

Homograft replacement of the pulmonary valve

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Pulmonary incompetence is a common sequel of pulmonary valvotomy, but many authors believe that the haemodynamic consequences of this regurgitation are well tolerated (Talbert, Morrow, Collins, and Gilbert, 1963). In this paper we report a patient who had severe pulmonary incompetence after valvotomy and who failed to maintain her initial clinical improvement. Because of the severe leak it was considered reasonable to correct the incompetence surgically. A homograft pulmonary valve was used, and we are unaware of any previous report of its use in man, though Ross, Cooper, and Brockenbrough (1961) described the experimental replacement of the pulmonary valve with a tricuspid semilunar prosthesis in dogs.

CASE REPORT

A 9-year-old white girl was seen in 1958. There was a history of sweating and listlessness during infancy. After the age of 1 year she began to tire easily, and at the age of 4 years cyanosis of the lips and nails was noted. She was breathless on moderate exertion.

She was undersized for her age and weighed 53 lb. (24 kg.). Central and peripheral cyanosis was present. The pulse was regular and the blood-pressure was 104/70 mm. Hg. There was a left parasternal heave of right ventricular hypertrophy, but no thrill could be felt. On auscultation a loud coarse pulmonary ejection systolic murmur with a single second sound was heard. Femoral pulses were present. The liver was not palpable.

Radiographs showed an increase in the transverse diameter of the heart with a cardiothoracic ratio of 55% and a prominent pulmonary artery segment. The right ventricle and right atrium were enlarged. The lung fields were under-vascularized (Fig. 1).

The electrocardiogram (Fig. 2) showed right ventricular hypertrophy and strain.

Cardiac catheterization was done under basal narcosis with rectal barbiturate and using local anaesthetic. The salient findings are listed in Table I.

The diagnosis was 'valvular pulmonic stenosis with intact ventricular septum; small right-to-left shunt, probably via a patent foramen ovale'.

In January 1959 open pulmonary valvotomy was performed under cardiopulmonary bypass using the disc oxygenator. The heart was arrested with potassium citrate. The pulmonary artery was opened and a cone-shaped pulmonary valve was encountered. No commissures were identifiable. The diaphragm was cut, converting it into a bicuspid valve with an estimated orifice diameter of 2.5 cm. A small patent foramen ovale was closed by direct suture.

Convalescence was uneventful.

Examination three weeks after operation revealed no cyanosis, and the systemic arterial pulse was collapsing in character. The blood-pressure was 120/60 mm. Hg. Jugular venous pressure was raised to the angle of the jaw due to tricuspid incompetence. The liver was not palpable. No adventitious sounds were audible in the lungs. On palpation of the precordium a systolic thrill under the left clavicle and a right ventricular left parasternal heave were felt. The first heart sound was loud. The pulmonary component of the second sound could not be heard. There was a coarse pulmonary ejection murmur (grade 4/6), stopping short of the second sound, and a rough pulmonary diastolic murmur.

The patient was seen again at the age of 15 years in February 1964. She had remained well and been able to swim and play tennis until about a month previously, when she had begun to feel continually tired. She was now a well-developed adolescent. There was no cyanosis or respiratory distress. Prominent 'a' waves were noted in the jugular venous pulsations. The marked left parasternal heave due to right ventricular enlargement persisted. There was a grade 2/6 pulmonary ejection systolic murmur and a grade 2/4 rough diastolic murmur of pulmonary incompetence commencing 0.08 sec. after aortic valve closure (Fig. 3).

TABLE I
CARDIAC CATHETERIZATION 1958

Site	Pressure (mm. Hg)		O ₂ Saturation (%) (oximeter)
	Systolic	Diastolic	
Right pulmonary capillary	5	3	96
Main pulmonary artery	15	8	35
Right ventricle	185	0-15	
Right atrium	a = 11 (mean = 6)		
Brachial artery	75	55	86

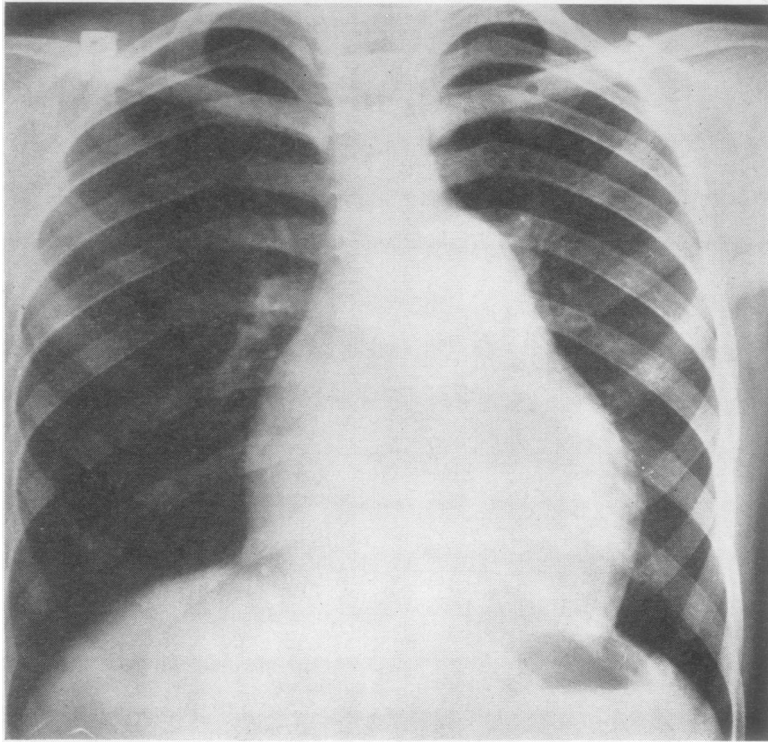


FIG. 1. *Postero-anterior chest radiograph 1958.*

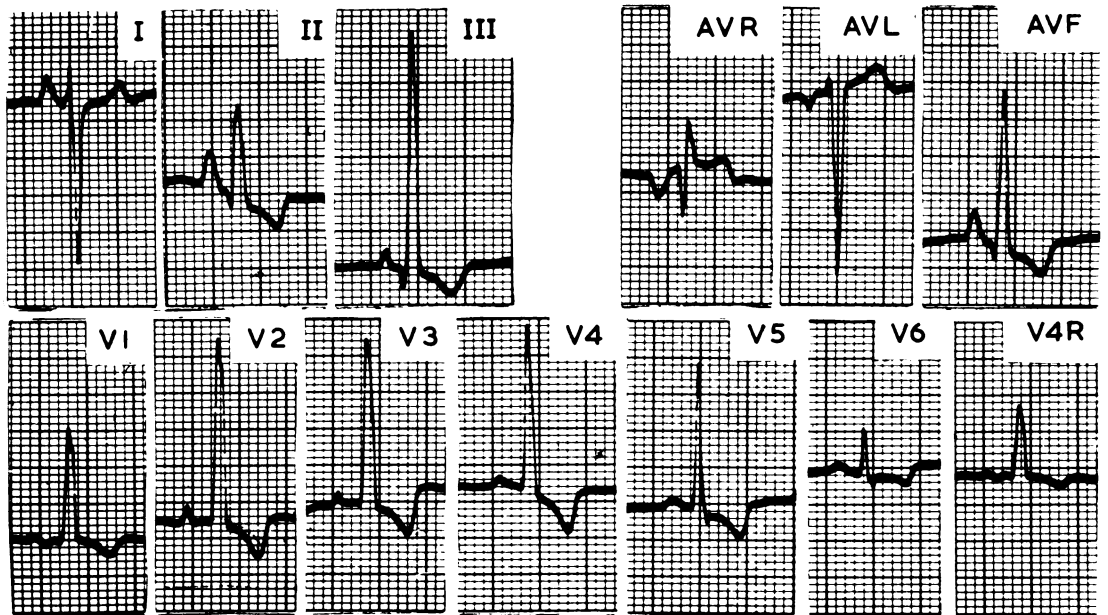


FIG. 2. *Electrocardiogram 1958. The chest leads are at half sensitivity.*

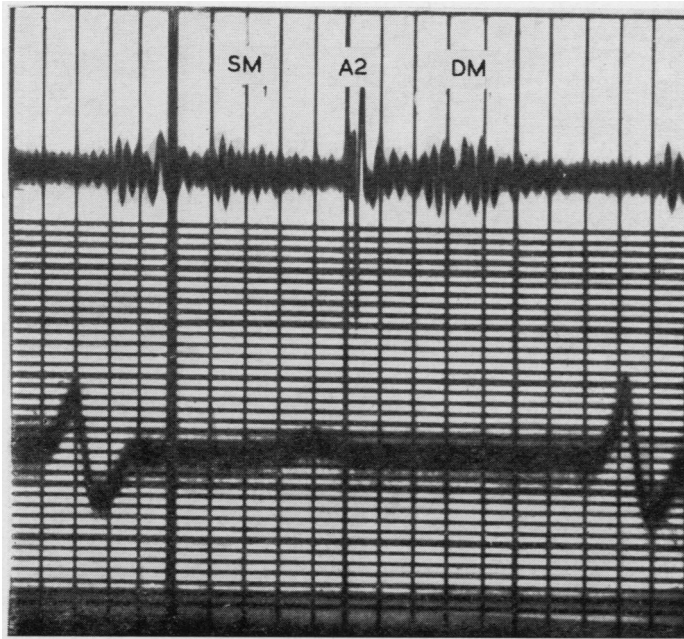


FIG. 3. *Phonocardiogram 1964. Recorded at pulmonary area (logarithmic tracing). SM, systolic murmur; A2, aortic component of second heart sound; DM, diastolic murmur.*

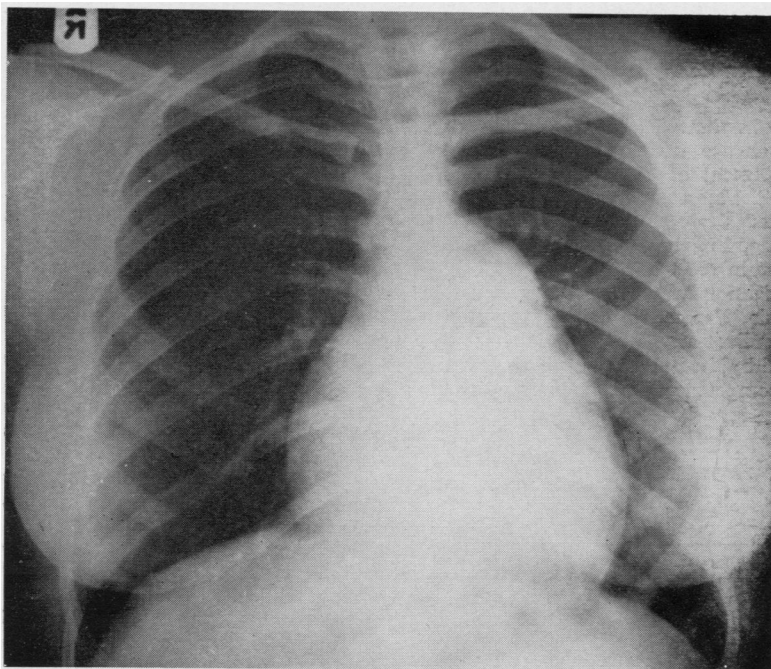
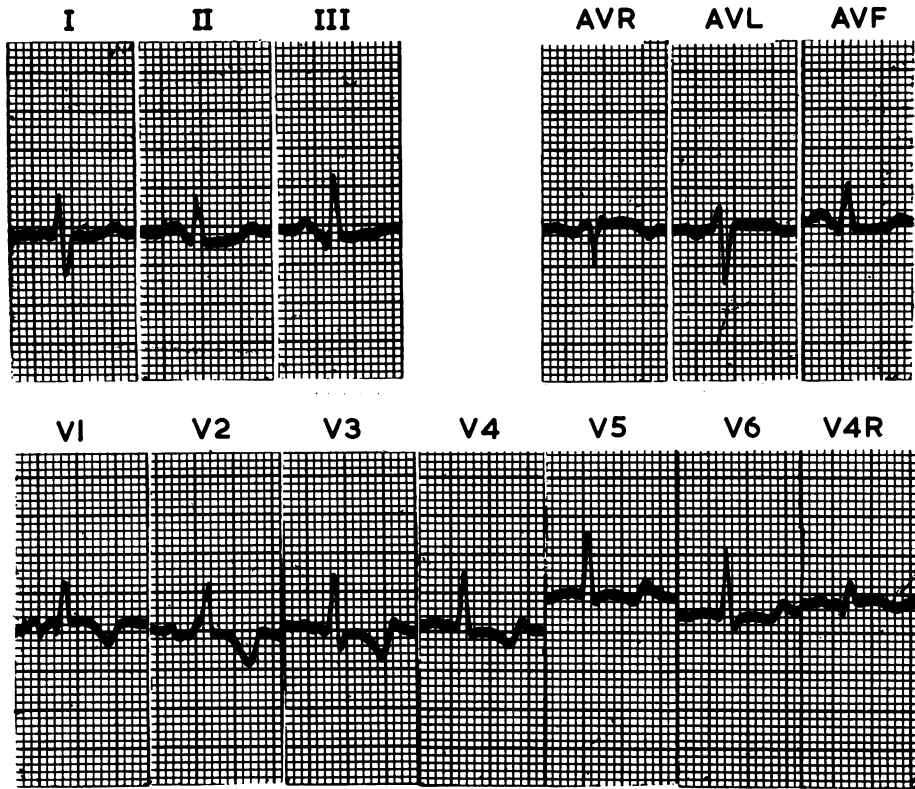


FIG. 4. *Postero-anterior chest radiograph 1964.*

FIG. 5. *Electrocardiogram 1964.*

Cardiac radiographs showed persistent enlargement with marked prominence of the pulmonary artery segment and enlargement of the right ventricle and right atrium (Fig. 4). The pulmonary vascularity was within normal limits.

An electrocardiogram showed right ventricular hypertrophy and strain (Fig. 5).

Cardiac catheterization was done under sedation and local anaesthesia. The salient findings are shown in Table II. The gradient across the pulmonary valve had been eliminated by the pulmonary valvotomy, but free pulmonary incompetence was present causing the

TABLE II
CARDIAC CATHETERIZATION 1964

Site	Pressure (mm. Hg)		O ₂ Saturation (%) (oximeter)
	Systolic	Diastolic	
Main pulmonary artery	35	5	72
Right ventricle	35	0-10	72
Right atrium	a = 12	v = 10	71
	(mean = 8)		
Brachial artery	125	75	89

Cardiac output 4.7 l./min. (measured by dye dilution technique).

pressure tracing in the main pulmonary artery to resemble that in the right ventricle. The end-diastolic pressure in the right ventricle and the right atrial pressure were raised. The arterial oxygen saturation of 89% is at the lower limit of normal for this altitude (5,760 feet) (1,755 m.).

In August 1965 she was admitted to hospital for observation and further assessment. Her symptoms, physical findings, electrocardiogram, and radiographs were unchanged.

The severe degree of pulmonary incompetence was considered to be partly responsible for her symptoms and it was felt that restoration of pulmonary valve competence was a reasonable approach to the problem.

On 2 September 1965 a homograft pulmonary valve was inserted at the pulmonary valve site under cardiopulmonary bypass.

The approach was through a vertical sternal-splitting incision. Pericardial adhesions were divided without difficulty. The external diameter of the base of the pulmonary artery measured 4.3 cm. The main pulmonary artery was opened longitudinally, the incision extending from the valve ring to the arterial bifurcation. In view of the large size of the vessel

excellent exposure was provided. It was possible to identify three blunt commissures, but the remnants of the cusps were thickened and retracted, forming narrow ledges which could not possibly approximate. They were excised at their bases. A freeze-dried pulmonary valve homograft taken from a youth who had been killed accidentally three months previously was rehydrated with a saline solution containing penicillin and streptomycin. The stretched diameter of the reconstituted homograft, measured with a Tubbs dilator, was 4.2 cm. and the internal stretched diameter of the host's pulmonary arterial ring using the same instrument was 3.8 cm. The arterial wall of the homograft had been cut about 2 mm. clear of the cusp bases and commissures, and below the cusps as much muscle as possible had been removed up to and following the curve of the cusp bases.

The insertion of the homograft in this position was technically easier than the equivalent replacement of an aortic valve because of the large size of both the host's artery and the homograft. Three 4-0 silk anchoring sutures were placed precisely below the site of the host's commissures. These were then brought through the base of the homograft in the same relative positions and were tied. The commissural pillars of the homograft were then fixed with mattress sutures passed through the pulmonary artery wall at the precise situation of the commissural remnants and were tied externally over teflon pledgets. The fringes of pulmonary artery left attached to the

homograft were then sewn to the remnants of the bases of the host's cusps with continuous 5-0 silk sutures. These remnants were not always clearly identifiable, and in parts the suturing had to be made directly to the pulmonary artery endothelium along an estimated curve. On completion the graft sat snugly in its bed and the cusps were free and mobile. The artery was then repaired.

Throughout the 70 minutes' bypass the heart remained in sinus rhythm and no difficulties were experienced when coming off the pump.

Pressures recorded by direct needle puncture of the pulmonary artery before bypass showed a close resemblance to ventricular pressure curves (Fig. 6A). After replacement of the pulmonary valve the pulmonary artery pressure tracing (Fig. 6B) revealed normal features but with a Venturi effect in early systole. There was a peak systolic gradient of 10 mm. Hg across the homograft valve.

Convalescence was rapid and free of complications.

Clinical examination three weeks after operation revealed persistence of the prominent "a" waves in the jugular venous pulsations and of the right ventricular enlargement. On auscultation (Fig. 7) there was a grade 3/6 pulmonary ejection systolic murmur, and both aortic and pulmonary components of the second heart sound were clearly audible. Movement of the components of this sound with respiration was normal.

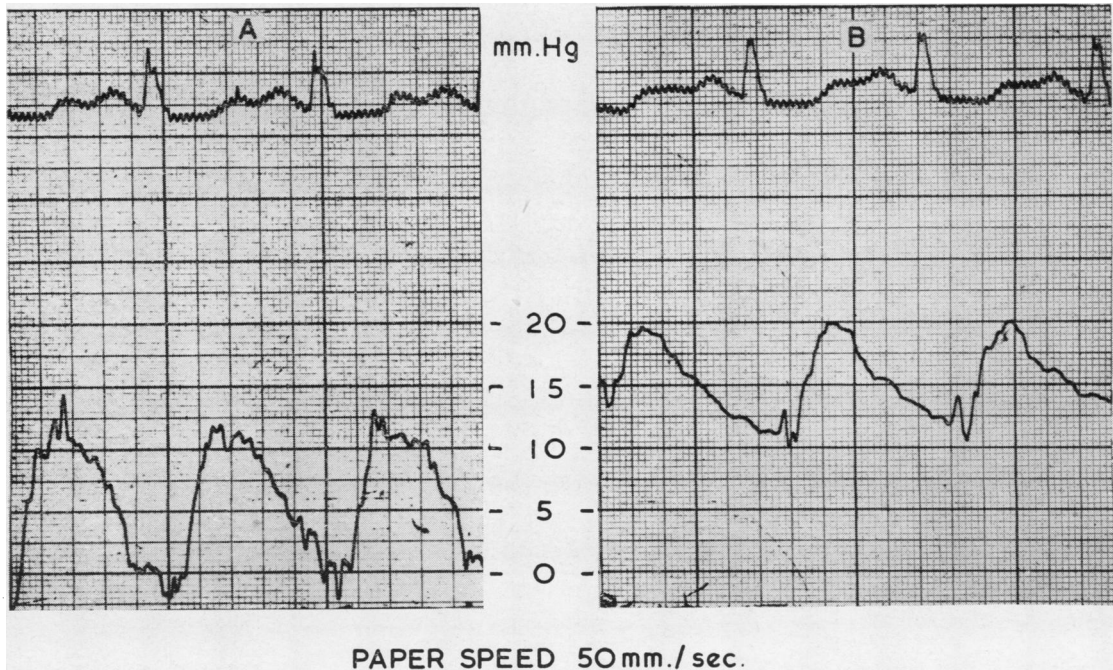


FIG. 6. Intra-operative pressure tracings by direct needle puncture of pulmonary artery. (A) Pulmonary artery pressure before valve insertion; (B) pulmonary artery pressure after valve insertion.

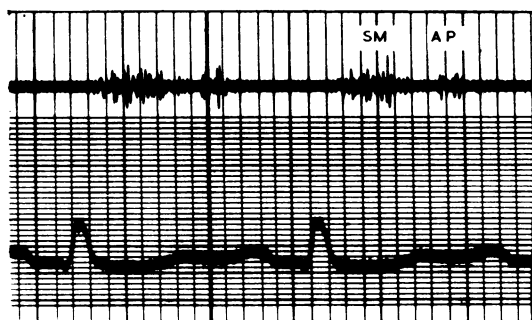


FIG. 7. Phonocardiogram September 1965, three weeks after surgery, recorded at pulmonary area (logarithmic tracing). SM, systolic murmur; A, aortic, and P, pulmonary component of the second heart sound.

The electrocardiogram and radiographs were unchanged.

SUMMARY AND CONCLUSIONS

A case of pulmonary incompetence following pulmonary valvotomy with treatment by insertion

of a pulmonary valve homograft is reported. The technique of the operation is described.

The operation was not difficult, particularly as coronary artery perfusion was not required. The pulmonary valve homograft was not particularly flimsy, and it held sutures without difficulty.

Clinical evidence to date suggests competence of the pulmonary valve.

The clinical and haemodynamic response will be fully assessed one year after operation.

We wish to express our thanks to Dr. H. Pretorius for permission to reproduce the electrocardiogram in Fig. 2, and to Mr. A. Shevitz for his assistance with all the reproductions.

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