Arrhythmias in Variant Angina Pectoris

Relationship of Arrhythmias to ST-segment Elevation and R-wave Changes

NICHOLAS Z. KERIN, M.D., MELVYN RUBENFIRE, M.D., MANSOOR NAINI, M.D., WALDEMAR J. WAJSZCZUK, M.D., ADELFO PAMATMAT, M.D., AND PHILIP N. CASCADE, M.D.

SUMMARY Twenty-six patients with variant angina pectoris (VAP) were studied 1) to determine whether the degree of ST-segment elevation and R-wave changes correlate with the development of arrhythmias; and 2) to evaluate the relationship between the prevalence of arrhythmias, the severity of coronary artery disease, left ventricular function and wall motion.

Serious arrhythmias were found in 12 patients (46%) (ventricular fibrillation in two, ventricular tachycardia in four, ventricular premature complexes [VPCs] [≥5 VPCs/min, multifocal and R-on-T phenomenon] in four, and second- and third-degree atrioventricular block in three). All twelve patients with arrhythmias had ST-segment elevation ≥ 0.4 mV during VAP (range 0.4–1.6 mV). R-wave amplitude was compared before and during episodes of VAP and expressed as %ΔR. An increase in R > 10% was seen in 10 of 12 patients with arrhythmias (group 1) and in only six of 14 patients without arrhythmias (group 2) (p < 0.05).

Twenty-three of the 26 patients underwent coronary angiography and ventriculography, and one was examined by autopsy. Sixteen patients in this group had single or multiple high-grade obstructive lesions, while the remaining eight had normal coronary arteriograms.

Arrhythmias were more common in the group with coronary obstructive disease (66%) than in the group with normal coronary arteriograms (44%). There was no significant difference between patients with arrhythmias (group 1) and those without arrhythmias (group 2) in the coronary arteriographic score or left ventricular ejection fraction.

The data suggest that arrhythmias occur frequently during VAP and correlate well with the degree of ST-segment elevation and %ΔR. In patients with VAP, arrhythmias are not contingent upon preexisting coronary artery disease or left ventricular ejection fraction, and are more commonly detected in patients with normal coronary arteriograms.

IN 1959 and 1960, Prinzmetal et al.1-2 delineated a variant form of angina pectoris (VAP) that primarily differs from Heberden's classic angina pectoris in that the pain is frequently spontaneous, occurs at rest, and is unrelated to physical activity or emotion. The pain in VAP generally lasts longer and may be more severe than in classic angina pectoris. Attacks tend to be cyclic, often recurring at a specific time of day. Episodes of pain are accompanied by transient ST-segment elevation with reciprocal changes in the standard leads, as opposed to typical ST-segment depression. VAP has been reported4-7 in patients with normal coronary arteriograms,1-2,8 as well as in those with angiographically demonstrated focal lesions that affect the proximal segment of the coronary arteries.

Abnormalities of rhythm and conduction may be associated with VAP.6-18 During pain, the wide spectrum of reported arrhythmias includes second- and third-degree atrioventricular block,6,7,9,10 ventricular tachycardia and ventricular fibrillation,10,11 ventricular premature complexes (VPCs),12,14 ventricular asystole15 and atrial fibrillation.18

We studied 26 patients with VAP to determine 1) how the degree of ST-segment elevation and R-wave changes relate to the development of arrhythmias; and 2) whether there is a correlation between the prevalence of arrhythmias, the location and severity of coronary obstruction, left ventricular function and wall motion.

Materials and Methods

Patients

Twenty-six consecutive patients (21 men and five women ages 32–76) seen from 1970–1978 were selected for analysis. Selection was based on the occurrence of chest pain at rest and ST-segment elevation on the ECG during pain. Although 15 patients had multiple clinical episodes of VAP, only episodes that were accompanied by electrocardiographic recordings were included in table 1.

ECG and Arrhythmias

The ST-segment elevation of each patient was measured on the ECG, with the TP segment as the isoelectric line. Deviation was measured in millivolts to the nearest point at 0.06 second after the nadir of the S wave (when present) or from the Q wave. Of the leads reflecting subepicardial injury, the one showing the highest ST-segment elevation was used for measurement. During VAP, 24 of the patients were monitored through serial ECGs and two (nos. 7 and 22) were monitored by continuous calibrated magnetic tape recording (Avionics Model 445 Electrocar-
diocorder). Leads V2 and V3 were monitored in patient 7 and lead MV5 was monitored in patient 22. When patients had repeated episodes of VAP, the greatest ST-segment elevation recorded during those episodes was used for statistical purposes in Table 1. The arrhythmias and the magnitude of ST-segment elevation reported in Table 1 represent data from the same episode of VAP. R-wave amplitude was measured in the leads showing the highest ST-segment elevation and, at its maximum change (during VAP), averaged from the isoelectric line to the peak of the R wave (in mm) for 10 consecutive beats. R-wave amplitude was compared before (R0) and during (R1) episodes of VAP and expressed as the percentage change in R wave (%ΔR)

\[
\frac{R_1 - R_0}{R_0} \times 100
\]

We considered ΔR ≥ 10% an increase, and negative change a decrease.

Left-heart Catheterization and Coronary Angiography

These procedures were performed using the Sones or Judkins technique.17, 18 Left ventricular angiography was performed in the 30° right anterior oblique (RAO) view. Left ventricular end-systolic and end-diastolic volumes were measured from the RAO left ventricular angiograms, according to the modified method of Dodge,19 and a left ventricular ejection fraction was calculated. The coronary arteries were separated into branches in accordance with the reporting system of the American Heart Association.20 Coronary artery anatomy was reviewed by at least two investigators.

A coronary arterial score was determined for each patient according to the method described by Friesinger et al.21 This method scores each of the three major coronary arteries on a scale of 0–5 as follows: 0 = no abnormalities seen on selective coronary arteriograms; 1 = minimal irregularity of the coronary artery lumen; 2 = localized narrowing > 50% but < 90% of cross-sectional area; 3 = multiple

Table 1. Clinical Data

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age/sex (years)</th>
<th>ST-segment elevation (mV)</th>
<th>Leads</th>
<th>Arrhythmias</th>
<th>R wave (mV) Before VAP</th>
<th>During VAP</th>
<th>After VAP</th>
<th>Leads</th>
<th>%ΔR</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39/M</td>
<td>1.6</td>
<td>L1; V1-V4</td>
<td>VT, VPCs</td>
<td>1.9</td>
<td>2.7</td>
<td>2.0</td>
<td>V3</td>
<td>42</td>
</tr>
<tr>
<td>2</td>
<td>59/M</td>
<td>0.8</td>
<td>V1-V4</td>
<td>VT, VPCs</td>
<td>2.0</td>
<td>2.4</td>
<td>2.2</td>
<td>V4</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>68/M</td>
<td>0.7</td>
<td>L1; V1-V4</td>
<td>VPCs</td>
<td>1.9</td>
<td>2.7</td>
<td>2.0</td>
<td>L1</td>
<td>42</td>
</tr>
<tr>
<td>4</td>
<td>39/M</td>
<td>0.6</td>
<td>L2-L3; aVF</td>
<td>2° AV block</td>
<td>0.4</td>
<td>1.2</td>
<td>0.6</td>
<td>L2</td>
<td>200</td>
</tr>
<tr>
<td>5</td>
<td>62/M</td>
<td>0.6</td>
<td>L1; aVL; V1-V4</td>
<td>VF</td>
<td>0.9</td>
<td>1.6</td>
<td>1.0</td>
<td>L1</td>
<td>78</td>
</tr>
<tr>
<td>6</td>
<td>54/M</td>
<td>0.6</td>
<td>L1; V1-V4</td>
<td>VT</td>
<td>1.1</td>
<td>1.0</td>
<td>1.0</td>
<td>V4</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>64/M</td>
<td>0.6</td>
<td>V2</td>
<td>2° AV block</td>
<td>0.2</td>
<td>0.7</td>
<td>0.2</td>
<td>V2</td>
<td>250</td>
</tr>
<tr>
<td>8</td>
<td>54/M</td>
<td>0.5</td>
<td>V1-V6</td>
<td>VPCs</td>
<td>0.6</td>
<td>1.2</td>
<td>0.7</td>
<td>V4</td>
<td>100</td>
</tr>
<tr>
<td>9</td>
<td>76/M</td>
<td>0.5</td>
<td>L1-L3; aVF</td>
<td>VPCs</td>
<td>0.7</td>
<td>0.75</td>
<td>0.7</td>
<td>L2</td>
<td>7</td>
</tr>
<tr>
<td>10</td>
<td>57/M</td>
<td>0.4</td>
<td>L2-L3; aVF</td>
<td>VF</td>
<td>0.7</td>
<td>0.8</td>
<td>0.8</td>
<td>L2</td>
<td>14</td>
</tr>
<tr>
<td>11</td>
<td>47/F</td>
<td>0.4</td>
<td>V1-V4</td>
<td>VT, VPCs</td>
<td>1.1</td>
<td>1.8</td>
<td>1.2</td>
<td>V4</td>
<td>64</td>
</tr>
<tr>
<td>12</td>
<td>58/F</td>
<td>0.4</td>
<td>L2-L3; aVF</td>
<td>VPCs</td>
<td>0.9</td>
<td>1.4</td>
<td>0.9</td>
<td>L2</td>
<td>56</td>
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<tr>
<td>13</td>
<td>32/M</td>
<td>0.3</td>
<td>V2-V4</td>
<td>—</td>
<td>0.6</td>
<td>0.7</td>
<td>0.6</td>
<td>V4</td>
<td>17</td>
</tr>
<tr>
<td>14</td>
<td>48/M</td>
<td>0.3</td>
<td>V1-V4</td>
<td>—</td>
<td>1.0</td>
<td>1.0</td>
<td>1.0</td>
<td>V3</td>
<td>0</td>
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<tr>
<td>15</td>
<td>67/M</td>
<td>0.3</td>
<td>V1-V4</td>
<td>—</td>
<td>0.2</td>
<td>0.2</td>
<td>0.2</td>
<td>V2</td>
<td>0</td>
</tr>
<tr>
<td>16</td>
<td>61/M</td>
<td>0.3</td>
<td>V1-V6</td>
<td>—</td>
<td>0.7</td>
<td>1.7</td>
<td>0.7</td>
<td>V4</td>
<td>143</td>
</tr>
<tr>
<td>17</td>
<td>51/M</td>
<td>0.3</td>
<td>V1-V5</td>
<td>—</td>
<td>0.9</td>
<td>0.9</td>
<td>0.8</td>
<td>V4</td>
<td>0</td>
</tr>
<tr>
<td>18</td>
<td>49/M</td>
<td>0.25</td>
<td>L2-L3; aVF</td>
<td>—</td>
<td>1.0</td>
<td>1.1</td>
<td>0.95</td>
<td>L2</td>
<td>10</td>
</tr>
<tr>
<td>19</td>
<td>57/M</td>
<td>0.25</td>
<td>L2-L3; aVF</td>
<td>—</td>
<td>1.0</td>
<td>1.1</td>
<td>1.1</td>
<td>L2</td>
<td>9</td>
</tr>
<tr>
<td>20</td>
<td>56/F</td>
<td>0.25</td>
<td>L2-L3; aVF</td>
<td>—</td>
<td>1.3</td>
<td>1.4</td>
<td>1.3</td>
<td>L2</td>
<td>8</td>
</tr>
<tr>
<td>21</td>
<td>55/F</td>
<td>0.2</td>
<td>V1-V4</td>
<td>—</td>
<td>0.5</td>
<td>0.6</td>
<td>0.7</td>
<td>V3</td>
<td>20</td>
</tr>
<tr>
<td>22</td>
<td>35/M</td>
<td>0.2</td>
<td>MV5</td>
<td>—</td>
<td>0.2</td>
<td>0.6</td>
<td>0.2</td>
<td>MV5</td>
<td>200</td>
</tr>
<tr>
<td>23</td>
<td>68/F</td>
<td>0.2</td>
<td>V2-V6</td>
<td>—</td>
<td>0.9</td>
<td>0.9</td>
<td>0.8</td>
<td>V4</td>
<td>0</td>
</tr>
<tr>
<td>24</td>
<td>44/M</td>
<td>0.2</td>
<td>L1; aVL; V1-V2</td>
<td>—</td>
<td>0.9</td>
<td>1.2</td>
<td>0.8</td>
<td>L1</td>
<td>33</td>
</tr>
<tr>
<td>25</td>
<td>38/M</td>
<td>0.2</td>
<td>L2-L3; aVF</td>
<td>—</td>
<td>1.1</td>
<td>1.2</td>
<td>1.1</td>
<td>L2</td>
<td>9</td>
</tr>
<tr>
<td>26</td>
<td>52/M</td>
<td>0.15</td>
<td>V2-V6</td>
<td>—</td>
<td>1.3</td>
<td>1.3</td>
<td>1.3</td>
<td>V4</td>
<td>0</td>
</tr>
</tbody>
</table>

Abbreviations: VT = ventricular tachycardia; VPCs = ventricular premature complexes; VF = ventricular fibrillation; %ΔR = percentage change in R wave amplitude; VAP = variant angina pectoris.
narrowings > 50% but < 90% in the same vessel; 4 = narrowing > 90% but not total obstruction; 5 = total obstruction of any coronary artery. The score of all major coronary arteries (right, left anterior descending and circumflex) are then added to obtain a score for the coronary angiogram. A score of 0–4 represents single-vessel disease, 5–9 double-vessel disease and 10–15 triple-vessel disease. The left ventricular angiogram was divided into five segments: 1 — posterior, 2 — diaphragmatic, 3 — apical, 4 — anterolateral and 5 — anterobasal. The systolic contraction patterns were described as hypokinetic when the movement was less than normal, akinetic when no movement was seen and dyskinetic when paradoxical movement was seen.

Statistical analysis was performed using the two-tailed t test for uncorrelated data and the chi-square test.

**Results**

**Electrocardiographic Studies**

At rest, 24 of the 26 patients had a normal 12-lead ECG in the absence of VAP. Patient 15 had complete left bundle branch block and patient 16 had complete right bundle branch block. During pain, concave ST-segment elevations with upright T wave and reciprocal ST-segment depression in the standard leads were found in all patients. Elevation of the ST segment was seen in anterior and lateral leads (L1, aVL, V1-V6) in three patients, anterior leads (L2 and/or V1-V6) in 12 and inferior leads (L3, L4 and aVF) in eight.

R-wave amplitude compared before and during episodes of VAP increased ≥ 10% in 10 of 12 patients with arrhythmias and in only six of 14 patients without arrhythmias (p < 0.05) (table 1). Within minutes after the cessation of pain and normalization of the ST segment, the R-wave amplitude reached the control levels. No statistical correlation was found between the magnitude of ST-segment elevation and %ΔR. During episodes of VAP, there was no change in the QRS axis on the 12-lead ECG among the 24 patients.

**ST-segment Elevation and Arrhythmias**

Of the 26 patients, 12 had severe arrhythmias during pain (group 1). The other 14 patients constituted group 2. In episodes of VAP, the ST-segment elevation ranged from 0.15–1.6 mV. All patients with ST-segment elevation ≥ 0.4 mV had arrhythmias. All patients with arrhythmias had an ST-segment elevation ≥ 0.4 mV (table 1). Six patients had malignant types of VPCs that were multifocal and frequent (5–10
VPCs/min). Three patients had the R-on-T phenomenon, which precipitated ventricular tachycardia in two. Those patients had ventricular fibrillation (fig. 1) and four had ventricular tachycardia (fig. 2) with heart rates of 136–154 beats/min. Three patients had high-grade atrioventricular block, 2:1 second-degree atrioventricular block, 3:2 Wenckebach conduction alternating with 2:1 and 3:1 block and a third-degree atrioventricular block. In two patients (nos. 4 and 9), the atrioventricular blocks were accompanied by ST-segment elevation in inferior leads, while in one (no. 7), the ST-segment elevation was seen in lead V₂ with reciprocal changes in V₅. Eight patients each had two to 19 episodes of VAP. Three of those patients (nos. 1, 2 and 7) had repeated episodes of arrhythmias with attacks of VAP. Both episodes of ventricular tachycardia recorded in patient no. 1 occurred with ST-segment elevation of 1 and 1.6 mV. Patient no. 2 had four episodes of VAP with ST-segment elevation of 0.2, 0.3, 0.35 and 0.8 mV, but only the episode with ST-segment elevation of 0.8 mV was accompanied by arrhythmias. In patient no. 7, the two episodes of second-degree atrioventricular block occurred at the same ST-segment elevation of 0.6 mV. Patients nos. 16, 20, 21, 24 and 26 had repeated episodes of VAP with ST-segment elevation < 0.4 mV that were not accompanied by arrhythmias. Ventricular irritability (fibrillation, tachycardia and VPCs) was found predominantly in patients who developed ST-segment elevation in anterolateral leads (seven cases) and in those developing ST-segment elevation in inferior leads (two cases). The high-degree atrioventricular blocks were seen with ST-segment elevation in inferior (two cases) as well as anterior leads (one case).

**Coronary Arteriography**

The results of coronary arteriography in 23 patients are given in table 2. Eight patients had normal coronary arteriograms and three developed coronary spasm before the selective injection of the coronary arteries. Of 12 patients with arrhythmias, nine had angiographic studies and one had a postmortem examination. Four patients had normal coronary arteries and six had significant proximal coronary artery obstructive disease (table 2). The arrhythmias were more commonly seen in the group with coronary artery obstructive disease (66%) than in the group with normal coronary arteriograms (44%). Sixteen patients had focal lesions (60–100% obstruction) that affected the proximal segments of the various coronary
arteries. Only patient no. 13 had collateral circulation.

There was no significant difference in the coronary score between groups (p = NS) (table 3). The lack of correlation was apparent in the total group as well as the subset with coronary obstructive disease (table 3).

**Left Ventriculography**

Table 4 lists the contractility pattern of myocardial segments from left ventricular angiograms and the results of left ventricular ejection fractions. Of nine patients from group 1, six (67%) had normal left ventricular segments and three (33%) had abnormal segments. Although there was no significant difference in the number of abnormal segments between group 1 and group 2 (table 4) (p = NS), the number of patients with abnormal segments was too small to rely on its statistical value. The mean ejection fraction in group 1 was 73 ± 10% and 68 ± 6% in group 2 (p = NS) (table 4).

**Discussion**

The prevalence and significance of ventricular arrhythmias have been shown to be related to the extent of coronary artery disease and wall motion abnormalities in patients with angina pectoris. We did not find a similar relationship between the extent of cor-
TABLE 3. **Coronary Angiographic Characteristics of Patients**

<table>
<thead>
<tr>
<th></th>
<th>Group 1†</th>
<th>Group 2‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal coronary arteries</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Coronary angiographic score</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Total group</em></td>
<td>2.50 ± 3.03</td>
<td>5.07 ± 4.83</td>
</tr>
<tr>
<td><em>Patients with obstructive disease</em></td>
<td>4.17 ± 2.86</td>
<td>7.1 ± 4.2</td>
</tr>
<tr>
<td>Single-vessel disease</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Double-vessel disease</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Triple-vessel disease</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Total patients</td>
<td>10</td>
<td>14</td>
</tr>
</tbody>
</table>

*p = NS (>0.05) comparing group 1 with group 2.
†Patients with arrhythmias.
‡Patients without arrhythmias.

Electrocardiographic and Hemodynamic Changes in VAP

According to several investigators, the degree of ST-segment elevation correlates with the severity of ischemia. Experimental work with dogs performed by Ekmekci et al.,26 showed that complete ligation of a major coronary artery produces the highest ST-segment elevation in the central part of the ischemic area. When the central area is rendered less ischemic, ST-segment depression occurs. As noted previously,1,23 ST-segment deviation reflects myocardial ischemia. ST-segment elevation indicates a more severe degree of ischemia than ST-segment depression. The degree of epicardial ST-segment displacement has been correlated with the degree of change in anaerobic tissue metabolism,24 membrane potential25 and reduction in coronary blood flow.26 The ST-segment elevation is often accompanied by marked increase in R-wave amplitude and diminution of the S wave.23

Hemodynamic abnormalities of the left ventricle during VAP have been documented by Guazzi et al.27 and Gaasch et al.28 During episodes of VAP, left ventricular impairment is manifested by increased end-diastolic pressure and decreased maximal dP/dt. The former is probably related to impaired relaxation and the latter to impaired contractility. This change results in reduction of mean systolic ejection rate, cardiac output and arterial hypotension. The appearance of a prominent R wave during episodes of VAP might be explained by changes in left ventricular volumes during ischemia. Brody29 postulated that the R-wave amplitude will decrease as left ventricular blood mass decreases. Although this study was performed in the open-chest dog with an epicardial electrode, the role of intracardiac blood mass upon R-wave amplitude was later confirmed in further animal experiments.30-32 These studies indicate that an increase or lack of change in R-wave amplitude during exercise stress testing reflects ventricular dysfunction with elevated left ventricular systolic and diastolic volumes, probably accompanied by severe and multiple coronary artery obstructive disease. A decrease in R-wave amplitude during exercise is associated with normal or minimal left ventricular dysfunction and is conspicuous in patients with no disease or single-vessel disease. No correlation was found between the magnitude of ST-segment elevation and R-wave changes, which suggests that different operational factors affect them.

Arrhythmias During VAP

There appears to be no correlation between arrhythmias, preexisting coronary artery disease and resting left ventricular function in VAP. Thus, the sudden, transient, severe coronary spasm producing a profound degree of ischemia may play the most important role in the genesis of the arrhythmias. However, the mechanism of arrhythmias in VAP is unknown. Transient ligation of the coronary artery produces significant ST elevation at epicardial sites, accompanied by profound metabolic derangement (including accumulation of high levels of lactate, depletion of ATP and creatine phosphate), which indicates a pronounced anaerobic stress.35 The ischemic myocardium has been shown to be the site of a temporal dispersion of refractoriness, thus predisposing to reentrant arrhythmias.36-38

It appears that an ST-segment elevation of ≥0.4 mV clearly separated patients with arrhythmias from those without arrhythmias. The results of our study must be interpreted with caution because all of the episodes of clinical VAP were not documented electrocardiographically. The existence of a correlation between the degree of ST-segment elevation and arrhythmias in patients with VAP did not seem to be fortuitous, as shown by the presence of arrhythmias in two of our patients with recurrent episodes of VAP. One patient developed arrhythmias with the same ST-segment elevation, while the second had ventricular tachycardia accompanied by a higher ST-segment elevation. By contrast, five patients without arrhythmias who had ST-segment elevation < 0.4 mV did not develop arrhythmias during repeated episodes of VAP with ST-segment elevation < 0.4 mV.

**TABLE 4. Functional Anatomy of Myocardial Segment and Ejection Fraction**

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ejection fraction</td>
<td>73 ± 10*</td>
<td>68 ± 6</td>
</tr>
<tr>
<td>Myocardial segments*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>6 (67%)</td>
<td>12 (86%)</td>
</tr>
<tr>
<td>Hypokinetic</td>
<td>1 (11%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Akinetic</td>
<td>2 (22%)</td>
<td>2 (14%)</td>
</tr>
<tr>
<td>Dyskinetic</td>
<td>0 (0%)</td>
<td>0 (0%)</td>
</tr>
<tr>
<td>Total patients</td>
<td>9</td>
<td>14</td>
</tr>
</tbody>
</table>

*p = NS (>0.05) compared with group 2.
*Group 1 patients had arrhythmias; group 2 patients did not have arrhythmias.
Selzer and co-workers\(^9\) reported that patients with VAP and ST-segment elevation in the inferior leads are likely to have normal coronary arteriograms, ischemia-dependent atrioventricular block and bradycardia; the anterolateral leads are involved when coronary disease and ventricular tachycardia or fibrillation are present. We and others\(^9\) have not determined the location of ST-segment elevation to be a predictive factor, having observed patterns with and without coronary artery disease.

We have not found that the specific types of arrhythmias are predictive in distinguishing patients with normal coronary arteriograms from those with significant coronary disease. Ventricular tachycardia or fibrillation was equally present in groups of patients with and without coronary disease. Advanced degrees of atrioventricular block are an infrequent complication in VAP.\(^1\) A high-degree atrioventricular block was reported in patients with electrocardiographic alterations limited to the diaphragmatic leads, thus implicating the right coronary artery.\(^1,8,9\) Patient no. 7 developed a high-degree atrioventricular block with ST-segment elevation in lead V\(_2\). Most likely, the anterior ischemia produced conduction dysfunction below the His bundle.

Clinical Implication

Coronary vasodilators such as nitroglycerin and the calcium antagonists represented by Nifedipine are helpful in preventing episodes of VAP.\(^4\) Alpha-adrenergic blockade with phenoxybenzamine has also been effective in controlling VAP.\(^4\) On the other hand, propranolol tends to aggravate attacks of VAP.\(^4\) Our study reveals that arrhythmias are related to the degree of ST-segment elevation, which may well reflect the severity of ischemia. Therefore, it is likely that nitroglycerin and Nifedipine will be most beneficial in preventing the arrhythmias as well as the episodes of VAP. The role of the antiarrhythmic agents in preventing arrhythmias in VAP has yet to be established.

References

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Performance of Conventional Orthogonal and Multiple-dipole Electrocardiograms in Estimating Left Ventricular Muscle Mass

Rosalie A. Dunn, Ph.D., Hubert V. Pipberger, M.D., John H. Holt, Jr., M.D., A.C.L. Barnard, D.Sc., and Hanna A. Pipberger, B.A.

SUMMARY For estimating left ventricular mass (LVM), ECG criteria for left ventricular hypertrophy (LVH) were selected from conventional 12-lead ECGs, orthogonal three-lead ECGs, and multiple-dipole ECGs (MDECG). The three cardiograms were recorded in 139 patients for whom the degree of LVH was independently determined from biplane ventriculograms.

Tested ECG criteria included Sokolow-Lyon measurements for the 12-lead ECG; for the orthogonal ECG, maximal QRS magnitude in the horizontal plane, R duration in the z-lead and J_{sys} (spatial magnitude of point J); and for the 126 leads of the MDECG, the dipole activity (DA) of the septum and the free left ventricular wall.

Correlation coefficients between LVM and the 12-lead ECG, three-lead ECG and MDECG were 0.61, 0.78 and 0.89, respectively, with corresponding errors of estimated LVM of 103, 82 and 60 g. More complex recording and analytic methods clearly led to increased accuracy in LVM estimates. However, the large error of estimate may limit practical applicability of such correlations. For classification of subjects into normal and above-normal categories, a likelihood ratio was also used and led to a maximum performance index of 86% with MDECG measurements.

RECENT ATTEMPTS to quantitate left ventricular mass (LVM) are part of the trend to refine and improve noninvasive diagnostic techniques. An easily obtained estimate of LVM would be particularly desirable for patients with left ventricular hypertrophy (LVH). The ECG is well recognized as a clinical tool for the diagnosis of hypertrophy and has been used to demonstrate an increase or decrease in the degree of LVH accompanying change in clinical status. However, previous efforts to determine a significant correlation between degree of LVH and ECG measurements have had only limited success. Holt et al. reported a wide range of performance of various ECG criteria for the recognition of LVH. This may be attributed in part to the use of too few or inappropriate ECG leads that may not provide all the electrical information which is obtainable from the body surface. In addition, comparative studies have been inconclusive either because the various lead systems may not have been recorded in the same patients, or because an independent and accurate measure of muscle weight was not available, or because of the lack of uniformity in the statistical methods used.

In this study we compare several lead systems —
Arrhythmias in variant angina pectoris. Relationship of arrhythmias to ST-segment elevation and R-wave changes.

N Z Kerin, M Rubenfire, M Naini, W J Wajszczuk, A Pamatmat and P N Cascade

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