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This information is current as of September 10, 2006

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The pulmonary autograft – a permanent aortic valve

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Abstract. Between 1969 and 1991, 339 patients had an aortic valve replacement with their own living pulmonary valve at the National Heart Hospital, Guy’s Hospital and the Harley Street Clinic, London. The longest follow-up is 24 years and cumulative follow-up is 3774 patient-years. No form of anticoagulation was used and there were no emboli. There were 25 hospital deaths (7.4%) but only 1 death since 1976. Late deaths occurred in 38 patients mainly from technical mal-insertion. Bacterial endocarditis occurred in 11 patients. Thirty-eight patients were re-operated upon and account for 15 of the late deaths. Freedom from re-operation was 85% and the actuarial patient survival was 80% at 20 years. There has been no evidence of primary tissue degeneration and explanted valves showed normal cusp cellularity. Accumulating evidence suggests that the cusps not only survive permanently but can grow with the patient making the operation ideal for children.

Key words: Pulmonary autograft – Pulmonary homograft

The first homograft valves were inserted in 1962 [15] and at that time, we were optimistic that we had achieved a permanent valve replacement since we thought they would be repopulated with cells from the host. However, the first explants at 3–5 years showed a uniformly acellular structure together with degenerative changes and calcification. Immunological reaction, although present, did not seem to play a major role in this process [2, 3].

In spite of a number of variations in sterilization and storage techniques, there is still a relentless process of tissue degeneration in these non-living cusps although the process is less florid than in glutaraldehyde tissue. It was clear that only a living and actively metabolizing cusp structure could offer the prospect of a permanent valve and this we felt could only be achieved by the use of living homograft cusps plus immunosuppression or by the use of living autologous cusps with identical structural characteristics. Only the pulmonary valve fulfills these criteria.

Consequently in 1967 we carried out the first pulmonary autograft procedure [17] restricting our indications initially to aortic valve disease in young adults between 30 and 40 years. Our chief anxiety related to the ability of the pulmonary valve to withstand systemic pressure, but we had backing from the experimental work of Lower and Shumway [13]. In fact, there has never been an acute cusp failure nor evidence of progressive failure over the 24 year follow-up period. The remarkable functional adaptability of the pulmonary cusps has been supported by biomechanical stress testing by Gorczynski et al. [8]. They showed that the pulmonary cusp has up to three times the tensile strength of the corresponding aortic cusp. This may relate to the fact that during foetal development, the right sided pressures are at systemic level.

It was anticipated that if a permanent aortic valve replacement could be achieved, this would have particular value in young people by avoiding a lifetime of anticoagulation or increasingly hazardous reoperations. The replacement of the right ventricular outflow tract has been with an aortic homograft [19] in 312 of the 339 patients. Again, the late results of this operation are well documented [11, 12] and more recently, a pulmonary homograft has been used routinely.

Material and methods

There have been 339 patients operated upon by the same surgeon. The median age was 29.3 years and the dominant lesion was aortic regurgitation (51%) while stenosis was dominant in 30% and 19%.
were mixed. Additional operative procedures were performed in 83 patients but without additional valve replacements. More recently, the tendency has been to operate on a younger age group because of the prospect of valve growth [4, 6, 16].

The surgical technique has been straightforward and consists of the well-established right ventricular reconstruction with a homograft plus replacement of the aortic valve with the autograft by exactly the same technique as described for aortic homografts [13, 18]. The only significant hazard is the first septal artery, but with a knowledge of the surgical anatomy [4] it is easy to avoid.

Since 1986, only pulmonary homografts have been used to reconstruct the right ventricular outflow. No anticoagulants or antiaggregants are used at any stage. Patients have been reviewed annually with the longest follow-up 24 years and a cumulative follow-up total of 3774 patient years. Event-free analysis was conducted using the methods of Kaplan and Meier [9].

**Results**

Hospital mortality accounted for 25 patients (7.4%). The majority of deaths occurred in the first 5 years from haemorrhage and arrhythmias secondary to involvement of the first septal artery. There has been only 1 operative death in the last 15 years. There were 38 late deaths again mostly confined to the first 5 years of this learning curve. Twenty-three were related to problems with the first septal artery and 15 followed reoperations for technical malinsertion. The actuarial survival rate including operative deaths in 80% at 20 years.

Twenty-three patients were reoperated upon during the period under review – 19 of these were for technical malinsertion usually resulting in prolapse of the non-coronary cusp at the time we were perfecting our homograft technique.

![Fig. 2A–E. Steps in the dissection of the pulmonary autograft. Note relationship of septal artery](image-url)
Fig. 3. 339 pulmonary autografts 1967–1991. Late deaths arose from problems with the septal arteries in 23 patients. The remaining 15 occurred during reoperations to correct regurgitation for malinsertion. Autograft reoperations (n = 33): technical failure was in 19, S.B.E. in 9, and "degeneration" in 5 patients. Freedom from reoperation at 20 years was by 85%.

Fig. 4. Actuarial patient survival including early operative deaths and related to life expectancy for this age group. Survival at 20 years was by 80%.

At surgery there was no evidence of thinning, overgrowth or calcification of the tissues.

The 5 valves classified as degenerative showed none of the features of primary tissue failure such as thinning, tearing, overgrowth or calcification and the consensus of opinion was that they probably represented healed or subclinical endocarditis. Proven endocarditis occurred in 11 patients (32%). Two were treated medically. Of the 9 reoperations, 2 were repaired quite simply and 7 needed replacement. There were no deaths in this group. Freedom from reoperation at 20 years was 85%. There have been no embolic episodes and a series of 96 postoperative clinical and catheter studies showed no evidence of progressive aortic regurgitation [1].

The right ventricular outflow tract has been routinely replaced with a pulmonary homograft [10] since 1986 and before that, an aortic homograft [19]. A small series of 14 patients had fascia lata or pericardial valve conduits and the majority of these stenosed and required replacement.

Application to children: over the past 20 years with growing confidence in the valve [6, 7] 34 children ranging from 3–6 years have been operated upon. There were 4 early and 4 late deaths included in the foregoing statistics. Survivors show no evidence of primary structural deterioration and all are in the NYHA functional class 1 without medication.

Histological examination of explanted valves confirm the presence of a full complement of living cells, preservation of the vulnerable endothelial cells and no evidence of calcification.

Discussion

The first autograft operation coincided with our early difficulties in achieving a competent homograft valve.
This plus the problems of the first septal artery made the learning curve (including early and late deaths) remarkably steep. Consequently the long term function of the autograft relates more accurately to the last 20 years of our experience during which time operative, late deaths and complications have been minimal. The pulmonary autograft incorporates all the functional advantages of the homograft with perfect design, structure and sterility without the need for damaging storage and preservation methods. The cusps are entirely non-antigenic and retain their full cellular complement.

The fact that the pulmonary cusp supports aortic diastolic pressure applied acutely and without evidence of progressive regurgitation attests to its strength and functional adaptability. The tissue slowly thickens almost imperceptibly to become indistinguishable from aortic cusp tissue. Sophisticated stress testing [8] confirms the superior tensile strength of the pulmonary cusps and almost certainly relates to the fact that during foetal development, the right sided pressures are at systemic level.

One difficulty in accepting the operation has been the belief that this represents a double valve procedure. However, homograft replacement of the right ventricular outflow is widely practised and with excellent results[11, 12]. Moreover, pulmonary dysfunction, if it occurs, presents virtually no risk or disability and is certainly not life-threatening. This is amply demonstrated in the many corrections of Fallot’s tetralogy where valve insufficiency is the reimplanted pulmonary autograft could grow nor- of Elkins [4] confirms this observation in children. In these patients, the autograft should be inserted as a free-standing root replacement to avoid any lateral compression and restriction to growth.

The prospect of growth and the retained viability together with an absence of calcification or degenerative change and coupled with earlier follow-up observations showing no evidence of progressive aortic regurgitation confirms our belief that this could be a permanent valve. Any functional deterioration would then be subject only to the normal aging processes.

References


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Discussion

Dr. C. A. Yankah (Berlin, FRG). I congratulate Mr. Ross on his pioneer work and his unique lecture on the state of the art regarding the pulmonary switch operation, which is heterotopic pulmonary autograft transplantation. I am indebted to Mr. Ross for the knowledge I have in this field of pulmonary autograft switch, and we are especially grateful to him for introducing this unique operative technique which, however, is still not popular but will be a challenge to many surgeons in the coming years, as was the case with arterial switch for d-TGA.

Mr. Ross has used this operative technique to circumvene the immunological problem that is encountered especially in young patients. He has demonstrated clinically, with the pulmonary autograft in the aortic position, that the aortic root is not a privileged site for the aortic valve alone.

The pulmonary switch operation is accomplished finally with concomitant pulmonary valve replacement and since this operation is a double valve operation, a meticulous implantation technique is very necessary to achieve patent valves and success. Your actuarial rate shows an 80% freedom from reoperation at 20 years, which is an impressive and encouraging result. Interestingly enough, the clinical findings of the autograft demonstrated concomitant growth with the native aortic ring, which suggests that children at an acceptable age, who need valve replacement, will benefit from this operation. In adults, we do not anticipate growth of their grafts, but instead long-term valve function.

Immune modulation by selective elimination of endothelial cells of fresh valves using cryopreservation techniques or tissue typing (ABO, HLA) of allovalvital valves, as has been shown in my earlier experiments, might solve the immunological problems in adult patients.

At this junction, I would like to ask you four questions. Is it justified to extend the indications to the adult age group since graft growth in this group of patients is not a major problem? If immune modulation is used, should a cryopreserved ABO-HLA-compatible allograft be generally accepted in the adult age group to achieve the same goal?

Do you have to do annuloplasty in cases such as tunnel-type left ventricular outflow tract obstructions? What are the results in this group of patients with respect to annular growth?

Which tricks do you have to manage cases with bicuspid valves?

Mr. Ross: Thank you, Dr. Yankah. You asked whether we were justified in using it in adults. We started off in young adults, but I think there is less justification for using a longer operating time in the older age group. The specific indication is in young patients, but we've used it in patients of 1 year of age and 60 years of age. In fact, it can be used in any age group, but its chief aim is to prevent repetitive operations and repeated dangers of anticoagulation and haemorrhage in the 30 or 40 year lifespan ahead of a patient.

As far as tunnel-type outflow tract reconstructions are concerned, of course we use the autograft as a root replacement after excision of the tunnel. We excise the root.

Dr. G. Ziemer (Hannover, FRG). I would like to add to the evidence that the pulmonary autograft will grow. To the best of our knowledge we have successfully operated on the only neonate to receive so far a pulmonary autograft.

A 12-day-old boy, 2.7 kg body weight, had a diagnosis of aortoventricular tunnel and critical aortic stenosis. He had undergone ineffective palliation on day 2 of life. With the aortic annulus severely hypoplastic and the tunnel wide open again, the patient required extensive aortic root replacement. The pulmonary valve and trunk were then replaced by an ABO-compatible 9-mm pulmonary homograft, conduit.

Now, two and a half years later, the pulmonary homograft has had to be replaced for anastomotic stenosis. The patient, however, is now five times heavier than at the time of the operation and the valve clearly grew. However, there is some regurgitation of the autograft valve. Before we can prove that this is a permanent valve replacement, however, the patient must be five times more than his current weight — still a long way to go.

Mr. Ross: I think it is very interesting that you've used it in a neonate and I think that might be the prime indication for the operation. I am not sure whether you used root replacement, free-standing root replacement, or the orthotopic insertion technique. I think if you are looking for growth, it must be done as a free-standing root so that there is no lateral restriction on the growth of the valve.

Incidentally, there has been very important work from Kawashima's unit in Osaka, Japan. He did autotransplantation of the pulmonary valve in puppies and showed definite growth while there was no growth in a control series of homografts.
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