pected and may be the underlying pathogenesis of occult renal insufficiency, as in the case presented. Furthermore, the onset of the syndrome may not be immediate and may be delayed following an invasive procedure. The proposed mechanism may be endothelial injury at the time of arterial manipulation (surgery) or catheter-related endothelial denudation that places the cholesterol-rich extracellular matrix in direct contract with the blood. This allows the turbulent blood to dislodge fragments from these now unroofed ulcerated plaques. It may also be possible that embolism may occur spontaneously as a result of endothelial injury related to rheologic factors.

Unfortunately, there are no ideal or effective treatment methods for the prevention or treatment of this syndrome. Anticoagulant therapy has been proposed as a possible preventive treatment, as the finding of superimposed thrombus is common. There are, however, no studies to support the use of anticoagulation and its potential adverse effects such as major hemorrhage in the elderly and the potential for plaque hemorrhage, as has been proposed in the past. If investigation of the aorta by TEE demonstrates significant atheromatosis with embolic potential, we recommend the substitution of noninvasive vascular testing such as contrast computed tomography (CT) or magnetic resonance imaging (MRI) when feasible. The information obtained during transesophageal study may also be valuable to the cardiovascular surgeon, who at the time of surgery may be able to perform endarterectomy, as has been suggested by Tunick et al.[5] In conclusion, the case presented is the first report of a patient in which aortic atheromas were documented in vivo and a subsequent tissue (renal) biopsy demonstrated arterial embolization of atherosclerotic debris. Thus TEE is useful for localizing aortic atheromatosis and may help identify patients who are at risk to develop cholesterol embolism associated with intraaortic catheter placement. Furthermore, as suggested by Tunick et al.,[7] protruding atheromas in the thoracic aorta may be an independent risk factor for systemic embolization.

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Percutaneous directional atherectomy for discrete coronary lesions in cardiac transplant patients

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Coronary arteriosclerosis of the cardiac allograft is a major cause of morbidity and mortality in late survivors of heart transplantation.[1-3] Because transplant patients usually do not have angina pectoris, most centers perform coronary angiography annually to detect the onset or monitor progression of allograft coronary artery disease. In addition to the typical diffuse luminal narrowing with distal vessel obliteration, cardiac transplant recipients with prolonged survival may have more sharply demarcated lesions.[2, 3] Percutaneous transluminal coronary angioplasty (PTCA) for focal proximal coronary stenoses has been performed to alleviate myocardial ischemia and prolong allograft function.[4, 5] Directional coronary atherectomy (DCA) in cardiac transplant recipients, however, has not been reported previously.

Patient A, a 37-year-old man with dilated cardiomyopathy, received the heart of a 34-year-old female donor in January 1985. After transplantation, cyclosporine and prednisone were given for immunosuppression. There was one episode of acute rejection. The patient resumed smoking; in the posttransplant years the serum cholesterol level averaged 5.9 mmol/L, high-density lipoprotein cholesterol level 1.8 mmol/L, and serum triglyceride level 1.5 mmol/L. Results of routine annual coronary angiography showed development of diffuse luminal irregularities of the epicardial arteries without significant stenosis. On the fifth annual coronary angiogram, however, a focal eccentric lesion was found in the middle segment of the left circumflex coronary artery. After ^{99m}Tc methoxyisobutyl isonitrile (MIBI) perfusion scintigrams had shown evidence of reversible ischemia of the left ventricular posterior wall, the patient was referred to the catheterization laboratory for a percutaneous intervention.

Patient B, a 28-year-old woman with postpartum cardiomyopathy, received the heart of a 26-year-old male donor in April 1986. There were five episodes of acute rejection after transplantation, which were treated with pulsed doses of methylprednisolone and polyclonal and monoclonal anti-T cell antibodies. Because of ongoing rejection, azathi-

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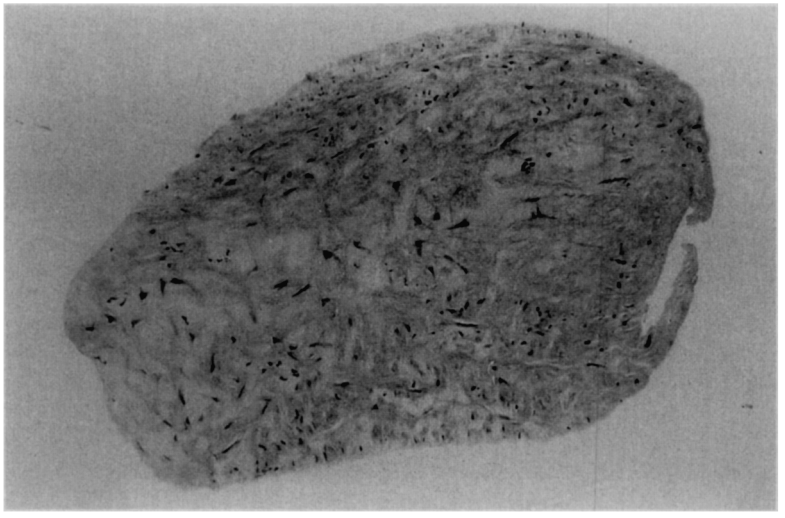

Fig. 1. Photomicrograph of atherectomy fragment from mid-left circumflex coronary artery of patient A showing moderate cellularity. Myofibroblasts are embedded in collagen bundles and extracellular matrix (hematoxylin-azofloxin stain; original magnification ×120).

Table I. Quantitative analysis of left circumflex (patient A) and left anterior descending (patient B) coronary segments before and after atherectomy and at follow-up

	Patient A			*Patient B*		
	Pre-DCA	*Post-DCA*	*Follow-up*	*Pre-DCA*	*Post-DCA*	*Follow-up*
MLD (mm)	1.8	3.0	2.6	0.8	2.4	1.8
REF (mm)	3.9	3.7	3.7	2.4	3.0	2.5
STEN (%)	54	19	30	67	20	28

DCA, Directional coronary atherectomy; *MLD*, minimal luminal diameter; *REF*, reference diameter; *STEN*, diameter stenosis.

oprine was added to the maintenance immunosuppressive therapy regimen which consisted of cyclosporine and prednisone. Hypertension was present, which was treated with vasodilators. The mean serum cholesterol level after transplantation was 6.0 mmol/L, and mean high-density lipoprotein cholesterol and triglyceride levels were 1.5 and 1.8 mmol/L, respectively. On the third annual coronary angiogram, a focal 50% stenosis was detected in the left anterior descending coronary artery just distal to a large diagonal side branch. One year later this lesion had progressed to 80% diameter stenosis on visual interpretation. After perfusion scintigraphy had shown reversible ischemia of the anterior myocardium, a percutaneous intervention was scheduled.

Because the geometry of the stenoses was considered to be ideal for atherectomy, that is, a localized eccentric lesion in a proximal to mid-coronary artery segment, DCA was performed in both patients with a device (Atherocath, Devices for Vascular Intervention, Inc., Redwood City, Calif.) developed by Simpson's group.[6] For anticoagulation and platelet inactivation, 10,000 IU of heparin and 250 mg of acetylsalicylic acid were given. To reduce vasomotor tone, intracoronary nitroglycerin was administered during the procedure and before the initial diagnostic and control angiograms. An atherectomy device with a 6F profile was used in both patients, resulting in a decrease in the diameter of stenosis to <50% on visual assessment. No adjunctive balloon dilatation was required, and 6 hours after the procedure the sheath was removed. There were no complications related to the procedure, that is, ECG changes, myocardial enzyme elevations, or embolic or bleeding events, and the patients left the hospital in good condition the following day. ^{99m}Tc MIBI perfusion scintigrams returned to normal in both patients, showing no evidence of myocardial ischemia after DCA.

For histologic examination, fragments of retrieved arteriosclerotic tissue were fixed in 10% buffered formalin, sectioned, and stained with hematoxylin-azofloxin and elastic van Gieson stain. Light microscopy of tissue collected from the left circumflex coronary artery of patient A showed moderate cellularity. Collagen bundles were separated by accumulations of homogeneous staining extracellular matrix (Fig. 1). Immunohistochemical staining with monoclonal antibodies specific for α-smooth muscle type actin was consistent with the myofibroblastic nature of the cells present. Results of histologic examination of retrieved tissue from the left anterior descending coronary artery of patient B showed mainly coarse collagen bundles with

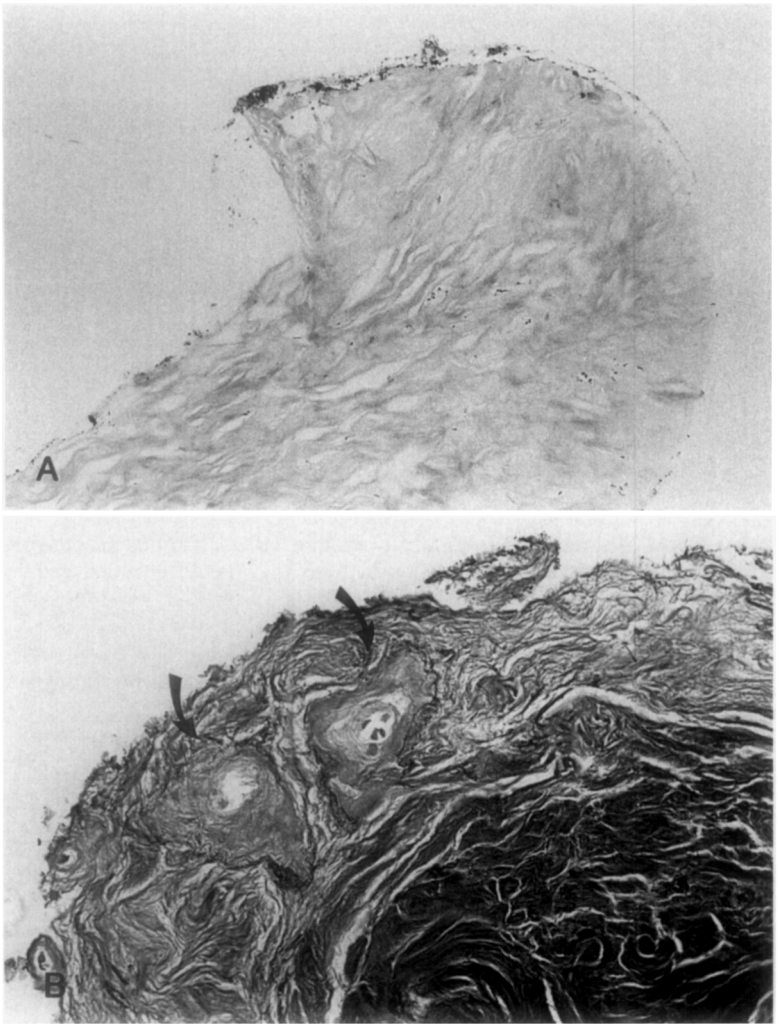

Fig. 2. Photomicrographs of arteriosclerotic tissue specimens collected from mid-left anterior descending coronary artery of patient B. **A,** Atherectomy fragment showing coarse collagen bundles with sporadic myofibroblasts. Black particles at periphery are considered to be abrasive dust from atherectomy device (hematoxylin-azofloxin stain; original magnification ×120). **B,** Collagenous tissue fragments with vasa vasorum *(arrows)* indicative of adventitia tissue (elastic van Gieson stain; original magnification ×240).

sporadic intermingled myofibroblasts (Fig. 2, *A*). Another atherectomy fragment from this lesion contained some intensely red-stained tissue incorporating vasa vasorum, which is indicative of adventitia (Fig. 2, *B*).

Quantitative analysis was performed on the treated coronary segments with the use of the computer-assisted Cardiovascular Angiography Analysis System described previously.[7] In brief, an optically magnified portion of a selected 35 mm cine frame encompassing the segment of interest is converted into video format by means of a cine video converter. The contours of the vessel segment are detected automatically and corrected for pincushion distortion caused by the image intensifier. The vessel diameter functions are determined in absolute millimeters by use of the guiding catheter as a scaling device. In the pre- and postatherectomy angiograms (Fig. 3) and follow-up angiogram, the reference diameter and the minimal luminal diameter were measured and averaged from multiple views. The reference diameter is a computer estimation of the expected normal diameter of the vessel at the site of obstruction. To determine the minimal luminal diameter the actual luminal contour is used. The percentage diameter stenosis can then be calculated. Results of quantitative coronary angiography are summarized in Table I. Results of follow-up angiography, 7 months (patient A) and 11 months (patient B) after the intervention, revealed no significant restenosis on the basis of various angiographic criteria.

Accelerated coronary artery disease of the cardiac allograft limits long-term survival (>1 year) after heart transplantation.[1-3] PTCA has been performed in cardiac transplant recipients with focal lesions in major epicardial coronary arteries or their branches.[4, 5] Halle et al.[5] recently reported the results of a multicenter retrospective experi-

ence among 51 PTCA procedures for 95 lesions in 35 patients 53 ± 5 months after transplantation. Angiographic success, defined as ≤50% residual diameter stenosis, was achieved in 88 of the 95 lesions (93%). The complication rate was comparable to that for routine PTCA in nontransplant patients. At follow-up 23 of the 35 patients (66%) were alive 13 ± 3 months after PTCA. Major events including death, retransplantation, repeat PTCA, myocardial infarction, and progressive coronary disease occurred in 19 patients within 6 months after PTCA and in 14 patients after 6 months. It was concluded that although PTCA may prolong cardiac allograft function, progression of coronary artery disease may be rapid requiring repeated interventions.

In recent years, percutaneous DCA[6, 7] has been applied as an attractive alternative for the treatment of coronary artery disease including complex eccentric stenoses. With the advent of this new intervention a percutaneous "biopsy technique" for collection of coronary arteriosclerotic tissue in selected heart transplant recipients has become available. Results of histopathologic examination of coronary arteries from cardiac allografts obtained at autopsy or retransplantation have shown a broad spectrum of abnormalities ranging from concentric fibrous intimal thickening to focal arteriosclerotic plaques that bear a close resemblance to spontaneous atherosclerosis.[3] Fibrofatty atheromatous plaques and so-called intermediate lesions are frequently found in cardiac allografts with survival times of 3 to 5 years or longer.[3] In a recently published study by Johnson et al.,[3] histopathologic features were correlated with the angiographic appearance of transplant coronary artery disease. It was demonstrated from the hearts of 10 cardiac transplant recipients who died or underwent retransplantation within 2 months of coronary angiography that two thirds of discrete proximal stenoses corresponded to intermediate or atheromatous disease. Our findings seem to be in agreement with these observations; geometry and histology of the resected lesions in the present patients resembled native coronary artery disease.

In previous studies from other groups and ours, it has been shown that DCA is a relatively safe procedure.[6-9] Our limited experience in two transplant patients indicates that DCA can be applied safely and effectively for discrete coronary lesions in allograft arteriosclerosis. The intervention was uncomplicated and successful in both patients as judged by angiography and control perfusion scintigraphy. Angiographic success, as confirmed by quantitative analysis, was maintained after 7 and 11 months of follow-up, respectively. This favorable long-term outcome after DCA is the result of actual removal of excess arteriosclerotic tissue. In matched patient populations with native coronary arteriosclerosis, however, DCA has been shown to have an incidence of restenosis comparable to that of PTCA,[8] despite a substantially better initial result.[8, 9] DCA has the advantage in that it enables histologic analysis of removed tissue acting as a biopsy technique. In one histologic study[10] a low smooth muscle cell density in arteriosclerotic tissue excised by DCA from native coronary arteries was associated with an eight times lower restenosis rate after 6 months than lesions with moderate cell density. The favorable long-term outcome after DCA in our two transplant patients, however, was not related to the moderate (patient A) and low cellularity (patient B) in retrieved arteriosclerotic tissue. Further studies are required to demonstrate whether DCA

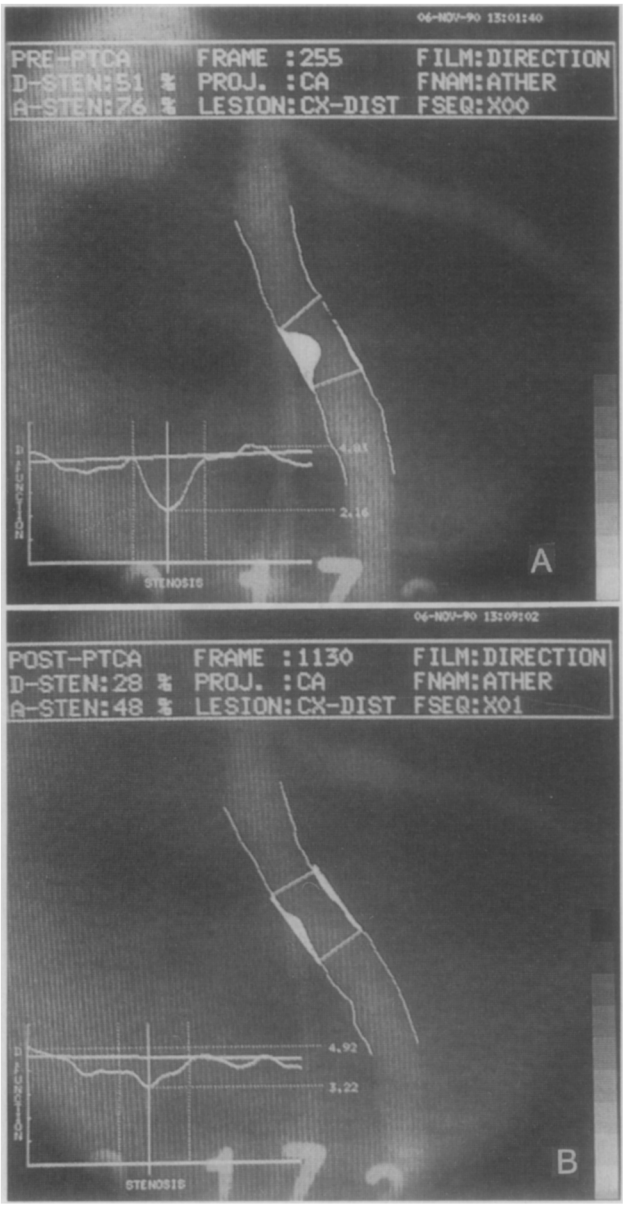

Fig. 3. Edge contour analysis of obstruction in middle segment of left circumflex coronary artery of patient A, filmed from caudal view before **(A)** and after **(B)** directional atherectomy. White area is measure for "arteriosclerotic plaque" and is derived from actual luminal contour and reconstructed reference contour. Diameter function and reference curves are shown at bottom. Minimal luminal diameter *(verticle line)* increased from 2.2 mm to 3.2 mm, corresponding to decrease in diameter of stenosis from 51% to 28% and area of stenosis from 76% to 48% in this projection.

of focal coronary lesions in cardiac transplant patients is followed by a lower restenosis rate as after PTCA.

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Self-limited acute pericarditis as initial manifestation of primary cardiac tumor

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The etiologic diagnosis of acute pericarditis is difficult and often has a low yield. Actually, the diagnosis of idiopathic pericarditis is accepted on the basis of excluding other specific causes.[1] There are no clinical features leading to specific diagnosis, but it is generally accepted that a short, self-limited clinical course favors a viral or idiopathic diagnosis and, by contrast, a prolonged, progressive course with clinical deterioration suggests a specific etiology of pericarditis, including malignancy.[2] On the other hand, a relapsing course with an initial onset followed by remission and a second outbreak is considered very suggestive of the benign forms of pericarditis (viral, idiopathic, autoimmune). A high degree of clinical alertness is needed for diagnosing cardiac tumors, as these disorders are uncommon[3] and are associated with protean manifestations. When the tumor involves the pericardium, pericardial effusion is the most common finding.[4] This effusion is usually hemorrhagic and is often associated with cardiac tamponade. Indeed, onset as acute pericarditis is very rare.[5,6] We report three cases of cardiac neoplasms that initially were diagnosed as acute pericarditis because they had a short, self-limited course but that, after a period of 3 weeks to 1 year, developed overt clinical manifestations of cardiac tumors.

Case No. 1. A 59-year-old woman with a 3-month history of constitutional symptoms and a 5 kg weight loss was admitted to our institution after 3 weeks of fever, pericardial pain, and progressive dyspnea. An echocardiogram (Fig. 1, *A*) disclosed a large pericardial effusion. Overt cardiac tamponade developed rapidly. Pericardiocentesis yielded hemorrhagic fluid (packed red blood cells 27%) whose cytology turned out to be nonspecific. Pericardial biopsy showed a fibrinous pericarditis without malignant cells. Thereafter the patient improved as fever, pericardial pain,

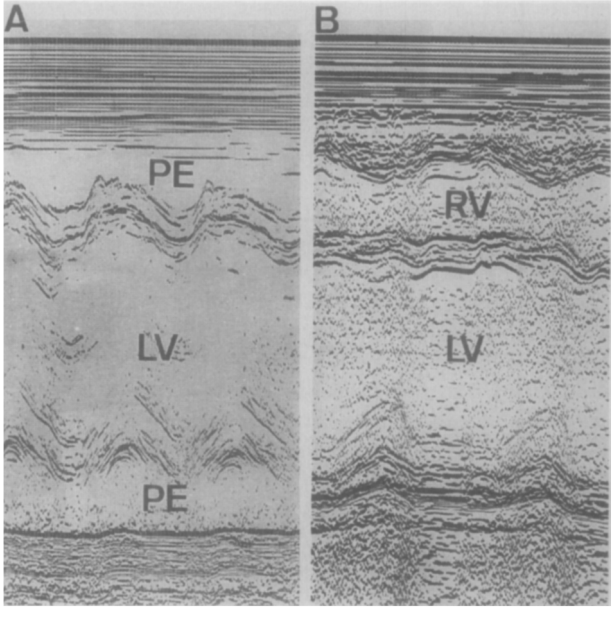

Fig. 1. Case No. 1. **A,** On admission, an M-mode echocardiogram showed a wide anterior and posterior free space corresponding to pericardial effusion *(PE)*. **B,** After 3 weeks, pericardial effusion had nearly disappeared and the recording just demonstrated a thickened pericar[illegible]. Left ventricle; *RV*, right ventricle.

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