Mitral valve replacement with stored inverted pulmonary homograft valve¹

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The fate is described of 11 patients who had a stored pulmonary valve homograft mounted on a frame or in a Dacron jacket used for mitral valve replacement. Mitral regurgitation due to holes in the attenuated cusps occurred in nine by one year, requiring re-operation or causing death. Only one patient remains well with moderate mitral regurgitation after two years.

Since January 1967, 118 patients have had mitral valve replacement with a preserved cadaver semilunar homograft valve mounted in a jacket or frame (Murray, Roschlau, and Loughed, 1956; Heimbecker *et al.*, 1962). In all except 11 patients the aortic valve was used.

However, in the 11 patients in whom an inverted pulmonary valve was used (Lower, Stofer, and Shumway, 1961) the clinical course has been different. The purpose of this report is to draw attention to the problems and discourage the use of this form of mitral valve replacement.

MATERIAL

Between September 1968 and April 1969, 11 patients aged 24 to 66 years had mitral valve replacement with an inverted stored pulmonary valve homograft. The valves in five were sterilized by gamma-irradiation and in five by ethylene oxide. These valves were flash frozen and stored at -79°C . There is no record of the method of preparation in one.

In five patients the valve was mounted on a Dacron or Teflon covered strut of polypropylene (one Ross-Kay, one Ionescu, one Cutter) and in six it was wrapped in a Dacron or Teflon jacket (Yacoub and Kittle, 1969).

All patients had severe and disabling symptoms at the time of operation and had had full medical treatment for a minimum of six months. Six had chronic congestive failure and three had had previous valve surgery. Five patients had homograft replacement of another valve at the same operation, and one 61-year-old had replacement of the ascending aorta for a dissecting aneurysm. There were no operative deaths. Clinical follow-up included regular documentation of the symptoms, physical signs, electrocardiogram, and chest radiograph. Recatheterization was undertaken on five after symptomatic deterioration. The gross appearance of the pulmonary valve was noted at death or re-operation, but histology was not available.

RESULTS

The follow-up of patients with a pulmonary valve in the mitral position varied from two weeks to

two and a half years and the clinical findings are shown in the Table. Some of these patients lived for a longer period after replacement of the pulmonary homograft by a Starr or fascia lata valve. Six patients required re-operation for mitral regurgitation two weeks to 15 months after the initial replacement, and one of these died. In the patient operated on at two weeks, the graft had become detached from its jacket and folded inwards, causing mitral obstruction as well as regurgitation.

Of the remaining five patients, three died from heart failure three months, 14 months, and 15 months after operation, one is in severe heart failure 22 months later and has had re-operation, and the other (R.D.), who has signs of mitral regurgitation, is ambulant with a limited effort tolerance two and a half years after operation. Clean holes in the thin central part of one or more cusps were found in seven patients, and in four, two cusps were attenuated and thinned. In one (C.M.) the strut split, and in another (J.K.) a cusp had split away from the jacket and there was a separate hole in an attenuated cusp; in another (A.R.) there was central regurgitation but no holes. The valve was well incorporated but there was an exposed area of Dacron covered with platelet thrombus which might have been the source of embolism.

One patient had been unconscious from the time of operation with a tetraplegia; the mitral valve at three months had a hole in one thinned cusp

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TABLE

SUMMARY OF CLINICAL DATA, FATE, AND MACROSCOPICAL PATHOLOGY: 11 PATIENTS WITH INVERTED PULMONARY HOMOGRAFT REPLACEMENT OF THE MITRAL VALVE

Case	Age (yr)	Disease	Op. Date	Sterilize	J or F	Other Procedure	Ant.	After Operation					
								Pan SM	LHF	Re-op. Date	Pathology Mitral	Complication	State Aug. 1971
J.K.	43	Calcific MS	Sept. 1968	γ Eth. O	J	—	1/12	2/12	9/12	Sept. 1969 St.	Holes + split from jacket	Pulm. cerebral embolus	Dec. 1969 Died
P.K.	46	Calcific MS	Oct. 1968	γ Fr.	J	—	+	1/52	6/12	Oct. 1970	Holes 2 thin cusps	—	AR mild Well
C.W.	45	TR, MR AR	Nov. 1968	γ	J	Ao.Ho.	+	1/12 TR	15/12	June 1971 3 St.	Holes 2 thin cusps	Ascites oedema haemolysis RHF	Well Haemo-lysis ill R + LHF
A.R.	54	MR, MS	Dec. 1968	Fr.	J	—	+	9/12 TR	22/12	Oct. 1971	Central leak. No holes	Cerebral embolus	Well
C.H.	43	Calcific MS, AR	Dec. 1968	Eth. O	J	Ao.Ho. 1966	+	2/52	2/52	Feb. 1969	Graft split from ring	Ao.Ho. Re-op. Aug. 1969	Syst. cerebral embolus
R.D.	61	MR, aneurysm	Feb. 1969	Eth. O	J	Repl. ascending aorta	—	4/12	—	—	—	—	Ambulant psychosis
J.E.	47	MR, TR	Feb. 1919	?	F	Ao.Ho. Tr.	—	4/12	14/12	—	No necropsy	Pneumonia Pulm. embolus	Died April 1970
I.R.	24	MR, AR	Mar. 1969	Eth. O Fr.	F	Ao.Ho.	1/12	1/12	3/12	July 1969 FL	Holes 2 thin cusps	Pulm. embolus	Well
A.M.	40	MR, MS, TR, TS	Mar. 1969	γ Eth. O	F	Pulm. Ho. Tr.	+	?	?	—	Holes 1 avulsion	—	Died May 1970
M.T.	66	Calcific MS	April 1969	Eth. O	F	—	+	1/12	?	—	Hole 1 cusp	Unconscious	Died July 1969
C.M.	36	MR, MS	Sept. 1969	γ Fr.	F	—	+	4/12	5/12	Sept. 1970 St.	Hole 1 cusp strut split	TR	Well

J=jacket; F=frame mounted; Ant.=anticoagulants started; Pan SM=pansystolic murmur; RLHF=right and left heart failure; γ=gamma sterilized; Eth. O=ethylene oxide; Fr.=flash frozen; St.=Starr replacement; FL=fascia lata valve replacement; TR=tricuspid regurgitation; Tr.=tricuspid valve; MS=mitral stenosis; MR=mitral regurgitation; AR=aortic regurgitation; Ao.Ho.=aortic homograft; TS=tricuspid stenosis.

and a regurgitant murmur had appeared at one month. The pathology of the valve was not known in two patients as no necropsy was done in one and the other (R.D.) has not had re-operation.

A clear pansystolic murmur was documented two weeks to four months after operation in nine patients, and in six, reliable auscultators had not noted evidence of mitral regurgitation in the early postoperative weeks. Severe left heart failure usually occurred one to 12 months later, although in two, pulmonary oedema occurred quickly after the appearance of the murmur.

Systemic emboli occurred in three patients one month, 18 months, and 22 months after operation and none was on anticoagulants at the time of embolism, although in one (A.R.) with the late embolus, anticoagulants had been given for the first postoperative month. The other eight patients had anticoagulants for a minimum of two months with no embolic problems.

DISCUSSION

The use of the pulmonary valve in the mitral position was tried because of its larger size in comparison with the aortic valve and also because of shortage of cadaver homografts. As can be

seen from this experience, the results have been unsatisfactory; there was deterioration in six patients before six months had elapsed and a further three were in left heart failure by one year. Two patients remain well after 18 months and only one after two years. The gross pathology of the valve was similar in all patients with attenuation of cusps and holes or splitting away from the ring. It is possible that the method of sterilization or storage may have weakened the valves as this group includes the early gamma-irradiated valves. However, of the 23 inverted aortic valves in the mitral position either jacketed or frame-mounted and sterilized in the same way, there have been four with mitral regurgitation needing re-operation or causing death. The usual cause was tearing of the valve away from the frame with attenuation of the cusps and holes in the thinned areas in only one.

It thus appears that the pulmonary homograft valve may not be adequate, as suggested by Angell, Wuerflein, and Shumway (1967), for use in the mitral position, although a small experience using a living pulmonary autograft for mitral valve replacement (Ross, 1967) has been promising over a two- to three-year period in three patients. Attenuation with holes has been seen in one

patient with a frame-mounted pulmonary autograft after three months. Experience with the living pulmonary autograft is too small to know whether its fate will be the same as that of the pulmonary homograft. Rigid frame-mounted valves in the mitral position impose unusual stresses, and if the valve is weakened by sterilization or in basic design, failure may often occur quickly. It is likely to be a combination of factors which causes the failure of the pulmonary homograft.

From the above experience it is suggested that the stored pulmonary homograft valve mounted or jacketed is not suitable for mitral valve replacement.

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