemodynamic predictors of cardiac death after acute closure. The multivariate analysis evaluating predictors with univariate correlation coefficients $p \leq 0.10$ was performed using data from the 62 patients (10 deaths) with complete data available. (The most common variables not available in all patients were the coronary vessel diameter, postangioplasty translesional gradient, which was not always measured, and closure and jeopardy scores because procedural angiograms did not always visualize nondilated arteries.) Data collection was 98% complete for the remaining variables.

Results

Clinical characteristics and predictors of death after acute closure. The clinical characteristics analyzed as potential risk factors are shown in Table 2. Selected clinical characteristics of the patients with fatal acute closure and nonfatal acute closure found to be related to subsequent death are shown in Table 3. The most outstanding difference between the two groups was the number of women with cardiac death. Twelve of 13 patients in whom a cardiac complication played a major role in their death were women ($p < 0.0001$). The presence of left ventricular hypertrophy by ECG ($p = 0.01$) and history of insulin-dependent diabetes mellitus ($p \leq 0.02$) were also more common in the group with fatal cardiac complications. Age, history of hypertension, smoking, hypercholesterolemia, unstable angina or previous coronary artery bypass graft surgery were not more common in the group with a fatal outcome. Lesser height and weight were slightly more common in the group with poor outcome ($p = 0.10$), but there were no differences in the height or weight of women who died and those who did not.

Angiographic characteristics and predictors of death after acute closure. The angiographic characteristics analyzed as potential risk factors are shown in Table 2. The characteristics found to be closely related to cardiac death are noted in Table 3. As might be expected, the presence of jeopardized collateral vessels ($p < 0.0001$), the amount of potentially jeopardized myocardium ($p \leq 0.003$) and the presence of multivessel disease ($p \leq 0.03$) were the most important predictors of death. The infarct score was higher for the group with fatal outcome ($p \leq 0.06$), but the jeopardy score was a somewhat more powerful predictor of fatal outcome ($p \leq 0.003$). Left ventricular function before the procedure, as assessed by the left ventricular ejection fraction, was not a predictor of outcome (patients who died, 52 ± 10%; survivors, 56 ± 10; p = NS). The vessel dilated (patients who died, left anterior descending artery 53%, left circumflex artery 30%, right coronary artery 17%; survivors, left anterior descending artery 55%, left circumflex artery 17%, right coronary artery 28%; p = NS) and the preangioplasty percent stenosis (patients who died, 79 ± 15%; survivors, 77 ± 14%; p = NS) were not predictors of outcome. Patients with a small vessel diameter more often had a fatal outcome (patients who died, 2.38 ± 0.29 mm; survivors, 2.82 ± 0.45;

Table 3. Univariate Predictors of Death After Acute Vessel Closure

	Patients Who Died: No. (%)*	Patients Who Survived: No. (%)*	p
Women	12 (92.3)	32 (32.0)	<0.0001
Collateral vessels from artery dilated	6 (50.0)	2 (2.4)	≤0.0001
Use of intraaortic balloon counter-pulsation	8 (61.5)	16 (16.2)	0.0002
Hypotension (SBP ≤90) after initial coronary arteriography and nitroglycerin and/or nifedipine	6 (54.5)	2 (4.0)	0.0003
Hypotension (SBP ≤90) after acute closure	10 (83.3)	28 (28.6)	0.003
Overall jeopardy score ≥2.5	10 (76.9)	25 (31.3)	0.003
LVH by ECG	5 (38.5)	4 (7.4)	0.013
Hypertension (SBP >150) in catheterization laboratory before PTCA	8 (61.5)	16 (18.6)	0.022
Diabetes mellitus	5 (38.5)	11 (11.0)	0.024
Multivessel disease	10 (76.9)	40 (41.2)	0.034

*Percent of patients for whom variable was known.
SBP = systolic blood pressure; other abbreviations as in Table 1.

Table 4. Multivariate Stepwise Logistic Regression Analysis in 13 Cases of Preprocedural Risk Factors for Cardiac Death After Acute Closure

Variable	Coefficient	p Value
Collateral vessels from beyond the site dilated	−1.256	0.0001
Women	−5.237	0.0014
Multivessel disease	−1.140	0.0020

$p = 0.05$), but this was largely accounted for by the fact that vessel diameter was smaller in women (Table 4).

Procedural characteristics and predictors of death after acute closure. The procedural characteristics were analyzed and those important in determining outcome are shown in Tables 2 and 3. A hypotensive response to routinely administered vasodilators (sublingual nitroglycerin, sublingual nifedipine or contrast dye) ($p \leq 0.0003$), a systolic blood pressure of <90 mm·Hg immediately after closure and arterial hypertension on arrival in the catheterization laboratory ($p \leq 0.003$) were significant univariate predictors of death after acute closure. The use of, or need for, postangioplasty intraaortic balloon pump placement was also associated with an increased risk of death ($p \leq 0.0002$). Year of coronary angioplasty, emergency angioplasty, number of vessels dilated, placement of a "bailout" catheter and emergency coronary artery bypass surgery were not predictive of a fatal outcome in this analysis. Similarly, the time to cardiopulmonary bypass was actually shorter for patients going to emergent bypass surgery who died than for those who did not die (83 ± 44 versus 143 ± 60 min, $p \leq 0.04$), presumably reflecting a more aggressive approach to those with graver hemodynamic compromise. The contractile reserve function for women who died was no different from that of women who had a better outcome (24 ± 3 versus 25 ± 7; p = NS), whereas the contractile reserve function for men was significantly higher (34 ± 10; $p \leq 0.05$).

Independent predictors of death. The risk factors found by multivariate analysis to be important predictors of death are shown in Table 4. The presence of collateral vessels emanating from beyond the site dilated, female gender and multivessel disease were the only significant independent preprocedural predictors of cardiac death. The use of intraaortic balloon pump support was also an independent predictor of death, but this was probably related to the hemodynamic severity of the ischemic insult.

Discussion

Cardiac death after elective coronary angioplasty in carefully selected patients with aggressive management of acute closure and capable surgical "backup" is fortunately rare in experienced centers, occurring in only 13 of 8,207 patients (0.16%). However, the number of cardiac deaths at these institutions in 1985 and the first half of 1986 exceeded that for the entire previous period, thus stimulating this analysis to attempt to define the risk factors for cardiac death after angioplasty.

Causative factors of acute postangioplasty closure. We reviewed 50 clinical, angiographic and procedural factors as possible risk factors for death with acute coronary closure. Multivariate analysis found three major independent preprocedural risk factors for death: collateral vessels emanating from the vessel dilated, female gender and the presence of multivessel disease. In addition, a jeopardy score ≥2.5 portended greater risk than the presence of multivessel disease, but its value was not available for all patients, thereby diminishing its power in the multivariate analysis.

Women versus men. The risk of coronary angioplasty previously has been noted to be higher in women than in men (12). It has been suggested that the early standard use of 3.0 mm balloons, which were sometimes oversized for women, predisposed them to acute closure and the consequent higher incidence of death. This analysis, however, demonstrates that other factors predispose women to an increased risk of death. Several mechanisms that unfortunately cannot be fully evaluated with these data emerge as possibilities. The female patients' more frequent hypotensive response to vasodilators and their poorer contractile reserve and the correlation of low body surface area (13) with increased risk of death suggest that volume depletion is more likely to occur in women because they have a smaller intravascular volume and, hence, are less able to respond to the stress of acute vessel closure. The correlation of left ventricular hypertrophy with risk of death might support this hypothesis, as patients with left ventricular hypertrophy are more preload dependent. In addition, women have smaller coronary vessels than do men (14), and the coronary artery diameter or the women who died was somewhat smaller than that of the women who did not have fatal complications. This suggests that the severity of disease may have been underestimated in these women, placing them at a higher risk of ischemia and death. Finally, women may have a less well conditioned left ventricle than do men, as reflected in their lower contractile reserve function. Mortality in women undergoing coronary bypass surgery has been found to be higher than for men undergoing the same procedure (15,16), although this difference appears to be decreasing with improved surgical techniques (17).

Jeopardy score and multivessel disease. The importance of the overall jeopardy score and the presence of multivessel disease, both of which reflect the amount of potentially ischemic myocardium, is not surprising. The jeopardy score also has been shown to be an independent predictor of outcome for patients with coronary artery disease who are treated medically (11).

Jeopardized collateral vessels. This factor was an even better predictor of risk than was the jeopardy score, perhaps

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because not all myocardial segments supplied by an artery with a ≥70% lesion would be expected to become ischemic in the event of an acute closure. In this series, there were two deaths among the four patients with collateral vessels who had dilation of the collateralized artery before dilation of the artery that closed. Of the four patients who had the collateralizing vessel dilated first, all four died. One might suspect that the former approach is safer, but this study has an insufficient number of patients to state this conclusively.

Hypotension after closure. Of the other predictors of risk, hypotension after vessel closure and the use of intraaortic balloon counterpulsation appear to reflect the amount of ischemic myocardium after closure. Hypotension after initial angiography may imply limited preload and propensity toward cardiogenic shock. The presence of left ventricular hypertrophy by ECG has been associated with a poorer coronary flow reserve (18) and a susceptibility to subendocardial ischemia (19). These factors and acute hypotension may contribute to the downward spiral of cardiogenic shock (20).

Risk factors. The risk factors for acute closure in the setting of coronary angioplasty were recently defined (4) as the presence of long stenoses, female gender, stenosis location at a bend or branch point, in situ thrombosis and other stenoses ≥50% in the vessel dilated. These factors, along with those identified in this study, may define a relatively high risk population for angioplasty.

Limitations of the study. This study is limited by the small number of end points (cardiac deaths), the lack of complete hemodynamic data including cardiac output and pulmonary capillary wedge pressures and the fact that newer methods of treating closure-induced ischemia were not available to most of these patients. The power of the three independent risk factors, however, is such that they are likely to continue to be risk factors for poor outcome.

Conclusions. We have defined the major factors that appear to predispose patients to cardiac death following acute vessel closure after coronary angioplasty. They are as follows: the amount of myocardium and collateral vessels in jeopardy beyond the site dilated, multivessel disease, a high jeopardy score and female gender. The risk of death occurring in experienced centers is currently very low. As the apparent indications for coronary angioplasty expand (21,22), it would seem prudent to proceed very carefully when dealing with such high risk patients.

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