Left Ventricular Function Following Replacement of the Aortic Valve
Hemodynamic Responses to Muscular Exercise

By John Ross, Jr., M.D., Andrew G. Morrow, M.D., Dean T. Mason, M.D., and Eugene Braunwald, M.D.

It is generally agreed that the decision regarding operative treatment in adult patients with aortic valve disease must be based primarily on the presence of a structural defect in the aortic valve, which is responsible for significant hemodynamic and clinical disturbances. Yet to be resolved, however, is the question whether or not coexistent disease of the myocardium, or of the coronary circulation, plays an important role in limiting cardiac function in patients with aortic stenosis or regurgitation. Moreover, if myocardial dysfunction is significant preoperatively, the extent to which it may impair ventricular performance following corrective surgery also remains uncertain. The development of satisfactory prosthetic aortic valves, and their widespread clinical application, now permit an approach to these problems, since the function of the left ventricle may be examined after its mechanical burden has been relieved; any residual impairment of ventricular performance may then be attributed solely to abnormalities of the myocardium per se, or to impaired myocardial function due to coronary arterial insufficiency.

In the present study, patients were selected in whom the aortic valvular defect was proved by hemodynamic studies to have been effectively corrected by a valvular prosthesis. Left ventricular performance was assessed directly during left heart catheterization by determining the response of the ventricle to the stress of muscular exercise.

Methods
Fourteen patients were studied in the postab-
sorptive state following premedication with 100 mg. of pentobarbital. All of the patients were men, ranging in age from 29 to 64 years; the pertinent clinical and hemodynamic findings are summarized in Table 1. Thirteen of the patients were in sinus rhythm at the time of study, and one (L.T.) was in atrial fibrillation.

Prior to operation, 10 of the patients had valvular aortic stenosis with or without associated aortic regurgitation. One patient had pure aortic regurgitation, one had aortic regurgitation associated with a ventricular septal defect, and in another, aortic regurgitation was associated with congenital subaortic stenosis. The ventricular septal defect was closed at operation in patient C.H., and complete closure was confirmed at the postoperative cardiac catheterization study. In the remaining patient, both aortic and mitral stenosis were present, and mitral commissurotomy was also performed at the time of aortic valve replacement; no residual end-diastolic pressure gradient across the mitral valve was present at the time of study. Thirteen of the patients were studied 4 to 10 months after the aortic valve had been replaced with a Starr-Edwards ball-valve prosthesis.1 In two of these patients (E.N. and W.L.) flexible Teflon valves of the Muller type2 had originally been inserted, but they became regurgitant and were replaced with ball-valve prostheses. One patient (R.H.) was studied 16 months after the insertion of a Muller valve, which was competent at the time of study.

All but one of the 14 patients were markedly improved following operation and had resumed essentially normal activity. Patient R.H. had dyspnea on moderate exertion, and he had experienced several attacks of congestive heart failure since operation. Two patients (R.S. and W.L.) had residual angina pectoris, although the pain occurred less frequently than prior to operation. Patient E.N. had mild dyspnea, considered to be secondary to chronic pulmonary disease. All of the patients except W.C. were receiving digitalis.

Catheterization of the left ventricle at the time of the postoperative study was performed

From the Cardiology Branch, and the Clinic of Surgery, National Heart Institute, Bethesda, Maryland.

Circulation, Volume XXXIII, April 1966 507
Table 1

Clinical Diagnoses and Hemodynamic Findings in the Patients Studied

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age (yr.)</th>
<th>Preop. diag.</th>
<th>Interv. (mo.)</th>
<th>Preoperative</th>
<th>Postoperative</th>
<th>O₂ Cons.</th>
<th>BSA, M²</th>
<th>Ex. fac.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LV pr. mm. Hg</td>
<td>BA pr. mm. Hg</td>
<td>L/min. M.²</td>
<td>LV pr. mm. Hg</td>
<td>BA pr. mm. Hg</td>
<td>L/min. M.²</td>
<td>HR</td>
<td>mL/min.</td>
</tr>
<tr>
<td>CC*</td>
<td>41</td>
<td>AS, MS</td>
<td>9</td>
<td>223/15</td>
<td>120/65</td>
<td>3.71</td>
<td>Rest</td>
<td>124/8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>162/7</td>
</tr>
<tr>
<td>RH†</td>
<td>52</td>
<td>AI</td>
<td>16</td>
<td>180/16</td>
<td>160/48</td>
<td>3.59</td>
<td>Rest</td>
<td>154/24</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>173/28</td>
</tr>
<tr>
<td>LB</td>
<td>49</td>
<td>AS</td>
<td>6</td>
<td>205/15</td>
<td>105/70</td>
<td>—</td>
<td>Rest</td>
<td>122/14</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>162/25</td>
</tr>
<tr>
<td>KW</td>
<td>41</td>
<td>Subv. AS with AI</td>
<td>4</td>
<td>215/40</td>
<td>108/58</td>
<td>1.79</td>
<td>Rest</td>
<td>160/14</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>186/12</td>
</tr>
<tr>
<td>GN</td>
<td>54</td>
<td>AS</td>
<td>6</td>
<td>200/13</td>
<td>140/58</td>
<td>—</td>
<td>Rest</td>
<td>109/6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>198/9</td>
</tr>
<tr>
<td>EN</td>
<td>49</td>
<td>AS, AI</td>
<td>8</td>
<td>125/38</td>
<td>128/54</td>
<td>3.68</td>
<td>Rest</td>
<td>122/5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>162/9</td>
</tr>
<tr>
<td>LT</td>
<td>50</td>
<td>AS, AI</td>
<td>7</td>
<td>180/13</td>
<td>110/50</td>
<td>2.90</td>
<td>Rest</td>
<td>120/7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>185/9</td>
</tr>
<tr>
<td>WL</td>
<td>51</td>
<td>AS, AI</td>
<td>10</td>
<td>163/15</td>
<td>122/32</td>
<td>2.89</td>
<td>Rest</td>
<td>114/2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>134/8</td>
</tr>
<tr>
<td>JH</td>
<td>29</td>
<td>AS, AI</td>
<td>6</td>
<td>199/11</td>
<td>142/75</td>
<td>2.70</td>
<td>Rest</td>
<td>145/3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>160/5</td>
</tr>
<tr>
<td>CW</td>
<td>53</td>
<td>AS, AI</td>
<td>6</td>
<td>192/13</td>
<td>107/60</td>
<td>3.51</td>
<td>Rest</td>
<td>143/6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>178/10</td>
</tr>
<tr>
<td>WC</td>
<td>39</td>
<td>AS</td>
<td>9</td>
<td>215/13</td>
<td>172/53</td>
<td>3.77</td>
<td>Rest</td>
<td>148/11</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>159/11</td>
</tr>
<tr>
<td>RS</td>
<td>64</td>
<td>AS</td>
<td>6</td>
<td>253/23</td>
<td>120/50</td>
<td>2.64</td>
<td>Rest</td>
<td>150/12</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>170/22</td>
</tr>
<tr>
<td>JR</td>
<td>29</td>
<td>AS</td>
<td>7</td>
<td>230/26</td>
<td>118/72</td>
<td>3.14</td>
<td>Rest</td>
<td>154/9</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>172/13</td>
</tr>
<tr>
<td>CH</td>
<td>31</td>
<td>VSD, AI</td>
<td>8</td>
<td>130/5</td>
<td>120/45</td>
<td>—</td>
<td>Rest</td>
<td>138/3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ex.</td>
<td>166/5</td>
</tr>
</tbody>
</table>

*End-diastolic mitral valve pressure gradient 15 mm. Hg, preoperatively, 0 mm. Hg, postop.
†Pulsus alternans during both cardiac catheterizations.
Interv., number of months following surgery that study was performed; AS, aortic stenosis; MS, mitral stenosis; AI, aortic insufficiency; Subv., subvalvular; VSD, ventricular septal defect; LV pr., left ventricular pressure, systolic/end-diastolic; BA pr., brachial arterial pressure; CI, cardiac index; HR, heart rate; Ex., measurements made during exercise; Ex. fac. (exercise factor), ml./min. increase in cardiac output per 100 ml./min. increase in O₂ consumption.
LEFT VENTRICULAR FUNCTION

509
either by the transseptal approach or by the percutaneous insertion of a short Teflon catheter through the ventricular apex. A needle was placed into the brachial artery for pressure determinations, and cardiac output was measured by the indicator-dilution technic, with injection of indocyanine-green dye into the left ventricle and brachial arterial sampling. Oxygen consumption was determined by measuring ventilation with a Tissot spirometer, and the expired gas was analyzed with a micro-Scholander apparatus. Left ventricular and systemic arterial pressures, oxygen consumption, and the cardiac output were measured in the resting state. The patients then pedaled a bicycle ergometer for 5 to 7 minutes in the supine position. In 12 patients, the workload was 500 foot lb./min., and in two patients (R.H. and R.S.) it was 250 foot lb./min. During the final minute of exercise, the measurements of left ventricular and arterial pressure, cardiac output, and oxygen consumption were repeated.

The “exercise factor” was defined as the increase in cardiac output in ml./min. per 100 ml./min. increase in oxygen consumption. The stroke work index of the left ventricle was calculated with use of the formula

\[ SV \times (LVS - LVED) \times 1.36 \]

\[
\frac{100 \times BSA}
\]

where \(SV\) = stroke volume in ml., \(LVS\) = mean left ventricular pressure during ejection, determined by planimetric integration, and expressed in mm. Hg, \(LVED\) = left ventricular end-diastolic pressure expressed in mm. Hg, and \(BSA\) = body surface area in M.².

Results

At the time of the postoperative study the cardiac indices in the resting state were normal in 11 patients (over 2.50 L./min./M.²) and slightly reduced in three (2.20 to 2.37 L./min./M.²). The left ventricular end-diastolic pressures were normal in 11 patients (below 12 mm. Hg) and elevated in three (14 to 24 mm. Hg); the latter three patients had normal cardiac indices. These left ventricular end-diastolic pressures contrasted with the findings of elevated end-diastolic pressures in all but two of the patients prior to operation (table 1).

The peak systolic pressure gradients between the left ventricle and the brachial artery averaged 12 mm. Hg in the resting state, and ranged from 0 to 25 mm. Hg; the mean gradients ranged from 0 to 15 mm. Hg and averaged 7 mm. Hg. No consistent changes in the gradients were induced by exercise, the gradients at peak systole during the exercise period ranging from 0 to 28 mm. Hg and averaging 14 mm. Hg. The alterations in heart rate induced by exercise are shown in table 1.

Exercise resulted in substantial increases in the cardiac indices in 12 patients. When the increases in cardiac output were related to the increases in oxygen consumption, the exercise factors were found to be within normal limits for this laboratory in nine of these patients (over 600), and mildly reduced in three (462 to 540) (fig. 1). In patient G.N. the cardiac index rose only slightly with exercise, the exercise factor being 255, and in R.H. the cardiac index declined.

The directional alterations in the left ventricular end-diastolic pressure that occurred during exercise consisted of reductions in two patients, no change in one, and increases in 11 patients; in eight of the latter 11 patients the increases exceeded 2 mm. Hg, and ranged from 3 to 11 mm. Hg. In nine patients, the left ventricular end-diastolic pressures remained below the upper limit of normal (12 mm. Hg) throughout the period of exercise, and in one patient it fell from 14 to 12 mm. Hg (fig. 1); the records of left ventricular and brachial arterial pressures obtained in one of these nine patients (W.L.) before and during exercise are reproduced in figure 2. In the remaining four patients, the left ventricular end-diastolic pressures were abnormally elevated to levels of 13 to 28 mm. Hg during exercise; in two of these patients the end-diastolic pressure was also abnormal at rest.

The effects of exercise on the relationship between the left ventricular end-diastolic pressure and left ventricular stroke work are shown in figure 3. In 12 patients the stroke work indices increased with exercise. In two patients there was a fall in the left ventricular stroke work during exercise. In both of these patients (R.S. and R.H.), the end-diastolic
Figure 1

Relationship between cardiac index and left ventricular end-diastolic pressure (L.V.E.D.Pr.) before (open circles) and during (closed circles) supine muscular exercise. The exercise factor (ml./min. increase in cardiac output per 100 ml./min. increase in O₂ consumption) for each patient is indicated adjacent to the closed circle.

Figure 2

Tracings of the electrocardiogram, brachial arterial (B.A.) and left ventricular (L.V.) pressures obtained in patient W.L. before (upper panel) and during supine muscular exercise (lower panel). The high sensitivity recordings of the left ventricular end-diastolic (L.V.E.D.Pr.) are shown in the right-hand portion of each panel.

Circulation, Volume XXXIII, April 1966
pressures were markedly elevated during exercise.

The relationship between the change in the left ventricular end-diastolic pressure and the change in the stroke volume induced by exercise is plotted in figure 4. The normal response to this level of supine exercise indicated by the cross-hatched area, is an increase in the stroke volume, and a change in the left ventricular end-diastolic pressure that ranges from −5 to +2 mm. Hg. In five patients the responses to exercise were normal by these criteria. In one patient, both the left ventricular end-diastolic pressure and the stroke volume fell. In the remaining eight patients, the left ventricular end-diastolic pressures increased abnormally during exercise, from 3 to 11 mm. Hg. In three of these eight patients the increase in end-diastolic pressure was associated with an augmented stroke volume, in three there was little change in the stroke volume, and in two patients a fall in the stroke volume occurred.

Discussion
The clinical improvement evidenced by 13 of the 14 patients in the present study was striking, and was unquestionably related to the nearly complete correction of the mechanical defect in aortic valve function. It is of interest that the small pressure gradients that existed between the left ventricle and the brachial artery at rest were not increased during exercise despite elevations in the cardiac output, an observation that has previously been made both in patients with valvular aortic stenosis and with aortic valve prostheses. This finding can probably be explained, in part, by the increase in the total systolic ejection time per minute that accompanies the tachycardia of exercise.

Judged by the relationship between the increase in oxygen consumption and the elevation of the cardiac index, cardiac performance during muscular exercise in the group as whole was remarkably good, the exercise factors being normal for this laboratory (over 600) in nine patients and over 450 in three others. Thus, it would appear, both from the clinical observations and from the exercise factors, that in the great majority of these

![Figure 3](http://circ.ahajournals.org/download)
patients residual myocardial or coronary artery disease did not impose serious limitations upon the over-all ability of the heart to meet the demands of the imposed stress. In contrast are the findings reported by Bristow et al.,9 who studied 10 patients following aortic valve replacement, all of whom exhibited marked clinical improvement; however, calculation of the exercise factors from their data reveals values of less than 600 in eight of the 10 patients. It should be appreciated that the present group of patients may not provide a representative view of the importance of myocardial dysfunction which may occur in aortic valve disease. Although the subjects were not selected, in the sense that the group included consecutive patients found to have satisfactory mechanical function of the prosthetic valve, it would seem likely that patients with more severe degrees of myocardial involvement either may not reach operation or may not survive the surgical procedure.

Although the exercise factors were normal in most of the patients, there was other evidence that in many patients the left ventricular myocardium was not normal, and in these subjects determination of the exercise factor alone would have been inadequate for detecting the abnormality. Left ventricular end-diastolic pressure measurements have been made during exercise in only a few patients without left ventricular disease, and in these subjects the left ventricular end-diastolic pressure either falls, or it rises by less than 3 mm. Hg during the relatively mild degrees of supine exercise used in the present study.7 This finding is consistent with cineradiographic studies of radiopaque metal markers sutured to the epicardium which have demonstrated that a reduction in the size of the left ventricle always occurs during exercise.10 The

Figure 4
Relationship between the change in the stroke volume index (SVI) and the change in the left ventricular end-diastolic pressure (L.V.E.D.Pr.) induced by exercise in the 14 patients studied. Each arrow points to the changes occurring during exercise, which are indicated by the closed circles. The shaded area indicates the range of values observed in other human subjects without disease of the left ventricle.7
responses in eight of the patients in the present study did not coincide with the normal patterns, the left ventricular end-diastolic pressure increasing by between 3 and 11 mm. Hg, and rising above normal levels in four patients. Increases in left ventricular end-diastolic pressure during exercise were also observed in five of the eight patients with prosthetic aortic valves studied by Bristow et al.9 It is of interest that the three patients in the present group who exhibited the largest increases in left ventricular end-diastolic pressure during exercise had normal exercise factors, emphasizing that determination of the relationship between the cardiac output and oxygen consumption alone may be insufficient in the evaluation of the left ventricular response to exercise.

In interpretation of the changes in left ventricular end-diastolic pressure, several pertinent experimental and clinical observations may be considered. First, it is now relatively certain, from recent studies in the intact dog heart and in the isolated papillary muscle, that alterations in sympathetic tone, and changes in heart rate, short of marked tachycardia, do not result in acute changes in the distensibility of the left ventricle.11–16 Secondly, the mean intrapleural pressure may diminish slightly during the hyperventilation that accompanies exercise,17, 18 and this effect alone would tend to lower the left ventricular end-diastolic pressure with reference to an external zero reference point. From these considerations it seems justifiable to conclude that any increase in the left ventricular end-diastolic pressure during exercise reflects an increase in the end-diastolic volume of the ventricle. Finally, while acute changes in distensibility probably did not occur, the possibility must be considered that chronic alterations in ventricular compliance existed consequent to long-standing aortic valve disease. Thus, while it may be assumed that in the eight patients in whom abnormal increases in left ventricular end-diastolic pressure occurred the left ventricular end-diastolic volume also increased, the elevations in pressure could have been magnified by increased stiffness of the ventricular wall."19

In only two patients did left ventricular stroke work fail to increase during exercise, and, in general, there was no correlation between the magnitude of the increase in the stroke work and the change in the left ventricular end-diastolic pressure. In the two patients in whom the stroke work fell, as well as in most of the others studied, the primary determinant of a change in the stroke work was an alteration in stroke volume, since changes in the mean systolic pressure in the left ventricle resulting from exercise were usually small.

In consideration of the stroke volume alterations in relation to the changes in the left ventricular end-diastolic pressure induced by exercise, it may be recalled that the response of the normal left ventricle to supine exercise usually consists of a small increase in the stroke volume,20–25 and, as mentioned previously, a slight fall, or a rise of less than 3 mm. Hg, in the left ventricular end-diastolic pressure.7 In five patients in the present study, this type of response was observed, suggesting that left ventricular function was normal in this group (fig. 4). This pattern is consistent with the concept that an improvement in left ventricular function occurred during exercise, a larger stroke volume being delivered from an essentially unchanged or lower end-diastolic volume.10 In one patient (K.W.) a fall in the stroke volume was accompanied by a reduction in the left ventricular end-diastolic pressure; it is possible that in this instance the reduction in stroke volume was related to the rather marked increase in the heart rate which occurred. In three of the eight patients in whom the left ventricular end-diastolic pressure increased by 3 to 11 mm. Hg, the increase in pressure was associated with an increase in the stroke volume, in three, the stroke volume remained unchanged, and, in two, the stroke volume fell. It has been well established both from experimental26–28 and clinical observations29–31 that the failing or depressed left ventricle responds to a change in end-diastolic pressure or volume
by an abnormally small alteration in stroke volume. Thus, as observed in five of these patients, an increase in left ventricular end-diastolic pressure, coupled with a reduction or little change in the stroke volume, suggests that a distinct depression of left ventricular function was present. In the three patients in whom both left ventricular end-diastolic pressure and stroke volume increased, the mechanism by which the stroke volume increased is uncertain, since it could have been mediated either by a sizable change in the end-diastolic volume, i.e., through the Frank-Starling mechanism, primarily through a positive inotropic effect, or through a combination of these mechanisms. A final point regarding the stroke volume changes may be made in reference to patient G.N. In this instance, a rather marked increase in the peak left ventricular systolic pressure was noted during exercise (to 198 mm. Hg), and a large increase in the left ventricular stroke work occurred. This was associated with some rise in the left ventricular end-diastolic pressure, but the stroke volume fell. Previous studies from this laboratory have shown that increasing the systemic arterial pressure by means of an infusion of angiotensin results in no change or a fall in the stroke volume in patients with left ventricular dysfunction, while in normal subjects an increase in stroke volume occurs.\(^{31}\) It is thus possible that in this patient the decrease in stroke volume may have been partly related to the increased pressure load on the left ventricle.

The findings in the present study indicate that direct determination of the left ventricular pressure, and of the stroke volume, before and during exercise permits the detection of abnormalities in left ventricular function that would not be apparent from assessment of changes in the cardiac output and oxygen consumption alone. The etiology of these abnormalities of the left ventricular myocardium must remain speculative. In all of the patients who exhibited abnormal responses, the left ventricular end-diastolic pressure was elevated prior to operation. In two patients, angina pectoris was present prior to opera-

tion, and in one (R.H.) pulsus alternans was evident during the initial cardiac catheterization, suggesting that underlying myocardial or coronary artery disease contributed to the preoperative disability in these three patients. In the remaining five patients, of whom two had aortic stenosis and regurgitation and three had aortic stenosis alone, four were 49 years of age or older (L.B., G.N., E.N., and C.W.), and patient J.R. had a left ventricular pressure of 230/26 mm. Hg prior to operation. Thus, it seems likely that a long-standing or unusually severe hemodynamic burden contributed to the abnormal left ventricular function observed in these patients postoperatively.

**Summary**

Evaluations of left ventricular myocardial function were carried out in 14 patients 4 to 16 months after stenotic or regurgitant malformations of the aortic valve had been corrected by valve replacement. The circulatory responses to exercise, judged by the increases in cardiac output in relation to the increases in oxygen consumption, were normal or only mildly reduced in 12 patients. In five patients, the relationships between the change in the left ventricular end-diastolic pressure and the alteration in the stroke volume were also normal, a fall or an increase in left ventricular end-diastolic pressure of less than 3 mm. Hg being accompanied by an increase in stroke volume. In eight patients, however, abnormal increases in the left ventricular end-diastolic pressure occurred during exercise, and in four the left ventricular end-diastolic pressure was increased to levels above 12 mm. Hg. Variable alterations in the stroke volume accompanied these increases in end-diastolic pressure. It is suggested that in the three patients who exhibited simultaneous increases in left ventricular end-diastolic pressure and stroke volume, the changes were either a manifestation of the Frank-Starling mechanism, or primarily the result of a positive inotropic influence. In the five patients who exhibited increases in left ventricular end-diastolic pressure, but no change or a fall in stroke volume, it is proposed that a distinct depression of left ven-

*Circulation, Volume XXXIII, April 1966*
tricular performance was present. Thus, while the cardiac output response was adequate to meet the stress of exercise in the majority of the patients studied following aortic valve replacement, determination of the relationship between the left ventricular end-diastolic pressure and the stroke volume permitted the detection of abnormalities in the function of the left ventricle.

References


---

On Watching a Heart Operation

This is the heart, but not as poets dreamed it,
Rich fountain of a thousand different flows,
This is the heart as Harvey's genius schemed it,
Four-chambered pump with petals like a rose.
This is the heart, but not as poets see it,
Insurgent centre of tumultuous joy,
This is the heart as surgeons' scalpels free it
And scientific stratagems employ.
This is the heart torn from its tender sac,
Denied its pulses, cheated of its blood,
Until the moment, rhythm given back,
It manages its own triumphant flood.
And looking in the theatre I view
A love as keen as any Venus knew.

Left Ventricular Function Following Replacement of the Aortic Valve: Hemodynamic Responses to Muscular Exercise

JOHN ROSS, JR., ANDREW G. MORROW, DEAN T. MASON and EUGENE BRAUNWALD

_Circulation_. 1966;33:507-516
doi: 10.1161/01.CIR.33.4.507

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1966 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:
http://circ.ahajournals.org/content/33/4/507

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/