have been reported up to 1980. The organisms causing the infections have been Mycobacterium, Pseudomonas, Streptococcus faecalis, hemophilus influenzae, Streptococcus viridans. Enterobacter, Staphylococcus edipimerida, Candida parapsilosis, Candida, Phycocyanus and Aspergillus. To the best of our knowledge, our case is the first reported of endocarditis upon a bioprosthesis produced by Brucella melitensis. One case of Brucella endocarditis on a Bjork-Shiley prosthesis has been reported in the literature.\(^6\)

Although brucellosis is an endemic disease in Spain, bacterial endocarditis is a very rare complication of the disease in our country or in other places.\(^7\) Brucella endocarditis usually affects the aortic valve. Anular and perianular abscesses, aneurysm of the Valsalva sinus and dissecting aortic aneurysm have been described.\(^8\) In our case, destruction of the aortic anulus was very extensive. There were three anular and septal abscesses as well as an aneurysm in one of the sinuses of Valsalva. The bioprosthesis itself was extremely altered by thrombosis, fibrin deposition, destruction of the cusps, intimal layer and infiltration with inflammatory cells.

The clinical history of the patient did not point to the diagnosis of brucellosis and this diagnosis was not made until all the blood cultures showed Brucella melitensis. Cultures taken from the perianular tissue were also positive for the same bacteria. Brucellosis is usually treated with cloramphenicol, tetracycline and sulfadiazine.\(^9\) Lezaun et al obtained good results with surgical treatment and tetracycline in their case of Brucella endocarditis on a Bjork-Shiley prosthesis. In our case, prosthetic valve replacement, wide debridement of the perianular infected tissue and medical treatment with sulfamethoxazole-trimethoprim, given by mouth, have controlled the disease. The patient is asymptomatic six months after the operation and free of infection.

**REFERENCES**

5 Magilligan DJ, Quinn EL, Davila JL. Bacteremia, endocarditis and the Hancock valve—Ann Thorac Surg 1977; 24:508
8 Grant GH. Rupture of the heart as a result of Brucella abortus endocarditis. Br Med J 1953; 1:914

**Aortic Dissection Following Coronary Arterial Bypass Graft Surgery**

**Long-term Survival of a Patient**

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A 60-year-old man underwent coronary arterial bypass graft surgery for unstable angina. After surgery, he developed a widened mediastinum on the chest x-ray film and a murmur consistent with aortic regurgitation. He refused study until the return of his anginal symptoms five years after surgery. At that time the patient had unchanged chest x-ray film and physical findings. Aortograms revealed a large type-I aortic dissection extending from the aortic root to the descending thoracic aorta. Long-term survival with an aortic dissection after coronary arterial bypass grafting is possible. The pathophysiologic and pathologic findings of a spontaneous type-I aortic dissection may differ from that of the dissection described herein, and these differences may relate to survival.

Coronary arterial bypass graft surgery has been successfully performed for an increasing number of indications, including intractable angina, left main coronary arterial disease, and triple-vessel coronary arterial disease.\(^1\) Despite advances in surgical technique, awareness and management of both the usual and unusual complications of the procedure remain essential.\(^2\) Postoperative aortic dissection, a rare complication of coronary arterial bypass graft surgery, has uniformly carried a poor prognosis.\(^3\) This report describes long-term survival of a patient with a well-documented aortic dissection after coronary arterial bypass grafting.

**CASE REPORT**

A 60-year-old white man was well without a history of hypertension or peripheral vascular disease until the onset of unstable angina in 1974. His angina was refractory to medical therapy, and he underwent cardiac catheterization in 1974. The study revealed normal left ventricular function with a 90 percent occlusion of his proximal left anterior descending coronary artery. A preoperative chest x-ray film was entirely normal (Fig 1).

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Coronary arterial bypass graft surgery was performed employing aortic cross-clamping but not aortic cannulation, and an end-to-side anastomosis of the left internal mammary to the left anterior descending coronary artery was done. The postoperative course was complicated by excessive bleeding. No significant hypertension was present in the postoperative period. The patient also developed a new early diastolic blowing murmur at the cardiac base, and on the chest x-ray film, his mediastinum was wider than on the preoperative film (Fig 2). The patient refused any further invasive procedures or investigation. He thereafter recovered uneventfully and enjoyed complete relief of angina.

Five years later, in July 1979, the patient was admitted to the intensive care unit with a prolonged anginal episode, possibly representing a small subendocardial myocardial infarction. Then and one month later, at repeat cardiac catheterization, the chest x-ray film still showed widening of the mediastinum identical to 1974. Ascending aortograms revealed a large type-I aortic dissection involving the right coronary cusp and extending from the aortic root to the descending thoracic aorta but not into the abdominal aorta (Fig 3). The site of reentry was not identified, but all major brachiocephalic vessels were opacified from the true lumen. Mild aortic regurgitation was also noted. The patient currently remains relatively asymptomatic with mild exertional angina while receiving medical therapy.

**DISCUSSION**

Aortic dissections are unusual among the complications of coronary arterial bypass surgery. There are three reported sites of aortic dissection: (1) the site of aortic or femoral cannulation;\(^1\) (2) the site of aortic cross-clamping;\(^2,4\) and (3) the graft insertion site.\(^5,6\) It has been assumed that patients with this complication have underlying pathologic abnormalities of the aortic root, usually secondary to atherosclerosis. These patients appear to have a uniformly poor prognosis.\(^3,4\) Usually, there is immediate and fatal hemodynamic decompensation. Occasional short-term survivors tend to develop severe complications, including congestive heart failure due to aortic regurgitation.\(^5\) Such a clinical course is like that of a spontaneous type-I aortic dissection, where underlying aortic pathologic abnormalities (ie, longstanding hypertensive changes, atherosclerosis, or cystic medial necrosis) are invariably present.
and where prompt surgery has been proven of benefit. The anatomic classification of aortic dissection developed by DeBakey (type-1 dissection, from the aortic root extending beyond the left subclavian artery; type-2 dissection, localized to the ascending aorta; and type-3 dissection, originating distal to the left subclavian artery) has proven extremely useful in making therapeutic decisions.

Extensive preoperative pathologic abnormalities of the aortic root may not be prerequisite for an aortic dissection in the patient with a coronary arterial bypass graft who experiences aortic trauma as part of his procedure. Therefore, the catastrophic hemodynamic course and the need for prompt surgery in patients with spontaneous type-1 aortic dissections may not be duplicated in all patients with postoperative dissections. While the relatively benign course in this patient and a similar patient with a more localized ascending aortic dissection and aneurysm (so far followed for six months) certainly does not suggest that medical treatment is the therapy of choice in a patient with an aortic dissection after coronary arterial bypass grafting, it does demonstrate that certain patients with this disease may be successfully treated with medical therapy and careful observation.

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REFERENCES


Ruptured Chordae Tendineae of the Tricuspid Valve due to Nonpenetrating Trauma*

Echocardiographic Findings

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A case of traumatic tricuspid insufficiency is presented. In the diagnosis of ruptured chordae tendineae of the tricuspid valve, two-dimensional echocardiography was an essential diagnostic procedure, while M-mode echocardiography showed no specific findings. Ruptured chordae tendineae of the tricuspid valve were confirmed at operation.

Although the echocardiographic findings of ruptured chordae tendineae of the mitral valve are widely known, those of the tricuspid valve have been reported rarely. We report a case of ruptured chordae tendineae of the tricuspid valve and discuss the echocardiographic findings.

CASE REPORT

A 49-year-old man was admitted to our hospital in August, 1979, for diagnosis of recently developed shortness of breath and palpitations on exertion. He had been well until three years before, when he fell off a precipice and sustained extensive injuries to the right side of his chest with multiple rib fractures. At that time he had no sign of cardiac damage.

On physical examination in our clinic, the pulse rate was irregular, 70-80 beats per minute, and the blood pressure was 92/65 mm Hg. The jugular venous pulse showed predominant V waves. A grade 2/6 holosystolic murmur was audible at the lower left sternal border. The liver was enlarged 5 cm below the right costal margin. There was pitting edema of the lower extremities.

The chest x-ray film showed an enlarged cardiac silhouetted on the right side. The lung fields were normal. The electrocardiogram revealed atrial fibrillation and left ventricular hypertrophy. An M-mode echocardiogram showed predominant right ventricular enlargement with paradoxical septal movement and asymmetric septal hypertrophy. The left cardiac chambers and the aortic and the mitral valves were normal. An M-mode tricuspid valve echocardiogram (Fig 1) showed diastolic irregular coarse fluttering and wide excursion of the anterior tricuspid valve leaflet, but showed no definite evidence of ruptured chordae tendineae of the tricuspid valve. A two-dimensional echocardiogram (Fig 2), (Toshiba Medical SSH-11A) showed marked systolic excursion of the tricuspid valve into the right atrium beyond the level of the tricuspid valve ring and loss of the usual coaptation point. Asymmetric septal hypertrophy and enlargement of the right atrium and ventricle were detected simultaneously. Two-dimensional echocardiographic findings were consistent with ruptured chordae tendineae of the tricuspid valve.

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