Indices Predicting Long-term Survival after Valve Replacement in Patients with Aortic Regurgitation and Patients with Aortic Stenosis


SUMMARY
The long-term results of aortic valve replacement were reviewed in all 88 patients with isolated aortic regurgitation and all 103 patients with isolated aortic stenosis who were operated upon at the National Heart and Lung Institute from 1963 to 1971. Survival curves were compared to determine whether any of 30 preoperative clinical and hemodynamic findings correlated with long-term survival. The indices that were of predictive value in patients with aortic regurgitation were found to be different from those in aortic stenosis. Symptoms, cardiac index, and cardiothoracic ratio did not influence survival in patients with aortic regurgitation. In these patients, survival was inversely correlated with the level of left ventricular end-diastolic pressure (LVEDP); six-year survival was 74% in patients with LVEDP ≤ 10 mm Hg, 41% with LVEDP 11-20 (P < .05), and 30% with LVEDP > 20 (P < .01). Survival also was lower in patients with aortic regurgitation who had elevated pulmonary arterial and left atrial pressures, and in patients with electrocardiographic evidence of severe left ventricular hypertrophy (LVH). Using an LVH point score method (Romhilt-Estes), 56% of patients with a score ≤ 6 survived six years; 29% with a score > 6 survived (P < .02). Survival in aortic stenosis did not relate to any of the above, but did correlate with preoperative functional class. Five-year survival was 70% in class II, but only 40% in class III-IV (P < .02). Moreover, cardiothoracic ratio in patients with aortic stenosis correlated with survival in an unexpected way. Eleven of 31 patients with cardiothoracic ratio ≤ .45 had sudden unexplained death postoperatively, compared to only six of 72 patients with cardiothoracic ratio > .45 (P < .01). This difference did not correlate with postoperative hemodynamic measurements, including magnitude of the transprosthetic gradient. We conclude that certain preoperative indices are of value in predicting long-term prognosis after valve replacement for aortic regurgitation and for aortic stenosis, but that the specific predictive indices for the two groups differ.

Additional Indexing Words:
Starr-Edwards valves
Ball variance
Operation
Sudden death

ONE OF THE most important unresolved problems in the management of patients with chronic aortic valvular disease is determination of the proper timing of operative intervention. The decision to operate is straightforward in the patient who is functionally incapacitated by his cardiac disease. However, a large group of patients who have significant valvular lesions have only moderate symptomatic impairment that can be managed reasonably well with medical therapy. It is in this group that the therapeutic dilemma arises: should operation be deferred until symptoms progress in the hope that, with time, operative techniques and prosthesis technology will improve further, or should operation be performed without delay in order to prevent the development of irreversible myocardial damage?

To judge optimal time for operative intervention, it would be of critical importance to know which changes in the patient’s clinical or hemodynamic status would place him in a higher risk group in relation to either the natural history of the disease or the long-term survival following valve replacement. Unfortunately, knowledge of the natural history of aortic valvular disease in the current era, in which patients are treated with potent oral diuretics as well as digitalis glycosides and antiarrhythmic drugs, is limited. Information is confined mainly to observations derived from retrospective studies that do not include hemodynamic data.1-4 Moreover, although several investigators have attempted to identify preoperative findings that can predict long-term sur-
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Survival following aortic valve replacement, few correlations have resulted. This might be attributable to two major shortcomings of these studies: patients with all types of aortic valve lesions were analyzed as a group, and relatively limited hemodynamic data were examined.

The present investigation is an attempt to determine whether any preoperative findings can be identified that provide information relating to long-term postoperative survival of patients who have had valve replacement for pure aortic stenosis or for pure aortic regurgitation. Our data indicate that several indices can be identified which predict long-term survival after operation, but the indices that predict survival for aortic regurgitation are considerably different from those that predict survival for aortic stenosis.

Methods

Patient Population

The study population was composed of all 88 patients who underwent Starr-Edwards aortic valve replacement for isolated pure aortic regurgitation, and all 103 patients who underwent Starr-Edwards valve replacement for isolated pure aortic stenosis at the National Heart and Lung Institute between 1963 and 1971. Patients with associated cardiac defects or clinically significant disease of other cardiac valves were excluded. "Pure aortic regurgitation" was defined as severe aortic regurgitation accompanied by a peak systolic left ventricular outflow gradient of less than 25 mm Hg. "Pure aortic stenosis" was defined as severe aortic stenosis with 1+ or less (out of a maximum of 3+) aortic regurgitation, or with no audible diastolic murmur if aortic root angiography was not performed. Ninety-nine patients with isolated mixed aortic valve disease were not analyzed in this study. The means and ranges of several of the important clinical and hemodynamic findings for each group are shown in table 1. Of note, although only 14 of the patients with aortic stenosis were 40 years of age or less, there were 33 patients with aortic regurgitation in this category.

Surgical Criteria

The results of operation undoubtedly are influenced importantly by the criteria used for operation. Our criteria are based almost entirely on symptoms. If a patient with aortic regurgitation or stenosis develops angina, acute left ventricular failure (as judged by paroxysmal nocturnal dyspnea or pulmonary edema), or presyncope or syncope, operation is considered urgent, regardless of whether or not the patient has been adequately treated medically. In the absence of these symptoms, decision for operation is based on whether or not the patient is able to lead a life in a style reasonably acceptable to him. Thus, if a patient has only mild to moderate exertional dyspnea or fatigue, and he is able to do most of the things he considers necessary to feel reasonably fulfilled, operation is deferred. On the other hand, if the patient feels that his life-style is too restricted even after institution of an optimal medical program, he is considered an operative candidate. Radiographic, electrocardiographic or hemodynamic abnormalities, in themselves, are not considered to be sufficient indications for proceeding with operative intervention when these symptomatic indications for operation are absent.

The presence and severity of preoperative symptoms were assessed from the clinical records. We recorded preoperative NYHA functional class and the presence or absence of those symptoms commonly associated with aortic valve disease — angina, syncope or presyncope, dyspnea, orthopnea, and paroxysmal nocturnal dyspnea. Eighteen patients with aortic regurgitation (20%) and 36 patients with aortic stenosis (35%) were in functional class II. The larger proportion of mildly symptomatic patients in the aortic stenosis group was due to the frequent occurrence of syncope and presyncope in patients with aortic stenosis who were otherwise mildly symptomatic.

Cardiothoracic ratio was measured from the posteroanterior chest X-ray taken just prior to operation. Electrocardiographic analysis was performed using the ECG obtained immediately prior to operation. A left ventricular hypertrophy (LVH) point score was determined using the criteria of Romhilt and Estes with the following modifications: since almost all patients were receiving digitalis, only one point was given for repolarization abnormalities, even in the occasional patient who was not taking digitalis; three points were given for left atrial enlargement; a score was assigned all patients, including those with left bundle branch block. Preoperative cardiac catheterization was carried out in all patients, and complete left and right heart data were available in most; however, several patients had abbreviated studies because of the severity of their illness. Since very few patients had coronary arteriograms and a considerable number did not have left ventriculograms or ventricular volume studies, these data were not included in the analysis.

Follow-up information was obtained in most of the patients by annual outpatient visits. In the few who were not seen in the clinic, follow-up was obtained by telephone conversation with either the patient or his personal physician. No follow-up data could be obtained in three patients with aortic regurgitation and two patients with aortic stenosis.

There were a sufficient number of patients under observation for meaningful description of survival probability for six years after operation. In the group of 88 patients with aortic regurgitation, 16 patients were alive at the sixth postoperative year, 27 are currently alive who have been followed for intervals ranging from two to five years, and 45 have died. In the group of 103 patients with aortic stenosis, 27 patients were alive six years after operation (three of these subsequently died), thirty-one are currently alive with a follow-up of less than six years, and 45 patients died before the sixth postoperative year.

Table 1

<p>| Several Descriptors of Patients with Aortic Regurgitation and Patients with Aortic Stenosis |
|----------------------------------------|----------------------------------------|
| Aortic regurgitation | Aortic stenosis |</p>
<table>
<thead>
<tr>
<th>Mean</th>
<th>Range</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>41</td>
<td>18 – 64</td>
<td>51</td>
</tr>
<tr>
<td>Cardiothoracic ratio</td>
<td>.59</td>
<td>.43 – .80</td>
<td>.49</td>
</tr>
<tr>
<td>Estes score</td>
<td>6</td>
<td>0 – 12</td>
<td>4</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>2.6</td>
<td>1.2 – 4.5</td>
<td>2.8</td>
</tr>
<tr>
<td>LVEDP</td>
<td>21</td>
<td>4 – 60</td>
<td>17</td>
</tr>
<tr>
<td>PA systolic pressure</td>
<td>33</td>
<td>14 – 83</td>
<td>30</td>
</tr>
</tbody>
</table>

Abbreviations: LVEDP = left ventricular end-diastolic pressure; PA = pulmonary artery.

Circulation, Volume 30, December 1974
Patients were segregated into subgroups according to the clinical and hemodynamic data described above; survival curves were constructed using the life-table method. Patients lost to follow-up were treated as having been withdrawn alive at the time of last observation. Significance of differences was tested by the Chi-square analysis of Mantell and Haenszel.

Results

Over-all Survival and Causes of Death

The survival curves for the two groups of patients are plotted in figure 1. Operative mortality was 17% in patients with aortic regurgitation and 10% in patients with aortic stenosis, but this difference was not statistically significant. Mortality rate after discharge was highest during the first two years after operation in both groups, with survival curve slopes tending to decrease after the second to third postoperative year.

Figures 2 and 3 show the causes of death in the two groups of patients. Deaths are separated into those that occurred in hospital and those that occurred later, after discharge. Many of the causes of hospital death (bleeding, infection, and technical errors) were not primarily cardiac in origin. Since these accounted for nearly half of the 25 hospital deaths, attempts to predict hospital mortality from analysis of preoperative clinical or hemodynamic findings in this small group of patients would be meaningless. However, of the 68 late deaths, 40 could be considered to be primary cardiac deaths that occurred despite successful mechanical correction of the patient’s valvular abnormality. A total of 33 patients in the two groups died suddenly, without explanation, presumably of arrhythmias. Routine postoperative catheterization had been performed in 18 of these patients. One patient had a 50 mm Hg transprosthetic gradient. The remaining patients had hemodynamically documented, technically successful prosthetic function. Seven other patients died in a setting of low cardiac output and congestive heart failure. The remaining 28 late deaths did not appear to be related to the patient’s underlying heart disease. Of these 28 late deaths, nine occurred as a result of prosthesis related events, including ball variance, coronary embolization, prosthetic thrombosis, and endocarditis. Thus, 13% of all late deaths (18% of late cardiac deaths — nine of 49) could be ascribed to the Starr-Edwards valve. This represents five percent of those who survived operation. Of the prosthesis related deaths not due to ball variance, one occurred in a patient with a model 1200 prosthesis, one with a model 2300, two with model 2310s, and one with a model 2320. Thus, other than the model 1000, no model appeared to be more failure prone than any other.

The noncardiac deaths were evenly distributed throughout the patient population, and did not cor-

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**Figure 1**

Percent survival of the 88 patients with aortic regurgitation and the 103 patients with aortic stenosis is plotted as a function of the time after operation. The legend in the lower left hand corner of the graph denotes the symbols used to describe the curve for each group and the number of patients in each group. The same general format is used in all plots of survival. The dashed lines denote that after the sixth postoperative year the number of patients under observation is too small for meaningful analysis.

**Figure 2**

Causes of hospital and late deaths in patients with aortic regurgitation.

**Figure 3**

Causes of hospital and late deaths in patients with aortic stenosis.
relate with any of the preoperative clinical and hemodynamic findings. Including or excluding these deaths did not alter the relation between any preoperative index and long-term survival. Accordingly, the deaths due to noncardiac causes are included in all survival analyses.

Aortic Regurgitation

Symptoms

In patients operated on for aortic regurgitation, there was no relation between the preoperative NYHA functional class and long-term survival. Similarly, there was no difference in survival when patients were grouped according to the presence or absence of angina, syncope, dyspnea, or orthopnea.

Age

Fifty-three percent of patients under 40 were alive six years after operation; 43% of patients between 40 and 49, and 30% of those 50 and over, survived six years. Although this trend is suggestive, the differences are not statistically significant.

Sex

An unexpected difference in long-term survival was found when the survival curves of men and women were compared (P < .02, fig. 4). Twenty percent of the women died within one year of operation; however, no deaths occurred subsequently. This contrasted with the continued attrition apparent in the survival curve for men. The reason for this finding is unclear. No statistically significant differences in any of the assessed variables were demonstrable (i.e., type of symptoms, age at operation, cardiothoracic ratio, ECG-LVH point score, left ventricular end-diastolic pressure, left atrial pressure, or pulmonary arterial pressure).

![Survival curves of patients with aortic regurgitation segregated by sex. The curves are significantly different (P < .02).](image)

ECG Point Score for Left Ventricular Hypertrophy

Figure 5 depicts the survival curves of patients with aortic regurgitation segregated by their LVH point score into those with scores of 6 or less and those with scores greater than 6. There is a significantly reduced survival (P < .02) in those patients with point scores greater than 6. This difference is due not only to operative mortality, but to the greater number of late deaths in the group with higher point scores. In analyzing the components of the point score, the most important contributor was found to be the P wave. Thus, even when the other components of the point score were neglected, patients with left atrial enlargement tended to have a poorer prognosis.

![Survival curves of patients with aortic regurgitation segregated by their preoperative electrocardiographic left ventricular hypertrophy score. The two curves are significantly different (P < .02).](image)

Catheterisation Data

Preoperative resting cardiac index did not correlate with survival. However, significant correlations were found between left heart pressures and survival. Figure 6 depicts the patients operated on for aortic regurgitation, segregated into three groups on the basis of left ventricular end-diastolic pressure (LVEDP). Patients with an LVEDP of 10 mm Hg or less had an excellent outlook — 74% survived six years. Survival was poorer — 41% at six years (P < .05) — in patients with LVEDP between 11 and 20, and the worst — 30% at six years (P < .02) — in those patients with LVEDP above 20. Subdivision of patients on the basis of their left atrial mean or pulmonary arterial pressure produced essentially similar results (figs. 7 and 8).

Interrelations between the ECG-LVH point score and LVEDP in predicting long-term survival are depicted in figures 9 and 10. Patients with modest elevations of LVEDP (11–20 mm Hg) and LVH point scores of 6 or less had a significantly (P < .05) better long-term survival than those with the same LVEDP.
but with LVH point scores above 6 (fig. 9). In patients with LVEDP greater than 20 mm Hg, prognosis was poor, regardless of the LVH point score (fig. 10). In patients with normal LVEDP, prognosis was excellent and only 3 of the 18 patients in this group had LVH point scores greater than 6. Similar results were observed when different LVEDP ranges were subgrouped according to pulmonary arterial pressure. Patients with modest elevations of LVEDP, but normal pulmonary arterial pressure, had a better prognosis than patients with the same LVEDP but elevated pulmonary arterial pressure. When LVEDP was greater than 20 mm Hg, prognosis was poor regardless of the level of pulmonary arterial pressure.

Finally, pulmonary arterial pressure was rarely elevated in patients with normal LVEDP.

Cardiothoracic Ratio

When considered as an isolated measurement, the preoperative heart size was not related to long-term survival. The results of further analysis of the influence of heart size on long-term survival are depicted in figures 11 and 12. In these figures patients with cardiothoracic ratios of .60 or less (fig. 11) and those with cardiothoracic ratios greater than .60 (fig. 12) are further stratified on the basis of their left atrial pressures. Of note, although patients with large hearts had elevated left atrial pressures more often than patients with normal or slightly enlarged hearts (P < .02), considerable overlap was present. For example, of the 46 patients with cardiothoracic ratios of .60 or less, six had left atrial pressures above 20; of the 30 patients with cardiothoracic ratios greater than .60, only seven had normal left atrial pressures. If it is assumed that a normal mean left atrial pressure indicates a well compensated left ventricle and that, conversely, an elevated filling pressure is more likely to occur when the ventricle is failing, the above results suggest that heart size per se does not provide an absolute index of whether or not left ventricular failure is present. Thus, it would appear that increased heart size could occur in the presence of 1) a well compensated left ventricle (with cardiomegaly caused by marked aortic regurgitation), or 2) a failing left ventricle (with cardiomegaly caused by a combination of decompensation and aortic regurgitation). The survival curves plotted in figures 11 and 12 are consistent with such a concept, insofar as they demonstrate that
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Survival curves of patients with aortic regurgitation whose LVEDP was 11-20 segregated on the basis of their electrocardiographic left ventricular hypertrophy score. The curve for patients with scores greater than 06 is significantly different from the curve for patients with scores of 06 or less (P < .05).

within each cardiothoracic ratio subgroup, survival is inversely related to mean left atrial pressure.

Change in Heart Size

Change in heart size as measured on X-ray over the first four to six months following operation did correlate with long-term prognosis (fig. 13). Thus, 85% of patients operated upon for aortic regurgitation whose cardiothoracic ratios decreased measurably (an absolute decrease of .03 or more) survived six years. However, only 43% of patients (P < .02) whose heart size did not change, or whose heart size increased, survived six years.

Preoperative left atrial pressure measurements were available in 52 of the 58 patients who had pre- and six months postoperative chest X-rays. A decrease in heart size occurred more frequently in patients with normal preoperative left atrial pressures (P < .05). Of the 22 patients whose heart size decreased, 11 (50%) had left atrial pressures of 10 mm Hg or less; of the 30 patients whose heart size did not decrease, only 9 (30%) had normal left atrial pressures. In addition, there have been no late deaths in the 16 patients with left atrial pressures of 20 mm Hg or less whose heart size decreased postoperatively. This contrasts with 11 deaths in the 26 patients with left atrial pressure ≤ 20 mm Hg whose heart size did not decrease (P < .02).

Aortic Stenosis

Symptoms

As in the patients with aortic regurgitation, there was no relation between the presence of individual symptoms (angina, syncope, dyspnea, or orthopnea) and survival. However, in contrast to patients with
aortic regurgitation, survival was significantly better \((P < .02)\) in those patients who were in a lower functional class (fig. 14).

Age

If age is divided into decades, the number of patients in each subgroup is too small for meaningful statistical analysis. Some interesting trends become apparent, however, which may or may not prove valid when a larger series of patients is studied. The greatest survival occurred in those patients operated upon in their fifth decade (75% surviving six years). Survival decreased progressively in older patients (52% of patients between 50 and 59 years and 28% of those 60 and over survived six years). However, those patients who came to operation before the age of 40 had a relatively poor outlook, with only 35% surviving six years.

Cardiothoracic Ratio

Whereas there was no relation between radiographically determined heart size per se and survival in patients with aortic regurgitation, an interesting and unexpected correlation was found between cardiothoracic ratio and survival in patients with aortic stenosis (fig. 15). Survival was lowest in the patients with normal sized hearts. Although differences in the survival curves did not achieve statistical significance, there was a statistically significant difference in the causes of death between the patients with normal sized hearts and those with cardiomegaly. Seventeen of the 31 patients without cardiomegaly have died. Eleven of 17 died suddenly, unexplained deaths. Of the 31 deaths in the group of 72 with enlarged hearts, only six died suddenly, a highly significant difference \((P < .001)\). As in patients with aortic regurgitation, radiographically measured reduction in heart size over the first four to six months following operation was associated with a better long-term prognosis (fig. 16). Seventy-eight percent of patients operated on for aortic stenosis whose cardiothoracic ratio decreased measurably survived six years following operation. However, only 48% of patients \((P < .02)\) whose heart did not change or increased in size survived six years.

ECG Point Score for Left Ventricular Hypertrophy

In contrast to patients with aortic regurgitation, there was no relation between the electrocardiographic LVH point score and long-term survival in patients with aortic stenosis. Survival also was not related to P wave morphology.

Survival curves of patients with aortic stenosis segregated by the change in cardiothoracic ratio (CTR) that occurred over the four to six months after operation. Patients in the group labeled with minus sign had an absolute decrease in cardiothoracic ratio of .03 or more. The curve of patients in whom a decrease in CTR occurred is significantly different \((P < .01)\) from the two curves depicted of patients with no change or increase in CTR.

Survival curves of patients with aortic stenosis segregated by their preoperative NYHA functional class. The two curves are significantly different \((P < .02)\).
In patients with aortic stenosis, none of the variables measured in the catheterization laboratory provided any useful prognostic information. This was true not only for cardiac index, which was also not predictive in patients with aortic regurgitation, but also for LVEDP, left atrial pressure, and pulmonary arterial pressures, each of which were good predictors of long-term survival in patients with aortic regurgitation.

Discussion

The results of this investigation demonstrate that preoperative indices can be identified that predict long-term survival of patients after aortic valve replacement. However, the indices of predictive value in patients with aortic regurgitation differ from those with aortic stenosis. Survival is lower in patients with aortic regurgitation who have electrocardiographic evidence of severe left ventricular hypertrophy (as quantified by the Romhilt-Estes criteria), or who have markedly elevated LVEDP, left atrial pressure, or pulmonary arterial pressure. However, these indices are of no value in predicting long-term survival of patients with aortic stenosis. In contrast, preoperative functional class has some predictive value in patients with aortic stenosis, while it has none in patients with aortic regurgitation.

The demonstration that clinical and hemodynamic predictors of postoperative survival differ markedly in patients with aortic regurgitation from those with aortic stenosis is not surprising, considering the very different type of hemodynamic burden each abnormality presents to the left ventricle. For example, the chronic pressure load imposed on the left ventricle by aortic stenosis results in a concentrically hypertrophied chamber that has decreased compliance. In contrast, the chronic volume load imposed on the ventricle by aortic regurgitation causes gross left ventricular dilatation, a change that shifts the left ventricular pressure volume curve to the right, indicating increased compliance. Thus, while one of the more important contributing factors leading to elevated LVEDP in patients with aortic stenosis is decreased compliance, very different mechanisms lead to elevated filling pressures in patients with aortic regurgitation.

It is of interest that cardiac size, as estimated by chest X-ray, did not correlate with long-term survival of patients with aortic regurgitation. Nonetheless, survival did correlate with postoperative change in heart size. These findings are compatible with the hypothesis that at least two different mechanisms are responsible for the left ventricle dilating in patients with aortic regurgitation. The chamber may be grossly dilated because of a large regurgitant volume, but compensation may be good (i.e., ejection fraction would be expected to be normal). In these patients, operative relief of regurgitation would cause left ventricular size to diminish appreciably. Alternatively, the left ventricle may be grossly dilated in the presence of only moderate regurgitation if the ejection fraction (and presumably myocardial function) is reduced. Operative relief of regurgitation in these patients would presumably cause less diminution of left ventricular size. Ventricular volume measurements were not available for the patients in this study. However, there was a subgroup of patients with aortic regurgitation who had very large hearts, but nevertheless had normal mean left atrial pressures. These patients had an excellent survival (fig. 12), a finding compatible with the concept that such patients had large regurgitant volumes and normal left ventricular ejection fractions. These patients may be contrasted with the patients who also had gross cardiomegaly, but in whom left atrial mean pressure was elevated. Since long-term postoperative survival was very poor in these patients, it is possible that they had comparatively smaller regurgitant volumes, and that cardiomegaly was caused largely by left ventricular dilatation consequent to cardiac decompensation. This hypothesis is strengthened further by the fact that a postoperative decrease in heart size occurred more commonly in patients with normal left atrial pressures.

Survival in patients operated on for aortic stenosis correlated with preoperative heart size in an unexpected way. Survival tended to be poorest in the subgroup with no radiographic evidence of cardiomegaly (cardiothoracic ratio of .45 or less) when compared to...
the subgroups with larger hearts. Although the survival curves among the subgroups were not significantly different statistically, the incidence of sudden unexplained deaths, presumably due to arrhythmias, was significantly greater in the subgroup with normal-sized hearts. This finding could not be explained by any other pre- or postoperative data. In particular, the patients with normal-sized hearts did not have larger transprosthetic gradients postoperatively, and there were no preoperative clinical or hemodynamic findings that distinguished the patients whose heart size was normal from those in whom it was increased.

Several potential mechanisms responsible for the higher incidence of sudden death in the patients with aortic stenosis and normal-sized hearts deserve further study. For example, it is possible that the myocardium of such patients is thicker than those with cardiomegaly, and that beyond a critical point, regression of left ventricular mass postoperatively does not occur. If this were so, muscle mass might outstrip the capacity of the coronary circulation to deliver the requisite amount of oxygen to all parts of the myocardium, thereby predisposing to the development of ischemia-induced arrhythmias. If the hypothesis of excessively thickened myocardium is correct, it also is possible that intraoperative myocardial perfusion is less adequate in patients with greatly thickened left ventricles than in those who have coexistent dilatation and less thickened hearts.

The patients who died suddenly and unexpectedly late postoperatively constitute one portion of the rather large subgroup of patients undergoing valve replacement who die after hospital discharge from (apparently) cardiac causes, despite successful mechanical correction of the hemodynamic abnormality. Thus, over half of all late postoperative deaths of patients prior aortic stenosis or regurgitation are sudden and unexplained, or are the result of progressive cardiac failure. One of the possible explanations for these deaths is that patients were operated upon too late in the course of their disease and that irreversible myocardial injury had already occurred. However, the possibilities cannot be ruled out that undetected intraoperative myocardial injury occurred, which disposed to late death, or that transient mechanical abnormalities of the prosthetic valve occurred, which could not be detected at necropsy. In this regard, 18% of late cardiac deaths (5% of all patients who survived aortic valve replacement) were due to known complications of the prosthetic valve (ball variance, coronary embolization, prosthetic thrombosis and endocarditis). Since the new model Starr-Edwards valves have a metallic rather than a silastic ball, ball variance no longer will be a cause of late death. Eliminating deaths due to ball variance from our analysis, the percentage of prosthesis-related death was about 10% of all late primary cardiac deaths (3% of all patients who survived aortic valve replacement).

Two other points derived from this study are of interest. First, we found that almost half of the patients who died before leaving the hospital died as a result of noncardiac complications. Hence, it is difficult to predict operative mortality from indices related primarily to cardiac function. Second, it would appear that left heart pressures are necessary to obtain the most complete information relating to long-term survival in patients with aortic regurgitation. For example, although the ECG-LVH point score is quite helpful in this regard, we found that even when the score was less than six, prognosis was poor if LVEDP was above 20 mm Hg. Although there was a tendency for patients with high ECG-LVH scores to have an elevated left ventricular end-diastolic pressure, half of those patients with an LVEDP greater than 20 mm Hg had ECG point scores less than six. That the analysis of the ECG is still of help in predicting survival even when LVEDP is known is indicated by analyzing the group of patients who have moderately elevated LVEDPs, i.e., in the range of 11–20 mm Hg. In this subgroup of patients, LVH point scores of six or less indicate a relatively good prognosis, while LVH point scores of greater than six indicate poor long-term prognosis.

The identification of subgroups of patients with aortic regurgitation or with aortic stenosis who have poor long-term survival postoperatively might be used to advance the argument that operation should be performed earlier in the course of the disease (e.g., as soon as a patient manifests any of the abnormalities indicating reduced long-term survival postoperatively). However, the patients studied in the present investigation were catheterized because of the occurrence of symptoms judged to be serious enough to warrant operative intervention. Serial hemodynamic and electrocardiographic studies were not performed. It is not known, therefore, how long a finding suggesting poor prognosis had been present. In other words, it is possible that early detection of an elevated left ventricular end-diastolic pressure, prior to the development of angina, left heart failure, or presyncope may be too sensitive an index to use as the indication to proceed with operation, and would result in premature operative intervention. Obviously, prospective studies are needed to determine whether the appearance of one of the indices predicting poor postoperative survival will also prove to be a harbinger of imminent symptomatic deterioration. Once this latter information is known, it then will be possi-
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able to develop a more rational approach to the decision of when operation should be performed in a given patient with aortic valve disease.

Acknowledgment

The authors would like to thank Ms. Cora Burn for her help in coordinating the efforts leading to completion of the manuscript, Mr. Fred Bullock and Ms. Ginny King for their assistance in assembling and verifying the hemodynamic data, and Mr. Ray Danner and Mrs. Catherine Staneck for assistance with data processing.

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JOHN W. HIRSHFELD, JR., STEPHEN E. EPSTEIN, ARTHUR J. ROBERTS, D. LUKE GLANCY and ANDREW G. MORROW

Circulation. 1974;50:1190-1199
doi: 10.1161/01.CIR.50.6.1190

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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