Introduction. The Ross procedure has become established as an appropriate method for aortic valve replacement in children and young adults. There is controversy regarding the results of this surgical technique depending on whether the aortic valve disorder is congenital or acquired. The objective of this study was to analyze the outcome of this technique in different etiologies.

Patients and method. We analyzed 61 patients who underwent the Ross procedure between November 1997 and November 2001. Age range was 6 to 54 years, and 44 patients (72%) were male. The mean duration of follow-up was 15.6 (10.6) months. The aortic valve lesion was stenosis in 17 patients, regurgitation in 22 and both in 22. The patients were divided into two groups: etiology was congenital in group I (40 patients) and acquired in group II (21 patients: 14 rheumatic, 2 degenerative, 2 endocarditis, and 3 other).

Results. Pre-intervention data showed significant differences in age, functional class and percentage of patients with previous cardiac surgery. In the last follow-up examination, autograft gradient and homograft gradient were similar in both groups. Diastolic and systolic diameters and left ventricle ejection fraction were normal in both groups, without differences. The patients needed stent implantation in the homograft in group I; 2 patients in group II underwent reoperation because of severe autograft dysfunction. There were no statistically significant differences between groups.

Conclusion. Short-term morbidity and mortality associated with the Ross procedure are low in patients with either congenital or acquired aortic valvulopathy.

Key words: Ross procedure. Etiology. Follow-up.
INTRODUCTION

In 1967, Donald Ross developed native pulmonary valve autograft for aortic valve replacement, in turn replacing the pulmonary valve with a homograft.1 The Ross procedure has since become one of the most successful techniques for aortic valve replacement in certain groups of patients, whether young adults or children.2,3 Pulmonary autograft uses live tissue from the patient’s own pulmonary valve. This means the valve grows as the patient grows and can function indefinitely.4 Moreover, patients do not need permanent anticoagulation. Pulmonary autograft permits central laminar flow5 and improves hemodynamic performance so that previous dilation and ventricular hypertrophy recede in most patients.6

The original indication for pulmonary autograft was treatment of aortic valve disease not susceptible to repair in young patients with life expectancy ≥20 years. This has gradually been extended to other indications.7,8

Contraindications for this procedure include coexistence of associated coronary disease, moderate or severe multiple valve pathology, connective tissue disease (genetic or autoimmune) and aortic and pulmonary annulus mismatch.9,10

However, worse outcomes have been described in patients with congenital characteristics6,11,12 such as bicuspid aortic valve,13,14 which is the most frequent congenital aortic valve condition. This is characterized by changes in the connective tissue, particularly elastic fiber fragmentation, which can extend to the ascending aorta with cystic necrosis of the media, and changes in smooth muscle cell orientation.15 It is also believed to affect the pulmonary autograft and can cause progressive neoaortic insufficiency.16-18 Although more recent studies have not supported this theory,19 worse outcomes have been described in patients with rheumatic etiology or with inflammatory disease of the aortic root as the underlying process, both of which may affect the live tissue autograft and cause postoperative progressive neoaortic insufficiency.20,21

The objective of this study is to compare a series of Ross procedure patients with congenital and acquired etiologies in a short-term follow-up at our hospital in Córdoba (Spain).

PATIENTS AND METHODS

We enrolled all patients undergoing Ross procedure in our hospital from November 1997 thru November 2001. Patients were divided into 2 groups by etiology: congenital (group I) or acquired (group II). The Ross procedure was performed on 61 patients aged 6-54 years: 44 (72%) male and 17 (28%) female. Congenital etiologies identified in 40 (66%) patients were: rheumatic (14, 23%), endocarditis (2, 3%), degenerative (2, 3%), and other (3, 5%). Twelve patients (20%) had bicuspid aortic valves. The valvular lesions were: aortic stenosis (28%), insufficiency (36%), and stenosis and insufficiency (36%). Four patients (6%) had associated coarctation of the aorta, 5 (8%) had subaortic membrane, and I had Shone syndrome.

During preintervention clinical interviews we recorded data on age, gender, previous history of cardiac surgery and New York Heart Association (NYHA) functional class: 23% were in NYHA class I, 43% in class II, and 34% in class III. Twenty patients (33%) had previously undergone operations, 12 (20%) with surgery and 8 (13%) percutaneous surgery. General data on the population appear in Table 1.

Surgical Technique

All operations were elective. Extracorporeal circulation with bicaval cannulation of the aorta was used in all patients. We evaluated the native pulmonary valve by transverse pulmonary arteriotomy and resected the diseased aortic valve and complete aortic root conserving right and left coronary artery buttons either freestanding or attached to the aortic wall. Later, we resected the right ventricular outflow tract pulmonary autograft (conserving the left anterior descending artery first septal branch) and implanting it in aortic position as free root tied with interrupted sutures on an annulus of autologous pericardium, anastomosing the coronary buttons to the autograft with running suture, and the autograft to the ascending aorta. We performed aortoplasty in patients with ascending aorta dilation. We carried out right ventricular outflow tract reconstruction with a cryopreserved pulmonary homograft with distal and proximal running sutures.

<table>
<thead>
<tr>
<th>TABLE 1. General Preoperative Characteristics of Study Sample</th>
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<tbody>
<tr>
<td><strong>Variable</strong></td>
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</tbody>
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532 Rev Esp Cardiol 2004;57(8):531-7
Echocardiography Protocol

Echocardiography was performed with Acuson Sequoia® equipment with M mode, 2D mode, second harmonic imaging, color Doppler, continuous wave Doppler, and pulsed Doppler. Echocardiographic data were collected pre- and intraoperatively, at 6-, 12-, 24-, 36-, and 48-month follow-ups and on presentation of clinical events (see appendices).

Clinical Follow-up

Patients were invited to attend clinical re-evaluation including NYHA functional status and occurrence of events at 6, 12, 24, 36, and 48 months and on presentation of clinical events. These were defined as all-cause death (including perioperative death), need for reoperation due to autograft dysfunction, need for stent implantation due to homograft stenosis or graft endocarditis. Mean follow-up was 15.6±10.6 months.

Chi-square was used to compare qualitative variables, Student t test to compare quantitative variables, and Kaplan-Meier to construct survival curves.

RESULTS

Average age of patients with congenital etiology (group I) was 24±11 years and 67.5% were males. Average age of patients with acquired etiology (group II) was 40±7 years and 81% were males. Table 2 presents preoperative data by etiology.

Age was significantly lower in group I (P<.001); differences in gender between groups were statistically nonsignificant. Group I patients had fewer preoperative symptoms but the difference was statistically nonsignificant (NYHA functional class I: group I 30.8% vs group II 5.6%; P=.03). Group I patients presented higher incidence of previous cardiac surgery (50% vs 5%; P=.001). Mean left ventricular ejection fraction was normal in both groups (70.7% vs 61.8%). Differences in transaortic gradient were statistically nonsignificant: left ventricular systolic diameter (19.3 mm/m² vs 20.2 mm/m²); interventricular septum (6.1 mm/m² vs 5.3 mm/m²); left ventricular posterior wall (6.6 mm/m² vs 5.9 mm/m²). Similarly, at follow-up ejection fraction was normal and similar in both groups (64.9% vs 60.2%). Annulus diameters produced similar data for autograft (24.8 mm vs 26.2 mm) and homograft (18.8 mm vs 20.7 mm). At final follow-up, differences between groups for peak autograft gradient (7.8 mm Hg vs 7.2 mm Hg), mean autograft gradient (4.4 mm Hg vs 4.1 mm Hg), and peak homograft gradient (19.9 mm Hg vs 18.1 mm Hg) were statistically nonsignificant. Final follow-up data on seriousness of autograft insufficiency (without insufficiency or with mild insufficiency: 93.4% vs 84.3%), and homograft insufficiency (without insufficiency or with mild insufficiency: 100% in both groups) were statistically nonsignificant (Figure 1).

At final follow-up, body surface area adjusted left ventricular dimensions were normal in both groups with statistically nonsignificant differences: left ventricular diastolic diameter (30.7 mm/m² vs 30.3 mm/m²); left ventricular systolic diameter (19.3 mm/m² vs 20.2 mm/m²); interventricular septum (6.1 mm/m² vs 5.3 mm/m²); left ventricular posterior wall (6.6 mm/m² vs 5.9 mm/m²). Similarly, at follow-up, differences between groups for peak autograft gradient (7.8 mm Hg vs 7.2 mm Hg), mean autograft gradient (4.4 mm Hg vs 4.1 mm Hg), and peak homograft gradient (19.9 mm Hg vs 18.1 mm Hg) were statistically nonsignificant. At final follow-up, NYHA functional class showed no statistically significant differences between groups and the majority of patients in both groups were asymptomatic (Figure 2). In group I, 78.6% were in class I, 17.9% in class II, and 3.5% in class III; in group II 92.3% were in class I and 7.7% in class II.

One patient died in hospital (1.6%) (group I). No patient died during follow-up and global final follow-up mortality was 1.6%. Comparison of the total or individual number of “events” did not differ between groups (Table 4). 4 (10%) events in group I (1 patient

TABLE 2. Patient Preoperative Characteristics by Etiology*

<table>
<thead>
<tr>
<th></th>
<th>Congenital</th>
<th>Acquired</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>24±11</td>
<td>40±7</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Male gender, %</td>
<td>67.5</td>
<td>81</td>
<td>NS</td>
</tr>
<tr>
<td>NYHA functional class</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>30.8%</td>
<td>5.6%</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>46.2%</td>
<td>33.3%</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>23.0%</td>
<td>61.1%</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>0%</td>
<td>0%</td>
<td>.03</td>
</tr>
<tr>
<td>Previous surgery</td>
<td>50%</td>
<td>5%</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Type of aortic lesion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>30.3%</td>
<td>6.3%</td>
<td></td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>33.3%</td>
<td>50%</td>
<td></td>
</tr>
<tr>
<td>Combined aortic lesion</td>
<td>36.4%</td>
<td>43.7%</td>
<td>NS</td>
</tr>
</tbody>
</table>

*NYHA indicates New York Heart Association.
Table 3. Comparison of Echocardiographic Data Between Groups at Final Follow-up*

<table>
<thead>
<tr>
<th></th>
<th>Congenital</th>
<th>Acquired</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body surface area adjusted LV dimensions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV diastolic diameter, mm/m²</td>
<td>30.7±6</td>
<td>30.3±5</td>
<td>NS</td>
</tr>
<tr>
<td>LV systolic diameter, mm/m²</td>
<td>19.3±4</td>
<td>20.2±4</td>
<td>NS</td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>64.9±12</td>
<td>60.2±7</td>
<td>NS</td>
</tr>
<tr>
<td>Autograft annulus diameter, mm</td>
<td>24.8±5</td>
<td>26.2±5</td>
<td>NS</td>
</tr>
<tr>
<td>Homograft annulus diameter, mm</td>
<td>18.8±3</td>
<td>20.7±4</td>
<td>NS</td>
</tr>
<tr>
<td>Peak autograft gradient, mm Hg</td>
<td>7.8±6</td>
<td>7.2±6</td>
<td>NS</td>
</tr>
<tr>
<td>Mean autograft gradient, mm Hg</td>
<td>4.4±3</td>
<td>4.1±3</td>
<td>NS</td>
</tr>
<tr>
<td>Peak homograft gradient, mm Hg</td>
<td>19.9±17</td>
<td>18.1±14</td>
<td>NS</td>
</tr>
<tr>
<td>Autograft insufficiency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No insufficiency, %</td>
<td>16.7</td>
<td>21.1</td>
<td></td>
</tr>
<tr>
<td>Mild insufficiency, %</td>
<td>76.7</td>
<td>63.2</td>
<td></td>
</tr>
<tr>
<td>Moderate/severe insufficiency, %</td>
<td>6.6</td>
<td>15.7</td>
<td>NS</td>
</tr>
<tr>
<td>Homograft insufficiency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No insufficiency, %</td>
<td>74.1</td>
<td>87.5</td>
<td></td>
</tr>
<tr>
<td>Mild insufficiency, %</td>
<td>25.9</td>
<td>12.5</td>
<td></td>
</tr>
<tr>
<td>Moderate/severe insufficiency, %</td>
<td>0</td>
<td>0</td>
<td>NS</td>
</tr>
</tbody>
</table>

*LV indicates left ventricle.
tified in those with bicuspid aortic valve\textsuperscript{15,16} due to
degenaration of the ascending aorta and secondary
autograft insufficiency.

The present series distinguished between patients
with congenital and acquired aortic pathologies be-
cause data in the literature are contradictory about re-

sults in these groups. In our evaluation of pre- and
postoperative data, echocardiography provides mea-
sures of left and right ventricular size and function,
valve annulus or graft annulus size, graft competence
and transvalvular gradients. Echocardiography also
identifies complications such as endocarditis and can
indicate need for reoperation or stent implantation due
to homograft stenosis. Consequently, echocardiogra-
phy is as essential to pre- and postoperative study of
Ross procedure patients\textsuperscript{18} as the clinical history and
clinical follow-up.

The majority of patients in both groups were in
NYHA class I at final follow-up. This corresponds to
other international series in which patients show low
follow-up morbidity.\textsuperscript{5}

Although the Ross procedure is technically more
complex, inhospital mortality was 1.6%. This is very
low by comparison with other aortic valve replace-
ment procedures that have inhospital mortality rates
of 10%.\textsuperscript{19} Events (death, need for surgery, need for
stent implantation or endocarditis) are equally fre-

\begin{table}[h]
\centering
\caption{Comparison of Total and Individual Events in the 2 Groups (Congenital and Acquired Etiology)}
\begin{tabular}{lccc}
\hline
 & Congenital & Acquired & \textit{P} \\
\hline
Total events & 4 (10\%) & 2 (9.5\%) & NS \\
Death & 1 & 0 & NS \\
Autograft surgery & 0 & 2 & NS \\
Autograft endocarditis & 1 & 0 & NS \\
Stent in homograft & 2 & 0 & NS \\
\hline
\end{tabular}
\end{table}
quent in the two groups: The only patient who died was in the congenital etiology group, had previously undergone surgery and had a complex congenital heart condition (Shone syndrome). This patient’s death may have been closely related to the associated condition than the etiology of the valve lesion. Patients with congenital aortic valve disease were younger and this may explain the concentration of stent implants due to homograft stenosis in this group, even though differences were statistically nonsignificant. We found no cases of homograft endocarditis. The one case of late autograft endocarditis occurred in a patient with congenital etiology, who presented moderate-severe autograft insufficiency. Again, differences between the two groups were statistically nonsignificant.

The need for reoperation of the autograft seems to have been concentrated in the group of patients with acquired etiology, although the difference was statistically nonsignificant. Earlier studies report development of progressive neo-aortic insufficiency in patients with rheumatic etiology. In our series, 2 patients with rheumatic etiology required autograft surgery for severe aortic insufficiency. These were two of the earliest patients in the series and technical inadequacies of the procedure may have been the cause as autograft reinforcement with pericardial annulus was not used and dysfunction occurred early in the first month. We have had no cases of severe aortic insufficiency in later operations. However, rheumatic conditions are not studied by us as short-term autograft dysfunction has not occurred in a patient with congenital etiology, who had previously undergone surgery and had a complex congenital heart condition (Shone syndrome). This patient’s death may have been closely related to the associated condition than the etiology of the valve lesion. Patients with congenital aortic valve disease were younger and this may explain the concentration of stent implants due to homograft stenosis in this group, even though differences were statistically nonsignificant. We found no cases of homograft endocarditis. The one case of late autograft endocarditis occurred in a patient with congenital etiology, who presented moderate-severe autograft insufficiency. Again, differences between the two groups were statistically nonsignificant.

APPENDIX. Echocardiographic Parameters Measured

1. Preoperation
   - Type of aortic lesion (stenosis, insufficiency, combined lesion)
   - Presence of associated disease.
   - LVEF calculated by Teichholz’s method in M mode
   - Body surface area adjusted size of left ventricle, in M mode
   - LV diastolic diameter
   - LV systolic diameter
   - IVS
   - LVPW
   - Pulmonary annulus diameter in 2D, parasternal projection of large vessels
   - Aortic annulus diameter in 2D, parasternal long axis projection
   - Peak and mean transaortic gradients
   - Degree of aortic insufficiency
   - Degree of autograft insufficiency
   - LV function
   - Degree of autograft insufficiency of RV outflow tract
   - 3. Echocardiographic follow-up (at 6, 12, 24, 36, and 48 months or at occurrence of any event)
      - LVEF in M mode
      - Body surface area adjusted LV dimensions, diastolic diameter in M mode
      - LV systolic diameter
      - IVS
      - LVPW
      - Peak and mean autograft gradients in aortic position
      - Maximum homograft gradient in pulmonary position
      - Degree of autograft insufficiency in aortic position
      - Degree of homograft insufficiency in pulmonary position

LVEF indicates left ventricular ejection fraction; LVPW, thickness of left ventricular posterior wall; IVS, thickness of interventricular septum; LV, left ventricle; “**” using continuous Doppler and modified Bernoulli AP formula (mm Hg)=4v² (m/s); “*” using semi-quantitative color Doppler (without mild, moderate or severe insufficiency).

We conclude that hemodynamic performance and short-term evolution of patients who undergo the Ross procedure for aortic valve disorders are similar whether patients have congenital or acquired conditions. Moreover, in patients with congenital etiology whether the aortic valve is bicuspid or tricuspid has no apparent influence on these parameters. Consequently, unless contraindicated, the procedure seems adequate in aortic valve disease of any etiology.

REFERENCES


