Prosthetic Aortic Stenosis
A Method to Prevent Its Occurrence by Measurement of Aortic Size from Preoperative Aortogram

By RONALD P. SENINGEN, M.D., BERNADE H. BULKLEY, M.D., AND WILLIAM C. ROBERTS, M.D.

SUMMARY

A cause of early death after aortic valve replacement with a caged-ball prosthesis is obstruction to left ventricular outflow because the prosthesis is too large for the aortic root. Of 68 patients dying within two months of aortic valve replacement, death in ten, each of whom had had intractable low cardiac output after operation, was attributed at necropsy to prosthetic aortic stenosis, despite the use of small sized (8A Starr-Edwards) prostheses in seven of them. The diameters of the aorta at the sinotubular junction, determined from the preoperative cineangiograms, in the seven patients with prosthetic stenosis were < 30 mm in all. Poppet clearances, defined as the differences between poppet and aortic root diameters, ranged from 4 to 12 mm (avg. 9). In contrast, the diameters of the aortas at the sinotubular junctions in eight control patients (unobstructed prosthetic aortic valves and early death from other causes) were > 30 in all but one, and the poppet clearances ranged from 12 to 19 mm (avg. 15). Thus, prosthetic aortic stenosis is likely to develop after aortic valve replacement with rigid-framed caged ball valves if the preoperative aortograms disclose aortic diameters at the sinotubular junctions to be < 30 mm. In such patients, either the aorta must be widened for a caged-ball prosthesis or a central flow valve must be used.

Additional Indexing Words:
Aortic valve
Cardiac valve replacement
Cardiac operation

WHENEVER THE MITRAL or aortic valve is replaced with a caged-ball prosthesis, some degree of stenosis is nearly always produced by the prosthesis.1-5 The obstruction is usually relatively mild, but on occasion the degree of stenosis is considerable.1-5 Anatomic features of fatal prosthetic mitral and prosthetic aortic stenosis have been described6-10 but fatal prosthetic stenosis has not been documented hemodynamically in the early postoperative period. If the prosthetic stenosis is extremely severe, cardiac function may not be restored in the operating room after valve replacement. Also, cardiac catheterization is infrequently performed during the first few days after operation. As a consequence, diagnosis of fatal prosthetic obstruction has been made almost exclusively11 at necropsy.

To help predict and perhaps prevent its occurrence, the sizes of the aortic roots measured by preoperative angiogram were compared to the sizes of the implanted prosthetic aortic poppets in patients with necropsy-diagnosed prosthetic aortic obstruction, and these measurements were compared to those in necropsy patients with replaced aortic valves but without prosthetic obstruction.

Patients Studied and Methods

Of 68 patients dying within two months of aortic valve replacement, death in ten was attributed at necropsy to prosthetic stenosis. Of the 10, seven who had preoperative aortic cineangiograms form the basis of this report (tables 1 and 2). Preoperatively, 5 patients had pure aortic regurgitation and 2 had aortic stenosis. Death occurred in the operating room in 1 patient, and in 6, from 1 to 8 days (avg. 4) after valve replacement. Size 8A to 11A (avg. 8.6) Starr-Edwards prostheses were used. In each of the latter 6 patients, death was attributed to the low cardiac output syndrome.* At necropsy, the prosthesis in each appeared to be too large for the aorta. At least 2 of the 3 struts of the cage came into contact with the wall of aorta which protruded into the cage and prevented adequate movement of the poppet (figs. 1 and 2).

*All patients had at one time direct systemic systolic pressures of < 90 mm Hg. Each also had evidence of underperfusion of at least one organ system: brain (disorientation or seizures); bowel (pain or bleeding); kidney (oliguria); lung (alveolar infiltrates = alveolar hemorrhage [shock lung]); liver (hyperbilirubinemia); and skin (peripheral cyanosis or coldness). The hypotension and organ-perfusion inadequacy responded poorly or not at all to therapy, including vasopressors. These signs of low organ perfusion were confirmed by necropsy examination in all patients.
Table 1

Data in Patients Studied

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (yrs)</th>
<th>MV/LV (PSG)</th>
<th>LV/LA (MM)</th>
<th>AR</th>
<th>MR</th>
<th>Internal Operation</th>
<th>Sinus Diameter</th>
<th>Diameter Index</th>
<th>Clearance Index</th>
</tr>
</thead>
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<td>1</td>
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<td>2-20</td>
<td>1-16</td>
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</tbody>
</table>

Of the remaining 58 necropsy patients who died < 2 months after aortic valve replacement, 8 were selected as controls on the basis of their having measurable aortic root cineangiograms (i.e., grids present on the frames), similar preoperative lesions and similar sized prostheses (Starr-Edwards) as those in the 7 patients with prosthetic stenosis. Preoperatively, 5 patients had pure aortic regurgitation, and 3 had aortic stenosis. Death occurred in the operating room in 3 patients and from 2 to 56 days (avg. 33) after valve replacement in the other 5 patients. Size 8A to 11A prostheses (avg. 8.7) were utilized. Death was due to clinical mishaps in 4 patients, unexplained low cardiac output in 2, unexplained sudden death in 1, and non-cardiac causes in 1. At necropsy, the aortic poppets moved freely in the cages and never did more than 1 of the 3 struts of the prosthesis contact the aortic wall (fig. 3).

The hearts in the 7 patients with prosthetic stenosis weighed 310 to 1040 g (avg. 670), and those in the 5 controls, 500 to 1100 g (avg. 650). None of the 15 patients had had the Marfan or Marfan-like syndrome, and none had had a portion of ascending aorta resected.

Cinecineangiogram Evaluation

The diameters of the aortic roots were measured from preoperative cinecineangiograms, corrected for geometric magnification, in all 15 patients. The internal diameter of each aorta was measured at the sinotubular junction (the most proximal portions of the tubular aortas or the most cephalad extensions of the sinuses of Valsalva), and at a level 2 cm above the sinotubular junction (fig. 4). Both measurements were taken from the same cine frame at maximal systolic aortic distention. These measurements were performed without prior knowledge of the clinical or necropsy findings. The diameters of the aortic prosthetic poppets (supplied by Edwards’ Laboratories) were compared to the diameters of the aortas and the differences (maximal clearances) were recorded (tables 1 and 2).

Table 2

Prosthetic Aortic Stenosis

<table>
<thead>
<tr>
<th>No. of Patients</th>
<th>Diameter (mm) of aorta at S-T junction</th>
<th>Diameter (mm) of aorta 2 cm above S-T junction</th>
<th>Size of prostheosis (Starr-Edwards)</th>
<th>Diameter (mm) of poppets utilized</th>
<th>Maximal clearance (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>7</td>
<td>20-29</td>
<td>20-29</td>
<td>8A-11A</td>
<td>15-18</td>
</tr>
<tr>
<td>II</td>
<td>8</td>
<td>28-34</td>
<td>29-39</td>
<td>8A-11A</td>
<td>15-18</td>
</tr>
</tbody>
</table>

Comments

Malfunction of an implanted prosthesis is a common cause of death in the early postoperative period after cardiac valve replacement. Of 133 patients dying early (<2 mo) after replacement of one or more cardiac valves with caged ball prostheses (mainly Starr-Edwards), 54% died from prosthetic dysfunction. Of the 68 necropsy patients with aortic prostheses, death in 10 resulted because the prosthesis was too large for the aorta into which it had been inserted. All 10 patients with prosthetic aortic stenosis had evidence of inadequate cardiac output postoperatively. Prosthetic stenosis in all 10 patients was diagnosed only at necropsy.

Studies in the patients with prosthetic stenosis demonstrated that measurement of aortic size from preoperative aortograms can predict those patients who are at high risk of developing prosthetic aortic stenosis. The critical diameter of the ascending aorta at the sinotubular junction is about 30 mm. This measurement was <30 mm (avg. 25) in each of the 7 patients with fatal prosthetic aortic stenosis but in only 1 of the 8 control patients with normally functioning aortic prostheses of similar size (avg. 31).
Prosthetic aortic stenosis in patient 4. The ascending aorta at 2 cm above (a), at 1 cm above (b), and at the sinotubular junction itself (c).

Moreover, prosthetic stenosis occurred in 5 of the 7 patients despite the use of small sized (8A) prostheses. Thus, some aortas are simply not large enough for even these small rigid-framed caged ball prostheses. Aortic diameters measured 2 cm above the sinotubular junctions did not correlate with the development of prosthetic aortic stenosis. Hence, at operation the aorta above the sinuses of Valsalva may appear of adequate size to readily accommodate a rigid-framed prosthesis, but at the same time, the size of aorta at the sinotubular junction may be too small to accept the prosthesis.

Thus, measurement of the diameter of aorta from the preoperative cineaortogram at the level of the sinotubular junction can be used to predict those patients in whom prosthetic stenosis is likely to develop after aortic valve replacement with a rigid-framed caged-ball prosthesis. Those patients with aortic diameters < 30 mm are at high risk, and in such patients widening of the aorta should be considered if caged-ball prostheses are implanted or central flow type prostheses should be used. Knowledge of this measurement recently proved helpful in a patient undergoing aortic valve replacement. The diameter of aorta at the sinotubular junction measured 29 mm on the preoperative cineaortogram. After valve replace-

Figure 2

Cineaortogram in the right anterior oblique view with corresponding line drawing. Measurement A represents the internal aortic diameter at the junction of the sinus of Valsalva and tubular aorta (sinotubular junction); measurement B, the internal aortic diameter 2 cm above the sinotubular junction.

Figure 3

Two examples of non-stenotic aortic prostheses. The aortic walls do not contact the poppets or the struts of the prostheses, and poppet movement is unimpeded.

References


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