Two-stage replacement of tricuspid valve in active endocarditis

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One month after a 21-year-old female drug addict with tricuspid staphylococcal endocarditis seemed to have been cured by antibiotic therapy she relapsed. Further antibiotic treatment for one month failed to control the infection. Tricuspidectomy was then performed and the infection was eliminated. A valve prosthesis was inserted 3 months later. She recovered and is now fully restored to health. We recommend this two-stage surgical technique in similar cases.

Right-sided endocarditis, mainly involving the tricuspid valve, is no longer a rare disease, its frequency increasing from less than 5 per cent (Thayer, 1931; Bain et al., 1958) to 10 to 20 per cent in the past decade (Roberts and Buchbinder, 1972; Arbulu et al., 1973); this can be ascribed largely to the increased incidence of narcotic addiction (Conway, 1969; Banks et al., 1973; Graham et al., 1973; Svanbom et al., 1973). Valve replacement during the active stage of endocarditis is now an accepted form of treatment in many places (Strandell et al., 1969; Manhas et al., 1972; Graham et al., 1973; Black et al., 1974), but not in all. Good results have been obtained in many cases of tricuspid valve endocarditis by means of valve resection alone (Arbulu et al., 1973; Graham et al., 1973, Michl et al., 1973) or with the insertion of an artificial valve at a second operation (Arbulu et al., 1973; Simberkoff et al., 1974).

Case report

The patient was a 21-year-old woman without known heart disease, who had taken narcotics intravenously for about 4 years. Some weeks before the onset of the present illness, she had an infection in one foot after treading on a piece of glass.

On 14 June 1974 during a holiday abroad, she fell ill with malaise and fever above 40°C and was treated with antibiotics. On returning to Sweden on 24 June, she was admitted to Roslagstull Hospital for persisting high fever.

On admission she was febrile, but had no signs of cardiac failure or peripheral emboli. A systolic murmur grade 2/6 was heard over the apex. The liver and spleen were enlarged. X-ray film showed infiltrations in the right middle and left lower lung lobes. Electrocardiogram was normal apart from sinus tachycardia. Phonocardiogram showed a faint systolic murmur of medium frequency, thought, however, to be consistent with a normal flow in the pulmonary artery.

On the third day in hospital a growth of penicillinase-producing Staphylococcus aureus from blood was reported and cloxacillin plus benzyl penicillin were administered. On the next day, she became dyspnœic and x-ray examination showed a pleural effusion. Despite changes in antibiotic therapy, the lung shadows increased with cavity formation in the right lower lobe.

On 15 July, transient murmurs and electrocardiographic abnormalities suggestive of pericarditis were noted and pulmonary scintigraphy indicated changes consistent with multiple small emboli. Right-sided cardiac catheterisation showed normal pressures. Angiocardiology defined a walnut-sized mass on the tricuspid valve, with extreme regurgitation.

After further antibiotic treatment, the patient was discharged on 1 October, afebrile and asymptomatic, but was readmitted on 28 October with fever and cough. A chest x-ray film showed further infiltration and scintigraphy revealed fresh infarction of the left lower lobe. The heart size had increased from 300 to 400 ml per m² body surface area. The electrocardiogram was initially normal but signs of right ventricular strain appeared later. Though repeated blood cultures were negative, carbenicillin
and gentamicin were given and the temperature subsided.

On 25 November, the tricuspid valve was resected under total cardiopulmonary bypass at the Thoracic Surgical Clinic, Karolinska sjukhuset, Stockholm. Large vegetations were seen on the posterior leaflet with acute inflammatory reaction. Bacteria were not seen on microscopy or cultured. Increased pulsation of the jugular veins and the liver was noted after operation. Right heart catheterisation showed a low stroke volume, normal pressures in the pulmonary capillary venous and pulmonary arterial positions, and a high right ventricular end-diastolic pressure. Heart size was now 510 ml per m² body surface area. She was dyspnoea on walking but otherwise felt well.

On account of the persistent right-sided failure, a 31 mm Björk-Shiley mitral valve prosthesis was inserted on 12 February 1975, and 15 days later she was discharged. One year after operation, she was in good health, working full-time, and had resumed water-skiing.

Discussion

The combination of narcotic addiction, staphylococcal septicaemia with right-sided endocarditis, and pulmonary embolism was first described by Hussey et al. (1944), and has often been reported since then (Hussey and Katz, 1950; Olsson and Romansky, 1962; Conway, 1969; Roberts and Buchbinder, 1972; Menda and Gorbach, 1973; Svanbom et al., 1973). These authors drew attention to the possibility of tricuspid valve involvement. In this form of endocarditis cardiac symptoms are initially often obscure (Bain et al., 1958; Conway, 1969; Arbulu et al., 1973; Graham et al., 1973), and the diagnosis and treatment may be delayed. Later in the course of the disease, typical signs of tricuspid regurgitation (Banks et al., 1973) and increasing heart size are frequent. These symptoms were observed in our patient and the diagnosis was also verified by right-sided ventriculography.

Staphylococci are still the most common agents in this connection (Roberts and Buchbinder, 1972; Banks et al., 1973; Graham et al., 1973; Menda and Gorbach, 1973), but in recent years other organisms, particularly Gram-negative bacilli, have also been implicated (Carruthers and Kanokvichayant, 1973; Graham et al., 1973; Rosenblatt et al., 1973). Mixed infections have also been reported (Menda and Gorbach, 1973; Simberkoff et al., 1974).

Staphylococcal tricuspid endocarditis may respond to antibiotic therapy alone (Arbulu et al., 1973; Menda and Gorbach, 1973), and initially this seemed to apply in our case as the patient was discharged apparently without active infection, after 10 weeks of antibiotic treatment. Her second illness with fever and pulmonary emboli suggested reactivation of her endocarditis. The signs of acute inflammatory reaction in the excised valve supported this suggestion but the aetiology of the relapse remained obscure since repeated blood cultures were negative and examination of the excised valve gave no indication of the causative agent.

Infectious foci in heart valves are difficult to treat. The bacteria may acquire resistance to the antibiotics, or L-forms of the bacteria may develop. In our case, a superinfection may have occurred when the patient may have been giving herself injections. A 'mixed' infection with Gram-negative bacteria in addition to staphylococci (Simberkoff et al., 1974) may have developed. The favourable effect of gentamicin and carbenicillin supports this.

The primary treatment of bacterial endocarditis is conservative. Surgical intervention was used only for patients with haemodynamic failure after healed endocarditis (Yeh et al., 1964), until Wallace et al. (1965) for the first time operated during the active stage of endocarditis when conservative treatment had failed. Since then several similar reports have been published (Strandell et al., 1969; Manhas et al., 1972; Graham et al., 1973; Black et al., 1974). The favourable results tally with the well-known fact that the elimination of infected foci is important for the successful treatment of septicaemia.

Valve replacement during active endocarditis is not always successful, and there are reports of paraprosthetic endocarditis and failure of sutures (Manhas et al., 1972). Consequently, a decision on surgical intervention must not be premature, and should only be undertaken when antibiotic treatment has been shown to be ineffective. On the other hand, the probabilities of left heart involvement and toxic myocarditis increase with time. Arbulu et al. (1973) recommend surgical intervention when antibiotic treatment has not healed the infection within 6 weeks.

The small pressure differences within the right heart make damage to its valvular system less detrimental than such damage to the left heart. Traumatic tricuspid damage, which persisted for several decades without any symptoms, has been described previously (Croxon et al., 1971; Morgan and Forker, 1971; Marvin et al., 1973). In the series of Arbulu (Arbulu et al., 1973) during a follow-up time of 3 to 27 months, only 1 of 10 survivors required insertion of a prothetic valve. However, experience from animal experiments (Spellman and Balkissoon, 1956; Michl et al., 1973), from traumatic tricuspid regurgitation, and from patients with valve resections (Michl et al., 1973; Robin et
ad., 1974), indicates that right heart failure may develop sooner or later.

Adaptation to tricuspid regurgitation in young and otherwise healthy persons with normal pulmonary resistance may be considerable, but the insertion of a prosthesis could result in completely normal haemodynamic function. Our patient is now working full time and water skis, which is indicative of good haemodynamic function.

We consider that tricuspid valve replacement is safe when a low profile prosthesis with optimal haemodynamic properties, such as the Björk-Shiley valve, is used.

References


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Two-stage replacement of tricuspid valve in active endocarditis.

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Br Heart J 1977 39: 1276-1278
doi: 10.1136/hrt.39.11.1276

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