judged not to be of hemodynamic significance in terms of left-to-right shunting, but was not fulfilling its intended purpose of supplying blood to the anterior wall of the left ventricle. There was no change in the native coronary artery anatomy from the preoperative study.

Subsequently, the patient underwent a second operation with removal of the graft to the coronary vein and placement of a saphenous vein graft from the aorta to the proximal left anterior descending coronary artery. The patient convalesced without incident and the continuous murmur was no longer present.

COMMENT

When heard, the murmur of aortocoronary bypass is systolic. The finding of a continuous murmur in the present case in the postoperative period led to restudy and the finding of inadvertent placement of a saphenous vein graft from the aorta to the coronary vein adjacent to the proximal left anterior descending coronary artery (Fig 2). Other causes of continuous murmur were excluded and the murmur was no longer present following the second operation.

Other possible causes of a continuous murmur emanating from a saphenous vein graft might be kinking or obstruction of the graft, as well as flow to the left ventricle during diastole; however, we have seen neither in our clinical experience.

Enthusiasm for arterIALIZATION of coronary venous blood has been revived through modification of the Beck procedure. An internal mammary artery is grafted to a coronary vein and the vein ligated in order to provide reversed flow. Whether a continuous murmur can be heard in this setting has not been reported.

REFERENCES


Diphtheroid Endocarditis after Aortic Valve Replacement*

Francis E. Wanat, M.D. and
Adam R. Wychulis, M.D., F.C.C.P.

Diphtheroid endocarditis after aortic valve replacement was cured with penicillin, gentamycin and erythromycin. Infections occur most commonly on the aortic valves of men patients within two weeks after prosthetic insertion. Management with combined drug therapy based on sensitivity and clinical response are suggested. Paravalvular leaks do not necessarily require valve replacement.

The successful treatment of diphtheroid endocarditis with antibiotics in a patient with a prosthetic heart valve is reported. In a review of the literature, another 15 cases of diphtheroid prosthetic endocarditis were found.

CASE REPORT

A 59-year-old white machinist underwent aortic valve replacement with a No. 9 cloth-covered Starr-Edward's prosthesis (model 2320) for severe calcific aortic stenosis on October 11, 1972. Preoperation, he experienced progressive symptoms of congestive heart failure, as well as syncope and angina. Cardiac catheterization had demonstrated a gradient of 100 mm Hg across the aortic valve. Cephalothin sodium (Keflin), 1 gm administered parenterally, was given every four hours as prophylaxis for eight hours prior to surgery and continued until the seventh day after surgery. Anticoagulant therapy with warfarin (Coumadin) was begun on the third postoperative day. He had an uneventful hospital course and was discharged 11 days after surgery on digoxin and warfarin.

On October 26, 1972, the patient was re-admitted with complaints of nocturnal fever to 38.7°C (101°F) and nasal congestion of two days duration. Vital signs were within normal limits except for an admission temperature of 37.5°C (99.6°F) (Fig 1). Cardiac auscultation revealed a grade 2/6 systolic murmur in the aortic area and normal sounds of the prosthetic ball. The following day he complained of a sore throat and his rectal temperature was 38.1°C (100.6°F). Nose and throat cultures revealed normal flora with no diphtheroids. Five of six blood cultures obtained on admission yielded gram-positive bacilli consistent with diphtheroids. The diphtheroid colonies obtained from plated blood agar showed nonsporing pleomorphic gram-positive rods, with the tendency to appear in palisades on gram stain. Biochemical testing resulted in the following: aerobic, nonhemolytic, catalase-positive, nonmotile, and no fermentation of glucose, lactose, maltose or sucrose. Treatment was begun with 40 million units of penicillin G potassium per day intravenously on October 30. The penicillin dosage was increased to 60 million units daily four days later since the organism was found to be resistant at 1:4 dilutions of serum with a serum bactericidal level against the organism of greater than 1:16.

On November 11, a petechial lesion was observed in the conjunctiva of the right eye. Five days later the right first metacarpophalangeal joint became painful and enlarged.

On November 25, penicillin V, 500 mg every six hours and probenecid, 0.5 gm every six hours was added to the antibiotic regimen. Two days later another right conjunctival petechia was noted and gentamicin, 3 mg per kg daily in divided doses was begun. The patient finally became afebrile on November 30.

Generalized abdominal pain and hypotension followed the passage of tarry stools on December 6. The hemoglobin was 4.5 gm. Parenteral vitamin K and blood transfusions were administered as anticoagulation was terminated. A duodenal ulcer was demonstrated on upper gastrointestinal series.

All medications were stopped on December 19 and erythromycin, 250 mg four times daily, was begun. The patient was discharged on December 26 and the erythromycin was continued for another week.

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During the subsequent 24 months, he has been observed closely and has shown no signs of recurrent endocarditis. A grade 1/6 diastolic murmur became audible along the left sternal border in January, 1973. He is asymptomatic on lanoxin 0.25 mg and chlorothiazide 1.0 gm per day.

**DISCUSSION**

In large series of bacterial endocarditis, diphtheroids represent a small percentage of infecting agents. They are also uncommon agents to infect prosthetic valves. The cardiopulmonary bypass apparatus has been incriminated as a source of diphtheroid infection, but the evidence is not conclusive. It is interesting that most organisms other than diphtheroids responsible for prosthetic infections have been sensitive to the antibiotic administered prophylactically, and thus it was postulated that these bacteria came from protected locations. On the other hand, diphtheroids, as in our case, have been relatively resistant to the antibiotics used prophylactically.

To our knowledge, 15 cases of diphtheroid prosthetic valve endocarditis have been reported. There were four drug treatment successes and five drug failures. Another five patients were successfully treated with replacement of the infected valve. One surgical failure has been reported. Our case is the fifth case cured with antibiotic therapy alone, and he is doing well 24 months after discharge, the longest reported survival. In cases where the sex was reported, nine of ten were men.

Eleven of 15 patients developed evidence of infection within 30 days after surgery. Our patient became symptomatic on the 12th postoperative day, while seven patients developed infection on the 14th postoperative day. Only two patients had infections very late following the operation, ie, 14 and 30 months respectively. One reported case had wound dehiscence and two had dental problems. In 12 cases there were no known precipitating factors.

Information regarding the presence or absence of a regurgitant murmur was available for nine cases. Seven patients had diastolic murmurs while the remaining two did not. The hemodynamic consequences rather than the simple presence of a regurgitant murmur dictates the mode of therapy.

Antibiotic therapy should be based on the clinical course and sensitivity studies. Intravenous aqueous penicillin 20 to 40 million units and 1 gram of streptomycin in divided doses per day is a common approach. A different drug program was employed in each of the four reported cases treated successfully with antibiotics.

Our patient had no clear response to penicillin and marked clinical improvement occurred with the addition of gentamicin at 3 mg/kg/day. If penicillin allergy is present, cephalothin, erythromycin, or vancomycin could be used based on sensitivities. Theoretically, one bactericidal drug should be sufficient therapy. However, it did appear that in most drug treatment successes, two or three drugs were used.

Signs of treatment failure are the appearance of a new hemodynamically significant murmur, fluoroscopic confirmation of valve dislodgement, persistent emboli, advancing heart failure or persistent spiking fever. Valve replacement is then suggested. Additional indications for valve replacement include failure to halt blood stream invasion by specific therapy, cardiovascular accidents due to emboli, mycotic aneurysms and yeast or fungus infections.

Block et al continued anticoagulation in their series of 12 patients with prosthetic infections, stopping it only if hemorrhagic complications occurred, as in our case. The prothrombin time was kept in the range of 1.5 to 2 times control values. King et al discontinued anticoagulation when endocarditis was diagnosed.

We feel that the prophylactic antibiotics oxacillin or cephalothin or both should be administered one day prior to valve replacement and seven days after surgery. If diphtheroid prosthetic endocarditis is diagnosed, treatment initially should consist of 20 million units of intravenous aqueous penicillin and 1 gram intramuscular streptomycin per day. Changes in therapy are based on disc sensitivity and the bactericidal effect of the treated patient's serum and the clinical course. Anticoagulants could be continued unless significant bleeding occurs. Reoperation is considered if drug therapy is unsuccessful.

![Graph showing patient's hospital course](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20970/ on 04/19/2017)
as previously defined. Repeat blood cultures during therapy are important to determine the effectiveness of treatment.

REFERENCES


Double Outlet Right Ventricle with Absent Aortic Valve*

Warren H. Toews, M.D.,** Randall H. Lortscher, M.D.,†
and Leslie L. Kelminson, M.D.;‡

A case of double outlet right ventricle with multiple associated cardiovascular anomalies, including total absence of the aortic valve, is reported.

Double outlet right ventricle (DORV) is the pathologic result of aberrant trunco-conal cardiovascular development. The basic anatomy of this unusual form of congenital heart disease, as well as the associated cardiovascular anomalies, have been well described.1-3 The purpose of this communication is to describe an associated absence of the aortic valve, which has not, to our knowledge, been previously reported.

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CASE REPORT

A 7 lb 6 oz term caucasian boy was transferred to Denver Children’s Hospital at 36 hours of life because of severe cyanosis and cardiomegaly. On admission, he was tachypneic and the peripheral pulses were diminished. Pertinent cardiac findings included right ventricular lift; split first heart sound; loud second heart sound; and a rough, low-pitched systolic murmur in the left axilla, associated with a softer, high-pitched diastolic murmur. At the lower left sternal border, the murmur became "to-and-fro."

A complete blood count, serum electrolytes, serum glucose, and blood urea nitrogen were normal. Arterial blood gas analysis on FiO2 80 percent revealed Po2 55 mm Hg, Pco2 27 mm Hg, and pH 7.41. The electrocardiogram showed a heart rate of 135 beats/min, mean frontal QRS axis +90°, P-R interval 0.14 sec, right and left atrial enlargement, and severe right ventricular hypertrophy. Chest x-ray examination demonstrated marked cardiomegaly and pulmonary vascular congestion. Echocardiogram was technically inadequate to support a specific anatomic diagnosis. However, it did demonstrate a dilated right ventricular (RV) and diminutive left ventricular (LV) cavity. The tricuspid valve (TV) had a very wide excursion, but the mitral valve (MV) appeared to have markedly restricted motion. A great vessel was anteromedially located, but the relationship to the interventricular septum and atrioventricular valve could not be demonstrated.

The infant subsequently underwent cardiac catheterization which demonstrated markedly elevated RV (90/0/10 mm Hg), right (RA) and left (LA) atrial pressures (a = 12, v = 8, m = 9 mm Hg in both). The catheter could not be made to enter either great vessel or the LV. Biplane cineangiograms were performed in the RV and LA which demonstrated complete passage of contrast medium from LA to RA and then to the RV. Filling of both great vessels from the RV occurred with aortic (Ao) opacification slightly delayed. A diagnosis of DORV with mitral atresia was made. During the procedure, the infant suffered respiratory arrest, requiring vigorous resuscitation.

Following the procedure, the infant’s condition further deteriorated and death occurred despite all supportive measures. Post-mortem examination confirmed the diagnosis of DORV. Specifically, the heart was markedly enlarged—principally the right atrium and right ventricle. The aorta arose completely from the right ventricle. The former was dis-

Figure 1. The ascending aorta showing one of the coronary ostia with the ridge beneath it. No aortic valvular tissue is seen.