Left Ventricular Performance Following
Correction of Free Aortic Regurgitation

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SUMMARY
Left ventricular (LV) myocardial contractility, or inotropic state, was characterized
in terms of the instantaneous relations between velocity of circumferential fiber shortening ($V_{CF}$),
determined cineangiographically, and LV wall tension (boop stress),
calculated from LV dimensions and pressure, in five patients before and 7 to 10 mo
after aortic valve replacement for free aortic regurgitation. Preoperatively the cardiac
index was reduced or the LV end-diastolic pressure was markedly increased (or both
occurred) in four patients, in each of whom depression of inotropic state was docu-
mented by a reduced $V_{CF}$ at maximum wall tension, ranging from 0.13 to 1.07 cir-
cumferences (circ)/sec (normal, > 1.40 circ/sec) at wall tensions of 318 to 464 g/cm²
(normal, 178 to 417 g/cm²). In one patient in whom LV end-diastolic pressure and
 cardiac index were normal preoperatively, $V_{CF}$ was 1.66 circ/sec at a maximum ten-
sion of 440 g/cm². Following operation, LV end-diastolic pressure fell in the four
patients with depressed inotropic state (average decrease, 23 mm Hg) and cardiac index
increased (average increase, 0.93 L/min/m²). However, no change in the tension-
velocity relation was observed postoperatively, $V_{CF}$ ranging from 0.27 to 1.14 circ/sec
in these patients, indicating that no change had occurred in the inotropic state.
In addition, a fixed abnormality in diastolic LV pressure-volume characteristics, determined
from preoperative and postoperative measurements of pressure and radius during
diastole, had occurred in patients with depressed myocardial function. In contrast, in
the patient with normal myocardial function, LV end-diastolic radius was reduced by
33% postoperatively while end-diastolic pressure was unchanged, suggesting reversal
of stress relaxation, or creep, following relief of volume overload.

Additional Indexing Words: Velocity of fiber shortening LV wall tension Inotropic state Cardiac index
Myocardial contractility LV end-diastolic pressure

Operative correction of valvular cardiac
lesions often results in striking symp-
tomatic and hemodynamic improvement, with
 reduction in left ventricular end-diastolic
pressure and increase in cardiac output.
However, whether these changes result simply
from relief of ventricular overloading, or
whether in fact changes in myocardial
inotropic state may occur following operation
has not been established.

Recently we demonstrated that measure-
ments of the force-velocity-length relation in
man make it possible to identify alterations
in ventricular performance determined by
changes in ventricular fiber length, afterload,
and contractile state.1–2 In the present study,
these technics have been employed to
characterize left ventricular performance in
five patients before and following replacement
of the aortic valve with a Starr-Edwards

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Circulation, Volume XLII, November 1970 773
prosthesis because of free aortic regurgitation. This approach has also permitted an examination of the mechanical properties of the ventricle during diastole before and after operation.

Methods

Five patients, ranging in age from 17 to 47 years, were studied before and 7 to 10 mo following replacement of the aortic valve with a Starr-Edwards prosthesis. In each patient free aortic regurgitation was documented angiographically at preoperative study; none had associated aortic stenosis or significant mitral valve disease.

Right and left heart catheterizations were performed in the postabsorptive state after premedication with sodium pentobarbital, 100 mg, intramuscularly. Preoperatively, a catheter-tip micromanometer* was introduced into the left ventricle from the right brachial artery, while a second angiographic catheter was positioned in the aortic root using the percutaneous retrograde femoral arterial technic. Systemic arterial pressure was recorded by means of an indwelling Cournand needle. With the patient placed in the right anterior oblique position, the left ventricle was then opacified by power injection of 50 to 65 ml of radiographic contrast material into the aortic root, while cineangiograms were exposed at 60 or 80 frames/sec. During the filming period, the full left ventricular pressure pulse via the micromanometer, as well as systemic arterial and high gain left ventricular pressures, were recorded at 200 mm/sec together with a signal indicating the moment of each radiographic exposure, thus permitting the correlation of angiographic and pressure events.

At postoperative study, following aortic valve replacement left ventricular pressure was measured by the transseptal technic3 employing a standard catheter-manometer system. Immediately following the pressure recordings, the catheter was withdrawn to the left atrium, and 50 to 60 ml of radiographic contrast material was injected through it; filming was done at 60 or 80 frames/sec in the right anterior oblique view. Again, the moment of each radiographic exposure was recorded together with the ECG and systemic arterial pressure in order to relate the angiographic and pressure events. On the basis of previous studies4 and our own observations indicating that the time lag in pressure pulse recording from well-flushed conventional catheter-manometer systems does not exceed 10 msec when compared with catheter-tip manometer recordings, no attempt was made to correct for the time delay in the pressure recordings at the postoperative study.

The injection of contrast material into the aortic root at preoperative study and into the left atrium at postoperative study avoided the arrhythmias frequently induced by ventricular injection. In each case, the second and third cardiac cycles following contrast material injection were employed to characterize left ventricular dimensions, thus minimizing the effects of this material on left ventricular performance.5

The technics employed to derive circumferential left ventricular myocardial wall tension (stress) and fiber shortening characteristics have been described in a previous communication.1 Briefly, the left ventricle was considered to be an ellipsoid of revolution, the long axis of which was represented by a line drawn from the mid-mitral valve plane to the ventricular apex and the diameter of the minor left ventricular circumference by a line constructed perpendicular to the long axis, at its midpoint (fig. 1). The radius of the minor left ventricular circumference, ri, and the long axis, L, were then measured frame by frame throughout systole from the cineangiogram, corrected for radiographic magnification and spherical distortion, and correlated with the left ventricular pressure pulse.

Myocardial wall tension in the plane of the

\[
\sigma_c = \frac{\rho \cdot r_1 \cdot (1 - \frac{2L^2}{L^2})}{h}
\]

Figure 1

A schematic representation of the left ventricle (LV), left atrium (LA), and aorta (AO) as projected in the right anterior oblique view is shown at the upper left, with the long axis (L), minor semi-axis (ri), and wall thickness (h) indicated as the corresponding dimensions of the idealized thick-walled model of the ventricle, at the upper right. The force (σc) acting at a tangent to the minor circumference in the slice of myocardium indicated at the ventricular equator is computed from the instantaneous ventricular dimensions and corresponding intracavitary pressure (P).
minor left ventricular circumference, that is circumferential hoop stress, was computed throughout contraction as:

\[
\text{Tension} = \text{P} \times r_1 \left(1 - \frac{2r_2}{L_2}\right)/h
\]

where \(\text{P}\) = intracavitary pressure in \(\text{g/cm}^2\), and \(h\) = left ventricular wall thickness in cm, also measured on the cineangiogram.

The velocity of circumferential fiber shortening was then calculated at the left ventricular midwall as the instantaneous rate of change of the midwall circumference, \(2\pi d (r_1 + h/2)/dt\), and expressed as a function of the corresponding instantaneous circumference, \(2\pi (r_1 + h/2)\), as cm/sec/cm, or circumferences (circ)/sec.

During diastole, left ventricular dimensions were also measured at 50-msec intervals throughout two consecutive filling periods and were correlated with the corresponding pressure to derive left ventricular pressure-radius curves both before and following operation.

**Results**

Hemodynamic findings and tension-velocity-length measurements before and following correction of aortic regurgitation (AR) are shown in table 1, together with ranges of normal values for left ventricular (LV) dimensions and tension-velocity measurements obtained in a previously studied group of 11 patients without valvular heart disease.\(^1\)\(^,\)\(^2\) The level of LV myocardial inotropic state was estimated in each patient from tension-velocity measurements.\(^1\) In one patient (V.R.), normal myocardial inotropic state was evidenced preoperatively by a velocity of circumferential fiber shortening at maximum wall tension \((V_{CR})\) of 1.66 circ/sec (normal, > 1.40 circ/sec), while in the remaining patients depressed myocardial function was reflected in reduced \(V_{CR}\), ranging from 0.13 to 1.07 circ/sec at levels of wall tensions which were normal or only slightly greater than normal.

**Hemodynamic and Clinical Findings**

Measurements of aortic diastolic pressure, LV end-diastolic pressure (LVEDP), and cardiac index (CI) before and after operation are shown in figure 2. Preoperatively LVEDP was elevated in each of the four patients with depressed inotropic state to levels that ranged from 20 to 45 mm Hg; postoperatively LVEDP declined to normal or near-normal levels in each instance. In addition, in the three patients in whom CI was reduced preoperatively, this measurement increased to normal levels. In the patient with normal contractility (V.R.) LVEDP and CI were normal at both preoperative and postoperative studies. Hemodynamic improvement was associated with clinical benefit in each patient; of four patients in functional class III (New York Heart Association) two improved to class I and two to class II, while the one patient in class IV improved to class II postoperatively.

**Left Ventricular Dimensions and Extent of Fiber Shortening**

The internal end-diastolic LV circumference was increased in all patients preoperatively, ranging from 21.6 to 26.8 cm (normal, < 17.3 cm).
cm). Postoperatively the end-diastolic internal circumference decreased by 0.9 to 3.8 cm (3.5 to 17.6%) in the patients with depressed inotropic state and remained abnormal in each of these patients. In the patient with normal inotropic state, LV circumference decreased by 7.6 cm (33%) after operation, returning to normal. The extent of circumferential shortening was markedly reduced in three of four patients with depressed contractility, and in these patients it was unchanged or increased slightly but remained abnormal postoperatively. In one patient with mild depression of inotropic state (R.G.), and in the patient (V.R.) in whom inotropic state was normal, the absolute extent of shortening decreased after operation, while the per cent of shortening was virtually unchanged, remaining within normal limits.

**Myocardial Wall Tension**

End-diastolic wall tension was increased in all patients preoperatively, principally reflecting the elevated LVEDP, since after operation end-diastolic tension returned to normal or near-normal levels even when LV circumference did not decline significantly. The time course of tension development was not altered by correction of AR. Thus, in patients with depressed inotropic state (fig. 3) LV tension showed little decline during the course of contraction either before or after operation.

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**Table 1**

<table>
<thead>
<tr>
<th>Patient</th>
<th>HR</th>
<th>CI (L/min/m²)</th>
<th>Pressure (mm Hg)</th>
<th>LV wall thickness (cm)</th>
<th>Internal ED circ. (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>R.G.</td>
<td>Preop</td>
<td>80</td>
<td>2.67</td>
<td>144/20</td>
<td>1.06</td>
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<tr>
<td></td>
<td>Postop</td>
<td>75</td>
<td>4.08</td>
<td>190/13</td>
<td>1.35</td>
</tr>
<tr>
<td>L.C.</td>
<td>Preop</td>
<td>96</td>
<td>1.94</td>
<td>98/35</td>
<td>0.99</td>
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<tr>
<td></td>
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<td>100</td>
<td>3.35</td>
<td>120/12</td>
<td>1.28</td>
</tr>
<tr>
<td>E.K.</td>
<td>Preop</td>
<td>96</td>
<td>1.89</td>
<td>162/45</td>
<td>1.22</td>
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<tr>
<td></td>
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<td>2.70</td>
<td>137/7</td>
<td>1.29</td>
</tr>
<tr>
<td>V.R.</td>
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<td>96</td>
<td>3.38</td>
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<td>0.94</td>
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<tr>
<td></td>
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<td>3.50</td>
<td>135/10</td>
<td>1.03</td>
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<tr>
<td>J.N.</td>
<td>Preop</td>
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<td>2.19</td>
<td>170/41</td>
<td>1.24</td>
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<tr>
<td></td>
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<td>2.76</td>
<td>138/16</td>
<td>1.14</td>
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<tr>
<td>Range of normal values</td>
<td></td>
<td></td>
<td></td>
<td>0.69</td>
<td>0.93</td>
</tr>
</tbody>
</table>

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**Figure 3**

The time course of left ventricular (LV) wall tension in g/cm² is shown in the upper panels, and velocity of circumferential fiber shortening (Vcf), in circ/sec, in the lower panels. Both are plotted on the respective vertical axes against time during contraction, on the horizontal axis and are from a patient with depressed inotropic state (E.K.). Measurements obtained at preoperative study prior to correction of aortic regurgitation are shown in the left panels, and those obtained following relief of aortic regurgitation in the right panels. Vertical dashed lines indicate the limits of the ejection period.

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Maximum wall tension was increased slightly in two patients and within normal limits in three preoperatively, averaging 464 g/cm². After operation it declined in each patient, reaching normal levels in all but one patient and averaging 370 g/cm². The maximum rate of tension development decreased in the patient with normal inotropic state from a level of 7,680 g/cm²/sec to 4,610 g/cm²/sec after operation (normal range for patients with normal circumferences, 3,800 to 6,700 g/cm²/sec). The maximum rate of tension development was unchanged in patients with depressed inotropic state, averaging 3,700 g/cm²/sec (range, 3,060 to 4,490 g/cm²/sec) preoperatively and 3,940 g/cm²/sec (range, 3,000 to 4,370 g/cm²/sec) postoperatively. The time required to achieve maximum tension was also unchanged in all patients, averaging 194 msec from the onset of contraction (range, 117 to 217 msec) preoperatively and 170 msec (range, 134 to 200 msec) postoperatively.

### Velocity of Circumferential Fiber Shortening (V CF)

Neither the time course of fiber shortening (fig. 3) nor maximum V CF (fig. 4) was altered by correction of AR, maximum V CF remaining normal in the patient with normal inotropic state, and reduced in patients with depressed inotropic state postoperatively, despite consistent reductions in wall tension to levels within the normal range.

**Tension-Velocity Relations and Inotropic State**

V CF at maximum wall tension, which can be assumed equal to V CE, is plotted together with

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<table>
<thead>
<tr>
<th>Shortening of internal</th>
<th>Maximum V CF (Midwall)</th>
<th>V CF Midwall</th>
</tr>
</thead>
<tbody>
<tr>
<td>% cm</td>
<td>cm/sec</td>
<td>c/sec</td>
</tr>
<tr>
<td>------</td>
<td>--------</td>
<td>-------</td>
</tr>
<tr>
<td>6.1</td>
<td>28.2</td>
<td>96</td>
</tr>
<tr>
<td>5.4</td>
<td>30.3</td>
<td>25</td>
</tr>
<tr>
<td>2.4</td>
<td>9.0</td>
<td>190</td>
</tr>
<tr>
<td>1.9</td>
<td>7.4</td>
<td>41</td>
</tr>
<tr>
<td>2.0</td>
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</tr>
<tr>
<td>2.8</td>
<td>11.1</td>
<td>51</td>
</tr>
<tr>
<td>8.1</td>
<td>35.0</td>
<td>60</td>
</tr>
<tr>
<td>5.5</td>
<td>35.9</td>
<td>35</td>
</tr>
<tr>
<td>1.7</td>
<td>5.5</td>
<td>158</td>
</tr>
<tr>
<td>3.2</td>
<td>13.3</td>
<td>51</td>
</tr>
<tr>
<td>3.4</td>
<td>27.4</td>
<td>35</td>
</tr>
<tr>
<td>6.9</td>
<td>46.9</td>
<td>51</td>
</tr>
</tbody>
</table>

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![Figure 4](http://circ.ahajournals.org/)

**Figure 4**

Velocity of circumferential fiber shortening at maximum wall tension (V CF), in c/sec, is plotted on the vertical axis together with corresponding values of maximum tension, in g/cm² on the horizontal axis for each patient in preoperative (closed circles) and postoperative study (open circles). The directional change in each patient is indicated by the arrow. Open squares represent tension-velocity measurements in 11 previously studied patients with normal left ventricular function without valvular regurgitation.
corresponding levels of wall tension at preoperative and postoperative studies in all patients in figure 5. While small increases in VCE occurred following operation in three patients with depressed inotropic state, this increase could be explained by the reciprocal decline in wall tension. In the patient with normal inotropic state before operation, the failure of VCE to increase despite the reduction of maximum wall tension may be explained by the concomitant substantial decrease in resting fiber length (internal circumference) after operation.

**Diastolic Pressure-Length Relations**

The relation between the radius of the minor circumference and LV pressure during diastole in the preoperative and postoperative studies is shown in a representative patient with depressed inotropic state in figure 6 and in the patient with normal inotropic state in

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**Figure 5**

Maximum velocity of circumferential fiber shortening (maximum VCE), circ/sec, is shown for each patient in preoperative (closed circles) and postoperative study (open circles). The lower limit of normal for maximum VCE observed in previously studied patients with normal left ventricular function without valvular regurgitation is shown by the horizontal dashed line.

**Figure 6**

Left ventricular (LV) internal radius is plotted on the horizontal axis with corresponding measurements of LV pressure on the vertical axis throughout the diastolic filling period in preoperative study (closed circles) and postoperative study (open circles) in a patient with depressed myocardial function. The crosshatched area defines the range of pressure-radius values observed in a previously studied group of patients with normal LV function without valvular regurgitation.

**Figure 7**

Left ventricular (LV) internal radius is plotted on the horizontal axis, with corresponding values of LV pressure on the vertical axis, in a patient with aortic regurgitation and normal myocardial inotropic state throughout diastole at preoperative (closed circles) and postoperative study (open circles). The crosshatched area indicates the range of diastolic pressure-radius measurements observed in patients with normal LV myocardial function without valvular regurgitation.
figure 7. In each patient with depressed myocardial performance, while LVEDP was markedly reduced postoperatively, the end-diastolic radius was only slightly smaller than before operation, and the pressure radius measurements throughout diastole fell on the same steep curve as the preoperative measurements. In contrast, in the patient with normal inotropic state the diastolic pressure-radius curve which preoperatively was displaced rightward, shifted leftward into the normal range, LVEDP being unchanged despite a substantial reduction in radius.

Discussion

Previous studies in this laboratory have shown that in patients with comparable degrees of severe aortic regurgitation (AR) hemodynamic evidence of impaired ventricular performance appears to occur as a consequence of depressed myocardial function and not as the result of the hemodynamic burden imposed by volume overloading per se.2 Thus, in patients with AR in whom depressed myocardial inotropic state was documented by a reduced velocity of circumferential fiber shortening (\(V_{CF}\)), LVEDP and CI were abnormal, while in patients with equally severe AR without myocardial dysfunction, LVEDP and CI were consistently normal.2 In the present study, each patient with depressed myocardial function exhibited substantial clinical and hemodynamic improvement following correction of AR by prosthetic aortic valve replacement, with CI returning to normal and LVEDP declining from markedly elevated to normal or near-normal levels. Despite dramatic hemodynamic improvement following operation, when myocardial function was assessed by means of tension-velocity-length measurements, it was evident that no change in myocardial inotropic state, or contractility, had occurred, maximum \(V_{CF}\) and \(V_{CF}\) at maximum tension (\(V_{CF}\)) remaining virtually unchanged despite consistent reduction in myocardial wall tension to levels well within the normal range, and despite persistence of an increased resting fiber length. The extent of circumferential fiber shortening and maximum rate of tension development was also unchanged, further evidence that the contractile properties of the myocardium were unchanged postoperatively. The improvement in CI could be ascribed, then, not to an improvement in inotropic state but simply to obviation of loss in effective stroke volume due to regurgitant leak.

The present study also demonstrated that a fixed abnormality in diastolic LV pressure-volume characteristics, determined from preoperative and postoperative measurements of LV pressure and radius during diastole, had occurred in patients with depressed myocardial function. Thus, because the ventricle was operating on a steep pressure-radius curve, a small decrease in end-diastolic dimensions after relief of AR resulted in a dramatic decline in LVEDP postoperatively (fig. 6). Although the LVEDP was normal or near-normal in these patients in the presence of substantially increased LV dimensions, consistent with increased LV compliance, the postoperative pressure-radius measurements fell on the same curve as the preoperative measurements. This observation is consistent with an alteration in LV pressure-volume characteristics in these patients that was not reversible within the 7 to 10 mo following operation. In contrast, in the patient with normal inotropic state, a marked leftward shift in the LV pressure-radius curve occurred after correction of volume overload (fig. 7). While it has been recognized that LV dilatation is a primary compensatory response in AR6-9 and that this dilatation may occur without increase in LVEDP,8,9 the mechanism by which this apparent increase in myocardial compliance occurs has not been defined. In the present study, the demonstration that the LV myocardium may become stiffer postoperatively, LVEDP being unchanged while LV dimensions decline, suggests that in patients with normal inotropic state, LV dilatation may be analogous to stress relaxation, or more properly, creep.10 This observation could not be explained by a reduction in LV mass with
regression of hypertrophy, since this change might be expected to result in an increase rather than a decrease in myocardial compliance.

The observation that marked depression of myocardial function may persist following correction of the mechanical defect despite a return to normal hemodynamic values emphasizes the importance of knowledge of ventricular geometry and dimensional changes in estimation of the level of myocardial function. The findings of the present study also have important implications regarding the approach to the management of patients with chronic valvular lesions. While postoperative studies were carried out within 7 to 10 mo after the operation, the observation that neither myocardial inotropic state nor diastolic compliance characteristics were altered by operation after this time in patients with depressed myocardial function points out the desirability of operative intervention in such patients prior to the development of evidence of depressed LV performance. With improvement in prosthetic devices, these considerations may provide justification for earlier operative intervention in patients with chronic mechanical overloading of the left ventricle.

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