ceed the dose recommended by AMA Drug Evaluations.11 Therefore, the presence of severe congestive heart failure in our patient could have resulted in a decreased dose requirement.12 Existing dose recommendations may occasionally place blood levels within the toxic range even in normal subjects, pointing to the need for close observation of all patients receiving this drug.13 In the settings of the coronary care unit where lidocaine is used so extensively, many patients, in addition to their cardiac arrhythmias, may evidence heart failure, liver decompensation, or renal insufficiency. Reports of increased sensitivity to the toxic effects of lidocaine in patients with heart failure, liver disease, or renal insufficiency have appeared in the recent literature.12-14 It is therefore urged that clinical assessment of the status of cardiac, hepatic, and renal function is an important consideration in selecting a therapeutic dose of lidocaine. All patients receiving this drug require observation for adverse effects, even if doses are within the currently recommended ranges because, as stated by James, "The overzealous therapist may abolish the arrhythmia only to find the sinus node unable to take over."15

Nonproprietary and Trade Names of Drugs
Furosemide--Lasix. Lidocaine hydrochloride--Xylocaine Hydrochloride.

References

Aortocoronary Bypass With Saphenous Vein Graft

Seven-Year Follow-Up

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A 42-year-old man had extensive occlusive disease of the coronary artery and angina pectoris. An autogenous saphenous vein bypass from the ascending aorta to the anterior descending coronary artery was performed on Nov 23, 1964. The patient suffered an asymptomatic anterior myocardial infarction during operation but made an uncomplicated recovery. Seven years after the operation, the graft functions with normal left ventricular hemodynamics, while the occlusive process has produced obstruction of the left main coronary artery and almost complete occlusion of the right coronary artery. To our knowledge, this is the first successful case of a saphenous vein-coronary artery bypass with the longest follow-up of a functioning coronary vein bypass graft.

AORTOCORONARY artery bypass with autogenous saphenous vein graft for the treatment of symptomatic coronary arterial disease has gained widespread acceptance in recent years. Combining the experience gained from both cardiac and peripheral vascular operations, surgeons in many centers have refined the surgical techniques and provided extensive clinical experience on which the current enthusiasm is based. It has been predicted that "... on the basis of current popularity vein grafting will become the most frequently performed operation in America..." (Med World News 13:39-50, 1972). Those critical of the operation await longer follow-up to evaluate such factors as long-term patency of the graft, prolongation of life, and progression of the underlying atherosclerotic disease.

The following case of a patient with a functioning saphenous vein bypass from the ascending aorta to the left anterior descending coronary artery, seven years after operation, is presented for two reasons: (1) to the best of our knowledge, it represents the first successful aortocoronary bypass with autogenous saphenous vein graft, and (2) it is the longest follow-up of a functioning aortocoronary saphenous vein bypass graft to date.

Report of a Case

A 42-year-old truck driver was first seen in March 1964 with severe angina pectoris. Since an attack of myocardial infarction in December 1963, he had been unable to work because of pain in the chest from minimal excitement or exertion. The patient was admitted to the Methodist Hospital on Aug 31, 1964. An electrocardiogram, made during one of the bouts of thoracic...
pain associated with sweating, revealed striking S-T segment depression. At bed rest, the patient continued to have frequent anginal pain in spite of administration of coronary vasodilators and anticoagulants.

The patient's mother had been a diabetic with heart disease and had had amputations of the legs for atherosclerotic occlusive disease.

On admission, the blood pressure, taken with the patient sitting, was 180/100 mm Hg in both arms. A loud bruit in the right subclavian artery and a soft, apical, systolic cardiac murmur were present. There was no lift of left or right ventricular hypertrophy. An abdominal bruit and reduced arterial pulsations in the legs were recorded. In the coronary arteriograms made on Sept 1, 1964, diffuse narrowing of the right coronary artery and stenosis of the left main coronary artery estimated at 85% were noted (Fig 1). The patient was discharged with instructions to continue medical treatment with coronary vasodilators, digoxin, anticoagulants, and restricted activity.

He was readmitted to the hospital on Nov 18, 1964, with little change in symptoms and virtual incapacity regarding physical tolerance. He had not worked for 1 year and could not walk without experiencing thoracic pain and dyspnea.

Operation was recommended. Accordingly, on Nov 23, left coronary endarterectomy was scheduled; the left main coronary artery and proximal segments of the anterior descending and circumflex branches were exposed. Since the lesion involved the entire bifurcation, endarterectomy with venous patch graft angioplasty was abandoned as too hazardous. The anterior descending coronary artery was softer distal to the bifurcation, although a posterior plaque was palpable. A 40 mm Hg gradient was measured from the ascending aorta to the anterior descending coronary artery. The decision to insert a reversed autogenous saphenous vein bypass from the ascending aorta to the anterior descending coronary artery was made. The vein graft was obtained and prepared, heparin sodium was administered, and the caval and femoral arteries were cannulated and attached to the pump oxygenator for use if necessary. A partial exclusion vascular clamp was used and the reversed vein graft was sutured to the ascending aorta with continuous 5-0 polyethylene suture technique. Vascular clamps were placed on the anterior descending coronary artery and vertical arteriotomy was performed. The graft was cut to an appropriate length and sutured end-to-side to the arteriotomy by similar suture technique. Since ventricular fibrillation was expected during the 23 minutes of occlusion of the anterior descending artery required to accomplish the distal anastomosis, cardiopulmonary bypass was immediately available. Satisfactory blood pressure was, however, maintained with normal sinus rhythm throughout this portion of the operation. The ECG by monitor showed severe S-T segment depression during occlusion, but reverted toward normal after circulation through the graft had been restored. The operation was accomplished without
incident, and convalescence after operation was uncomplicated. Serial ECGs showed the development and evolution of an operative or postoperative anterior myocardial infarction, although the patient remained asymptomatic. On Dec 18, coronary arteriography revealed diffuse narrowing of the right coronary artery, severe stenosis of the left main coronary artery, and virtual obliteration of the proximal anterior descending branch. The venous graft was not catheterized, but arterial root injection was performed. There was suggestion of a patent graft, but, unfortunately, patency was not absolutely confirmed by this study.

The patient was seen periodically during the ensuing four years. Symptoms of aortoiliac occlusive disease prompted readmission to the hospital on Sept 17, 1968. He was working again and during the three months before admission had had only one episode of thoracic pain, which was promptly relieved with nitroglycerin. Blood pressure was 135/80 mm Hg, and the soft, apical, systolic, cardiac murmur was heard. The ECG showed evidence of the previous anterior myocardial infarction. On lumbar arteriography almost complete obstruction of the abdominal aorta above the bifurcation was noted. On Oct 1, endarterectomy of the abdominal aorta with patch graft angioplasty was accomplished with restoration of pulses in the legs.

The patient was hospitalized for general evaluation on Sept 10, 1971. He had been working full time with virtually no angina, except on extreme effort. He had gained 18 kg (40 lb) above ideal weight. Blood pressure, taken with the patient sitting, was 190/100 mm Hg in the left arm and dropped to 160/90 mm Hg when taken in the supine position. A soft, precordial, systolic, cardiac murmur of doubtful hemodynamic significance was again heard. The cholesterol value was 278 mg/100 ml; total serum lipids, 699 mg/100 ml; and fasting blood glucose, 71 mg/100 ml. An ECG demonstrated the residual of an old anterior myocardial infarction slightly altered since the previous tracing. Coronary arteriography performed on Sept 9, 1971, showed left ventricular pressure of 145/7 mm Hg. There was 96% narrowing of the right coronary artery with several areas of poststenotic dilatation. There was collateral filling of the distal anterior descending and circumflex branches by right coronary artery injection. The left main coronary artery was completely occluded, as demonstrated by flush injection of the ascending aorta. The catheter was inserted into the vein graft, and opaque material was introduced. The anterior descending and circumflex branches were visualized, and retrograde filling was seen back to the completely occluded left main coronary artery (Fig 2 and 3). In roentgenograms of the chest the pulmonary fields were clear, and the heart was not enlarged.

The patient was discharged with instructions to follow a weight reduction program and to continue medical treatment, consisting of coronary vasodilators and digoxin. Since he was virtually asymptomatic, right coronary arterial bypass was not recommended at that time.

Comment

Definite conclusions cannot be drawn on the basis of a single case of long-term follow-up, although certain features of this case are of interest in light of more recent experience. Prolonged patency of the graft is possible with aortocoronary bypass without the functioning of the graft being jeopardized by deterioration of the graft or worsening of the atherosclerotic disease. In our patient, disease has continued to evolve in the right and left main coronary arteries, but there is no evidence of worsening of disease in the anterior descending coronary artery, even though the anastomosis was performed at the site of a known posterior plaque. In addition, it is likely that continued patency of the graft accounts for satisfactory left ventricular function and prolongation of life in this particular patient.

Myocardial infarction after aortocoronary bypass is reported to occur in from 10% to 20% of patients having multiple bypass grafts.** In most instances in which the graft continues to function, the infarction causes no symptoms and does not prevent a satisfactory result, as in this case. Continued progress in surgical efforts to combat occlusive coronary arterial disease has been due to introduction of multiple bypass grafts, anoxic arrest, or elective ventricular fibrillation during performance of coronary anastomosis, improved suture material, and refinements in anesthetic induction.

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References