

nique (Table I). The differences of 2 mm Hg and 1 mm Hg in peak and mean gradients, respectively, were not considered to be of clinical significance. Subsequent cardiac catheterization performed 48 hours later confirmed a mean mitral prosthetic diastolic gradient of 13 mm Hg. The calculated mitral valve area was 0.7 cm². The excised mitral valve prosthesis revealed extensively thickened and calcified leaflets that were restricted in motion (Fig. 3).

This case report represents a feasibility study of transesophageal continuous-wave Doppler in the evaluation of mitral prosthetic stenosis. Transesophageal echocardiography is extremely well suited for the evaluation of mitral valve prosthetic structure and function.⁴⁻⁶ Acoustic shadowing from the metallic components of prosthetic valves can create great difficulties in imaging the atrial surface of the prosthesis and the left atrium from the traditional transthoracic approach. These problems are clearly evident in the evaluation of mechanical prostheses, but the metallic frame of bioprostheses is a strong reflector of ultrasound as well. The advantages of transesophageal echocardiography over transthoracic echocardiography in the evaluation of mitral prostheses include the ability to identify small vegetations or thrombi, perivalvular or valvular regurgitation, spontaneous echo contrast of the left atrium, and left atrial appendage thrombi.⁴⁻⁶ Left atrial spontaneous echo contrast in this study was not seen on transthoracic examination, and has been implicated as an indicator of increased thromboembolic risk.⁶ Transesophageal continuous-wave Doppler now also allows for accurate measurement of the transvalvular gradients that were calculated from the modified Bernoulli equation. This may be especially valuable when a satisfactory transthoracic study cannot be obtained, such as in the immediate postoperative period or in patients with poor transthoracic windows. Transesophageal continuous-wave Doppler is a promising new technique in the evaluation of prosthetic mitral stenosis. Further studies are needed to define its broader diagnostic role in cardiovascular disease.

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Delayed rupture of right coronary artery after directional atherectomy for bail-out

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In interventional cardiology there are now two major approaches to the treatment of obstructive coronary disease—"dilating" and "debulking." Dilatation was initially accomplished by percutaneous transluminal coronary angioplasty (PTCA) and more recently by implantation of coronary stents. Debulking is a process that physically removes tissue from the diseased vessel wall (e.g., laser, rotational and directional atherectomy, thermal angioplasty).¹ Little is known about the early pathologic changes associated with directional atherectomy. Recently, the Mayo Clinic group² has reported that aneurysm formation at the site of previous atherectomy is uncommon. In fact, up to 30% of atherectomy specimens contained adventitial tissue without angiographic evidence of extravasation, aneurysm formation, and without clinical sequelae. In this report we document for the first time rupture of a coronary artery that occurred 2 days after an uneventful atherectomy. This procedure had been performed to remove an intimal flap resulting from a complicated conventional balloon angioplasty.

A 71-year-old man with a history of essential hypertension was admitted to our department with complaints of progressive angina at rest and with minimal exertion. The coronary angiogram showed single-vessel disease with a discrete, severe stenotic lesion of the midportion of the right coronary artery (RCA) (Fig. 1, A). Following the initial dilatation with a conventional 2.5 mm balloon angioplasty, a spiral medial dissection appeared that severely compromised antegrade flow (Fig. 1, B). Due to the unstable clinical situation, we attempted a "bail-out" procedure with a Simpson Coronary Atherocath (DVI, Redwood City, Calif.) (Fig. 1, C), which successfully restored flow although a fusiform dilatation was now evident at the site of the previously described dissection (Fig. 1, D). The patient remained asymptomatic until 2 days later, when he suddenly collapsed and died of electromechanical dissociation. At autopsy, approximately 300 ml of fresh blood and clots were found in the pericardial sac. In the pericardial fat tissue surrounding the RCA, a hematoma was present that was in communication with the pericardial sac via a small channel. A postmortem barium angiogram of the RCA revealed a fusiform dilatation at the site of the previous bal-

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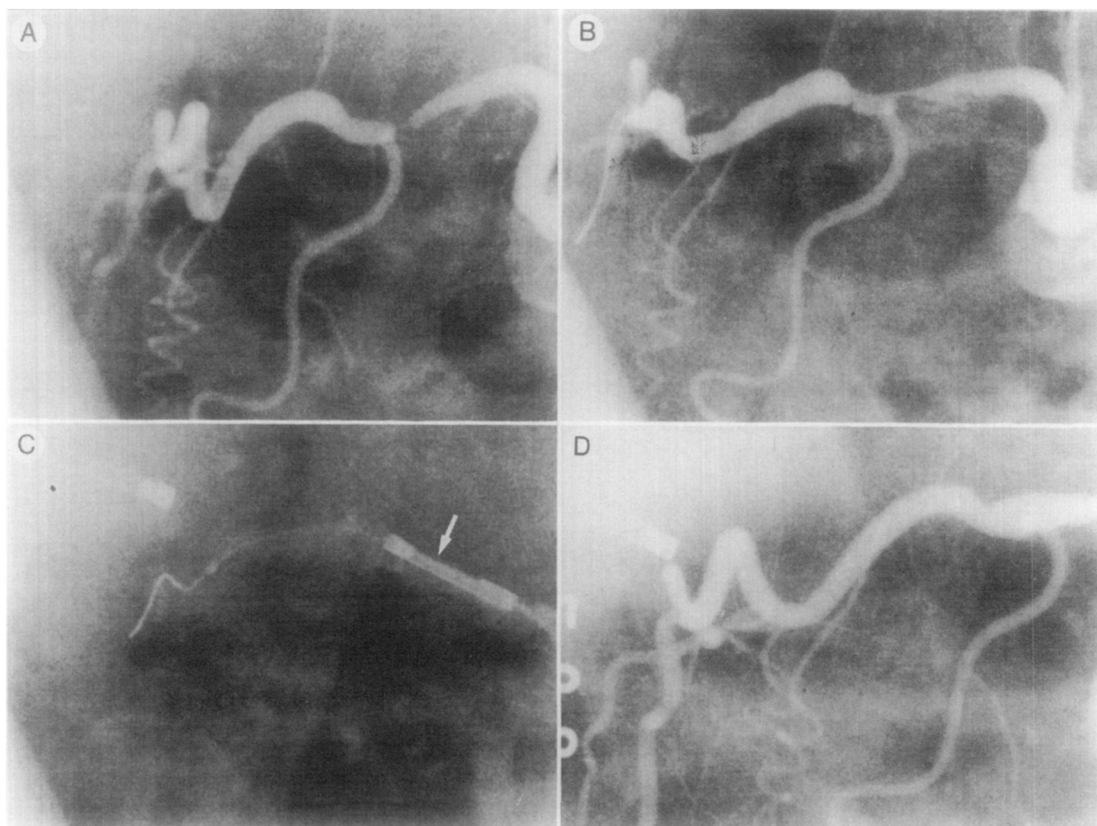


Fig. 1. **A**, Coronary angiogram of the right coronary artery (right anterior oblique view) showing a stenotic lesion proximal to the right ventricular side branch. **B**, Coronary angiogram following conventional balloon angioplasty showing tear and intraluminal defect. **C**, Cine frame showing the housing of the atherectomy device with its open window directed toward the posterior wall of the right coronary artery (*white arrow*). **D**, Post-atherectomy angiogram showing a fusiform dilatation at the site of the previously described dissection.

loon dilatation and atherectomy (Fig. 2). Histologic cross sections were obtained at four levels (Fig. 3). In the proximal part of the RCA not involved in the procedure, a moderate narrowing was present due to a classical atherosclerotic plaque (*level A*). Immediately proximal to the pseudoaneurysm (*level B*) a dissection was evident, which was induced by the balloon dilatation. The tear had occurred at the edge of the atherosclerotic plaque and separated it from the underlying media. At the level of the pseudoaneurysm (*level C*) a perforation of the coronary artery was evident. This perforation involved the full thickness of the normal arterial wall opposite the atherosclerotic plaque. The extent of the perforation increased at the most distal part of the pseudoaneurysm. The localized perforation was contained by the pericardial fat tissue (*level D*). The myocardium showed patchy fibrosis, which is a common feature in chronic ischemic heart disease. There were no indications of recent myocardial infarction in the distribution of the RCA. The immediate cause of death appeared to be related to sudden drainage of the hematoma and coronary artery into the pericardial sac, causing acute tamponade.

Atherectomy has been advocated as a bail-out technique for failed or complicated angioplasty, although the long-term results have not yet been reported. The rationale for

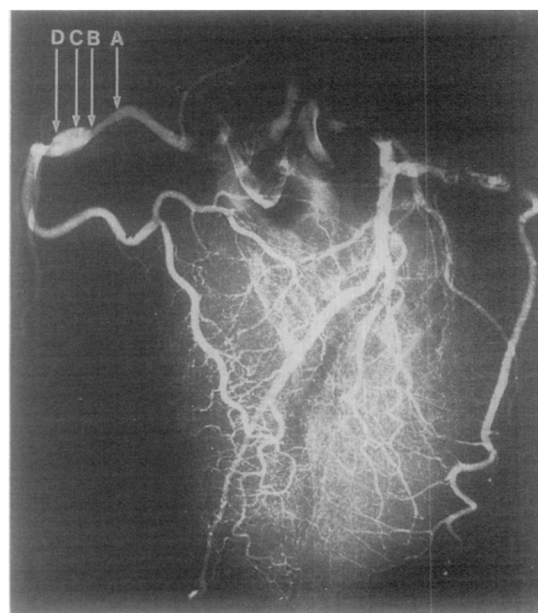


Fig. 2. Postmortem barium coronary angiogram showing a fusiform dilatation of the RCA at the site of the atherectomy. *A, B, C, and D* mark the level of the histologic cross sections.

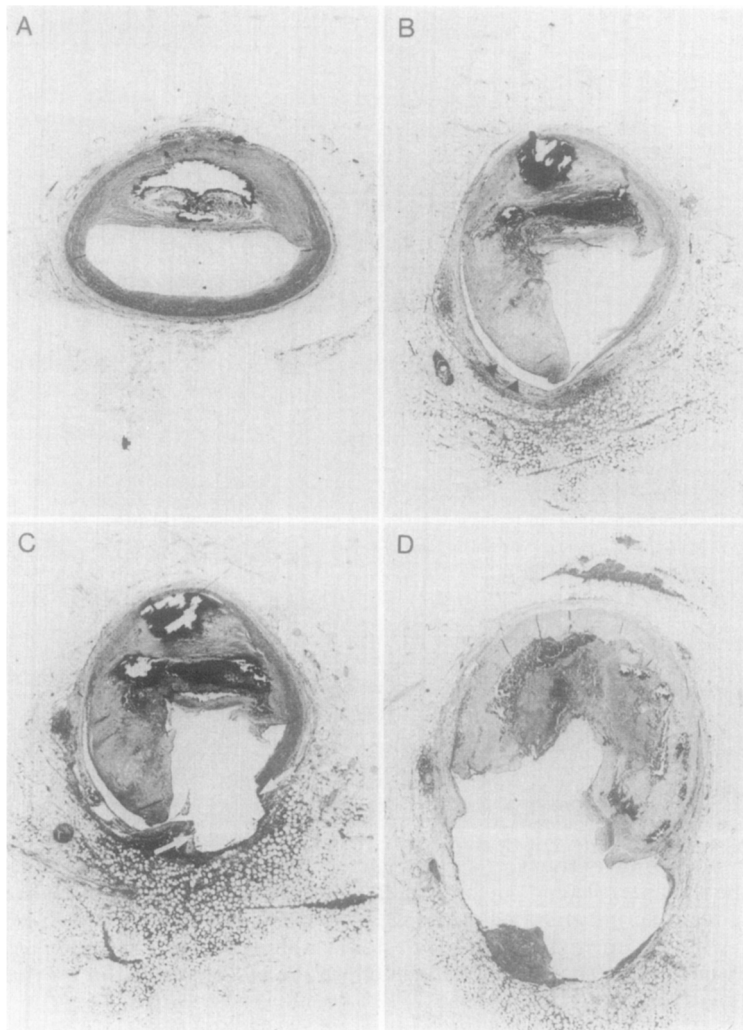


Fig. 3. **A**, Histologic cross section (*level A*), showing a classical atherosclerotic plaque causing moderate obstruction. (Hematoxylin-Azophloxine stain; original magnification $\times 12$.) **B**, Histologic cross section (*level B*), showing a dissection between media and atherosclerotic plaque (*arrow*). (Hematoxylin-Azophloxine stain; original magnification $\times 12$.) **C**, Histologic cross section (*level C*), showing that a part of the intima, media, and adventitia tissue has been resected by the atherectomy device (*arrows*). (Hematoxylin-Azophloxine stain; original magnification $\times 12$.) **D**, Histologic cross section (*level D*), showing that a major part of the wall of the pseudoaneurysm is formed by pericoronary fat tissue. (Hematoxylin-Azophloxine stain; original magnification $\times 12$.)

atherectomy in areas dissected by the balloon is the removal of the intimal flap, which may obstruct the vessel lumen. Although atherectomy seems to be a very appealing and attractive bail-out technique, the present experience calls for a word of caution. Two potential problems may occur with the directional atherectomy in the setting of acute dissection. First, recrossing the lesion with the guide wire may potentially result in a false route for the atherectomy device. The loss of the anatomic landmarks due to the dissection of the atherosclerotic plaque renders the directional use of the atherectomy device less dependable. Second, the eccentric nature of the coronary lesions, particularly in the presence of extensive dissection, may increase the risk of atherectomy when aggressive débridement is attempted. As demonstrated by the histologic cross sections, the rotating cutter did not affect the bulk of the

plaque but resected the intima, media, and adventitia of the nonatherosclerotic part of the vessel wall. Considering the high incidence of adventitial tissue present in the removed atherectomy specimens, it is surprising that the incidence of perforation is so rare. Further technical development, such as a "dumb-bell" configuration of the balloon of the atherectomy device, is currently under investigation to prevent unwanted damage to the media.

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