Improved Results in 336 Patients with the Isolated Mitral Beall Valve Replacement

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In the four year study of 336 mitral Beall valve replacements there was an early operative mortality of 8.6 percent (29 patients). The late mortality was 7.4 percent (25 patients). Only five (1.5 percent) of these died as a result of thromboembolism from the prosthesis. Withdrawal of anticoagulants may have been a factor in causing clotting on the prosthesis in two of these patients. There were 26 patients (6 percent) who developed nonfatal postoperative emboli, mostly transient in nature. This low embolism incidence may be due to a lack of exposed valve metal. Residual valvular leak occurred in 18 patients but only in five was it severe enough to warrant reoperation. The late results in the 273 survivors were gratifying; 92 percent (252) were in excellent or good condition at the end of the study period. Most of these survivors resumed a normal active life.

During the last decade there has been a gradual improvement in the results associated with prosthetic valvular replacement. This has been partly due to improvements in the design and development of valvular prostheses which have resulted in better hemodynamic results4 and lower incidence of postoperative complications. In particular, thromboembolism, one of the main complications of valve replacement, has decreased drastically since the introduction of the completely cloth-covered prosthesis58. This paper analyzes our postoperative results with the use of the Dacron-cloth covered Teflon-disc mitral prosthesis of Beall68 with special attention to the incidence of emboli, leaks, and infection.

CLINICAL MATERIALS

During the period from May 1967 to June 1971, 336 patients have undergone isolated replacement to their mitral valves. Two have required rereplacement with another Beall valve prosthesis. Follow-up information, as of September 1971, was available in 327 patients (97 percent). Nine

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![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21546/ on 03/31/2017)
IMPROVED RESULTS WITH ISOLATED MITRAL BEALL VALVE

Table 1—Indications for Mitral Valve Replacement.

<table>
<thead>
<tr>
<th>Findings at Operation</th>
<th>No.</th>
<th>Patients</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combined stenosis and regurgitation</td>
<td>200</td>
<td>50.5</td>
<td></td>
</tr>
<tr>
<td>Isolated stenosis</td>
<td>99</td>
<td>20.5</td>
<td></td>
</tr>
<tr>
<td>Isolated regurgitation</td>
<td>50</td>
<td>14.9</td>
<td></td>
</tr>
<tr>
<td>Previous mitral prosthesis</td>
<td>17</td>
<td>4.1</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>336</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ments analyzed. Eighteen (5.3 percent) of these had permanent residual neurologic damage from cerebral embolus at the time of the initial mitral surgery. Four had an embolus lodged in the femoral artery necessitating femoral embolectomy.

The clinical diagnosis of mitral valve disease was confirmed by cardiac catheterization in the majority of cases. Coronary angiography was also frequently utilized to assess the presence of coronary artery disease.

There were 175 (52 percent) Class II and 116 (34 percent) Class III patients in cardiac function according to the New York Heart Association Classification. Twenty (6 percent) and 25 (7.5 percent) were Class I and IV respectively. All exhibited cardiomegaly to various degrees upon clinical and laboratory examination.

In 334 (90.5 percent) the valvular lesions appeared to be of rheumatic etiology. There were two patients with pure mitral regurgitation caused by changes described as typical of "flabby" mitral valve. In four patients there were lesions of old superimposed subacute bacterial endocarditis.

Replacement of the mitral valve with a prosthesis was carried out only when the pathologic findings were such that no conservative procedure could restore adequate function.

At operation (Table 1) the most common indication for valve replacement was the presence of combined mitral regurgitation and stenosis in 200 patients (59.5 percent), mitral regurgitation as a predominant lesion in 30 patients (8.5 percent), and pure mitral stenosis, usually calcific, in 69 patients (20.5 percent).

Mallfunction or embolic complications of a previous mitral prosthesis was the indication in 17 patients (5.1 percent). In addition to these patients with previous mitral prostheses there were 63 (19.2 percent) who had had a mitral commissurotomy in the past, most of them as a closed procedure, 10 to 15 years previously.

Our surgical approach to the mitral valve has been previously described. Adequate exposure of the valve is obtained through a left posterolateral thoracotomy approach in patients who did not have previous operations on the left chest. If there has been a previous left thoracotomy, either a midternal incision in the supine position or an intercostal approach through the right side in the lateral position is utilized.

Postoperative anticoagulation was achieved within one week of surgery, using warfarin orally. An attempt was made thereafter to maintain the prothrombin time between 25 and 30 seconds.

RESULTS

Hospital Mortality

There were 29 hospital deaths (8.6 percent) up to one month postoperation (Table 2). The most common cause of early death was heart failure in seven cases and myocardial infarction also in seven, usually in the operating room or in the intensive care unit. There were five cases of postoperative

Table 2—Analysis of Operative Deaths.

<table>
<thead>
<tr>
<th>Cause of Death</th>
<th>No.</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mural arioration</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Acute heart failure</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>Postoperative bleeding</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Ventricular failure</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Cerebral anoxia or air embolism</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Postoperative shock</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Septicemia</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Mesenteric thrombosis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Clotted prosthesis</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>29 (8.6%)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 2. Operative mortality according to age in 336 patients. Numbers within the columns represent number of deaths per number of patients.

Figure 3. Operative mortality according to functional class in 336 patients. Numbers within the columns represent number of deaths per number of patients.
hemorrhage within the first 24 hours following surgery. Four of these patients were reexplored in an attempt to control the bleeding and the fifth developed massive upper gastrointestinal tract hemorrhage.

Cardiac irritability manifested by a variety of arrhythmias that led to uncontrolled ventricular fibrillation occurred in three cases. There were three patients who never regained consciousness and died within five days, presumably from air embolism or cerebral anoxia.

The remaining causes of the early mortality included one case of each of the following: shock, septicemia, mesenteric thrombosis, and thrombosis of the prosthesis. This last patient was the only early mortality due to thromboembolism (0.3 percent). He was a 55-year-old man who developed paralytic ileus and mild gastrointestinal hemorrhage postoperatively. Anticoagulants were therefore discontinued. Three weeks following the operation he developed refractory congestive heart failure. At autopsy a clotted prosthesis was found.

Correlation of the hospital mortality with age, functional class, and pathology found at operation and previous operative procedure showed that death was significantly more frequent in the 51-60 and 60-70 age groups (Fig 2). It was also higher in the patients with Class III (11.2 percent) and Class IV (20 percent) of the functional classification (Fig 3). Those patients with mitral stenosis and regurgitation had a 6.5 percent mortality as opposed to 14 percent in those with predominant mitral regurgitation (Table 3). In those patients who did not have previous operations upon the heart, it was significantly lower (Fig 5). There were 248 primary cases with 17 deaths or 6.8 percent mortality. This is in contrast with those who had had previous operations, 12 deaths (14 percent) among this group of 88 patients.

Late Mortality (Table 3)

During the follow-up period extending to September of 1971 there were 25 late deaths (7.4 percent). Four of these were due to thromboembolic complications of the prosthesis (Table 4). Three of these were late cerebrovascular accidents based on clinical information since they died at home without warning at ten, seven and one months postoperation. Autopsy was not performed. The fourth late mortality was a 58-year-old woman.

Table 3—Analysis of Late Deaths.

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>7</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>7</td>
</tr>
<tr>
<td>Irreversible anemia</td>
<td>1</td>
</tr>
<tr>
<td>*Paravalvular leak—reoperation</td>
<td>1</td>
</tr>
<tr>
<td>Accidental death</td>
<td>1</td>
</tr>
<tr>
<td>Uremia</td>
<td>1</td>
</tr>
<tr>
<td>Hepatitis</td>
<td>1</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>1</td>
</tr>
<tr>
<td>Carcinoma of the lung</td>
<td>1</td>
</tr>
<tr>
<td>Cerebrovascular accident</td>
<td>3</td>
</tr>
<tr>
<td>Clotted prosthesis</td>
<td>1</td>
</tr>
</tbody>
</table>

Total: 25 (7.4%)

*Death due to cerebral anoxia, possibly from air embolism.
The remaining causes of late mortality were related to the progression of the underlying myocardial disease leading to congestive heart failure in seven patients, and acute myocardial infarction in another seven patients. One patient developed cerebral anoxia at reoperation for repair of a paravalvular leak. Other late deaths unrelated to the prosthesis included accidental death, uremia, hepatitis, pneumonia and carcinoma of the lung—one of each.

**Postoperative Morbidity**

There were 273 (84 percent) long-term survivors to September 1971.

**Thromboembolism (Table 4)**

There were 20 patients (6 percent) who developed a variety of neurologic complications attributed to emboli from the prosthesis. Two of these were within the first month after the operation, the remaining 18 between 3 to 27 months postoperatively.

**The Early Emboli**

One patient, age 57, had his original Beall valve prosthesis removed after two years because of defective closure of the disc. The prosthesis was found well covered with pseudointima and the disc showed slight wearing and notching. Two weeks after his second operation he developed a mild left hemiparesis. He is now 15 months after surgery and much improved. The other case occurred in a 63-year-old man who suddenly developed aphasia two weeks postoperation. This episode was transient and he has recovered completely.

**The Late Emboli**

All of the 18 patients who developed late embolic episodes of nonfatal nature recovered completely.

**Table 5—Incidence of Paravalvar Leak in 273 Survivors.**

<table>
<thead>
<tr>
<th>No. Patients</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>No symptoms and no anemia</td>
<td>11 (4%)</td>
</tr>
<tr>
<td>Mild anemia</td>
<td>2 (7%)</td>
</tr>
<tr>
<td>Significant symptoms</td>
<td>4 (1.4%)</td>
</tr>
</tbody>
</table>

The following 17 (6.1%) patients were explored and other procedures were done:

- 1—Valve replaced
- 1—Valve explored

- 1—Valve replaced
- 1—Valve explored
The neurologic manifestations varied from mild cerebral symptoms, blurring of vision, to frank hemiparesis. Among this group of patients were three who were reoperated on for their mitral prosthesis. Two had replacement of their prosthesis with another Beall valve because of thrombi attached to the struts of the prosthesis. This was accomplished successfully via an aortotomy incision leaving the well-endothelialized prosthesis in situ.

Paravacular or Valvular Leak (Table 5)

Among the 273 survivors there were 11 asymptomatic patients (4 percent) with apical systolic murmurs, but without anemia. Cardiac catheterization was not done since all seemed to have minor mitral regurgitation. All are doing well with medical treatment. There were two patients who had a more severe degree of valvular leak as demonstrated by left ventriculography. They have required iron medication and folic acid and occasional blood transfusions; however, they may need reoperation in the near future.

In contrast, the other four patients (1.4 percent) had significant symptoms and clinical evidence of residual mitral regurgitation. This was confirmed with the left ventriculography in all four. Two were reoperated on for closure of the paravalvular leak. The two patients are alive and well, without anemia, but one still has a minor apical systolic murmur. Of the other two patients, the defective valve was replaced with another Beall valve in one. This patient was already discussed as having an embolic complication after his second operation. In the fourth patient, the valve was explored but no leak or thrombi could be identified. It is believed that this patient has intermittent valvular dysfunction. If we add the late death due to cerebral anoxia after the reoperation for a paravalvular leak (Table 3), the overall incidence of valvular leaks in 336 patients was 5.3 percent (18 patients).

Late Survivors

The late clinical results of the 298 patients who survived one month after operation can be appreciated better in actuarial curves. The survival rate of these patients, not counting those lost to follow-up, is represented graphically in Figure 7A. At the end of four years of follow-up there were still 273 patients alive. Similarly, Figure 7B represents actuarially the percentage of patients still living without the specific complications studied at the end of each year postoperation. Thus, there were 226 patients (93.5 percent) free of embolic complications and 226 patients (97.5 percent) who did not require reoperation at four years postoperation.

None of the survivors developed bacterial endocarditis or any other serious infection. Few patients developed superficial wound inflammation and these resolved with local treatment.

At the time of interviewing the patients, 200 (73 percent) were considered in excellent condition. All had returned to their previous levels of exercise. Fifty-two patients (19 percent) were asymptomatic but still not fully recovered. Some had returned to part-time work. Eighteen (7 percent) had not changed considerably; many still had signs and symptoms of congestive heart failure. In this group many had associated nonmitral valvular disease or pulmonary hypertension or both. Three patients were considered in a worse condition as compared to their preoperative status.

Discussion

The early mortality in 29 patients or 8.6 percent in this series correlated well with the risk factors analyzed, such as age, severity of cardiac disability, previous operations and type of mitral lesion. Only one early death occurred as a result of thrombosis of the prosthesis. Recent modification of the seat of the prosthesis (turtle-neck) has allowed us to implant the valve with a continuous running suture in shorter time, thereby decreasing the bypass time.
We hope that this improved technique will result in a lower operative mortality.

The most common cause of late deaths was progression of the underlying myocardial or coronary disease. With current advances in coronary angiographic techniques, selection of patients for concomitant coronary surgery may result in lowering the number of late deaths.

There were only four late embolic deaths related to the prostheses; did the leak appeared within the first year postoperation. No fatal emboli have occurred after one year even though the follow-up of most patients in this series extends over four years. The incidence of nonfatal emboli in 20 patients (8 percent) seems to be relatively low, considering the duration of our follow-up. Furthermore, the actuarial curves indicate that the frequency of emboli decreased gradually after the second follow-up year. This observation is in keeping with our findings on the reoperated on cases, and that of others,14 that these cloth-covered prostheses probably become encapsulated after the first year, thereby decreasing the tendency for local thrombus formation. This experience reinforces the original thesis of Palmer,6 Davila and associates,15,16 and Braunwald and co-workers'17 which suggested that clot coverage of the prosthesis allows tissue to grow into the interstices of the fabric until eventual “encapsulation” occurs.

Thus the incidence of only 1.5 percent fatal and 8 percent nonfatal embolic complications with the Beall cloth-covered prosthesis contrasts favorably with our earlier experience with the use of Starr-Edwards and Cross-Jones prostheses. A 28.6 percent incidence of embolic complications occurred with 30 Starr-Edwards mitral valve replacements from 1963 to 1967 and 43.8 percent among 35 mitral valve replacements using the Cross-Jones prosthesis from 1966 to 1967.

The efficacy of prophylactic use of anticoagulants is a question that has not been settled.18,19 The two deaths that occurred in our series as a result of clotting of the prosthesis had been given anticoagulants and they were abruptly discontinued within three months of the operation. This may be a particularly dangerous sequence as the valve is probably not fully endothelialized before one year. Probably withdrawal of anticoagulants should be avoided if possible.

Only in one of the 18 patients with residual mitral regurgitation did a leak appear to be related to the prosthesis. At reoperation there was disc wearing and notchching with resultant cooking. Since March 1969 the manufacturer4 has made the Teflon disc thicker and more highly compressed to decrease wear. Disc variance has been reported by Robinson.5 Three patients required reseating of the paravalvular leak with one mortality and the other two may require surgery in the near future because of their associated hemolytic anemia. Paravalvular leak is a complication common to all types of prostheses.20-24

The clinical condition in 252 patients out of 273 survivors was quite satisfactory. Most of them were in good or excellent condition, leading active lives. Although we have not performed postoperative cardiac catheterizations routinely in the asymptomatic patients, we believe that significant hemodynamic improvement also occurs in them as it has been reported in other series.1,2 We therefore continue to use this prosthesis as first choice for replacement of the mitral valve.

REFERENCES
12 Nichols HT, Blanco G, Urechico JD, et al: Open-heart

Surgitec Inc, Pittsburgh, Pennsylvania.
surgery for mitral regurgitation and stenosis. Arch Surg 82:125, 1961
commissurotomy. JAMA 182:208, 1960
14 Nichols HT, Morse DP, Blanco G, et al: Open Heart
Surgery for Mitral Stenosis. Springfield, Illinois, Charles
C Thomas, 1961
16 Davia JC: Prosthesis and living tissue: Conflict compat-
17 Braunwald ES, Detmer DE: A critical analysis of the
status of prosthetic valves and homografts. Prog Cardio-
vase Dis 1:113, 1968
18 Beall AC Jr, Bricker DL, Meumier BJ: Results of mitral
valve replacement with Dacron velour-covered Teflon-disc
and the Beall mitral prosthesis. Ann Thorac Surg 10:22-
26, 1970
20 Robinson MJ, Hildner FJ, Greenberg JJ: Disc variance of
21 Fishman NH, Edmunds LH, Hutchinson JC, et al: Five-
year experience with the Starr-Edwards aortic mitral prosthesis.
leaks and hemolytic anemia following insertion of Starr-
Edwards aortic and mitral valves. J Thorac Cardiovasc
of leaking prosthetic heart valves. Ann Thorac Surg 3:503-
513, 1967
24 Najafi H, Dye WS, Javid H, et al: Mitral valve replace-
ment. Review of seven years experience. Am J Cardiol
44:396-400, 1969

Oxides of Sulfur as Air Pollutants

Of the 3,000 chemicals found in the air, oxides of
sulfur, byproducts of modern technology, have been
recognized as harmful atmospheric agents since their
massive onslaught on human health in London, England
(1958) and New York City (1953 and 1966). Recurrent
exposures and mortality attributable to them in other
metropolitan and industrial communities the world over attest to
the persistence of this serious envi-
rionmental problem. These oxides result from the com-
bustion of sulfur contained in coal and oil of which huge
quantities are being used currently. Coal cleaning, coke
production, petroleum refining, public utilities, paper
mills, aluminum, copper, lead and zinc smelting, a great
many other manufacturing processes, transportation in-
dustry (air planes, diesel engines), heating and air condi-
tioning of residential and commercial buildings are
sources of enormous amounts of oxides of sulfur. Oil
found in California, Pennsylvania and in the South are
relatively low in sulfur, sometimes well under 1 percent.
Venezuelan oil contains 2.3 percent, Near Eastern oil 3-
4 percent or more. During the past several years, author-
itative research studies in humans and experimental
animals have offered persuasive pertinent information.
Sulfur oxides in the ambient air may result in pulmonary
ergorgement, increased capillary permeability, intersti-
tial inflammation and thickening of alveolar septa. The
latter is enhanced by swollen alveolar epithelial and
mononuclear cells, lymphocytes and fibroblasts. These
changes interfere with alveolar gas diffusion. Also, there
may be an interference with lung tissue metabolism and
slowing of enzyme function. Thus, decrease in cholin-
terase is conducive to bronchospasm which, in turn,
hinders ventilatory mechanics of the lung by increased
resistance to air flow. Slowing or transient cessation of
bronchial ciliary motion impairs pulmonary homeostasis.
Some individuals show allergic response to sulfur diox-
ide; the latter, in others, increases the severity of bron-
chial asthma. Experimental animals, whose lungs were
rendered emphysematous by aerosolized papain, mani-
fested increased lung compliance and reduction of lung
recruit following daily inhalations of sulfur dioxide. Ac-
cording to reliable estimates, 28 million tons of sulfur
dioxide are emitted into the atmosphere in the United
States annually. Sulfur dioxide may cause severe bron-
chitis, pneumonia and pulmonary edema. Pathogenic
microorganisms contribute to the severity of these
pneumonic changes. As reemphasized by Motley
(Aerosp Med 42:1108, 1971), air pollution aggravates
preexisting respiratory and circulatory diseases. More-
over, Hodgson (Environ Sci Technol 4:569, 1970) un-
derlines the fact that mortality from respiratory and
heart diseases is significantly related to the level of air
pollution. This challenge has led to the establishment of
NAPCA (National Air Pollution Control Administra-
tion) and EPA (Environmental Protection Agency).
The latter's primary standards for the protection of pub-
lic health were set for sulfur oxides in April 1971:80
micrograms per cubic meter (0.03 ppm) annual arith-
metic mean and 95 micrograms per cubic meter (0.14
ppm) as maximum 24-hour concentration not to be
exceeded more than once a year.

Andrew L. Banyai, M.D.