Differentiation of Posterior Myocardial Infarction from Right Ventricular Hypertrophy and Normal Anterior Loop by Echocardiography

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SUMMARY The differentiation of postero-basal myocardial infarction (PMI) from either right ventricular hypertrophy (RVH) or normal subjects displaying an anterior loop (AL) by electrocardiography (ECG) or vectorcardiography (VCG) is difficult. M-mode echocardiography (echo) via the anterior and subxiphoid methods has been helpful in defining cardiac chamber size and wall motion abnormalities. We tested whether this relatively more direct method would better separate these entities compared with the other two techniques. ECG and VCG using established criteria failed to distinguish the three conditions effectively. By echo, distinguishing characteristics were observed in each of the groups. Thus, right ventricular diastolic dimension and wall thickness were significantly increased only in the RVH group, echo dimensions and wall motion were normal in the AL group and the posterior left ventricular systolic thickening response and ejection phase indices were significantly reduced only by the subxiphoid method in the PMI group. To test the specificity of the latter finding, posterior wall motion in three infarction groups (posterior, combined posteroinferior and inferior) were examined and suggested that the target of the subxiphoid beam focuses on a more superior postero-basal left ventricular segment than the anteriorly placed transducer.

Echocardiography can differentiate PMI from either RVH or AL more directly than ECG or VCG, and may be of practical clinical importance.

THE PRESENCE OF A PROMINENT R WAVE in the right precordial leads of the ECG frequently creates a clinical dilemma. In the adult a distorted anteriorly directed vector may occur in postero-basal infarction as a result of loss of posterior forces, in right ventricular hypertrophy as a result of increased anterior forces and in at least 20% of normal individuals.1,2

In considering the electrocardiographic diagnosis of posterior infarction,3 additional precordial criteria have been tested, with disappointing results.3-7 The diagnosis can be further clouded by the occurrence of either right bundle branch block or preexcitation syndrome. While the development of an associated inferior infarction pattern supports the diagnosis, right ventricular hypertrophy may still coexist. The vectorcardiogram (VCG), which initially appeared to improve the sensitivity of the ECG in identifying posterior infarction,5,8-10 has also revealed suboptimal results in the simple differentiation of the above-mentioned entities.11-14

Recently, sonomicrometry and M-mode echocardiography have been shown experimentally and clinically to evaluate abnormal wall motion in acute and chronic coronary artery disease.15-22 The latter method also allows determination of cardiac chamber dimensions in a variety of conditions.22 Accordingly, we evaluated the relative capabilities of separating these three entities by echocardiography compared with electrocardiography and vectorcardiography.

Methods

Six groups of 10 individuals each comprised the study population. There were 10 normal subjects (mean age 32.1 years) as determined clinically, electrocardiographically and radiographically. A second group of 10 patients (mean age 39.2 years) with normal cardiovascular status were indistinguishable from the first group except for the presence of a prominent R wave in the right precordial leads of the ECG. Another 10 patients (mean age 56.6 years) were clinically and radiographically determined to have right ventricular hypertrophy of varying etiologies (mitral stenosis in four, congenital in two, and pulmonary disease in four). Cardiac catheterization performed in seven of these patients demonstrated right ventricular systolic hypertension (mean 55 mm Hg). The fourth group included 10 patients (mean age 49.1 years) who had previously sustained an isolated posterior myocardial infarction documented by clinical presentation, serial electrocardiographic changes in the right precordial leads and/or enzymatic determinations. Review of the selective coronary arteriograms performed in four patients from this group revealed severe obstruction of the left circumflex artery alone or in combination with other lesions. Additionally, two groups consisting of 10 patients with documented healed combined posteroinferior infarction (mean age 51.5 years) and 10...
patients with isolated inferior wall infarction (mean age 53.1 years) were evaluated in order to compare specific location of wall motion abnormalities by echocardiography. Patients with either preexcitation syndrome or right bundle branch block were excluded.

All 60 patients underwent standard 12 lead scalar electrocardiography. The ECG parameters evaluated included: the frontal axis, the R/S ratio in leads V1, V2 and V6, and the R wave duration and T wave polarity in the right precordial leads. A prominent R wave was considered to be present when the R/S ratio in leads V1 or V2 was $\geq 1$, and a prolonged R wave was noted if it was $\geq 0.04$ seconds. Additionally, the presence of pathologic Q waves in the inferior leads were determined.

The VCGs were recorded in 46 of the patients using the Frank lead reference system on a Hewlett-Packard model 1507A vectorcardiograph. Calibrations of 0.5 mV per 1, 2, 5 and/or 10 cm deflection were used, depending on QRS loop. The VCG trace was interrupted each 2.5 msec by means of dash markers. Polaroid photographs of the frontal, horizontal and left sagittal planes were taken from the oscilloscope screen. Measurements were made manually from the Polaroid prints obtained. The vectorcardiographic parameters included: the mean half area axes in the frontal and horizontal plane, the location of the 40 msec vector, the magnitude of the terminal rightward force and the direction of the QRS loop in the horizontal plane as described by Mathur and Levine. Additionally, the direction and location of the initial 30 msec QRS vector with respect to the E point in the frontal plane was noted.

The echocardiograms were obtained with a commercially available Ekoline 20A Ultrasonoscope in conjunction with a 1.9, 2.5 or 3.5 MHz focused transducer. Recording was accomplished on a Honeywell 1856 visicorder. Quality anterior and subxiphoid data were obtained on all patients either initially or upon subsequent examination. Echocardiographic measurements for three consecutive cardiac cycles were measured and averaged when individual variation was encountered.

The left ventricular end-diastolic dimension (LVDd) was determined at the level of the chordae tendinae simultaneously with the R wave of the ECG (fig. 1). The left ventricular end-systolic dimension (Ds) was measured at the peak of the anterior movement of the posterior left ventricular endocardium. The right ventricular internal dimension (RVD) and wall thickness (RVT) were carefully recorded only by the anterior method at end-diastole using proper damping adjustments and transducer selection. The fractional shortening response (%FS), or the percent change in dimension during systole, was calculated by the formula:

$$% FS = (Dd - Ds) \times 100 / Dd.$$  

The mean velocity of circumferential fiber shortening (VCF) was derived by dividing the left ventricular ejection period, obtained from the aortic valve echo into the %FS according to the modified method of Cooper et al. The percent systolic thickening of the left ventricular posterior wall and septum, (%ST and %ST's, respectively) was taken as the difference of the left ventricular thickness between end-systole and mid-diastole (DT) divided by the mid-diastolic thickness $\times 100$.

The electro-vectorcardiographic and echocardiographic data were calculated each by a cardiologist.

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**Figure 1.** Echocardiographic parameters. LVDd, Ds = left ventricular end-diastolic and end-systolic dimensions, respectively; RVd, RVt = right ventricular end-diastolic dimension and wall thickness, respectively; DT = left ventricular mid-diastolic thickness; %ST = % systolic thickening response; AV = aortic valve; ET = ejection time; Ao = aortic root; LA = left atrium.
unaware of the classification of the patients. Additionally, interobserver echocardiographic variation was tested by a separate cardiologist and revealed no important differences in either group or individual patient responses. The ECG and VCG were tested for differences among the groups by the chi square test with a correction for continuity included, due to the small sample sizes. Statistical analysis of the echocardiographic data was aided by means of a one-way analysis of variance. The least significant differences test was used to distinguish among means.

Results

Electrocardiographic and Vectorcardiographic Responses

The frequency of electrocardiographic and vectorcardiographic responses for the posterior infarction, right ventricular hypertrophy and normal anterior loop groups are presented in table 1.

There were no statistical differences noted among the three groups for any of the scalar ECG parameters evaluated. However, right axis deviation occurred relatively more often and a prominent S wave in V6 occurred selectively, although infrequently, in the right ventricular hypertrophy group. In general, the ECG failed to clearly distinguish the three conditions.

Using the VCG, a normal frontal axis occurred significantly less often in the right ventricular hypertrophy vs the normal anterior loop group (P < 0.005). All other comparisons were not significant. By combining vector parameters 2 and 4, as suggested by Mathur and Levine, six posterior infarction criteria were correctly satisfied in eight of nine cases. However, when these criteria were applied to the right ventric-

<table>
<thead>
<tr>
<th>Table 1. Electrocardiographic and Vectorcardiographic Responses for the Posterior Infarction, Right Ventricular Hypertrophy and Anterior Loop Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrocardiographic criteria</td>
</tr>
<tr>
<td>A) Frontal axis -15° to +45°</td>
</tr>
<tr>
<td>B) Frontal axis &gt; 90°</td>
</tr>
<tr>
<td>C) R/S ≥ 1 lead V1 or V2</td>
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<tr>
<td>D) R/S ≤ 1 lead V6</td>
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<tr>
<td>E) R wave ≥ 0.04 sec. lead V1</td>
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<td>F) T wave inverted lead V1</td>
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</table>

Vectorcardiographic criteria

1) Frontal QRS axis 350° – 90° | 6 of 9 | 3 of 10* | 10 of 10 |
2) Mean horizontal axis 350° – 90° | 8 of 9 | 7 of 10 | 10 of 10 |
3) Horizontal 40 msec vector 350° – 60° | 7 of 9 | 8 of 10 | 10 of 10 |
4) Terminal R wave < 1.0 MV | 9 of 9 | 10 of 10 | 10 of 10 |
5) Horizontal clockwise rotation QRS | 1 of 9 | 2 of 10 | 0 of 10 |
6) Both 2 and 4 | 8 of 9 | 7 of 10 | 10 of 10 |

*P <0.005 (right ventricular hypertrophy vs normal anterior loop). All other comparisons are not significant. Abbreviation: MI = myocardial infarction.

Echocardiographic Responses

Table 2 lists the mean echo responses for all patient groups. Figure 2 shows the individual patient responses for the important distinguishing parameters of the posterior infarction, right ventricular hypertrophy and anterior loop groups.

The left ventricular internal dimension was significantly larger in the posterior infarction group compared with the right ventricular hypertrophy group by both echo methods (anterior P < 0.05; sub-xiphoid, P < 0.01) (fig. 2A). This dimension was also larger (although not significantly so) in the posterior infarction group compared with the anterior loop group by both echo methods.

The right ventricular internal dimension and RVT as determined by the anterior echo method was significantly higher (P < 0.01) and above the normal range only in the right ventricular hypertrophy group (fig. 2B). The latter displayed less overlap of individual patient responses of the two parameters.

The %FS was nearly identical in the posterior infarction, right ventricular hypertrophy and anterior loop groups by the anterior method. With the sub-xiphoid approach, patients in the posterior infarction group demonstrated a significantly lower (P < 0.05) fractional shortening response compared with the right ventricular hypertrophy group (fig. 2C). Overlapping responses between these groups,
Table 2. Group Echocardiographic Results (Means)

<table>
<thead>
<tr>
<th>Group</th>
<th>LVDD (cm)</th>
<th>RVD (cm)</th>
<th>RVT (cm)</th>
<th>%ST (Post)</th>
<th>%ST (Sept)</th>
<th>VCF (circ/sec)</th>
<th>LVDD (cm)</th>
<th>%ST (Post)</th>
<th>%ST (Sept)</th>
<th>VCF (circ/sec)</th>
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</thead>
<tbody>
<tr>
<td>Anterior</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Normal</td>
<td>4.70</td>
<td>1.59</td>
<td>0.55</td>
<td>73.7</td>
<td>61.3</td>
<td>37.7</td>
<td>1.50</td>
<td>4.96</td>
<td>66.5</td>
<td>53.6</td>
</tr>
<tr>
<td>Anterior loop</td>
<td>4.62</td>
<td>1.53</td>
<td>0.63</td>
<td>63.7</td>
<td>54.6</td>
<td>39.2</td>
<td>1.50</td>
<td>5.03</td>
<td>65.3</td>
<td>49.4</td>
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<tr>
<td>Posterior</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right ventricular</td>
<td>4.35</td>
<td>2.56</td>
<td>1.10</td>
<td>58.3</td>
<td>50.4</td>
<td>32.4</td>
<td>1.32</td>
<td>4.50</td>
<td>73.5</td>
<td>46.8</td>
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<tr>
<td>Right hypertrophy</td>
<td>0.17</td>
<td>0.13</td>
<td>0.15</td>
<td>4.3</td>
<td>6.3</td>
<td>2.2</td>
<td>0.08</td>
<td>0.31</td>
<td>3.6</td>
<td>5.6</td>
</tr>
<tr>
<td>Posterior myocardial</td>
<td>5.13</td>
<td>1.59</td>
<td>0.70</td>
<td>78.4</td>
<td>57.6</td>
<td>36.5</td>
<td>1.37</td>
<td>5.34</td>
<td>13.7</td>
<td>65.0</td>
</tr>
<tr>
<td>Posterior myocardial</td>
<td>0.30</td>
<td>0.15</td>
<td>0.04</td>
<td>9.6</td>
<td>9.5</td>
<td>2.2</td>
<td>0.08</td>
<td>0.31</td>
<td>3.6</td>
<td>5.6</td>
</tr>
<tr>
<td>Inferior</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior myocardial</td>
<td>5.70</td>
<td>1.51</td>
<td>0.56</td>
<td>19.9</td>
<td>45.1</td>
<td>22.7</td>
<td>0.76</td>
<td>5.26</td>
<td>25.2</td>
<td>25.8</td>
</tr>
<tr>
<td>Inferior myocardial</td>
<td>0.37</td>
<td>0.20</td>
<td>0.04</td>
<td>3.8</td>
<td>6.3</td>
<td>1.7</td>
<td>0.06</td>
<td>0.21</td>
<td>5.9</td>
<td>5.9</td>
</tr>
</tbody>
</table>

Values are mean ± SEM.
Abbreviations: LVDD = left ventricular end-diastolic dimension; RVD = right ventricular internal dimension; RVT = right ventricular wall thickness; %ST = percent systolic thickening; Post = posterior; Sept = septal; %FS = fractional shortening response; VCF = velocity of circumferential fiber shortening.

however, were observed. Similar results were obtained with mean VCF determinations.

The systolic thickening response of the left ventricular posterior wall by the anterior method was significantly higher in the posterior infarction group compared with the right ventricular hypertrophy ($P < 0.01$) and anterior loop groups ($P < 0.05$). However, using the subxiphoid method, this parameter was significantly reduced ($P < 0.01$) and below the normal range only in the posterior infarction group (fig. 2D). No overlap of responses between this group and the remaining two groups was observed. The septal systolic thickening response was significantly higher in the posterior infarction group by the subxiphoid method compared with the remaining two groups (table 2; $P < 0.05$). Septal responses were similar with the anterior method.

Echocardiographic Correlations — Infarction Groups

The dramatic reduction in posterior left ventricular systolic thickening response in the posterior infarction group by the subxiphoid and not the anterior method suggested that two different areas of the left ventricle may have been visualized by these two different approaches, as schematically depicted in figure 3.

In order to test this hypothesis, we next compared the posterior thickening responses of the posterior, combined posterior-inferior and isolated inferior infarction groups, as shown in figures 4A and B. With the anterior approach, only the groups with inferior wall involvement (combined and inferior infarction groups) showed significantly reduced posterior systolic thickening ($P < 0.01$). Using the subxiphoid method, only those groups with posterior wall involvement (posterior, combined infarction groups) demonstrated significantly reduced posterior wall motion ($P < 0.01$). Comparing each group by the two echo methods, posterior wall motion was reduced in the posterior infarction group only by the subxiphoid method ($P < 0.01$), and in the inferior infarction group only by the anterior method ($P < 0.01$). Posterior wall motion in the combined infarction group was reduced by both echo approaches. Similar trends were noted using the fractional shortening and the derived mean VCF determinations. No significant difference in the left ventricular diastolic dimension was observed between the infarction groups by the two echo techniques (table 2).

Discussion

The scalar ECG has been determined to be an insensitive and nonspecific tool in the recognition of proven posterobasal infarction,4a and its differentiation from right ventricular hypertrophy or a normal anterior loop variant. With the addition of the VCG sensitivity has improved somewhat, but false positive diagnoses remain a practical clinical problem, especially in the adult population.4a, 5, 10-14

Overall analysis of our electrocardiographic and vectorcardiographic data revealed disappointing results in the clear separation of these three mentioned entities (table 1). Furthermore, the combination of vectorcardiographic parameters (nos. 2 and 4) suggested by Mathur and Levine4 resulted in the misclassification of seven of 10 right ventricular hyper-
trophy patients as true posterior infarction. Likewise, all anterior loop patients would have been considered as having sustained a posterior infarction by this same combination.

The differentiation of posterior infarction from normal anterior loop individuals probably is even more important in the adult population where right ventricular hypertrophy is uncommon. Ha et al., using multiple established vector parameters, determined that no significant vectorcardiographic differences were noted between these two groups. Their results paralleled the vectorcardiographic responses noted in table 1. Obviously, caution must be exercised in interpreting and basing clinical judgments on the VCG in these situations.

Echocardiography, in contrast to either electrocardiography or vectorcardiography, more effectively separated these three conditions by direct analysis of chamber dimensions and wall motion. Thus, in the right ventricular hypertrophy group, the right ventricular dimension and wall thickness by the anterior method were significantly increased and provided an easy means of separating this condition from the remaining two groups (fig. 2B). In the anterior loop group, the cardiac chamber dimensions and parameters of left ventricular function were within the normal range, and were not significantly different from a separate group of 10 normal subjects not dis-


thickening, of similar magnitude to our inferior infarction patients, was noted in 65% of their acute inferior infarction group by the anterior method. Additionally, in that study systolic thinning was helpful in detecting septal involvement but was insensitive in inferior infarction. Systolic thinning by either the anterior or subxiphoid method was not observed in our inferior or posterior infarction patients.

The reduction in posterior wall motion noted only by the subxiphoid method in our posterior infarction group prompted us to test the specificity of this finding. We did this by comparing the posterior %ST in three infarction groups (posterior, combined posterior-inferior, isolated inferior) by both echo methods (fig. 4). With the anterior method, infarction groups with inferior wall involvement showed reduced wall thickening, while with the subxiphoid method infarction groups with posterior involvement showed a diminished response. The combined infarction group showed reduced systolic thickening posteriorly by both techniques, reflecting more extensive disease. Based on this analysis and the initial work of Chang et al., it appears that the subxiphoid-placed transducer locates a more superior and probably lateral segment of the left ventricle, while the anteriorly oriented transducer identifies a more inferior segment (fig. 3).

Alternatively, the possibility that transducer angulation spuriously produced the subxiphoid abnormalities should be considered. This possibility seems unlikely, however, as posterior wall thickening by this same method in those groups without posterior involvement (anterior loop, normal, right ventricular hypertrophy and inferior infarction) appeared normal.

Although most of our data were derived from initial echocardiographic examinations, a small percentage of cases required a subsequent examination to obtain additional information. This is not unexpected in the population studied, which included patients with coronary artery disease, pulmonary disease and normal subjects with vertical hearts. Evaluation of the posterior left ventricular wall by both echo techniques was accomplished in all cases without difficulty. Septal evaluation by the subxiphoid approach was more difficult, with adequate visualization of this structure in only 70% of the patients surveyed. This limitation did not represent a drawback, since posterior wall thickening was the only subxiphoid parameter which clearly distinguished posterior infarction. Finally, the identification of the right ventricular epicardial surface can be difficult due to chest wall reverberations, near field transducer blurring and anterior intervening gaseous lung tissue. We minimized this problem by using lower anterior gain settings in conjunction with variable reject settings, held respiration and several transducer trials as necessary.

In summary, it would appear that echocardiography by more directly assessing chamber dimensions and left ventricular function may more practically distinguish the conditions associated with an anterior electrical force than either electrocardiography or vectorcardiography.
Acknowledgment

The authors express their appreciation for the technical expertise of Mrs. Dorothy Gore and Mrs. Sadie Rochon and the expert secretarial assistance of Mrs. Evelyn Hunt.

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Physiologic Correlates of Echocardiographic Pulmonary Valve Motion in Diastole

DAVID J. POCOSKI, M.D. AND PRavin M. SHAH, M.D.

Assisted by Linda J. Sylvester

SUMMARY It has been shown that the echocardiographic motion of the posterior aortic wall in diastole is closely related to the underlying left atrial events, possibly due to the anatomical proximity of the two structures. We observed that the pulmonary artery shares a similar close anatomical relationship with the left atrium. The present study in 55 consecutive patients with adequate echocardiographic recordings of the aortic root and the pulmonary valve demonstrates that the diastolic waveforms of the pulmonary valve and the posterior aortic wall are nearly identical in early diastole. The pulmonary valve e-f slope correlated with the posterior aortic wall O-R slope \( (r = 0.95) \) and the S-f interval (second heart sound to f on the pulmonary valve) correlated closely with the S-R interval (second heart sound to R on the posterior aortic root) \( (r = 0.94) \). No significant correlation was found between the pulmonary valve e-f slope and the pulmonary artery pressures. The presence and amplitude of the maximum “a dip,” on the other hand, correlated with the presence of pulmonary hypertension, with some notable exceptions. In addition, the “a dip” on the pulmonary valve and the depth of the A wave on the posterior aortic wall were significantly correlated \( (r = 0.85) \). It appears that the “a dip” on the pulmonary valve is influenced by dual mechanisms: pressure differences between the pulmonary artery and the right ventricle in late diastole and the left atrial events. Thus, the early diastolic waveform of the pulmonary valve, like the posterior aortic wall waveform, may primarily reflect underlying left atrial events and is not a measure of pulmonary artery pressure. The late diastolic waveform “a dip” has a dual mechanism, related in part to the underlying left atrial events, and also reflecting the instantaneous pressure differences across the pulmonary valve following atrial systole.

Echocardiographic evaluation of the pulmonary valve was first described by Grawia and associates. Subsequently, clinical and hemodynamic correlates of the valve motion have been reported. The systolic opening of the valve is readily explained by the right ventricular-pulmonary arterial pressure events during ventricular systole. However, the diastolic motion pattern remains largely unexplained. Strunk et al. have recently shown that the posterior aortic wall motion in diastole is closely related to left atrial volume changes. This was attributed to a close anatomic relationship between the two structures. We observed a qualitative similarity between the waveforms of pulmonary valve and the aortic root during diastole. We therefore reasoned that the pulmonary artery, also having a close anatomical relationship to the left atrium, may be influenced by the left atrial volume changes in diastole. This hypothesis was tested by quantitative measurements in a series of cases.
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