left ventricular outflow obstruction in patients with obstructive asymmetric septal hypertrophy (idiopathic hypertrophic subaortic stenosis). Am J Cardiol 35: 337, 1975

Electrocardiographic Findings in Patients with Obstructive and Nonobstructive Hypertrophic Cardiomyopathy

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SUMMARY One hundred and thirty-four patients with hypertrophic cardiomyopathy were evaluated by standard 12-lead electrocardiography. Normal electrocardiograms were extremely uncommon, occurring in less than 7% of each subgroup of patients (i.e., those with or without either symptoms or obstruction to left ventricular outflow), with the exception of those who were both asymptomatic and had no left ventricular outflow obstruction. Even in this subgroup, however, normal electrocardiograms occurred in only 27% of patients. Repolarization abnormalities and left ventricular hypertrophy were the most common abnormalities, occurring in 81% and 62%, respectively, of the total population. A broad spectrum of other electrocardiographic abnormalities was found, but none was unique to hypertrophic cardiomyopathy.

Patients with vs those without electrocardiographic left ventricular hypertrophy or left atrial abnormality had significantly (P < 0.005) greater mean ventricular septal thickness (22 ± 0.6 vs 19 ± 0.6 mm) and left atrial dimension (48 ± 1 vs 40 ± 1 mm) measured by echocardiography, and significantly (P < 0.01) higher mean pulmonary capillary wedge pressure (16 ± 1 vs 10 ± 1 mm Hg) and left ventricular end-diastolic pressure (20 ± 1 vs 15 ± 1 mm Hg). The high prevalence and diverse nature of electrocardiographic abnormalities suggest that any patient with an unusual and unexplained electrocardiogram should be suspected of having hypertrophic cardiomyopathy even if the physical examination is normal, as is often the case in patients without obstruction.

IN THE DECADE PRIOR TO 1968 there were numerous reports of electrocardiographic findings in patients with hypertrophic cardiomyopathy.1–10 Most of the patients were reported to have obstruction of left ventricular outflow, since the resulting murmur heard on physical examination and the gradient

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recorded at catheterization were the major criteria used to establish the diagnosis at that time. In the early 1970s Henry et al.16 and Abbasi et al.17 utilized asymmetric septal hypertrophy, detected by echocardiography, as a disease marker. This technique led to the recognition of a broad pathophysiologic spectrum of hypertrophic cardiomyopathy, including patients with or without either symptoms or left ventricular outflow obstruction. Little data have subsequently been published regarding the electrocardiographic findings in this group of patients.

This investigation examined the prevalence and diagnostic usefulness of electrocardiographic abnor-
malities in 134 patients with hypertrophic cardiomyopathy, and related the electrocardiographic abnormalities to symptomatic status as well as echocardiographic and hemodynamic findings.

Methods

Patient Population

One hundred thirty-four patients with hypertrophic cardiomyopathy were studied. Diagnosis was established by the echocardiographic demonstration of a ventricular septal-to-posterobasal left ventricular free-wall thickness ratio ≥ 1.3."16

Ninety-six of the patients were male and 38 were female. We are following several hundred patients with hypertrophic cardiomyopathy in our clinic, and decided for this investigation to select and study a representative sample of the patients. We examined our records and entered into the study the first 85 patients we found who had hypertrophic cardiomyopathy unequivocally identified by echocardiography. Another 49 patients were selected primarily to provide a more representative cross section of the clinical and hemodynamic subgroups of patients with this disease than was provided by the first 85 patients. Of these 49 patients: 1) 16 were selected because they had large left atrial dimension on echocardiogram, 2) 19 were studied primarily because of a history of syncope, 3) seven came from families in which premature death (i.e., < 50 years of age) had occurred in two or more first degree relatives who had hypertrophic cardiomyopathy19, and 4) seven patients had a history of paroxysmal atrial fibrillation.

Fifty of the 134 patients underwent cardiac catheterization within one week of the electrocardiogram. In these 50 patients the resulting hemodynamic data were used as the primary method for determining the presence of obstruction. Patients were considered to have obstruction if the gradient measured under basal conditions or with provocation (i.e., isoproterenol infusion or Valsalva maneuver) was 30 mm Hg or more. Those with smaller gradients or no gradient were considered not to have obstruction.

Echocardiography was used to estimate the magnitude of obstruction in 78 of the 84 patients without recent catheterization. This estimation was based on the degree of approximation of the anterior mitral valve leaflet to the ventricular septum.19,20 Patients were classified as having obstruction if the gradient estimated by echocardiography was 30 mm Hg or more under basal conditions or with provocation (Valsalva maneuver or amyl nitrite inhalation). Finally, six patients who had not undergone recent catheterization and in whom mitral valve echocardiograms were not of sufficiently good quality to estimate the magnitude of obstruction, were classified as either obstructed or nonobstructed on the basis of the presence or absence of a systolic ejection murmur of grade III/VI or louder under basal conditions and/or with provocation (Valsalva maneuver or amyl nitrite inhalation).

Patients were considered symptomatic if they had one or more of the following symptoms: chest pain, dyspnea on exertion, fatigue, orthopnea, paroxysmal nocturnal dyspnea, lightheadedness, or syncope. Of the 55 symptomatic patients with obstruction, 36 (65%) were in functional class II (New York Heart Association classification), 18 (33%) were in class III and one (2%) was in class IV. Of the 40 symptomatic patients without obstruction, 34 (85%) were in functional class II, and six (15%) were in class III. Mean age of the total group of patients was 38 ± 2 years (SEM) (range 9–65 years). Ages of patients in all subgroups were similar, with the exception of obstructed patients without symptoms who were significantly younger than obstructed patients with symptoms (26 ± 3 years vs 41 ± 2 years, \( P < 0.001 \)). None of the patients had undergone cardiac operation before the electrocardiogram.

Electrocardiographic Procedure and Criteria

Patients taking cardioactive medications had these drugs discontinued 36–48 hours before the electrocardiogram, with the exception of four patients who had chronic atrial fibrillation and continued to take digoxin. Standard 12-lead electrocardiograms were performed with the patients in the supine position during quiet respiration. Either a Sanborn heat sensitive paper or a Marquette ink writer electrocardiographic machine was used. The electrocardiogram was recorded from each patient within one week of the most recent electrocardiographic study.

Reprilization abnormalities21 were diagnosed if any of the following was present: 1) ST segment displacement ≥ 1 mV from the isoelectric line in the limb leads, 2) ST segment displacement upward with upward convexity in the precordial leads, 3) ST segment displacement downward in the precordial leads, or 4) widening of the angle between the mean spatial QRS vector and the mean spatial T vector (i.e., > 45° in the frontal plane, > 60° in the anteroposterior plane in those over 30 years of age, or > 90° in those under 30 years of age). These criteria were supplemented by those outlined by Friedman21 for abnormal T waves in adults and children.

Left ventricular hypertrophy was diagnosed using the point-score system of Romhilt and Estes.22 Four or more points were considered evidence of left ventricular hypertrophy. Point scores were not calculated for children less than 16 years of age (\( N = 8 \)), patients with QRS duration ≥ 0.12 seconds (\( N = 19 \)), patients with atrial fibrillation (\( N = 7 \)), or patients with indeterminate axis (\( N = 2 \)).

Left atrial abnormality23 was diagnosed if the product of the depth and duration of the negative portion of the P wave in lead V1 was greater than –0.03 mV-sec.

Abnormal Q waves21 were defined by the presence of any of the following: 1) a Q wave in leads I, II, or aVF ≥ 0.04 seconds wide, > 2 mV deep and > 25% of the following R wave, 2) a Q wave in lead aVF, ≥ 0.04 seconds wide, > 2 mV deep, and > 50% of the succeeding R wave, 3) a Q wave in lead III ≥ 0.04 seconds wide and > 6 mV deep or associated
with Q waves in leads II and aV_{F}, 4) a Q wave in the left precordial leads (V_{1}–V_{6}) \geq 0.04 \text{ seconds wide, } > 2 \text{ mV deep and } > 15\% \text{ of the succeeding R wave, 5) qR, QR or Qr patterns in the right precordial leads (V_{1}–V_{6}), or 6) a QS pattern in one or more of the following leads: I, II, V_{5}–V_{6}. 

Right atrial enlargement\textsuperscript{a} was diagnosed if there were peaked P waves in leads II and III or V_{1} \geq 2.5 \text{ mV in amplitude with a mean axis of more than } +60^\circ. 

Left anterior hemiblock\textsuperscript{a} was defined by: 1) left axis deviation (mean QRS frontal plane axis of \(-30^\circ \text{ to } -90^\circ\), 2) a small initial Q in lead I and R in lead III, and 3) a QRS duration < 0.12 seconds if this was an isolated conduction defect. 

Left posterior hemiblock\textsuperscript{a} was diagnosed if there was: 1) right axis deviation (\( \geq +120^\circ \)), 2) a small initial R wave in lead I and Q wave in lead III, 3) QRS duration < 0.12 seconds if this was an isolated conduction defect, and 4) no additional evidence for right ventricular hypertrophy. 

Left bundle branch block\textsuperscript{a} was defined by: 1) a QRS duration \geq 0.12 \text{ seconds in the extremity leads, 2) delayed onset of the intrinsoid deflection in } V_{6}, 3) broad slurred R waves in the left precordial leads without a Q wave and 4) ST segment displacement downward and T wave inversion in the left precordial leads. 

Right bundle branch block\textsuperscript{a} was defined by: a QRS duration \geq 0.12 \text{ seconds and terminal QRS forces directed to the right and anteriorly with normal early QRS forces.} 

Intraventricular conduction defect\textsuperscript{a} was diagnosed if the QRS duration was \geq 0.12 \text{ seconds but did not meet the additional criteria for either left or right bundle branch block.} 

Preexcitation (Wolff-Parkinson-White pattern)\textsuperscript{a} was defined by: 1) a short P-Q interval (\leq 0.10 \text{ sec}), 2) a wide QRS (0.11–0.14 \text{ sec}) and 3) slurred onset of the QRS (\Delta wave). 

Right ventricular hypertrophy\textsuperscript{a} was defined by: 1) QRS duration of < 0.12 seconds, 2) rightward mean QRS axis in the frontal plane (between +110^\circ \text{ and } +180^\circ) \text{ and an R or R' in lead } V_{1} \text{ of } 5 \text{ mV or greater in amplitude, with an R/S ratio in lead } V_{1} \text{ of 1 or greater.} 

\begin{table}[ht]
\centering
\caption{Frequency of Common Electrocardiographic Abnormalities in Patients with Hypertrophic Cardiomyopathy}
\begin{tabular}{|l|c|c|c|c|}
\hline
\textbf{ECG Finding} & \textbf{Obstructed} & \textbf{Asymptomatic} & \textbf{Nonobstructed} & \textbf{Asymptomatic} \\
\hline
Abnormal ECG & 54/55 (98) & 13/13 (100) & 37/40 (93) & 19/26 (73)* \\
Repolarization abnormalities & 48/55 (87) & 11/13 (85) & 34/40 (85) & 15/26 (58)* \\
Left ventricular hypertrophy & 31/38 (82) & 8/10 (80) & 19/33 (58) & 7/21 (33)† \\
Left atrial abnormality & 37/50 (74) & 5/12 (42) & 14/38 (37)* & 5/26 (19)* \\
Left axis deviation \((0 \text{ to } -90^\circ\)) & 25/53 (47) & 8/13 (62) & 12/40 (30) & 5/26 (19)† \\
Abnormal Q waves & 23/55 (42) & 5/13 (38) & 8/40 (20) & 8/26 (31) \\
Right atrial enlargement & 9/50 (18) & 3/13 (23) & 3/38 (8) & 1/26 (4) \\
Right atrial enlargement and right ventricular hypertrophy & 7/38 (18) & 2/10 (20) & 3/33 (9) & 0/21 (0) \\
P-R interval > 0.20 sec & 5/50 (10) & 2/13 (15) & 4/38 (11) & 1/26 (4) \\
\hline
\end{tabular}
\textsuperscript{a}Numbers in parentheses indicate percentages. 
\textsuperscript{*Comparison with obstructed asymptomatic patients \((P < 0.05)\).} 
\textsuperscript{†Comparison with obstructed and nonobstructed symptomatic patients \((P < 0.05)\).} 
\textsuperscript{\#Comparison with obstructed symptomatic and asymptomatic patients \((P < 0.05)\).}
\end{table}

\begin{table}[ht]
\centering
\caption{Frequency of Less Common Electrocardiographic Abnormalities in Patients with Hypertrophic Cardiomyopathy}
\begin{tabular}{|l|c|c|c|c|}
\hline
\textbf{ECG Finding} & \textbf{Obstructed} & \textbf{Asymptomatic} & \textbf{Nonobstructed} & \textbf{Asymptomatic} \\
\hline
Atrial fibrillation & 5/55 (9) & 0/13 (0) & 2/38 (5) & 0/26 (0) \\
Left anterior hemiblock & 6/55 (11) & 1/13 (8) & 4/40 (10) & 1/26 (4) \\
Left posterior hemiblock & 1/55 (2) & 1/13 (8) & 1/40 (3) & 0/26 (0) \\
Left bundle branch block & 3/55 (5) & 0/13 (0) & 1/40 (3) & 0/26 (0) \\
Right bundle branch block & 1/55 (2) & 0/13 (0) & 2/40 (5) & 1/26 (4) \\
Intraventricular conduction defect & 5/55 (9) & 1/13 (8) & 1/40 (3) & 1/26 (4) \\
Pre-excitation (WPW) & 0/55 (0) & 0/13 (0) & 1/40 (3) & 2/26 (8) \\
Atrioventricular dissociation & 0/55 (0) & 2/13 (15) & 0/40 (0) & 0/26 (0) \\
Right ventricular hypertrophy & 0/55 (0) & 0/13 (0) & 1/40 (3) & 0/26 (0) \\
\hline
\end{tabular}
\textsuperscript{a}Numbers in parentheses indicate percentages. 
\textsuperscript{Abbreviation: WPW = Wolff-Parkinson-White pattern.}
\end{table}
Echocardiographic Measurements

Echocardiographic studies were performed using an Aerotech transducer (2.25 MHz, 1.25 cm diameter, unfocused) with a modified Ekoline 20A or a Hoffrel 201 ultrasound unit using methods previously described. The ultrasound signal was connected via a custom-built video amplifier to a Honeywell Visicorder and was recorded continuously on light sensitive paper. Thickness of the ventricular septum was measured below the tips of the mitral valve leaflets before atrial systole but after rapid ventricular filling. Posterobasal left ventricular free wall thickness was measured at the level of the tips of the mitral valve leaflets during the same phase of the cardiac cycle. One hundred twenty-one patients had echocardiograms of sufficiently good quality to measure left atrial transverse dimension. This dimension was taken as the maximal distance between the posterior aortic root wall and the posterior left atrial wall and was measured in the damped portion of the record when the ultrasonic beam passed through the aortic leaflets.

Where appropriate, the t test or the χ-square test was used to assess statistical significance.

Results

Prevalence of Electrocardiographic Abnormalities

Abnormal electrocardiograms were found in 123 of 134 (92%) patients. Repolarization abnormalities and left ventricular hypertrophy each occurred in more than 70% of patients and were the two most common electrocardiographic abnormalities in each subgroup of patients (tables 1 and 2). The left ventricular "strain pattern" was present as part of the criteria for left ventricular hypertrophy in 59 of the 65 (91%) patients with left ventricular hypertrophy. Asymptomatic patients without left ventricular outflow obstruction (when compared to symptomatic patients with outflow obstruction) had significantly lower frequencies of abnormal electrocardiograms, repolarization abnormalities, left ventricular hypertrophy, left atrial abnormality and left axis deviation. The combination of right atrial enlargement and left ventricular hypertrophy, described in a previous report of electrocardiographic findings in patients with hypertrophic cardiomyopathy, was included among the more common findings in our patients.

More leftward QRS frontal plane axes were found in patients with left ventricular outflow obstruction compared with those without obstruction (fig. 1). Two patients had indeterminate QRS axes, including one with low QRS voltage (< 5 mV) in all limb leads. The prevalence of electrocardiographic abnormalities, including left ventricular hypertrophy and abnormal Q waves, was similar in younger and older patients (fig. 2). In addition, the prevalence of electrocardiographic abnormalities in the 49 patients selected because they were in specific clinical subgroups was similar to the prevalence of such abnormalities in the other 85 patients.

Figure 1. Mean electrical axes in the frontal plane of 132 patients with hypertrophic cardiomyopathy. The arrow from the closed circle = overall mean for obstructed patients (N = 66); the arrow from the open circle = overall mean for nonobstructed patients (N = 66).

Figure 2. Prevalence of electrocardiographic abnormalities in younger and older age groups. Frequencies of abnormalities were not significantly different in the two age groups.
Comparison of Electrocardiographic Findings with Echocardiographic and Hemodynamic Measurements

Patients with abnormal electrocardiograms had significantly greater mean echocardiographic septal thickness and mean left atrial dimension than patients with normal electrocardiograms (table 3). Similarly, patients with the specific electrocardiographic findings of left ventricular hypertrophy, left atrial abnormality or repolarization abnormalities had greater mean septal thickness and left atrial dimension by echocardiography than patients without these electrocardiographic findings. In contrast, the presence of prominent abnormal Q waves, including those suggesting prominent septal forces on electrocardiogram, did not significantly correlate with increased septal thickness or septal-to-free wall ratio. Abnormal electrocardiograms, including abnormal Q waves, were found in 60% (three of five) of the asymptomatic patients without obstruction who had only slight increases in both septal thickness (14–16 mm) and septal-to-free wall ratio (1.3 to 1.4).

Significantly higher mean pulmonary capillary wedge pressure (or left atrial mean pressure) and higher mean left ventricular end-diastolic pressure were found in patients with electrocardiographic left ventricular hypertrophy (table 4). Left atrial abnormality on electrocardiogram was also associated with significantly higher mean left ventricular end-diastolic pressure.

Discussion

The prevalence of abnormal electrocardiograms in patients with hypertrophic cardiomyopathy has been reported previously as 10%, 18% 18, 28 and almost 100%. 14, 20 Our study indicates that normal electrocardiograms are rare in patients with hypertrophic cardiomyopathy, particularly if they are asymptomatic or if they have obstruction to left ventricular outflow.

Although patients in our study were not chosen because of known electrocardiographic abnormalities, it is possible that the observed prevalence of these abnormalities may have been influenced by selection factors. The prevalence of electrocardiographic abnormalities in the 49 patients selected because they were in specific clinical subgroups was similar to the prevalence of such abnormalities in the other 85 patients. Moreover, even asymptomatic subjects without obstruction had a high prevalence of abnormalities (73%), indicating that selection of patients on the basis of severe symptoms could not account for our results.

A broad spectrum of electrocardiographic abnormalities was seen in patients with hypertrophic cardiomyopathy, but no abnormality appeared to be unique to this disease. Furthermore, no electrocardiographic abnormality that was found frequently occurred exclusively in obstructed or nonobstructed patients. This was consistent with the findings in a previous study 20 of children with obstructed and nonobstructed forms of this disease. In that study the prevalence of abnormal electrocardiograms in asymp-

<table>
<thead>
<tr>
<th>ECG Findings</th>
<th>Number of patients</th>
<th>Mean age in years ± SEM</th>
<th>Septal thickness (mm)</th>
<th>Free wall thickness (mm)</th>
<th>Septal-to-free wall ratio</th>
<th>Left atrial dimension (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal ECG</td>
<td>123</td>
<td>38 ± 1</td>
<td>22 ± 0.4*</td>
<td>13 ± 0.2</td>
<td>1.7 ± 0.03</td>
<td>46 ± 0.9† (112)</td>
</tr>
<tr>
<td>Normal ECG</td>
<td>11</td>
<td>35 ± 4</td>
<td>16 ± 0.6*</td>
<td>11 ± 0.2</td>
<td>1.5 ± 0.05</td>
<td>34 ± 2† (9)</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Present</td>
<td>65</td>
<td>39 ± 2</td>
<td>22 ± 0.6*</td>
<td>13 ± 0.2</td>
<td>1.9 ± 0.1</td>
<td>48 ± 1† (59)</td>
</tr>
<tr>
<td>Not present</td>
<td>37</td>
<td>38 ± 2</td>
<td>19 ± 0.6*</td>
<td>12 ± 0.3</td>
<td>1.6 ± 0.05</td>
<td>40 ± 1† (33)</td>
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<tr>
<td>Left atrial abnormality</td>
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<tr>
<td>Present</td>
<td>61</td>
<td>39 ± 2</td>
<td>24 ± 0.6*</td>
<td>13 ± 0.2</td>
<td>1.8 ± 0.04</td>
<td>49 ± 1† (56)</td>
</tr>
<tr>
<td>Not present</td>
<td>65</td>
<td>35 ± 2</td>
<td>19 ± 0.5*</td>
<td>12 ± 0.2</td>
<td>1.6 ± 0.03</td>
<td>40 ± 0.9† (58)</td>
</tr>
<tr>
<td>Repolarization abnormalities</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>Present</td>
<td>108</td>
<td>39 ± 1</td>
<td>22 ± 0.5*</td>
<td>13 ± 0.2</td>
<td>1.7 ± 0.03</td>
<td>46 ± 0.9† (97)</td>
</tr>
<tr>
<td>Not present</td>
<td>26</td>
<td>34 ± 3</td>
<td>17 ± 0.6*</td>
<td>12 ± 0.3</td>
<td>1.5 ± 0.03</td>
<td>40 ± 2† (24)</td>
</tr>
</tbody>
</table>

Numbers in parentheses = number of patients with echocardiograms of sufficiently good quality to measure left atrial dimension.

*P < 0.001. 
†P < 0.001.

Table 4. Comparison of ECG Findings with Hemodynamic Data

<table>
<thead>
<tr>
<th>ECG Findings</th>
<th>LVEDP in mm Hg (mean ± SEM)</th>
<th>PCW or LA mean pressure in mm Hg (mean ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left ventricular hypertrophy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>20 ± 1 (27)*</td>
<td>16 ± 1 (24)*</td>
</tr>
<tr>
<td>Not present</td>
<td>15 ± 1 (10)*</td>
<td>10 ± 1 (10)*</td>
</tr>
<tr>
<td>Left atrial abnormality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>22 ± 1.5 (26)*</td>
<td>17 ± 1 (29)*</td>
</tr>
<tr>
<td>Not present</td>
<td>16 ± 1 (19)*</td>
<td>13 ± 1.5 (15)*</td>
</tr>
</tbody>
</table>

Numbers in parentheses = number of patients.

*P < 0.01.
†P = not significant.

Abbreviations: LVEDP = left ventricular end-diastolic pressure; PCW = pulmonary capillary wedge; LA = left atrial.
tomatic children (average age 10 ± 1 years) without left ventricular outflow obstruction was low (30%), and contrasts with the 73% prevalence found in the same, but older (average age 38 ± 3 years), subgroup in the present study. This suggests that the frequency of abnormal electrocardiograms may increase as children with hypertrophic cardiomyopathy reach adulthood.

The combination of right atrial enlargement and left ventricular hypertrophy was surprisingly common. Goodwin et al.6 have suggested that identification of this combination is suggestive of hypertrophic cardiomyopathy, since it is said to be rare in valvular aortic stenosis, which is often included in the differential diagnosis of this disease. Prominent septal forces in young patients are another helpful diagnostic echocardiographic finding, because they suggest hypertrophic cardiomyopathy. However, similar to Halpern et al,20 we could not demonstrate a significant relationship between the presence or absence of prominent septal forces and the relative thickening of the ventricular septum compared with the free wall.

In a recent study24 we reported a high prevalence of arrhythmias in asymptomatic patients who had only slightly increased septal-to-free wall ratios and had no obstruction to left ventricular outflow. The high prevalence of electrocardiographic abnormalities on the routine electrocardiogram in this subgroup lends further credibility to the concept that such patients have clinically important disease25 and are not just part of a normal distribution curve of echocardiographic findings.38

The correlations of electrocardiographic findings with echocardiographic and hemodynamic measurements in our study are generally similar to those reported by Joyce et al.29 who also found that normal electrocardiograms were associated with less septal thickening and that electrocardiographic left ventricular hypertrophy was not associated with higher left ventricular outflow gradients. However, in contrast to their study, we did not find that electrocardiographic left ventricular hypertrophy was accompanied by increased left ventricular free wall thickness.

This study demonstrates that normal electrocardiograms are unusual in all subgroups of patients with hypertrophic cardiomyopathy. In addition, significant correlations exist between electrocardiographic findings and echocardiographic as well as hemodynamic measurements. However, the degree of overlap of electrocardiographic findings among the subgroups of patients limits the usefulness of the electrocardiogram as a predictor of specific echocardiographic or hemodynamic abnormalities in the individual patient with this disease.

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THE EFFECTIVE MANAGEMENT of ventricular arrhythmias is a difficult therapeutic challenge. Sudden death is usually due to ventricular fibrillation, and ventricular ectopy has been considered as a major risk factor. Identification of ventricular ectopy in the ambulatory patient is not easy, however, since arrhythmias often occur sporadically, and patients are not always aware of their presence. The availability of recorders that can monitor the electrocardiogram continuously in ambulatory patients has greatly enhanced our ability to detect and understand the nature of ventricular arrhythmias beyond that possible with standard resting electrocardiography (45 seconds) or electrocardiographic rhythm strips (several minutes to 1 hour). Twenty-four-hour long-term electrocardiographic monitoring has further documented cardiac arrhythmias as a cause of disability and death and has found widespread application as a means of quantitating ventricular ectopy and guiding antiarrhythmic therapy.

The evaluation of antiarrhythmic agents has led to increased awareness of the limitations of present methods of arrhythmia detection. Previous studies have noted the inconstant relation of ventricular ectopy to patient physical activity, diurnal, neural and psychological factors, and current monitoring recommendations have increased from 8–24 hours to better account for these physiologic variables.

The use of exercise testing has been considered complementary to 24-hour ambulatory electrocardiographic monitoring. Recently, however, the reproducibility of arrhythmia detection by this technique has been questioned by Sheps et al. They demonstrated that, in two successive exercise tests performed only 45 minutes apart, there was a marked variability of arrhythmia frequency, thus limiting the usefulness of paired exercise tests in evaluating antiarrhythmic therapy.

SUMMARY Variations in the frequency of ventricular premature depolarizations (VPDs) were evaluated with three consecutive 24-hour long-term electrocardiograph monitor recordings from 15 clinically stable patients with various cardiac disorders. Mean hourly VPD frequencies ranged from 37–1,801 per hour. Data were subjected to 4 and 5 factor nested analyses of variance. The extent of spontaneous variation in arrhythmia frequency that occurred in individual patients from day to day was 23%, between 8-hour periods within days was 29%, and from hour to hour was 48%. In addition, the variability between repeated three-day monitoring periods over time was quantified in five patients and found to be 37%. This analysis determined that to distinguish a reduction in VPD frequency attributable to therapeutic intervention rather than biologic or spontaneous variation alone required a greater than 83% reduction in VPD frequency if only two 24-hour monitoring periods were compared, and greater than 65% reduction if two 72-hour periods were compared. The limitations of routine 24-hour electrocardiographic monitoring must be considered in diagnostic and therapeutic decision-making.

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