New Vectorcardiographic Criteria for Diagnosing Right Ventricular Hypertrophy in Mitral Stenosis: Comparison with Electrocardiographic Criteria

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SUMMARY Frank-lead vectorcardiograms (VCGs) and standard 12-lead electrocardiograms (ECGs) were analyzed to develop simple, linear, quantitative criteria for the diagnosis of right ventricular hypertrophy (RVH). The study subjects included a population with definite RVH (84 patients with mitral stenosis proved by cardiac catheterization and pulmonary arterial systolic pressure > 40 mm Hg) and a population with minimal likelihood of RVH (173 young, healthy volunteers and 151 normal subjects proved by cardiac catheterization). VCGs were evaluated to identify criteria that provided maximum sensitivity and at least a 95% specificity: the maximum QRS amplitude of the QRS loop was 3 mV and either (1) the amplitude at $-45^\circ$ (transverse plane) had to be $< 0.3$ mV or (2) the maximum anterior amplitude plus the maximum rightward amplitude minus the amplitude at $-45^\circ$ must be $\geq 0.5$ mV.

Application of these criteria achieved 60% (50 of 84) sensitivity in patients with RVH, similar to that of several electrocardiographic criteria. Each of these criteria was more sensitive but significantly better ($p < 0.01$) than the best specificity of any ECG criteria ($78\%$, $5, 95\%$). The specificity of the proposed criteria was $96\%$ (310 of 324), significantly better ($p < 0.001$) than the specificity of the criteria (252 of 324) of existing VCG criteria. Thus, linear measurements of the QRS complex displayed on the VCG identify 60% of patients with moderate-to-severe RVH and falsely indicate RVH in only 4% of normal subjects.

THE VECTORCARDIOGRAM (VCG) is an excellent supplement to the standard electrocardiogram (ECG) in the diagnosis of right ventricular hypertrophy (RVH). Many criteria for RVH have been proposed that rely primarily on measurements of the QRS complex. Such measurements are very time-consuming and are subject to inaccuracies from errors in planimetry. Other studies have made use of complicated linear measurements, such as QRS deflection times, instantaneous vector measurements or computer-aided methods. These methods are prone to error because of the complexity of the measurements. Computer-aided methods are often clinically unavailable.

Other investigators have examined the sensitivity of VCG criteria, but the specificity has rarely been documented. The purpose of this study was to develop a set of simple, linear VCG criteria based on the QRS complex for the diagnosis of RVH. Characteristics required of the criteria were that they be quantitative, easily reproducible and have a 95% specificity.

Methods

Patient Selection

During 1970–1977, VCGs and ECGs were performed on all patients who underwent cardiac catheterization at Duke University Medical Center and the Durham Veterans Administration Hospital and on a group of healthy volunteers. Three groups of patients were studied. The first group, with a high likelihood of having isolated RVH, included patients with mitral stenosis diagnosed by cardiac catheterization. Two groups had a low likelihood of RVH: normal volunteers and patients with chest pain who were evaluated by cardiac catheterization and were found to be normal. No patients were included if the VCG indicated complete bundle branch block (QRS $\geq 120$ msec).

The 143 patients with mitral stenosis were selected retrospectively. Only patients who were found to have no significant mitral insufficiency, no other valvular disorders, normal coronary arteriograms and normal left ventriculograms were included.

A correlation between hemodynamic findings and the presence of RVH has been demonstrated. Patients with RVH secondary to mitral stenosis have increased right-sided cardiac pressures relative to the normal heart. For the purpose of this study, only patients with pulmonary arterial systolic (PAS) pressures greater than 40 mm Hg measured during resting conditions were considered to have definite RVH.

The normal controls for this study consisted of 324 patients divided into two groups. The first group consisted of 173 normal volunteers 20–29 years of age with no history of hypertension or heart disease. The second group consisted of 151 patients who had undergone cardiac catheterization for evaluation of chest pain. All patients in this group had normal coronary arteriograms and left ventriculograms, no valvular heart disease, and normal PAS pressures (< 30 mm Hg).
Data Collection

Standard 12-lead ECGs were recorded in the supine position using a Hewlett-Packard automatic cardiograph (1515B). The VCGs were recorded using either a Hewlett-Packard 1507A vectorcardiograph or an Instruments for Cardiac Research VCG-1B vectorcardiograph modified with a Hewlett-Packard 7806B oscilloscope and camera. Photographs of the frontal, transverse and left sagittal planes were recorded from the oscilloscope screen on Polaroid type 107 film. Chest electrodes (A, C, E and I) were placed at the fourth intercostal space as recommended for the supine position. A calibration of 1 mV per 2-4-cm deflection was used, depending on the size of the VCG loop. The initial QRS forces were enlarged with a calibration of 1 mV per 10-cm deflection and photographed, with P and T loops excluded. The VCG trace was interrupted every 2.5 msec (Hewlett-Packard) or every 2.0 msec (Instruments for Cardiac Research). The VCGs were recorded within 1 week before cardiac catheterization. All measurements were made manually from Polaroid prints of the VCG loops.

Previous VCG studies for the diagnosis of RVH have emphasized increased areas of the QRS loop initially in the left anterior quadrant and terminally in either the right posterior or the right anterior quadrant of the transverse plane. VCG studies for the diagnosis of left ventricular hypertrophy (LVH) have demonstrated increased amplitude of mid-QRS forces in the left posterior quadrant of the transverse plane. Two hypotheses were tested: first, that RVH might be optimally identified by criteria that indicate deviations of the quantitative amplitude of the QRS in a direction opposite to that of LVH, i.e., decreased maximum transverse magnitude; second, that the midpoint in the left posterior quadrant of the transverse plane (-45°) might provide a satisfactory location to measure the decrease in loop amplitude of mid-QRS forces, indicating RVH. Various algebraic combinations of the following linear measurements were also tested to maximize sensitivity while retaining the 95% specificity.

The following variables were measured by planimetry on all QRS loops, as suggested by previously published criteria, in the transverse plane: anterior QRS loop area, rightward QRS loop area, and QRS loop area in the left posterior quadrant; and in the frontal plane, QRS loop area in the right inferior quadrant. The following linear measurements (fig. 1) were made in the transverse plane to develop the proposed criteria: maximum rightward amplitude (R), maximum leftward amplitude (L), maximum anterior amplitude (A), and maximum amplitude at -45°. If no forces were present at -45°, zero was used for the computation. No measurements were made in the left sagittal plane.

Patients were initially evaluated for the presence of VCG evidence of RVH using any one of the criteria of Chou et al. (1) QRS loop in transverse plane directed clockwise; (2) anterior and rightward QRS loop area in the transverse plane is greater than 70% of the total, or the area in the left posterior quadrant is less than 30%; (3) QRS loop area in the right posterior quadrant (transverse plane) is greater than 20% of the total; and (4) QRS loop area in the right inferior quadrant (frontal plane) is greater than 20% of the total.

Standard 12-lead ECGs of all subjects were evaluated to determine the presence of RVH by the ECG criteria of Sokolow and Lyon and by the following point-scoring criteria used in the International Business Machines (IBM) automated ECG analysis program.

The frontal QRS axis must be 60-270°. (1) R + S in V6 < 0.6 mV — 3 points; (2) S in V6 > 0.3 mV — 3 points; (3) frontal axis 100-240° — 2 points; (4) R in V1 > S in V1 — 2 points; and (5) Rmax in V leads < 0.6 mV — 1 point. An ECG that received five or more points was considered diagnostic of RVH. For the purposes of this study, any ECG that received four points or less was considered normal.

Results

The first hypothesis tested was that RVH could be characterized by an absolute decrease in the maximum transverse magnitude, because Romhilt et al. found that LVH increased the maximum transverse magnitude. There is no discriminating magnitude of
the maximum transverse amplitude that is an accurate predictor of RVH (fig. 2). However, observation of the maximum transverse amplitude is an excellent method for screening patients without RVH who would be considered falsely positive by more sensitive criteria. Only one of the 84 patients (1%) with definite RVH had a maximum transverse amplitude ≥ 1.8 mV, but this magnitude was exceeded by 31 of the 324 patients (10%) in the control groups. Six of these 31 would have been considered falsely positive for RVH by the subsequently developed criteria.

The second hypothesis tested stated that the QRS-loop amplitude in the left posterior quadrant would be significantly less in patients with definite RVH than in normal controls. Figure 3 shows the results of the measurements of the amplitude at -45° in the transverse plane. A criterion of less than 0.3 mV was selected because of the high sensitivity achieved (44%, 37 of 84 patients) with a 95% specificity. Increasing the amplitude resulted in unacceptable specificity, while lowering it severely affected the sensitivity.

A further hypothesis suggested that some patients with RVH who failed to fulfill the requirement of diminished amplitude at -45° might have sufficient increases in initial anterior and terminal rightward forces that the sum of these might exceed their -45° amplitude by a critical amount (fig. 4). The best results were not significantly different from those achieved by observing only the amplitude at -45° in the transverse plane. RVH could be diagnosed with 48% accuracy in patients with definite disease if the anterior plus the rightward forces minus the amplitude at -45° was greater than or equal to 0.5 mV. This was achieved with an acceptable 96% specificity. Increasing or decreasing the fraction of the -45° amplitude yielded worse results by decreasing either the specificity or the sensitivity. Forty patients fulfilled this criterion, 13 of whom had not satisfied criterion 1 (table 1). Addition of criterion 2 to criterion 1 therefore improves the sensitivity from 44% (37 of the 84 patients) to 60% (37 + 13 of the 84 patients). Ten of the patients who met criterion 1 failed to meet criterion 2 (table 1).
the sensitivities obtained with the ECG criteria of Sokolow and Lyon (27%) or the IBM automated diagnostic program (23%), nor was there a significant difference between the sensitivity achieved using the proposed VCG criteria (60%, 50 of 84) and the Chou VCG criteria (52%, 44 of 84). There was a significant difference ($p > 0.01$) between the sensitivity of both VCG criteria (60% and 52%) and the best results obtained with the ECG (27%).

The specificity of the four criteria in the control groups are also evaluated in table 2. Both ECG criteria yielded identical results, a 98% (317 of 324) specificity. This was not significantly greater than the specificity of the proposed VCG criteria, 96% (310 of 324). Chou’s VCG criteria were accurate in only 252 of the 324 cases, a 78% specificity, which was significantly less ($p < 0.001$) than the results using either the ECG criteria or the proposed VCG criteria.

The percentages of diagnosed true positives by either ECG (Sokolow and Lyon) or VCG methods (proposed and Chou criteria) are compared in figure 5. In no instance did the VCG criteria fail to recognize RVH if it had been diagnosed by ECG methods. The proposed criteria, though not significantly more sensitive than the criteria of Chou et al., revealed 18% (16 of 84) of the total population with definite RVH not diagnosed by the Chou criteria. However, the Chou criteria recognized 10% (nine of 84) not diagnosed as true positives by the proposed criteria. In 25 of the 84 cases RVH was not diagnosed by any method.

The percentage of diagnosed true negatives is shown in figure 6. In no case did the ECG criteria (Sokolow and Lyon) fail to recognize a true negative diagnosed by either VCG criteria, nor did the proposed criteria fail to recognize a true negative diagnosed by the criteria of Chou et al. In only seven of the 324 patients (2%) was RVH falsely diagnosed.

The sensitivity of the proposed VCG criteria and the Sokolow and Lyon ECG criteria was tested in subgroups of patients with mitral stenosis with varying pulmonary arterial systolic pressures (table 3). All criteria have low sensitivity in patients with minimally elevated pressure ($< 30$ mm Hg). However, there is an increasingly greater difference between the capabilities of the ECG and the VCG criteria in the subgroups with increasingly severe pulmonary arterial hypertension. The exception occurs in the small group of 19 patients with systolic pressure greater than 80 mm Hg.

**Discussion**

The proposed VCG criteria yield a sensitivity that equals current VCG criteria and is significantly greater than current ECG diagnostic methods. The specificity of the proposed VCG criteria is significantly higher than that of current VCG criteria and is similar to that of ECG criteria.

The ECG lacks acceptable sensitivity in patients with RVH. In no instance did the VCG fail to recognize a patient with RVH if it had been indicated by the ECG. The VCG identified 20 additional patients with PAS pressures greater than 51 mm Hg.
TABLE 2. Evaluation of Electrocardiographic and Vectorcardiographic Criteria in Right Ventricular Hypertrophy and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>RVH group</th>
<th>Control groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 84)</td>
<td>83</td>
<td>293</td>
</tr>
<tr>
<td>Pts meeting VCG screening criterion (n)</td>
<td>37</td>
<td>9</td>
</tr>
<tr>
<td>Pts meeting VCG criterion 1 (n)</td>
<td>40</td>
<td>13</td>
</tr>
<tr>
<td>Pts meeting VCG criterion 2 (n)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sensitivity of proposed VCG criteria</td>
<td>50 (60%)</td>
<td>310 (96%)</td>
</tr>
<tr>
<td>Sensitivity of Chou VCG criteria</td>
<td>44 (52%)</td>
<td>252 (78%)</td>
</tr>
<tr>
<td>Sensitivity of Sokolow and Lyon ECG criteria</td>
<td>23 (27%)</td>
<td>317 (98%)</td>
</tr>
<tr>
<td>Sensitivity of IBM ECG criteria</td>
<td>19 (23%)</td>
<td>317 (98%)</td>
</tr>
</tbody>
</table>

Specificity of proposed VCG criteria | 50 (60%)  | 310 (96%)      |
Specificity of Chou VCG criteria | 44 (52%)  | 252 (78%)      |
Specificity of Sokolow and Lyon ECG criteria | 23 (27%)  | 317 (98%)      |
Specificity of IBM ECG criteria | 19 (23%)  | 317 (98%)      |

Abbreviations: VCG = vectorcardiographic; ECG = electrocardiographic; RVH = right ventricular hypertrophy.

TABLE 3. Sensitivity of Proposed Vectorcardiographic and Sokolow and Lyon Electrocardiographic Criteria with Progressively Higher Pulmonary Artery Systolic Pressures

<table>
<thead>
<tr>
<th>PAS pressure (mm Hg)</th>
<th>&lt; 30</th>
<th>30-50</th>
<th>51-80</th>
<th>&gt; 80</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>25</td>
<td>69</td>
<td>30</td>
<td>19</td>
</tr>
<tr>
<td>VCG</td>
<td>3 (12%)</td>
<td>27 (30%)</td>
<td>23 (77%)</td>
<td>14 (74%)</td>
</tr>
<tr>
<td>ECG</td>
<td>3 (12%)</td>
<td>12 (19%)</td>
<td>9 (30%)</td>
<td>8 (42%)</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>&lt; 0.01</td>
<td>&lt; 0.005</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>

Abbreviations: PAS = pulmonary artery systolic; VCG = vectorcardiographic; ECG = electrocardiographic.

and an additional 14 patients with pressures lower than 51 mm Hg.

The goal of this study was to develop simple, linear VCG criteria. Previous VCG criteria for the diagnosis of RVH have relied on complicated calculations or volumetric measurements. These measurements have been made either with the aid of computers or by planimetry. Linear measurements require no special equipment other than that used in evaluating standard ECGs. The calculations require no computing devices so that the VCG evaluation is in its simplest form. The results in table 2 demonstrate that there is no signifi-

FIGURE 5. Relative percentages of true-positive diagnoses of right ventricular hypertrophy (RVH) by electrocardiographic (ECG) and vectorcardiographic (VCG) methods in patients with definite RVH. The ECG criteria identified only 27% of patients. An additional 15% of patients met both the Chou and the proposed VCG criteria. Each of these criteria uniquely identified an additional 10-18% of patients. None of the criteria identified RVH in 30% of patients.

FIGURE 6. Relative percentages of true-negative diagnosis of right ventricular hypertrophy (RVH) by electrocardiographic (ECG) and vectorcardiographic (VCG) methods in normal control patients (n = 324). Application of the Chou VCG criteria yielded 22% false-positive diagnosis of RVH. The proposed VCG criteria yielded only 4% incidence of false-positive diagnosis, compared with the 2% obtained by application of ECG criteria.
significant difference in the sensitivity of either the proposed VCG criteria or the widely accepted criteria of Chou et al. However, sensitivity is significantly higher for the proposed criteria than for those of Chou et al.

In this study, RVH was considered definitely present if PAS pressure was ≥40 mm Hg in patients with proved mitral stenosis. Thus, RVH secondary to either obstructive or fibrotic pulmonary disease was not evaluated. The presence of such pulmonary disease, particularly emphysema, may distort the QRS complex.24 Thus, in studying patients with both lung disease and RVH, either ECG or VCG measurements may appear to be diagnostic of RVH, although they are actually related only to the pulmonary disease. This may explain the apparent discrepancy between the reported sensitivity of the IBM ECG criteria (51%) and the present analysis of these criteria (23%). The population used in defining the IBM criteria was obtained from routine autopsies and would be expected to contain many patients with QRS changes secondary to pulmonary disease.

It is important to contrast the results achieved using the proposed criteria for the diagnosis of RVH with the Romhilt criteria for LVH. Romhilt et al.20 found by comparing VCG results before death with autopsied hearts with LVH, that the VCG of the hypertrophied hearts had a maximal QRS amplitude in the transverse plane greater than or equal to 1.8 mV in patients over 50 years of age and greater than or equal to 2.2 mV in patients under 50 years of age. These criteria yielded a 61% sensitivity and a 100% specificity. The increase of specificity of the VCG LVH criteria over the VCG RVH criteria occurs because most electrical potentials are directed toward the left ventricle in a normal heart. Thus, a QRS loop indicating LVH will contain increased forces in the left posterior quadrant that will increase the maximal QRS magnitude. Alternatively, the hypertrophied right ventricle will produce electrical forces that are directed anteriorly and/or rightward. These forces continue to dominate with increasing pressure until the loop is eventually “inverted” and has a clockwise rotation that Chou and Helm labeled type A right ventricular overload.

These proposed VCG criteria for RVH provide a unifying concept for understanding the contrasting influences of LVH and RVH on the QRS complex. Since the VCG depicts this QRS complex in a single picture for each of the principal body planes, it permits a more quantitative evaluation. Future studies may provide improved quantitative criteria from the 12-lead ECG that will approach those proposed here for the VCG in sensitivity. Isolated mitral stenosis was selected for this study because of its measurable effects on the right ventricle. However, it is also important to study the effects of other clinical problems that overload the right ventricle on the QRS complex displayed on both the VCG and ECG.

Acknowledgment

The authors thank Conger Williams, M.D., for his advice in developing the concepts presented in this study. Also, we acknowledge the important contributions of Abe Walston, M.D., and Paul Gallentine, M.D., in a formative stage of the study. We also express appreciation to Virginia Utley and Jo Ella Martinez for preparation of the manuscript.

References

Is Postextrasystolic Potentiation Dependent on Starling’s Law?

Biplane Angiographic Studies in Normal Subjects

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SUMMARY

The cineangiograms of 26 normal subjects were analyzed to study the effect of Starling’s mechanism on postextrasystolic potentiation. The end-diastolic volumes (single plane and biplane) of the left ventricle were similar in the regular sinus beat before an extrasystole and sequential sinus beats after an extrasystole. However, the ejection fraction, mean normalized systolic ejection rate, mean velocity of fiber shortening and long-axis shortening were consistently larger in the first sinus beat after an extrasystole. We conclude that postextrasystolic potentiation is independent of left ventricular end-diastolic volume in normal human hearts and the compensatory pause after an extrasystole does not result in increased end-diastolic volume.

SINCE its first description by Langendorff1, postextrasystolic potentiation has become a well recognized phenomenon. Many investigations5-12 based on papillary muscle or heart-lung preparations have shown that postextrasystolic potentiation results in increased cardiac contractility regardless of changes in preload and afterload.

In the intact human heart, however, the nature of postextrasystolic potentiation is unclear. Cranefield, in a historical review, commented, “Starling’s law of the heart had come into the picture, and its widespread acceptance led many people more or less casually to attribute the increased force of the postextrasystolic beat to increased filling.” Braunwald, in reference to the intact ventricle, stated that it is independent of variations in diastolic filling of the ventricle; it has been demonstrated in the isovolumetrically contracting heart and in isometrically contracting cardiac muscle. But he also stated, “In the ejecting ventricle, when the premature beat is followed by a compensatory pause, the ventricular end-diastolic volume is augmented, and this increased preload contributes to the enhanced performance

which characterizes the postextrasystolic contraction.” Results of several recent reports15-18 support this concept. However, this view is not shared uniformly. Other investigators19,20 found that the left ventricular end-diastolic volume was not augmented after extrasystolic beats. To clarify this issue, we studied the relationship between the postextrasystolic potentiation and the left ventricular end-diastolic volume in normal subjects.

Materials and Methods

From 1974-1977, 2089 cardiac catheterizations were performed in the Clayton Foundation for Research Laboratory. Among these, the hemodynamics and coronary arteries were normal in 125 patients (6%). The cineangiograms of these patients were reviewed carefully. Only those with left ventriculograms of good quality that included an isolated premature ventricular contraction were selected for analysis. Twenty-six patients satisfied the criteria. All were premedicated with oral administration of 5 mg of diazepam, 25–50 mg of meperidine hydrochloride i.m. and 25 mg of promethazine hydrochloride.

At cardiac catheterization, the left ventricle was entered retrogradely from the brachial artery. Left ventricular images were recorded at 60 frames/sec simultaneously in the right anterior oblique (RAO) 30° and left anterior oblique 60° positions by injecting 50 ml of Renovist II (28.5% diatrizoate meglumine, 29.1% diatrizoate sodium and 31% bound iodine) into the left ventricle over 3–4 seconds. The peak of the R
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Circulation. 1980;62:1026-1032
doi: 10.1161/01.CIR.62.5.1026
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1980 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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