medical records department, Auckland Hospital Board, prepared the data sheets, while analysis of results was greatly helped by the co-operation of Mr. K. E. Bensley, of the medical statistics unit.

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Too Ill for Cardiac Surgery ?

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Rapid advances in open heart surgery and the achievements of individual surgeons have created new possibilities for patients with advanced valve disease. This paper is written to pay tribute to my surgical colleagues Donald N. Ross and Magdi H. Yacoub at the National Heart Hospital and to draw attention to the dramatic results which can be obtained even in very ill patients.

Four patients are reported, all of whom would have died in a matter of days or weeks without valve replacement. They are now well and have returned to their normal occupations. Three had advanced aortic valve disease, which was probably congenital in two. The fourth had rheumatic heart disease with severe destruction of mitral, aortic, and tricuspid valves. All four were so ill that one was hesitant to advise surgery at such a late stage, but it was evident that medical treatment had nothing further to offer.

The operations reported were carried out at the National Heart Hospital between 17 December 1965 and 5 October 1967 and comprised 4 of the 461 open heart procedures during this 22-month period.

Case 1

The patient, a man aged 42, had severe calcific congenital aortic stenosis. Heart disease had been diagnosed at the age of 12 years, when a murmur was heard at a routine school examination. He was asymptomatic until he was 41, when he developed dizziness on exertion. By June 1965 he was getting effort dyspnoea with severe angina, and in the autumn was having recurrent nocturnal dyspnoea. When admitted to hospital on 3 December 1965 he was in status anginosus and was deteriorating mentally. The physical signs were those of severe calcific aortic stenosis with a very low cardiac output. The limbs were cold and the peripheral pulses almost imperceptible. Blood pressure was 90/75 mm. The venous pressure was raised 8 cm. above the sternal angle with a dominant "a" wave. The heart was grossly enlarged and there was an aortic systolic murmur and thrill, also a soft immediate diastolic murmur of aortic regurgitation. The electrocardiogram (E.C.G.) confirmed sinus rhythm, with extreme left ventricular hypertrophy, and there was a small P mitrale. The chest radiograph showed gross left ventricular enlargement with very heavy calcification of the aortic valve and a dilated ascending aorta. The upper-lobe pulmonary veins were dilated (Fig. 1A). Moderate pulmonary hypertension was demonstrated at cardiac catheterization with a mean pulmonary artery wedge (indirect left atrial) pressure of 25 mm. Hg, pulmonary artery 75/25 mm. Hg, mean 40 mm. Hg. The aortic pressure varied from 95/60 to 105/75

mm. Hg. It was not possible to manoeuvre the catheter across the aortic valve, so left ventricular pressures were not obtained. Arterio-venous oxygen difference was 85 ml./l. with a cardiac output 4 l./min.

The patient's condition deteriorated in spite of full medical therapy, the urinary output fell, and the blood urea rose from normal to 140 mg./100 ml. Mr. Donald Ross operated as an emergency on 17 December and decided to insert a Magovern valve rather than a homograft to minimize the time of the operation. The aorta was opened by a long oblique incision, showing a heavily calcified disorganized bicuspid valve with a central irregular slit-like orifice 1 mm. in width by 2 cm. in length. It was completely excised, and large pieces of calcium which ran down into the anterior cusp of the mitral valve were also removed. A purse-string suture was placed round the bases of the aortic cusps and a size 5 Magovern valve was fixed in position, the claws being positioned to engage with the purse-string suture. After closure of the aortotomy the heart resumed its beat without difficulty. The left atrial pressure fell from a pre-valve replacement mean of 40 mm. Hg to 10 mm. Hg and the aortic pressure was well maintained at 110/80 mm. Hg. The total time on cardiopulmonary bypass was 34 minutes.

There were no postoperative problems; the blood urea returned to normal, the heart diminished in size, and the dilatation of the upper lobe veins disappeared. The left bundle-branch block which developed at operation just before insertion of the Magovern valve resolved spontaneously in the fourth postoperative week. The patient left hospital on 25 January 1966. When first seen as an outpatient after the operation on 17 March he was already going for long walks without symptoms and there had been a considerable reduction in heart size (Fig. 1B). He was last seen on 21 September 1967, when he was asymptomatic and again doing a full-time job as a traveller. The Magovern valve was competent and his systemic pressure was 130/80 mm. His only treatment was anticoagulants, which he has been advised to continue owing to the thromboembolic risk associated with prosthetic valves.

FIG. 1.—Case 1. Chest radiographs. A, Before aortic valve replacement (10 December 1965). B, After surgery (17 March 1966).

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Case 2

A married woman aged 46 had rheumatic, mitral, aortic, and tricuspid valve disease, with severe incompetence and some stenosis at each valve. She had two attacks of chorea, at the ages of 9 and 11 years, and heart disease was diagnosed after the second attack. Her activities were always restricted, but she had a normal pregnancy and delivery at the age of 23 and a second pregnancy complicated by toxæmia at 27 years. She developed bacterial endocarditis at the age of 37 years; the organism was *Streptococcus viridans*;

FIG. 2.—Case 2. Penetrated chest radiograph showing triple valve replacement. Mitral and tricuspid valves replaced by Starr-Edwards prostheses. Aortic valve replaced by Magovern valve.

treatment with penicillin was successful. She remained well, managing her household duties until June 1964, when at the age of 44 she developed cardiac failure, with increasing effort dyspnoea and peripheral oedema. She deteriorated, and four months later was investigated at the National Heart Hospital. From the investigation, which included left and right heart catheterization, left ventricular angiography, and aortography, it was apparent that aortic, mitral, and tricuspid valves were all badly disorganized and leaking severely. In addition there was moderate pulmonary hypertension (P.A. 80/35 mm. Hg, mean 50 mm. Hg). Triple valve replacement was clearly indicated, but at the time of these investigations in the autumn of 1964 we were not in a position to do this. She returned home on full medical treatment and was advised to rest as much as possible. In spite of a very inactive life she became

steadily worse until she was unable to leave the house owing to dyspnoea and severe peripheral oedema.

She was readmitted in December 1965. During the preceding four weeks she had noticed dark stools, and on admission her haemoglobin was 61%—8.9 g./100 ml. After investigation it was thought that her intestinal bleeding was due to anticoagulants rather than from oesophageal varices. In spite of a very large liver and ascites the serum alkaline phosphatase and total bilirubin were only slightly raised. She was severely hyponatraemic, with repeated serum sodium levels between 120 and 125 mEq/l., but there was a total exchangeable sodium of 3,170 mEq (normal for her build 1,920–2,940 mEq). There was never any hypokalaemia though the total exchangeable potassium was only 1,850 mEq (normal for her build 1,920–3,260 mEq). She continued to deteriorate in spite of medical treatment, and on 7 February 1966 Mr. Donald Ross operated.

The aorta was opened, showing a stenotic rigid triangle, and hypothermic coronary artery perfusion was started. The left atrium was then opened by a long oblique incision above the mitral ring, exposing a rigid, rolled, and thickened mitral valve with an orifice about 2.5–3 cm. This was excised together with the shortened fibrotic chordae and papillary muscles. The mitral valve was replaced with a size 2 mitral Starr-Edwards prosthesis. The left atrium was closed and the aorta opened more widely. A typically tricuspid rheumatic type of aortic valve was excised and a size 2 Magovern valve inserted. The aorta was closed and the right atrium opened. The tricuspid valve was regurgitant through a central jet, and there was fusion of all three commissures, leaving a triangular central orifice of about 2 sq. cm. This valve was also excised, being replaced with a size 3 Starr-Edwards prosthesis. Hypothermic arrest of the heart was used for the mitral replacement, but the aortic and tricuspid replacements were carried out with full-flow normothermic perfusion and a co-ordinated ventricular beat. The heart resumed its work-load without difficulty at the end of the procedure. The total time on cardiopulmonary bypass was 105 minutes.

The postoperative course was slow but without complications, and she left hospital to start her convalescence on 15 March 1966. At the time of writing she was leading a normal active life looking after her two young children. She was free from dyspnoea and had had no ascites or peripheral oedema since the operation. This case was one of the first successful triple valve replacements to be carried out in this country (Fig. 2).

Case 3

A man aged 29 with aortic valve disease had had heart disease in childhood but he led an unrestricted life until 1962, when at the age of 25 he developed angina and progressive effort dyspnoea. In October 1965 cardiac catheterization showed a peak systolic gradient of 100 mm. Hg and in January 1966, in Bristol, the aortic valve was replaced with a Starr-Edwards prosthesis (Fig. 3 A). His initial recovery was satisfactory, but within a month of operation

FIG. 3.—Case 3. Chest radiographs. A, 24 February 1966, with Starr-Edwards prosthesis in correct position. B, 16 May 1966, showing change in axis of the prosthesis with considerable cardiac enlargement and failure. C, 5 September 1966; heart again smaller after replacement of infected prosthesis by a reconstructed freeze-dried homograft.

he became pyrexial and early in February the diagnosis of bacterial endocarditis was made. Blood cultures were negative, presumably due to the antibiotics which had been used to cover his operation. He was treated with penicillin and streptomycin for seven weeks and rapidly became afebrile, but during this time a loud aortic diastolic murmur developed. Two weeks after antibiotics were stopped he again became pyrexial and developed severe cardiac failure. He was transferred to the National Heart Hospital on 16 May with gross heart failure, pulmonary oedema, and bilateral hydrothoraces, all of which failed to respond to medical treatment. There was evidence of severe aortic regurgitation as well as bacterial endocarditis, for he was still clubbed and pyrexial. Examination of the chest radiographs showed a change of about 30° in the axis of the Starr-Edwards prosthesis (Fig. 3 B), suggesting that it had become partially detached and this was the cause of the aortic regurgitation.

Medical treatment failed to improve either his failure or infection, so Mr. Donald Ross operated on 23 May. He found a grossly infected Starr-Edwards prosthesis, the infection having involved the whole valve ring. Half the circumference of the valve was detached. There was a large aneurysm posteriorly below the aortic ring which had caused detachment of the mitral leaflets from their aortic origin. In addition there was a second aneurysmal dilatation anteriorly in the region of the right coronary ostium. All infected tissue was excised and both the posterior and anterior aneurysms were obliterated, with interrupted sutures. The mitral cusps were reattached to the aortic root. The Starr-Edwards prosthesis was removed and a 2.5-cm. reconstructed freeze-dried homograft was then fixed in place. The heart remained in sinus rhythm throughout with bilateral coronary perfusion, and at the end of operation resumed its work-load without difficulty. The total time on cardiopulmonary bypass was 120 minutes.

He had a good diuresis, which started before he left the theatre, and the immediate postoperative course was satisfactory. In view of previous infection he was kept on 40 mega units of penicillin, 10 g. of cloxacillin, and 1 g. of streptomycin per day. At the beginning of the second postoperative week he developed a pyrexia of 102° F. (38.9° C.) and again became toxic. Blood cultures showed a heavy growth of *Candida albicans*. Treatment with amphotericin B was started, all other antibiotics being discontinued. He responded at first, but after five weeks the temperature rose to 104° F. (40° C.) and he was having rigors. Blood cultures were positive, but this time contained a coagulase-positive *Staphylococcus aureus* which was resistant to penicillin. He was then treated with cloxacillin 6 g./day, later increased to 18 g./day; the amphotericin B was continued. In all he had 10 weeks' treatment with cloxacillin (1,040 g.) and eight weeks' treatment with amphotericin B (2,580 mg.). In the sixth postoperative week he developed pain and swelling in the right calf and a pleural rub due to a small pulmonary embolus, so anticoagulants were started. He was afebrile from the twelfth postoperative week, but anticoagulants were continued for a further month, finally being withdrawn on 15 September 1966.

The subsequent course was entirely satisfactory, and all infection appeared to have been eradicated. He was symptom-free and back at work. He remained in sinus rhythm with a blood pressure of 110/60 mm. Hg. The heart was slightly enlarged, owing to the left ventricle, and there was a soft aortic diastolic murmur, but no peripheral signs to suggest important aortic regurgitation (Fig. 3 C).

Case 4

This patient was a man aged 41 with aortic valve disease. He had been well until 1962, when he first noticed that he had exertional dyspnoea, which was progressive. In November 1965 he was admitted to the Royal Victoria Hospital, Belfast, where cardiac catheterization showed a peak systolic gradient across the aortic valve of 84 mm. Hg, with an end diastolic pressure (pre "a" wave) in the left ventricle of 24 mm. Hg and an "a" wave in the left ventricle of 48 mm. Hg. Left ventricular angiography and an

aortogram showed a rigid valve with both stenosis and regurgitation. There was no mitral valve regurgitation. In January 1967 his effort dyspnoea became very much worse and he developed orthopnoea with occasional nocturnal dyspnoea. He was admitted to the National Heart Hospital, London, in September 1967, by which time his activities were minimal and he was having frequent nocturnal dyspnoea. In spite of medical treatment he deteriorated and developed progressive renal, hepatic, and cardiac failure. The blood urea rose from normal to 230 mg./100 ml., with a serum sodium of 117 mEq/l. and a serum potassium of 5.6 mEq/l. The liver was distended below the umbilicus and he became jaundiced; the serum bilirubin was 4.5 mg./100 ml. He lapsed into coma between 4 and 5 October with a systolic blood pressure of 90 mm. Hg.

Mr. Magdi Yacoub operated on the night of 5 October as an emergency. The aortic valve was bicuspid and heavily calcified, and was replaced by a Starr-Edwards prosthesis. The total time on cardiopulmonary bypass was 65 minutes. The blood pressure improved and by the time he left the theatre was in the region of 130/70 mm. Hg. He regained consciousness quickly and the post-

FIG. 4.—Case 4. Chest radiographs. A, Before aortic valve replacement (27 September 1967). B, Four and a half weeks after surgery (30 October 1967).

operative period was uneventful. Jaundice disappeared rapidly. The liver became impalpable and both the blood urea and the serum bilirubin returned to normal within 10 days of operation. The heart became smaller (Fig. 4) and he left hospital to start his convalescence on 31 October. When seen again on 21 November, before returning to Belfast, he was asymptomatic.

Summary

The cases of four patients are reported, all of whom had advanced congenital or rheumatic valve disease and whose life expectancy without surgery could not have been more than a few days or weeks. After valve replacement (single or multiple) all are now well and have returned to their previous occupations.

No patient with valve disease should be considered too ill for surgery, assuming that a skilled surgical team orientated to this type of problem is readily available.

I should like to express my thanks not only to the surgeons but to all members of the medical, nursing, and technical staff who were associated with these cases. Case 1 was referred by Dr. M. J. Goldberg, consultant cardiologist, the Groby Road Hospital, Leicester. Case 2 was under the care of Dr. P. Traub, of London. Case 3 was referred by Dr. D. W. Barritt, consultant physician, Bristol General Hospital, Bristol. Case 4 was under the care of Dr. D. Boyle, consultant cardiologist, Royal Victoria Hospital, Belfast.