Correlation of Echocardiographic Wall Stress and Left Ventricular Pressure and Function in Aortic Stenosis

NICHOLAS L. DEPACE, M.D., JIAN-FANG REN, M.D., ABDULMASSIH S. ISKANDRIAN, M.D., MORRIS N. KOTLER, M.D., A-HAMID HAKKI, M.D., AND BERNARD L. SEGAL, M.D.

SUMMARY Previous studies have suggested that left ventricular pressure (P) can be predicted in patients with aortic stenosis by the equation \( P = 235h/r \), where 235 is a constant peak wall stress (\( \sigma \)), h is end-systolic wall thickness, and r is end-systolic dimension/2; h and r are measured by M-mode echocardiography. In 73 patients with aortic stenosis (valve area < 0.7 cm\(^2\)), measured and predicted left ventricular pressure correlated poorly (\( r = 0.17 \)). The measured wall stress in our patients varied from 120 to 250 mm Hg in patients with normal left ventricular function and from 250 to 550 mm Hg in patients with abnormal function. The correlation between \( \sigma \) and h was only fair (\( r = 0.53 \)), because many patients had inappropriate left ventricular hypertrophy. There was a statistically significant correlation between ejection fraction and \( \sigma \) (\( r = 0.62 \)) and between ejection fraction and end-systolic dimension (\( r = -0.70 \)), but there was considerable scatter of ejection fractions for any given end-systolic dimension.

We conclude that \( \sigma \) is not constant in aortic stenosis, and the use of a constant \( \sigma \) to predict left ventricular pressure is unreliable; inappropriate left ventricular hypertrophy may explain why \( \sigma \) is not constant. M-mode echocardiography is not reliable in assessing the severity of aortic stenosis in adults; such assessment requires precise measurements of pressure gradients and flow by cardiac catheterization.

SEVERAL studies\(^1-3\) of cardiac muscle preparation, both isolated and in the intact heart, have shown that the extent and the velocity of fiber shortening are inversely related to the force; this finding emphasizes the importance of afterload on cardiac performance. According to Laplace’s law, the stress (or the force per unit area) along the circumference of the left ventricle at its minor axis is directly related to wall thickness. The chronic pressure overload in patients with aortic stenosis results in depression of left ventricular function unless compensatory hypertrophy develops.\(^4\) Therefore, measurement of wall stress in these patients provides a useful index to evaluate cardiac performance.

Gunther and Grossman\(^6\) and Carabello et al.\(^5\) found that in some patients with aortic stenosis, poor cardiac performance was the result of inappropriate hypertrophy rather than depression of myocardial contractility. Determination of wall stress from left ventricular pressure and angiographic or echocardiographic measurement of left ventricular dimension and wall thickness requires invasive procedures that are time-consuming and carry some risk. Several investigators have estimated the left ventricular peak pressure and the pressure gradient across the aortic valve in both children and adults with aortic stenosis by assuming a constant wall stress and measuring the radius and wall thickness of the left ventricle by echocardiography.\(^6-11\)

In this study, we examined the relation among left ventricular ejection fraction, wall stress, left ventricular pressure, cavity dimension, and wall thickness in a large number of patients with aortic stenosis to ascertain (1) whether the assumption of constant wall stress is valid in patients with aortic stenosis; (2) the relation between wall stress and degree of left ventricular hypertrophy; and (3) what determines the observed correlation between ejection fraction and wall stress in these patients.

Methods

We received the records of the cardiac catheterization laboratory in our institution between 1979 and 1981, and identified 91 patients with severe aortic stenosis (aortic valve area < 0.7 cm\(^2\)). The diagnosis was established by standard cardiac catheterization with measurement of the left ventricular and aortic pressures and the cardiac output, as previously reported.\(^12\)

Patients with more than trivial regurgitation were excluded, as were patients with previous aortic valve surgery and those with concomitant mitral stenosis or mitral regurgitation of more than mild degree. Eventually, 18 of the 91 patients were excluded because left ventricular ejection fraction measurements were not available or the echocardiograms were of poor quality.

The remaining 73 patients included 25 women and 48 men, ages 32–76 years (mean 59 years). Thirty of the 73 patients had concomitant coronary artery disease (defined as 70% or more diameter narrowing of one or more vessels) (table 1).

M-mode echocardiograms\(^13\) were available in each of these patients within 48 hours of the catheterization studies. In brief, we performed echocardiographic studies using an ultrasonoscope with a 2.5-MHz, medium (7.5-sonometer), internally focused transducer. Permanent records were obtained with a recorder at paper speeds of 50 and 100 mm/sec. The end-systolic diameter of the left ventricular cavity and end-systolic posterior wall thickness were measured at the smallest
left ventricular dimension. The remaining measurements were made, according to the recommendation made by the American Society of Echocardiography, from leading edge to leading edge.\textsuperscript{14} The percent systolic shortening of the left ventricular minor axis was calculated as (end-diastolic internal diameter − end-systolic internal diameter)/end-diastolic internal diameter × 100.

The end-systolic wall stress was calculated by two methods. The first method measured the end-systolic meridional wall stress ($10^3$ dyn/cm\(^2\)), as described by Grossman et al.\textsuperscript{15} and Reichek et al.\textsuperscript{16}:

$$0.334 \frac{P \times ESD}{h (1 + \frac{h}{ESD})}$$

where P is left ventricular peak pressure, h is the end-systolic posterior wall thickness, and ESD is the end-systolic internal dimension. With the second method, we measured the circumferential peak wall stress (mm Hg) using the method of Quinones et al.\textsuperscript{17}:

$$\frac{Pr}{h (1 - [2r^2/L^2])},$$

where r is equal to end-systolic dimension divided by 2 and L is the long axis of the left ventricle assumed to be equal to twice the end-systolic dimension. This formula can be simplified to

$$\frac{P \times r}{h}.$$

We measured the left ventricular ejection fraction from the single-plane contrast ventriculogram in the right anterior oblique projection using the area-length method.\textsuperscript{18} The results in patients with aortic stenosis (13 men and seven women, ages 17–73 years; mean 36.5 years) were compared with those in 20 normal subjects who had high-quality echocardiograms, who were free from underlying heart disease according to their history, physical examination, ECG, chest x-ray, and who had echocardiographically determined ejection fraction of 55% or greater. The blood pressure in these patients was obtained by the cuff method.

**Prediction of Left Ventricular Systolic Pressure**

Previous investigators\textsuperscript{5–11} have used a constant wall stress 225–245 mm Hg to predict left ventricular systolic pressure in patients with aortic stenosis, using the formula

$$P = \frac{\sigma \times h}{ESD},$$

where P is left ventricular pressure, $\sigma$ is wall stress, h is end-systolic posterior wall thickness, and ESD is end-systolic dimension. We used the same formula and a wall stress of 235 mm Hg, which is the average of the values used by other investigators, to predict the left ventricular pressure in our patients.

**Table 1. Patients with Aortic Stenosis and With and Without Associated Coronary Artery Disease**

<table>
<thead>
<tr>
<th></th>
<th>No CAD (n = 43)</th>
<th>With CAD (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59.8 ± 11.2</td>
<td>62.6 ± 9.7</td>
</tr>
<tr>
<td>LV peak pressure</td>
<td>203.9 ± 26.5</td>
<td>201.9 ± 38.7</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>24.3 ± 13.7</td>
<td>25.83 ± 11.72</td>
</tr>
<tr>
<td>Aortic pressure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>125.4 ± 23.1\textsuperscript{*}</td>
<td>141.3 ± 25.6</td>
</tr>
<tr>
<td>Diastolic</td>
<td>71.8 ± 14.8</td>
<td>71.7 ± 12.2</td>
</tr>
<tr>
<td>LV-AO mean gradient</td>
<td>80.1 ± 30.5\textsuperscript{*}</td>
<td>60.5 ± 24.4</td>
</tr>
<tr>
<td>AVA (cm(^2))</td>
<td>0.49±0.16</td>
<td>0.55±0.19</td>
</tr>
<tr>
<td>Associated MVD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trivial</td>
<td>16 (37%)</td>
<td>14 (47%)</td>
</tr>
<tr>
<td>Mild</td>
<td>3 (7%)</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>49.8 ± 19.1</td>
<td>47.9 ± 17.6</td>
</tr>
<tr>
<td>CAD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-vessel disease</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>2-vessel disease</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>3-vessel disease</td>
<td>0</td>
<td>13</td>
</tr>
</tbody>
</table>

\textsuperscript{*}p < 0.01.

Abbreviations: CAD = coronary artery disease; LV = left ventricular; EDP = end-diastolic pressure; AO = aortic; AVA = aortic valve area; MVD = mitral valve disease; EF = ejection fraction.

**Statistical Analysis**

The t test or analysis of variance was performed when appropriate. Linearity of relationship between two variables was assessed by linear regression analysis; $p < 0.05$ was considered significant. Results are expressed as mean ± SEM or ± SD.

**Results**

**Normal Subjects**

The echocardiographic measurements in the normal subjects are shown in table 2. The upper limit for peak circumferential wall stress in the normal population was 195 mm Hg.

**Patients with Aortic Stenosis**

In patients with aortic stenosis, meridional wall stress and circumferential wall stress correlated well ($r = 0.98$). This was expected, for both measurements were derived from the same original data.

**Correlation Between Predicted and Measured Peak Left Ventricular Function**

The correlation between the predicted and the measured peak left ventricular systolic pressure is shown in figure 1. The correlation is poor ($r = 0.17$). The correlation was better in the 24 patients with depressed fractional shortening (< 23%; $r = 0.57$) than in the remaining patients with normal fractional shortening ($r = 0.14$). The wall stress and left ventricular ejection fraction also differed in these two groups of patients;
TABLE 2. **Echocardiographic Measurements and Calculated Systolic Wall Stress in Normal Subjects**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Systolic pressure (mm Hg)</th>
<th>EDD (cm)</th>
<th>ESD (cm)</th>
<th>h(D)</th>
<th>h(S)</th>
<th>SCWS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>26</td>
<td>126</td>
<td>5.0</td>
<td>3.5</td>
<td>0.9</td>
<td>1.7</td>
<td>129</td>
</tr>
<tr>
<td>2</td>
<td>32</td>
<td>135</td>
<td>3.8</td>
<td>2.6</td>
<td>0.8</td>
<td>1.3</td>
<td>135</td>
</tr>
<tr>
<td>3</td>
<td>44</td>
<td>136</td>
<td>4.1</td>
<td>2.7</td>
<td>1.3</td>
<td>2.0</td>
<td>92</td>
</tr>
<tr>
<td>4</td>
<td>29</td>
<td>110</td>
<td>5.2</td>
<td>3.5</td>
<td>1.0</td>
<td>1.6</td>
<td>120</td>
</tr>
<tr>
<td>5</td>
<td>30</td>
<td>132</td>
<td>5.0</td>
<td>3.3</td>
<td>1.0</td>
<td>1.5</td>
<td>145</td>
</tr>
<tr>
<td>6</td>
<td>31</td>
<td>140</td>
<td>4.8</td>
<td>3.2</td>
<td>0.8</td>
<td>1.3</td>
<td>172</td>
</tr>
<tr>
<td>7</td>
<td>54</td>
<td>140</td>
<td>4.7</td>
<td>3.2</td>
<td>1.0</td>
<td>1.5</td>
<td>150</td>
</tr>
<tr>
<td>8</td>
<td>23</td>
<td>120</td>
<td>4.0</td>
<td>2.2</td>
<td>0.9</td>
<td>1.4</td>
<td>94</td>
</tr>
<tr>
<td>9</td>
<td>31</td>
<td>130</td>
<td>4.3</td>
<td>3.3</td>
<td>1.1</td>
<td>1.7</td>
<td>126</td>
</tr>
<tr>
<td>10</td>
<td>34</td>
<td>130</td>
<td>4.3</td>
<td>2.6</td>
<td>0.9</td>
<td>1.5</td>
<td>112</td>
</tr>
<tr>
<td>11</td>
<td>47</td>
<td>96</td>
<td>5.2</td>
<td>3.6</td>
<td>0.9</td>
<td>1.5</td>
<td>115</td>
</tr>
<tr>
<td>12</td>
<td>17</td>
<td>130</td>
<td>4.4</td>
<td>2.8</td>
<td>1.0</td>
<td>1.7</td>
<td>107</td>
</tr>
<tr>
<td>13</td>
<td>30</td>
<td>130</td>
<td>5.0</td>
<td>3.2</td>
<td>1.2</td>
<td>1.9</td>
<td>109</td>
</tr>
<tr>
<td>14</td>
<td>58</td>
<td>140</td>
<td>5.4</td>
<td>3.6</td>
<td>0.9</td>
<td>1.3</td>
<td>195</td>
</tr>
<tr>
<td>15</td>
<td>73</td>
<td>140</td>
<td>4.8</td>
<td>3.3</td>
<td>1.0</td>
<td>1.5</td>
<td>154</td>
</tr>
<tr>
<td>16</td>
<td>30</td>
<td>120</td>
<td>4.8</td>
<td>3.4</td>
<td>0.8</td>
<td>1.5</td>
<td>136</td>
</tr>
<tr>
<td>17</td>
<td>59</td>
<td>130</td>
<td>5.0</td>
<td>2.8</td>
<td>1.1</td>
<td>1.8</td>
<td>101</td>
</tr>
<tr>
<td>18</td>
<td>23</td>
<td>140</td>
<td>5.0</td>
<td>3.6</td>
<td>1.2</td>
<td>1.8</td>
<td>140</td>
</tr>
<tr>
<td>19</td>
<td>37</td>
<td>135</td>
<td>4.2</td>
<td>2.8</td>
<td>0.9</td>
<td>1.4</td>
<td>130</td>
</tr>
<tr>
<td>20</td>
<td>23</td>
<td>120</td>
<td>4.8</td>
<td>3.3</td>
<td>0.9</td>
<td>1.4</td>
<td>141</td>
</tr>
<tr>
<td>Mean</td>
<td>36.55</td>
<td>127.5</td>
<td>4.69</td>
<td>3.07</td>
<td>0.98</td>
<td>1.57</td>
<td>1297</td>
</tr>
<tr>
<td>± SD</td>
<td>14.66</td>
<td>11.78</td>
<td>0.45</td>
<td>0.41</td>
<td>0.14</td>
<td>2.0</td>
<td>18.9</td>
</tr>
<tr>
<td>± SEM</td>
<td>3.28</td>
<td>2.54</td>
<td>0.10</td>
<td>0.09</td>
<td>0.03</td>
<td>0.05</td>
<td>5.78</td>
</tr>
</tbody>
</table>

Abbreviations: EDD = end-diastolic dimension; ESD = end-systolic dimension; h(D) = wall thickness at diastole; h(S) = wall thickness at systole; SCWS = systolic circumferential wall stress.

Wall stress was 209 ± 7 mm Hg and ejection fraction 55 ± 3% in patients with shortening > 23%, compared with wall stress of 310 ± 9 mm Hg and ejection fraction of 35 ± 3% in patients with shortening < 23% (both p < 0.01). The peak left ventricular pressure was overestimated or underestimated by more than 30 mm Hg in most patients.

**Correlations Between Left Ventricular Ejection Fraction, Wall Stress, End-systolic Dimension, End-systolic Posterior Wall Thickness and Peak Systolic Pressure**

There was a fair correlation between left ventricular ejection fraction and meridional wall stress (r = −0.65) and between the left ventricular ejection fraction and the peak circumferential wall stress (r = −0.62; fig. 2). The peak stress in our patients with left ventricular ejection fraction ≥ 50% was 120–250 mm Hg, and in those with abnormal ejection fractions it was 250–550 mm Hg. The correlation between wall stress and ejection fraction in the 30 patients with aortic stenosis and concomitant coronary artery disease (r = −0.61) was similar to that in patients without associated coronary artery disease. Patients with normal circumferential wall stress (≤ 195 mm Hg) had a normal ejection fraction (≥ 50%), as did one-third of the patients with wall stress of 200–250 mm Hg; all patients with wall stress > 250 mm Hg had an abnormal ejection fraction (< 50%).

The correlations of left ventricular ejection fraction with end-systolic dimension, end-systolic posterior wall thickness and left ventricular peak systolic pressure are shown in figure 3. A statistically significant correlation was only present between ejection fraction and end-systolic dimension (r = −0.70). Thus, 26 of 33 patients with normal end-systolic dimensions had normal ejection fractions, but patients with large end-systolic dimensions had abnormal ejection fractions. A patient with an end-systolic dimension of 3.5 cm may have an ejection fraction of 30–90% (fig. 3A). Therefore, it is difficult to believe that the end-systolic dimension could be clinically useful in predicting ejection fraction.

The peak circumferential wall stress, the end-systolic dimension, end-systolic posterior wall thickness and left ventricular systolic pressure in patients with a normal (≥ 50%) or abnormal (< 50%) ejection fraction are shown in table 3. The end-systolic dimension and wall stress are significantly lower in patients with normal ejection fractions than in patients with abnormal ejection fractions, whereas the end-systolic posterior wall thickness was significantly higher in patients with a normal ejection fraction.

**Discussion**

Wall stress depends on the left ventricular geometry in addition to the pressure, radius and thickness. Taka-
LV FUNCTION IN AORTIC STENOSIS/De Pace et al.

suring wall stress is independent of the long axis of the chamber. However, as shown in this study, meridional wall stress correlates well with circumferential wall stress. Systolic wall stress was approximated from echocardiographic data obtained at end-systole and catheterization-measured peak left ventricular pressure.

The wall stress measured by this method has been referred to as peak or end-systolic stress by different investigators; but, as suggested by Quinones et al., the peak stress occurs early in systole. Therefore, wall stress measured by our method may reflect end-systolic stress more accurately. In addition, the peak systolic pressure occurs at a time corresponding to the smallest dimensions of the left ventricle when assessed by simultaneous echocardiographic measurement.

To avoid confusion, we refer to the measured stress as systolic stress alone. Several investigators have used a constant wall stress, which ranges between 225 and 245 mm Hg, to predict left ventricular peak pressure and the pressure gradient across the aortic valve in patients with aortic stenosis. Studies in both children and in adults have shown good correlation between the measured and the predicted left ventricular pressures, provided the left ventricular systolic function was normal. It has been argued that left ventricular failure increases wall stress because of left ventricular dilation, thereby nullifying the basic assumption of this technique. Schwartz et al. concluded that patients with left ventricular failure can be identified, because the percentage of systolic shortening of the left ventricular minor axis as measured by echocardiography is reduced in these patients.

Gewitz et al. found that noninvasive techniques are not reliable in patients after aortic valve surgery, probably because of the time lag between reduction in the increased afterload and regression of left ventricular hypertrophy. The assumption that wall stress is constant in patients who have aortic stenosis and normal systolic function is not supported by the findings in our study. Thus, the systolic stress in our patients with left ventricular ejection fractions ≥ 50% was 120–250 mm Hg, and in those with an abnormal ejection fraction it was 250–550 mm Hg.

All patients with aortic stenosis and a normal circumferential wall stress (established from data in our normal group) had normal left ventricular ejection fractions (fig. 2). Also, one-third of the patients with a wall stress of 200–250 mm Hg had a normal ejection fraction, whereas the remaining two-thirds had depressed ejection fractions and all patients with wall stress > 250 mm Hg had abnormal ejection fractions. The overall correlation between the ejection fraction and wall stress was fair ($r = -0.62$).

Therefore, in some patients with aortic stenosis, the ejection fraction may remain normal despite higher-than-normal wall stress; in other patients the degree of depression in ejection fraction is disproportionate to the wall stress. These findings indicate some depression in the inotropic state of the left ventricle, as suggested by other investigators.

The variability in wall stress in our patients with

---

**Figure 1.** Correlation between measured and predicted left ventricular peak systolic pressure in 73 patients with aortic stenosis. FS = fractional shortening of the left ventricular minor axis.

**Figure 2.** Correlation between left ventricular ejection fraction and circumferential wall stress in 73 patients with aortic stenosis. CAD = coronary artery disease.
aortic stenosis may explain why our predictions of left ventricular pressure differed from those of other investigators.\textsuperscript{5,11} In most of our patients, we over- or underestimated the peak pressure considerably. This disparity between the predicted and the measured peak left ventricular pressure was greater when patients with fractional shortening $> 23\%$ were considered (fig. 1). This is contrary to the findings of other investigators.\textsuperscript{6} Therefore, the use of M-mode echocardiography is not reliable in assessing the severity of aortic stenosis in adults; such assessment requires precise measurements of pressure gradients and flow by cardiac catheterization.

Gunther and Grossman\textsuperscript{4} suggested that inappropriate left ventricular hypertrophy in some patients with aortic stenosis may result in high wall stress and compromise the left ventricular function. We analyzed the degree of left ventricular hypertrophy based on the end-systolic posterior wall thickness in relation to the wall stress in our patients, and found considerable scatter. Many of our patients with high wall stress had normal end-systolic wall thickness.

Thus, inappropriate hypertrophy in some patients with aortic stenosis may result in high wall stress; therefore, a constant wall stress cannot be assumed in all patients with aortic stenosis. The reasons for inappropriate hypertrophy in some of these patients are not well understood. When we analyzed the correlation between ejection fraction and constituents of the wall stress in our patients, we found that the ejection fraction correlated best with the end-systolic dimension. Further analysis of the correlation between the left ventricular ejection fraction and the end-systolic dimensions revealed that 26 of 33 patients with normal end-systolic dimensions had normal ejection fractions, but patients with large end-systolic dimensions had abnormal ejection fractions. However, a patient with an end-systolic dimension of 3.5 cm may have an ejection fraction of 30–90\% (fig. 3A). Thus, it is difficult to believe that the end-systolic dimension could be clinically useful in predicting ejection fraction.

Left ventricular pressure in this study was not measured during echocardiography. Quinones et al.\textsuperscript{17} found a discrepancy between the systolic blood pressure measured during echocardiography and that measured during catheterization in approximately one-third of these patients. However, they emphasized that the correlation between the measured and the predicted peak left ventricular pressure was almost unchanged.

Precise definitions of the end-systolic dimensions and the end-systolic posterior wall thickness are necessary for accurate estimation of the wall stress, because slight inaccuracies may result in important differences. High-quality echocardiograms may not always be available in patients with aortic stenosis. In fact, some patients were excluded from our study because their echocardiograms were not of high quality.

Our data suggest that in adult patients with aortic stenosis the use of a constant wall stress to predict left ventricular peak systolic pressure results in considerable variation simply because the wall stress is not constant in patients with normal or abnormal left ventricular ejection fractions. Therefore, precise assessment of the degree of aortic stenosis requires measurements of pressure gradients and flow by cardiac catheterization.

\begin{table}
\centering
\begin{tabular}{cccc}
\hline
 & Normal EF & Abnormal EF & $p$ \\
 & (n = 33) & (n = 40) & \\
\hline
LV ESD (cm) & 3.2$\pm 0.09$ & 4.3$\pm 0.13$ & $< 0.001$ \\
LV PWT (cm) & 1.7$\pm 0.04$ & 1.5$\pm 0.04$ & $< 0.01$ \\
LV pressure (mm Hg) & 207$\pm 4$ & 196$\pm 5$ & NS \\
$\sigma$ (mm Hg) & 193$\pm 8$ & 286$\pm 12$ & $< 0.001$ \\
\hline
\end{tabular}
\caption{Left Ventricular Wall Stress, End-systolic Dimension, Posterior Wall Thickness and Pressure in Patients with Aortic Stenosis and Normal or Abnormal Ejection Fractions}
\end{table}

Values are mean $\pm$ SEM.

Abbreviations: EF = ejection fraction; LV = left ventricular; ESD = end-systolic dimension; PWT = end-systolic posterior wall thickness; $P$ = peak systolic pressure; $\sigma$ = peak wall stress.
References
Correlation of echocardiographic wall stress and left ventricular pressure and function in aortic stenosis.
N L DePace, J F Ren, A S Iskandrian, M N Kotler, A H Hakki and B L Segal

_Circulation_. 1983;67:854-859
doi: 10.1161/01.CIR.67.4.854

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1983 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/67/4/854

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/