Autologous Pulmonary Valve Replacement of the Diseased Aortic Valve

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SUMMARY
Living tissue has the potential to replace and reestablish its structure and, therefore, offers the best hope for permanent replacement of diseased cardiac valves. The autologous pulmonary valve has been used to replace the aortic valve in 97 patients since June 1967. An aortic homograft valve, and more recently an autologous conduit of fascia lata, containing a valvular structure, has been used to reconstruct the right ventricular outflow tract. Only four deaths have occurred among the last 50 patients, an 8% operative mortality. Histologic examination of valves, 1 and 13 mo after implantation, revealed normal architecture of cells and collagen fibers with normal living structures.

This initial experience has encouraged the authors to pursue this method of aortic valve replacement in selected cases.

Additional Indexing Words:
Living structure Biological tissue Autologous tissue

PULMONARY autograft replacement of the aortic valve offers all the characteristics inherent to the homograft valve and, in addition, the advantage of being a living tissue with the potential to replace and reestablish its structure. The clinical application of this procedure, initiated in June 1967, is the basis of this report.

Methods
Clinical Material
Ninety-seven patients have undergone aortic valve replacement with an autologous pulmonic valve up to March 1970. This procedure has been electively performed in young patients and only recently extended to include those in the sixth decade. The majority of the patients were in the third, fourth, and fifth decades. Forty-three patients (44.3%) were in functional class III, and 22 (22.7%) were in class IV prior to surgery.

The dominant lesion was aortic stenosis in 45 patients (46.4%) and regurgitation in 37 (38.1%). In the other 15, in addition to the aortic valve, one or more other valves were involved. A history of rheumatic fever or bacterial endocarditis or both was prevalent in 56% of the patients. Three patients had undergone a previous operation on the aortic valve; three others had a previous resection for a coarctation of the aorta.

Surgical Technic
Through a midline sternotomy the pericardial cavity is opened. Cannulation of the ascending aorta and right atrium is performed and normothermic, high flow, cardipulmonary bypass is instituted with a bubble oxygenator and hemodilution. The aortic valve is exposed through an oblique incision in the ascending aorta. Excision of the diseased valve (fig. 1A) is followed by cannulation and perfusion of both coronary arteries.

A transverse incision is made over the pulmonary artery just above the anterior commissure of the pulmonary valve; the artery is transected at this level. The valve is dissected down to the junction of the pulmonary artery and right ventricular muscle posteriorly; this is followed by anterior dissection and excision of the valve (fig. 1B). The pulmonary valve ring is

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usually the same size, or slightly larger, than the aortic annulus. It is markedly elastic and, in most cases, is suitable for replacement of the aortic valve.

In the majority of the patients, reconstruction of the right ventricular outflow tract is performed with a reconstituted aortic homograft (fig. 1C). A pulmonary valve homograft was used in six patients in our series, and a tube of autologous fascia lata with a tri-leaflet valve inside was placed in the last eight. Insertion of the graft for the pulmonary valve is accomplished with a running stitch of 4-0 Mersilene for the proximal and distal suture lines.

The pulmonary valve autograft is then prepared by excision of redundant muscle from the lower end of the cusps. It is inserted into the aortic annulus with two suture lines, as previously described. The lower suture line is placed with multiple interrupted sutures of 4-0 Mersilene after matching the pulmonary sinuses with those of the aortic annulus. Three commissural stitches are placed and the wall of the pulmonary artery is then tailored and scalloped around the coronary orifices. The upper suture line is placed with a running stitch of 4-0 Mersilene attaching the upper margin of the pulmonary valve to the wall of the aortic sinuses (fig. 1D).

The average bypass time for this procedure has been 2 hours.

Results

Mortality and Morbidity

Death occurring within 30 days of surgery has been considered as hospital mortality.
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Figure 2
Comparison of mortality figures for the first 47 patients operated on and the last 50 patients. Refinements in surgical technic and improvements in postoperative care have contributed to patient survival.

Fifteen (15.4%) of the 97 patients undergoing pulmonary autograft replacement of the aortic valve died in the hospital. Deaths were attributed to ventricular dysrhythmias, low cardiac output, neurologic damage, an error in respiratory management, and bleeding. Only four of the last 50 patients operated on died, a 8% operative mortality (fig. 2).

Refinements in the surgical technic and improvements in the postoperative care of these patients, especially electrical pacing, contributed to patient survival and to diminution of postoperative complications. The frequency and magnitude of postoperative complications are now comparable to those in patients undergoing prosthetic or homograft replacement of the aortic valve (table 1).

Follow-up
The status of the 82 patients surviving surgery has been ascertained. Systemic thromboembolism has not been encountered in spite of no anticoagulation therapy.

Seventy-seven (93.9%) were asymptomatic and had resumed normal activities. Normal heart sounds with a normal splitting of the second sound was present in 55 (67%) of the patients. A decrease in size of the cardiac silhouette has been noted 3 to 6 months after surgery in 80% of the patients. Hemodynamics are normal in those patients without murmurs who were studied postoperatively.

As with the homograft valve, the appearance of signs of aortic regurgitation following pulmonary autograft replacement has followed a definite pattern. Seventeen (20.7%) of 82 patients had a diastolic murmur at the time of discharge from the hospital; four more developed a murmur by 6 weeks after surgery, and at 5 mo a total of 26 patients had diastolic murmurs. One more patient developed a diastolic murmur after an episode of bacterial endocarditis 1½ years postoperatively.

The degree of aortic regurgitation in these 27 patients has been evaluated (fig. 3). The regurgitation was trivial in 19 (70.4%), being characterized only by the presence of a faint diastolic murmur. In three patients (11.1%) the regurgitation was mild; in addition to the diastolic murmur, they had a slightly jerky pulse, pulse-pressure less than one half the systolic pressure and, by angiography, a faint outlining of the left ventricle. In two (7.4%) the regurgitation was moderate with a diastolic murmur, a jerky or collapsing pulse, widening of the pulse-pressure and, by angiography, a rapid outlining of the left ventricle with elimination of the dye in 10 sec. Aortic regurgitation was severe in three patients with a frankly collapsing or water-

Table 1
Postoperative Complications of Autologous Pulmonary Valve Replacement of the Aortic Valve: June 1967 to March 1970

<table>
<thead>
<tr>
<th>Complication</th>
<th>Initial 47 patients</th>
<th>Last 50 patients</th>
<th>Total</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrhythmias</td>
<td>9</td>
<td>6</td>
<td>15</td>
<td>15.5</td>
</tr>
<tr>
<td>Persistent bleeding</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>5.2</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>4.1</td>
</tr>
<tr>
<td>Septicemia</td>
<td>2</td>
<td>1</td>
<td>3</td>
<td>3.1</td>
</tr>
<tr>
<td>Wound infection</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>3.1</td>
</tr>
<tr>
<td>Transient renal failure</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1.0</td>
</tr>
</tbody>
</table>

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hammer pulse and a persistence of dye in the left ventricle beyond 10 sec. These three patients have been considered to have failures of the autograft.

Failed Autografts

All three patients with autograft failures had severe aortic regurgitation immediately after surgery and two have undergone reoperation after 1 and 13 mo, respectively. The regurgitation in both instances was found to be due to malposition of the pulmonary valve autograft with a prolapse of the noncoronary cusp. The grafts were replaced with fascia lata valves. The leaflets of the excised autografts appeared thin, pliable, and intact with some cross striations. Histologic sections revealed normal architecture of cells and collagen fibers which indicated normal living structures.

The homograft valves in the right ventricular outflow tract were well incorporated with adjacent tissue. The outside of the grafts were covered with a fine layer of fibrous tissue.

The third patient had moderately severe aortic regurgitation and died suddenly while waiting for surgery.

Late Deaths

Three late deaths occurred, one 2 mo after surgery from an intractable pyocyanase infection in both lungs, a terminal result of renal failure, and peritoneal dialysis. One other patient died unexpectedly 4 mo after surgery, probably due to an arrhythmic episode. The other patient, aforementioned, died while awaiting reoperation for a failed pulmonary valve autograft. Thus, with a 3.1% late mortality, an overall mortality of 18.4% is realized among the 97 patients undergoing replacement of their aortic valve with an autologous pulmonic valve.

Discussion

Biologic valves, namely the homografts, offer a realistic alternative to prosthetic valves in the aortic position. There is concern, however, about their long-term durability. Current methods of sterilization and preservation of the homograft valve alter its structure. Some workers have collected the valve under sterile conditions to avoid the need of subsequent sterilization. These valves are fresh but not necessarily living, and, although the structure may be preserved, long-term evaluation is necessary to demonstrate its superiority over the conventional methods of homograft valve preparation. The limitations of this method regarding the availability and collection of sterile valves are obvious.

To overcome these problems the use of living autologous tissue for valve replacement has an immediate appeal. The patient's own pulmonary valve, which has the same design characteristics as the aortic valve, is a living autologous tissue, sterile and ready for implantation. Being an elastic structure, it adapts well to the aortic annulus and ensures excellent hemodynamics.

The deficiency in the outflow tract of the right ventricle is repaired by a homograft aortic valve and, although this has potential disadvantages, it has worked satisfactorily in cases of pulmonary atresia. The closing pressure of the valve is low thus reducing the risk of trauma. Late failure, should it occur, is unlikely to have serious consequences. More recently autologous fascia lata has been used to overcome this theoretical problem.
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References

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