Angina in Idiopathic Hypertrophic Subaortic Stenosis

A Clinical Correlate of Regional Left Ventricular Dysfunction: A Videometric and Echocardiographic Study

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SUMMARY  Angina, dyspnea and syncope often occur in idiopathic hypertrophic subaortic stenosis (IHSS), but have not hitherto been correlated with conventional measures of left ventricular (LV) function. We assessed regional and global LV function in 18 patients with IHSS by roentgen videometric analysis of biplane left ventriculograms and M-mode echocardiography to investigate 1) whether any relationship could be demonstrated between symptoms and global or regional LV function, and 2) how normal or supernormal global function could be maintained in the presence of severe septal hypokinesis. Syncope occurred in four patients with IHSS, angina in seven and dyspnea in 11. No correlation was demonstrable between any symptom and any measure of global LV function, or between syncope and dyspnea and regional LV function. However, a correlation was shown between angina and regional function in that peak rates of systolic thickening and diastolic thinning of the anterior LV wall in these patients were significantly ($p < 0.01$) less than in patients without angina. In view of the documented relationship between myocardial ischemia and regional wall dynamics, we conclude that angina in IHSS in the absence of coronary artery disease is strongly associated with impaired wall dynamics, although which is cause and which is effect is unresolved. Angina may therefore be useful as an indicator of regional LV dysfunction. Furthermore, the normal pump function is maintained by increased cavity emptying.

THE STRUCTURAL ABNORMALITIES in idiopathic hypertrophic subaortic stenosis (IHSS) that permit a definitive diagnosis can be readily shown by echocardiographic and angiocardiographic techniques. Little is known, however, of the physiologic effects these abnormalities impose on left ventricular (LV) function and less still of their role in the production of symptoms. In this disease, in which the septum makes little or no contribution to changes in cavity volume during filling and emptying, global function is reported to be normal or supernormal and must, therefore, be maintained by either enhanced free-wall dynamics or increased shortening of the long axis of the LV cavity or both.

The purpose of this study was to determine 1) how global LV function can remain normal in the presence of such severe septal abnormalities, and 2) whether a relationship between symptoms and any measure of global or regional LV function could be shown. We therefore examined both global and regional LV function, using roentgen videometric analysis of LV angiograms in 18 patients with IHSS. The septal function was evaluated by echocardiography.

Methods

Patient Population

The 38 patients in this study were divided into two groups. Group 1 consisted of 20 normal patients (eight females and 12 males) 24–64 years old (mean 46 years) investigated for atypical chest pain, all of whom had normal ECGs, negative treadmill exercise ECGs and normal hemodynamics and LV and coronary angiograms. Group 2 consisted of 18 consecutive patients with IHSS (eight female and 10 male) 19–73 years old (mean 50 years) in whom LV angiograms and echocardiograms were technically satisfactory for analysis and in whom intrinsic aortic and mitral valve disease was excluded. Coronary arteriograms in 13 patients showed no luminal stenosis greater than 50%. The diagnosis of IHSS was established echocardiographically by the presence of at least two of the following criteria: 1) asymmetric septal hypertrophy (18 of 18 patients), 2) systolic anterior motion of the mitral valve (16 of 18) and 3) mid-systolic closure of the aortic valve (10 of 18), and hemodynamically by the presence of a LV outflow tract gradient at rest (13 of 18) or upon pharmacologic provocation (17 of 18). The only patient who fulfilled the echocardiographic criteria for IHSS without having a provokable gradient had disproportionate septal thickening involving the apical part of the septum only, which may have accounted for the absence of outflow tract obstruction.

All patients underwent diagnostic catheterization in the fasting state. Fourteen patients were taking propranolol (Inderal) in dosages of 60–400 mg/day (mean 119 mg/day) and four patients were receiving no therapy, two with angina and two without angina. No patient had complete relief of angina with pro-
pranolol, so no patient was transferred from the angina group to the nonangina group. In addition, no patient had taken any medication known to influence LV function within 12 hours of catheterization.

**Angiographic Analysis**

Simultaneous biplane left ventriculograms were obtained in all patients in the 30° right anterior oblique and 60° left anterior oblique projections and recorded on cine film and videotape. Ventricular opacification was achieved with 45–55 ml of meglumine diatrizoate (Renografin-76) injected at 15 ml/sec during held deep inspiration. Biplane video images were recorded as a single split-screen image at 60 fields/sec, as previously reported.9–13 Coronary arteriography was performed by the Sones or Judkins technique.

Ventricular volumes (end-systolic and end-diastolic), calculated stroke volume, ejection fraction and cardiac indices were determined in each patient from the biplane left ventriculograms using the roentgen videometry system. This operator-interactive video-computer system has been described.9–14 The volume-computing program was based on Simpson’s rule.16

Percent shortening of the long axis of the LV cavity and also of the long axis of the total left ventricle was assessed by using as reference points the junction of the aorta and left ventricle — not the midpoint of the aortic valve, which was not often so easily visualized — and the endocardial and epicardial apices, respectively, in systole and diastole (fig. 1). Measurements were made from the end-systolic and end-diastolic frames of the right anterior oblique LV angiograms by means of the videometer and a manually operated electronic cursor that defined these reference points. XY coordinates were generated for these three reference points, and the distances (z) between two points were computed according to the formula

\[ z = 2 \sqrt{(x^2 + y^2)} \]

LV cavity long-axis shortening and total LV long-axis shortening were then expressed as percent reduction of their respective end-diastolic dimensions

\[
\text{long axis (diast)} - \text{long axis (syst)} \times 100% \\
\text{long axis (diast)}
\]

In some patients with IHSS, the free wall and the septum coapt during late systole and obliterate the LV cavity so that the true endocardial apex cannot be visualized angiographically because of almost complete ejection of contrast medium. We therefore designated the apex of the LV cavity as the point that was angiographically outlined most distal to the junction of the aorta and left ventricle, and this represented the effective apical limit of the functional LV cavity at end-systole. The distance between this point and the junction of the aorta and left ventricle was taken as the LV cavity long axis at end-systole and was used to calculate percent shortening of the long axis of the LV cavity (fig. 1). Because this represents functional rather than true cavity long-axis shortening, it was designated as “effective cavity long-axis shortening.”

The videometry system provided automatic recognition of the endocardial and epicardial surfaces indicated by the highlighted endocardial and epicardial ventricular surfaces in the stop-action video display of the LV angiograms (fig. 2). XY coordinates were generated at the intercepts between the television raster lines and the brightened endocardial and epicardial surface lines. Thus, two strings of XY coordinates

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Left ventricular cavity (LV) cavity long axis and total LV axes at end-diastole and end-systole in normal subjects and in patients with idiopathic hypertrophic subaortic stenosis (IHSS). See text.

![Figure 2](https://example.com/figure2.png)

**Figure 2.** Determination of left ventricular wall thickness by stop-action video display of biplane roentgen angiogram with simultaneous recognition of epicardial and endocardial borders. The patient, a 59-year-old woman, was given an injection of 50 ml of Renografin-76. (A magnified view is shown on the right). (From St. John Sutton, MG, Frye RL, Smith HC, Chesebro JH, Ritman EL: Relation between left coronary artery stenosis and regional left ventricular function. Circulation 58: 491, 1978. By permission of the American Heart Association.)
were obtained — one for the endocardial surface and one for the epicardial surface of the region of the midanterior LV wall selected for analysis. Wall thickness was then computed as the average of multiple estimates of the shortest distance between epicardial and endocardial XY coordinates. The distance between these coordinates measured along the television raster lines is equal to wall thickness only if the wall is imaged at right angles to the television raster lines. For this reason, the video camera was rotated through 45° so that the angle of incidence between the television raster lines and the anterior LV wall on the videotape angiograms in the right anterior oblique projection was approximately a right angle.

The region of anterior wall analyzed was the same in all patients, i.e., midway between two reference points: the junction of the aorta and left ventricle and the apex of the heart. Plots of continuous anterior LV wall dimension were obtained in which minimum and maximum thicknesses were identified by computer, and percentage systolic wall thickening was calculated. The maximum positive and negative rates of change of wall thickness were obtained after a running 3-point-average smoothing routine was applied to the continuous wall-thickness plot, which had 60 data points per second (one data point every 16.7 msec). The computer program identified the positive and negative peak rates of change of anterior wall thickness that represent the peak rate of anterior wall thickening and thinning, respectively, and constructed tangents at these points and superimposed them on the wall-thickness plots3 (fig. 3). These two measurements together with percentage systolic wall thickening were used to assess regional LV function. The reproducibility of this technique has been reported.9–11

The ratio of diastolic pressure-time index to systolic pressure-time index (DPTI/SPTI) has been shown to correlate with the ratio of subendocardial to subepicardial coronary blood flow, and this was calculated in the IHSS patients according to the method of Hoffman and Buckberg16 to establish whether a relationship existed between angina and DPTI/SPTI.

Echocardiographic Analysis

Echocardiograms was used only to establish the diagnosis of IHSS and to assess systolic and diastolic septal thicknesses and percent change in thickness. Echocardiograms with simultaneous ECGs were obtained with patients in the left semilateral recumbent position. Echoes of the right and left sides of the septum were obtained at the level of the chordae tendineae to assess end-systolic (VSs) and end-diastolic (VSd) septal thicknesses, from which percentage systolic change in thickness was calculated by means of the formula

\[
\frac{V_{Ss} - V_{Sd}}{V_{Sd}} \times 100%.
\]

Statistical Analysis

Statistical tests of significance were the t test and the rank-sum test. The latter was used to compare differences when the variance of the samples was dissimilar, in which case the ranges and mean values were tabulated.

Results

Normal Subjects (Group 1)

Global Function

The range and mean values of LV end-systolic and end-diastolic volume indices, stroke volume index, ejection fraction, end-diastolic pressure and cardiac index are shown in table 1. Percent systolic shortening of the LV cavity long axis and the total LV long axis are shown in table 1 and figure 4.

Regional Function

The range and mean values for maximum systolic and minimum diastolic anterior wall and septal thicknesses and their respective percent changes in thickness are shown in table 2. Peak rates of anterior wall systolic thickening and diastolic thinning are also shown in table 2 and figure 5.

IHSS Patients (Group 2)

Global Function

End-systolic and end-diastolic volume indices were significantly smaller than normal (p < 0.01), but stroke volume index was normal and ejection fraction was significantly increased (p < 0.01) (table 1). The percent effective shortening of the long axis of the LV cavity was significantly greater than normal (range 21–70%, mean 40%, p < 0.01), whereas percent shortening of the long axis of the total left ventricle
TABLE 1. **Global Measurements of Left Ventricular Function and Hemodynamics**

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Diastolic volume index (ml/m²)</th>
<th>Systolic volume index (ml/m²)</th>
<th>Stroke volume index (ml/m²)</th>
<th>Ejection fraction (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>20</td>
<td>59.1-93.4</td>
<td>14.6-37.9</td>
<td>44.4-78.8</td>
<td>64-87</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(79.8)</td>
<td>(23.2)</td>
<td>(53.5)</td>
<td>(70)</td>
</tr>
<tr>
<td>IHSS</td>
<td>18</td>
<td>44.5-94.2*</td>
<td>8.4-28.3‡</td>
<td>36.1-79.6</td>
<td>64-94‡</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(58.1)</td>
<td>(13.1)</td>
<td>(50.3)</td>
<td>(70)</td>
</tr>
</tbody>
</table>

Values are given as a range, with mean value in parentheses.

*Only 15 patients with IHSS and 15 normal subjects.

†Only 10 patients with IHSS and 12 normal subjects.

§p < 0.01.

Abbreviations: LV = left ventricular; IHSS = idiopathic hypertrophic subaortic stenosis; R = rest; I = isoproterenol.

TABLE 2. **Regional Measurements of Left Ventricular Function**

<table>
<thead>
<tr>
<th>Group</th>
<th>Anterior left ventricular wall</th>
<th>Septum</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic thickness (mm)</td>
<td>Diastolic thickness (mm)</td>
</tr>
<tr>
<td>-------</td>
<td>--------------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>Normal</td>
<td>18 ± 3</td>
<td>10 ± 2</td>
</tr>
<tr>
<td></td>
<td>(45)</td>
<td>(55)</td>
</tr>
<tr>
<td>IHSS</td>
<td>26 ± 6*</td>
<td>16 ± 4*</td>
</tr>
<tr>
<td></td>
<td>(53)</td>
<td>(43)</td>
</tr>
</tbody>
</table>

Values are given as mean ± sd or as a range, with mean value in parentheses.

*p < 0.01.

Abbreviation: IHSS = idiopathic hypertrophic subaortic stenosis.

was significantly less than normal (range 2–16%, mean 6%, p = 0.02) (table 1 and fig. 4).

LV end-diastolic pressure varied widely, with a mean value significantly greater than normal (p < 0.01). LV outflow tract pressure gradients were present in 14 of 18 patients (range 14–100 mm Hg, mean 32 mm Hg); in 17 of 18 patients undergoing provocation with isoproterenol, pressure gradients ranged from 30–170 mm Hg (mean 90 mm Hg).

DPTI/SPTI varied from 0.21–1.20 (mean 0.74),

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**Figure 4.** Plots of computed percent systolic shortening of angiographically visualized ventricular chamber and epicardial silhouette long axis in normal subjects and in patients with idiopathic hypertrophic subaortic stenosis (IHSS) as determined by roentgen videometry.

**Figure 5.** Plots of peak rates of systolic thickening and diastolic thinning of the midanterior left ventricular wall determined by roentgen videometry from right anterior oblique left ventriculograms in normal subjects and in patients with idiopathic hypertrophic subaortic stenosis (IHSS) show no significant difference from normal.
and in 60% of patients it was less than 0.8 (table 3), which indicates ischemic subendocardial muscle, according to established criteria,\textsuperscript{18} while in the normal subjects it ranged from 1.07–1.43 (mean 1.27).

**Regional Function**

End-systolic and end-diastolic anterior wall and septal thicknesses were significantly greater than normal ($p < 0.01$) (table 2). There was greater-than-normal variation in the peak rates of anterior wall systolic thickening and diastolic thinning, but the mean values were normal (table 2 and fig. 5).

**Clinical Symptoms**

**Angina Pectoris**

Angina (functional classes III and IV, New York Heart Association) occurred in seven of 18 patients, all of whom had normal coronary arteriograms. There was no demonstrable relationship between the presence of angina and any measure of global LV function, i.e., end-diastolic pressure, outflow tract pressure gradient, cavity volume indices, ejection fraction or cardiac index. Furthermore, DPTI/SPTI was less than 0.8 in 60% of the patients with IHSS, suggesting subendocardial ischemia; however, this ratio in patients with angina (0.21–1.20) was not different from that in patients without angina (0.35–1.16) (table 3 and fig. 6).

Wall and septal thicknesses and the respective percent changes in thicknesses were different in patients with and in those without angina. The range and mean values for peak rates of systolic thickening of the anterior LV wall in patients with angina were also different from normal but were significantly ($p < 0.01$) less than the values in patients without angina (table 3 and fig. 7). In addition, the range and mean values for peak rates of diastolic thinning in patients with angina were significantly ($p < 0.01$) less than normal (table 2), and also less ($p < 0.01$) than in patients without angina (table 3 and fig. 7). The differences in systolic and diastolic wall dynamics in patients with and those without angina could not be explained by any differences in dosage of propranolol, for the mean dosage in patients with angina (120 mg/day) was not significantly different from that in patients without angina (118 mg/day). There was no demonstrable relationship between systolic or diastolic wall dynamics and DPTI/SPTI.

**Syncope, Dyspnea and Palpitations**

Syncope or near-syncpe, dyspnea and palpitations occurred in 12, 11 and eight of the 18 patients, respectively, and there was no apparent relationship between any of these symptoms and any measure of either global or regional LV function.

**Discussion**

Symptoms of angina, dyspnea, and syncope occur frequently in patients with IHSS but have not correlated with conventional measures of LV function. This may be due in part to the fact that systolic pump function is well preserved in IHSS until late in the course of the disease, whereas abnormalities of ventricular function in diastole are manifest earlier,\textsuperscript{6, 17, 18} as in prolongation of isovolumic relaxation.\textsuperscript{6, 17, 18}

Global parameters of LV function in IHSS present a paradox in that ejection fraction and peak velocity of circumferential shortening are increased, indicating supernormal function,\textsuperscript{7, 8} whereas LV end-diastolic pressure is usually elevated,\textsuperscript{3, 4, 6} indicating increased stiffness and reduced compliance. Of all the

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Peak rate of diastolic thinning (mm/sec)</th>
<th>Peak rate of systolic thickening (mm/sec)</th>
<th>DPTI/SPTI</th>
<th>Dosage of propranolol (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IHSS with angina</td>
<td>7</td>
<td>23–44 ( (36) )</td>
<td>30–66 ( (36) )</td>
<td>0.21–1.20 ( (0.89) )</td>
<td>0–240 ( (120) )</td>
</tr>
<tr>
<td>IHSS without angina</td>
<td>11</td>
<td>35–72 ( (53)* )</td>
<td>30–102 ( (78)* )</td>
<td>0.35–1.16 ( (0.78) )</td>
<td>0–400 ( (118) )</td>
</tr>
</tbody>
</table>

Values are given as a range, with mean values in parentheses.

*\( p < 0.01 \).

Abbreviations: IHSS = idiopathic hypertrophic subaortic stenosis; DPTI = diastolic pressure-time index; SPTI = systolic pressure-time index.
parameters of global LV function, end-diastolic pressure has been considered to be the most reliable long-term prognostic index. In the resting state, however, severe abnormalities of regional LV function may escape detection when cavity function alone is assessed. Therefore, we also quantitated regional function in terms of peak rates of systolic thickening and diastolic thinning of the anterior LV wall, parameters that have proved sensitive to regional myocardial performance.

During ejection, the normal heart decreases cavity volume by simultaneous reduction in transverse and longitudinal LV cavity axes. These respective reductions are brought about by muscle fiber shortening that results in 1) concentric inward movement due to free wall and septal thickening, almost equal contributions being made by the septum and free wall, and 2) approximation of the apex and the base of the heart by contraction of the spirally arranged myocardial fibers. Normally the decrease in LV cavity volume is achieved to a greater extent by reduction in the minor axis than by reduction in the long axis. By contrast, in IHSS, changes in minor axis are achieved almost completely by the free wall because there is little or no contribution from the hypertrophic, hypokinetic septum. Because LV cavity volumes in IHSS were small, a normal stroke volume could only be ejected by more complete cavity emptying. This more complete cavity emptying was achieved by greater-than-normal systolic reduction in both transverse and effective longitudinal LV cavity axes, thus accounting for the normal cardiac output and increased ejection fraction. The significantly increased effective shortening of the cavity long axis in IHSS was not accomplished by greater-than-normal true long-axis shortening (i.e., retraction of the apex toward the base of the heart), but rather by cavity obliteration due to coaptation of the free wall and septum, or effective long-axis shortening (fig. 1). Frame-by-frame, stop-action video display of the LV angiograms showed that the anterior LV wall came into apposition with the septum initially at the mid-cavity level and progressed from there to the apex, obliterating rather than truly shortening the long axis of the LV cavity. Thus, the normal, highly integrated, concentric inward motion of the free wall and septum during ejection was lost, and the left ventricle behaved more like a bellows. The septum acted as the im-mobile base plate of the bellows while the blood was expressed from the cavity as the free wall became contiguous with the septum.

Although effective shortening of the long axis of the LV cavity in IHSS was greater than normal, that of the long axis of the total ventricle (junction of aorta and left ventricle to epicardial apex) (fig. 1) was consistently and significantly reduced. A possible explanation for the latter finding is that the septum, which was two to three times normal thickness, splinted the long axis of the left ventricle and prevented apical retraction. This inability to elevate the apex toward the base of the heart in IHSS may have functional significance, as it may contribute to the abnormal changes in shape conferred on the LV cavity during contraction.

**FIGURE 6.** Plot of ratio of diastolic pressure-time index (DPTI) to systolic pressure-time index (SPTI) in patients with idiopathic hypertrophic subaortic stenosis (IHSS) with and without angina.

**FIGURE 7.** Plots of peak rates of systolic thickening and diastolic thinning of midanterior left ventricular wall determined by roentgen videometry from right anterior oblique left ventriculograms in patients with idiopathic hypertrophic subaortic stenosis (IHSS) with and without angina.
In IHSS there was a wider-than-normal variation of peak rates of systolic thickening and diastolic thinning of the anterior wall, and the highest rates of systolic wall thickening occurred in patients who had the highest peak rates of diastolic wall thinning. Thus, increased systolic and diastolic wall dynamics in IHSS may represent the maximal compensatory response for septal hypokinesis by an anterior wall in which mechanical function is unimpaired, with only minimal involvement with fiber disarray and fibrosis. The reduced peak rates of systolic thickening and diastolic thinning in other patients may be explained by a greater involvement of the free wall with fiber disarray and fibrosis, resulting in change in the properties of the LV wall.

Syncope and dyspnea occurred in a large proportion of the patients with IHSS but varied independently of any measure of global or regional LV function. In contrast, a correlation between angina and regional wall dynamics was shown, although not with global LV function. Angina occurred in seven patients with IHSS, all of whom had normal coronary angiograms. Peak rates of systolic thickening and diastolic wall thinning in patients with angina were both significantly lower than in the IHSS patients without angina. Even so, the mean value for systolic wall thickening in the patients with angina remained within the normal range, indicating preservation of systolic function until late in the course of the disease, whereas there was significant reduction in the mean value for peak rate of diastolic wall thinning. Thus, reduction in peak rates of diastolic wall thinning precedes the reduction in peak rates of systolic wall thickening. The greater impairment of wall dynamics in patients with angina than in those without could not be explained by differences in wall thickness or in the dosage of propranolol, which were similar in both groups.

Furthermore, systolic and diastolic wall dynamics in patients with angina varied independently of LV end-diastolic pressure, outflow tract pressure gradient, cavity volume, cardiac output and ejection fraction.

To investigate whether the reduction in the peak rate of diastolic wall thinning might result in angina by reducing subendocardial coronary blood flow by slower-than-normal diastolic decrease in wall tension and coronary vascular resistance, we determined the diastolic and systolic pressure-time indices. These indices reflect blood supply and oxygen demand, respectively, and a good correlation has been shown between DPTI/SPTI and the ratio of subendocardial to subepicardial blood flow. A pronounced decrease in DPTI/SPTI to less than 0.8 at rest has been associated with subendocardial ischemia and increased production of lactate. Reduction of DPTI/SPTI below 0.8 was shown in 60% of the patients with IHSS, but this occurred equally in patients with and in those without angina and also could not be explained by any difference in wall thickness between these two groups. In addition to the lack of relationship between the presence of angina and DPTI/SPTI, there was no correlation between the peak rates of diastolic wall thinning and DPTI/SPTI. Despite the lack of apparent correlation between subendocardial coronary blood flow (DPTI/SPTI) and diastolic LV wall dynamics, in view of the significant association between angina and wall dynamics and also the strong relationship between myocardial ischemia and decreased wall dynamics, angina may be due to the reduction in peak rates of diastolic thinning of the LV wall, although precisely which is cause and which is effect is unresolved.

We conclude that in IHSS 1) despite small LV cavity volume, normal or supernormal pump function is maintained by more complete cavity emptying achieved by systolic coaptation of the free wall and the septum that obliterates the cavity and thereby greatly reduces its effective long axis; 2) regional myocardial muscle dysfunction may be detected with this technique when cavity function is still normal; 3) angina in the absence of coronary artery disease is strongly associated with and may be due to severely impaired LV diastolic wall dynamics; and 4) angina might therefore be useful as a clinical indicator of regional LV dysfunction.

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Intrapericardial Abnormalities in Patients with Pericardial Effusion

Findings by Two-dimensional Echocardiography

RANDOLPH P. MARTIN, M.D., ROBERT BOWDEN, M.D., KATHERINE FILLY, B.S.,
AND RICHARD L. POPP, M.D.

SUMMARY Ten patients with pericardial fluid who also had striking band-like intrapericardial echoes by two-dimensional ultrasonic sector scanning are reported. Four patients had prior mediastinal radiation and four patients had severe renal disease. One patient had purulent pericarditis and one patient had traumatic hemopericardium. The two-dimensional images permitted recognition of loculation of fluid and led to the suspicion of thickened pericardial membranes. Three postradiation patients had both tamponade and constriction; tamponade alone was present in four additional patients. These data are preliminary but suggest that such findings by two-dimensional echocardiography should alert us to consider an effusive-constrictive form of pericardial disease.

ECHOCARDIOGRAPHY has proved extremely useful as a noninvasive method for diagnosing pericardial effusion. M-mode echocardiography can reliably establish the presence of pericardial effusion, but the narrow field of view and lack of spatial orientation of echoes make assessment of the size and distribution of the fluid difficult. Wide-angle, two-dimensional echocardiography provides dynamic images of the myocardium and pericardium in multiple cross-sectional planes, permitting assessment of the size, distribution and postural shift of the fluid within the pericardial space. In this article, we describe adhesive or fibrinous pericardial bands producing loculation of pericardial fluid, pericardial thickening, and a probable intrapericardial thrombus recognized during echocardiographic studies in 10 patients with suspected or proved pericardial effusions.

Methods

Patients

Ten patients studied over 30 months in the Stanford Medical Center Echocardiographic Laboratory for suspected pericardial effusions had echo-producing intrapericardial structures on the original examination (not seen in other patients with pericardial effusions). The 10 patients ranged in age from 17–56 years. Pericardial effusion was suspected on the basis of their clinical situation and chest x-ray. The 10 patients reported presented within three general clinical settings (table 1).

Four patients had oncologic processes and had received anterior mediastinal radiation, in excess of 5000 rads, from 1–2 years before the echocardiographic evaluation. Two patients had oat cell carcinoma and two patients had Hodgkin's lymphoma. All four patients had attempted pericardiocentesis for suspected cardiac tamponade before their two-dimensional echocardiographic examinations. In three
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