Cardiac arrest during cardiac catheterization is uncommon, and fatal arrest is rare. In the coronary artery surgery study (CASS), which includes 7,553 patients, the mortality rate of coronary arteriography was 0.2 percent. In the past 11 years, we have studied at necropsy 12 patients with severe angina pectoris who died during or shortly after coronary angiography. Ten of the 12 patients were described elsewhere and in a recent one-month period, an additional two patients with unstable angina and fatal cardiac arrest during cardiac catheterization were studied at necropsy. Because the necropsy findings in patients with angina pectoris and fatal cardiac arrest during cardiac catheterization were studied at necropsy. Because the necropsy findings in patients with angina pectoris and fatal cardiac arrest in the peri-cardiac catheterization period are predictable, yet poorly recognized, a brief description of the latter two patients appears justified.

The patients, a 58-year-old woman (patient 1) and a 73-year-old woman (patient 2), had had stable angina for eight years, and unstable angina for three weeks (patient 1) and two weeks (patient 2), respectively. Neither had electrocardiographic or enzymatic evidence of myocardial necrosis immediately before catheterization. Both patients had a history of systemic hypertension, patient 2 had diabetes mellitus, and both had had acute myocardial infarcts three and six years, respectively, before death.

In each patient, cardiac catheterization was performed from the femoral artery by the Judkins technique. In patient 1, left ventricular angiography was performed first and then the Judkins catheter was placed directly into the left main (LM) coronary artery and 4 ml of contrast medium injected. Chest pain occurred immediately and the heart rate and systemic blood pressure fell. Despite endocardial pacing, electromechanical dissociation occurred and unsuccessful resuscitation. At necropsy, the heart of patient 1 weighed 400 g, and that of patient 2, 460 g. No foci of myocardial necrosis were evident; patient 1 had a transmural scar (anterior wall) and patient 2, a subendocardial scar (posterior wall). The four major (right, LM, left anterior descending, left circumflex) epicardial coronary arteries from each patient were excised, examined by x-ray, cut into 5-mm long segments, processed in alcohol and xylene, embedded in paraffin, stained by

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**Figure 1.** Bar graph showing the number and percentage of 5 mm segments of the four major epicardial coronary arteries narrowed to varying degrees by atherosclerotic plaque.
Angina Pectoris + Cardiac Arrest During Cardiac Catheterization

Non-fatal Arrest

Fat/Arrest

LM Narrowing? LAD, LC Right Narrowing? LM Narrowing 76-100% XSA LAD, LC Right Narrowing 76-100% XSA

Figure 2. Status of the four major epicardial coronary arteries in patients with angina pectoris having fatal and non-fatal cardiac arrest during cardiac catheterization.

Movat technique, and examined histologically. The amount of luminal cross-sectional area (XSA) narrowing by atherosclerotic plaque of each 5 mm segment was determined (Fig 1): narrowing 76-100 percent in XSA was present in all four major epicardial coronary arteries in both patients. Of 48 segments in patient 1, 12 (25 percent) were narrowed 76-95 percent and one (2 percent), 96-100 percent; and of 67 segments in patient 2, 14 (21 percent) were narrowed 76-95 percent and five (8 percent), 96-100 percent. Extravasated erythrocytes were present in plaque in the LM coronary artery of patient 1, but the lumen of this artery did not appear to have been narrowed by this mechanism.

Discussion

Several, but not all, investigators have suggested that patients with, compared with those without, severe narrowing of the LM coronary artery, are at greater risk of dying during cardiac catheterization. Many reports, however, suggest that catheterization of patients with unstable angina pectoris is no more hazardous than in patients with stable symptoms. Both of our patients had unstable angina and neither had evidence of acute myocardial infarction immediately before catheterization.

Clinical and necropsy findings in our two patients confirm previous morphologic observation in patients having fatal cardiac arrest during or shortly after cardiac catheterization for angina. Of our ten patients previously examined at necropsy, nine had narrowing of the LM>75 percent in XSA (by atherosclerotic plaque in seven, and by thromboembolic material superimposed on plaque in two). In addition, at least three of four major epicardial coronary arteries were narrowed 76-100 percent in XSA in all ten patients. Severe narrowing of the LM coronary artery is often indicative of severe narrowing of the right, left anterior descending and left circumflex coronary arteries. Of 35 necropsy patients with fatal coronary heart disease and narrowing 76-100 percent in XSA of the LM coronary artery previously reported from our laboratory, 94 percent also had similar narrowing of the right, left anterior descending and left circumflex coronary arteries.

In patients with angina pectoris suffering cardiac arrest during cardiac catheterization with successful resuscitation (Fig 2), the amount of narrowing of each of the four major coronary arteries has not been described. In contrast, of patients having cardiac arrest during cardiac catheterization and unsuccessful resuscitation, the previous study from this laboratory and the present one, strongly suggest that the LM and all three other major coronary arteries will be narrowed 76-100 percent in XSA. Therefore, in patients with angina pectoris who cannot be resuscitated from cardiac arrest during cardiac catheterization, emergency aortocoronary bypass grafting, if performed, should include insertion of conduits to all three major coronary arterial systems because this event is usually a marker of severe quadruple vessel disease.

References


Fatal Cardiac Arrest during Catheterization (Warnes, Kishel, Roberts)