Quantitative Detection of Regional Left Ventricular Contraction Abnormalities by Two-dimensional Echocardiography

II. Accuracy in Coronary Artery Disease

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SUMMARY The quantitative approaches to the assessment of regional left ventricular (LV) function described in the preceding paper were applied in a well-defined population of patients with coronary artery disease. Two groups were chosen by electrocardiographic and angiographic criteria: group 1 had infarction and regional wall motion abnormalities and group 2 had no infarction and normal wall motion. Sensitivity to detect wall motion defects, specificity to correctly categorize normal segments, and overall predictive accuracy were evaluated for each two-dimensional echocardiographic approach. In addition, the ability of each method to localize regional contraction defects properly was evaluated. Area methods yielded better predictive accuracy than linear methods (87–95% vs 76–84%). No significant differences in accuracy were noted between quadrant and octant approaches. The fixed external-axis system was superior to a floating one for localizing contraction defects. We conclude that an area-based method, using a fixed-axis system and either octant or quadrant image subdivision, provides the best combination of predictive accuracy in categorizing LV segments as normal or abnormal and the greatest ability to localize LV regional abnormalities.

REGIONAL ABNORMALITIES of left ventricular (LV) contraction are a hallmark of both acute and chronic coronary artery disease (CAD). Clinical studies of LV contraction abnormalities by two-dimensional echocardiography (2-D echo) have reported regional contraction defects using qualitative recognition.1–4 Quantifying the degree and extent of regional wall motion abnormalities permits a systematic assessment of the effects of CAD on myocardial performance and provides an objective basis for examining LV function serially in the course of illness or after therapeutic interventions. Quantitative techniques for regional wall motion evaluation have been demonstrated to be superior to qualitative ones in the analysis of angiographic images,4 and several approaches to the problem have been evaluated.5–8 No standard quantitative approach has been established for the regional analysis of 2-D echo images.

This study was designed to evaluate the approaches to the regional analysis of 2-D echo images for sensitivity to detect localized wall motion abnormalities in CAD patients with remote myocardial infarction and angiographically documented contraction defects, and specificity to identify regions in patients without antecedent infarction and normal regional function proved by LV cineangiography.

Methods

The patients studied each had 12-lead ECGs, 2-D echo examination and LV and coronary angiography performed by standard techniques during the same hospitalization.

Acquisition and Digitization of Echocardiographic Images

Two-dimensional echocardiographic studies were performed with a Varian V-3000 phased-array sector scanner. All examinations included short-axis views at the levels of the mitral valve leaflet tips and the mid-papillary muscles and apical four- and two-chamber views.

Studies were recorded and traced as described previously, without knowledge of electrocardiographic or angiographic results. Technically adequate images for 2-D echo measurements were obtained in 74% of the CAD patients. Because of the superiority of the area-shrinkage method and the potential sensitivity of octant subdivision, this analytic system was initially applied to a series of well-defined subjects to evaluate its merits and limitations. Fixed- and floating-axis system frameworks were examined concurrently as well as quadrant subdivision of the area approach. Finally, hemiaxis and perimeter methods were used in parallel to provide additional information bearing on the conclusions of the preceding paper.

Electrocardiographic and Angiographic Data

Electrocardiograms without patient identification were analyzed independently by two observers for evidence of antecedent transmural myocardial infarction. Inferior infarction was defined as the presence of abnormal (0.04 second) Q waves both in leads 3 and in
aVF; anterior infarction was defined by the presence of abnormal Q waves in three or more adjacent precordial leads.

Quantitative ventriculography and ejection fraction determinations were performed from 35-mm right anterior oblique (RAO) cineangiograms using the area-length method. Regional function was quantified using the hemiaxis approach of Herman et al.7 All angiographic outlines were traced by one investigator who was kept blinded to electrocardiographic and echocardiographic results.

Selection Criteria

To provide unequivocal reference groups for evaluation, patients were grouped by the following criteria after all studies were completed.

Group 1 — Abnormal Regional Contraction

(1) Electrocardiographic evidence of transmural infarction defined as initial QRS forces on standard 12-lead ECG consisting of either abnormal (0.04) Q waves in inferior leads (III and aVF) or abnormal Q waves in three adjacent precordial leads.

(2) A visually evident area of regional contraction abnormality on RAO left ventriculography corresponding in location to the ECG area of infarction.

(3) Regional hemiaxis shortening of 20% or less defined by the Herman/Gorlin axis system in the region identified in (2) above.

Patients had to meet all three criteria to be included in group 1.

Group 2 — Normal Regional Contraction

(1) No ECG evidence of transmural infarction as defined above; no ST- or T-wave changes suggestive of nontransmural infarction or ischemia.

(2) Visually normal angiographic LV contraction pattern in the RAO projection.

(3) Regional shortening of greater than 20% in all hemiaxes defined by the Herman/Gorlin axis system.

Patients had to meet all three criteria to be included in group 2.

Echocardiographic Evaluation Criteria

The performance of 2-D echo for quantifying regional function was evaluated by the following criteria: (1) Sensitivity for detecting abnormally contracting segments. An abnormal segment was identified as one that fell more than 2 standard deviations below the mean contraction established on 10 normal subjects studied as described in the preceding paper. Eight octants were examined at the mitral valve level and eight octants at the papillary muscle level; in addition, the apex was analyzed separately, bringing the number of segments evaluated to a total of 17 in any given patient. (2) Specificity for identifying normally contracting segments. A normal segment fell within 2 standard deviations of the normal mean. Once again, 16 octants and the apical segment were separately evaluated. (3) Accuracy of different approaches for correctly categorizing regions as normal or abnormal.

Group 1 was used to assess sensitivity. An infarct was considered to be correctly identified when any of the four anterior regions (octant analysis) was abnormal for acute myocardial infarctions or any one of the four posterior segments was abnormal for inferior myocardial infarction at either mitral valve or papillary muscle levels. Sensitivity was defined as the number of infarcts correctly detected divided by the number of group 1 patients.

Group 2 was used to assess specificity. These patients were considered to be correctly identified by an approach only if all eight segments at both levels plus the apical segment fell within the 2 standard deviation bands of our normal range. Specificity was defined as the number of patients with all segments normal divided by the number of group 2 patients.

When comparing methods, accuracy was defined as the number of patients correctly categorized divided by the number of patients studied. The statistical significance of differences between the methods in sensitivity, specificity and accuracy was tested by chi-square analysis, with Yates’ correction for small sample size. Differences in regional contraction between groups and between methods were tested for statistical significance by unpaired and paired t tests, respectively.

Results

Twenty male patients, ages 38–70 years (mean 53 years), met the criteria for inclusion in group 1; 11 had inferior and nine anterior infarctions (table 1). Eighteen male patients, ages 32–68 years (mean 52 years), met the criteria for group 2.

Sensitivity to Detect Regional Contraction Abnormalities

Nineteen of 20 group 1 patients (95%) had at least one octant (mean 5.3, range 1–10) of abnormal area shrinkage corresponding to their region of contraction abnormality. The mean percent contraction for these subjects compared with the prior established normal range is shown in figures 1 and 2. The only patient who was not detected by quantitative criteria had an isolated anterolateral contraction defect. Apical area shrinkage was abnormal in eight of the 20 infarct patients. Six of these contraction defects occurred in the nine patients with anterior infarcts and two occurred in the 11 patients with inferior infarcts. The distribution of abnormally contracting segments in group 1 patients is displayed in figure 3. Patients with anterior infarcts had predominantly anterior contraction defects and patients with inferior infarcts had primarily inferior contraction defects; contraction abnormalities were detected less frequently in continuity with the involved zones in both patient subsets. In particular, unsuspected contralateral abnormalities occurred in 11% of anterior infarct patients and in 27% of inferior infarct patients (fig. 3).
### Table 1. Summary of Electrocardiographic and Angiographic Findings

<table>
<thead>
<tr>
<th>Pt</th>
<th>Infarct location</th>
<th>Angio EF</th>
<th>Abnormal contraction</th>
<th>Diseased vessels (lesions $\geq 70%$)</th>
<th>Group 2—no prior myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>AF</td>
<td>IMI</td>
<td>0.58</td>
<td>D</td>
<td>RCA, LAD, LCx</td>
<td>KL 0.58</td>
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<td>AB, AL, A</td>
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<td>AB, AL</td>
<td>RCA, LAD, LCx</td>
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<tr>
<td>JD</td>
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<td>GM</td>
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<td>LB 0.61</td>
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<td>LAD</td>
<td>AB 0.61</td>
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<tr>
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<td>AB, AL, A, D</td>
<td>LAD</td>
<td>RW 0.59</td>
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<tr>
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<td>0.39</td>
<td>AB, AL, A</td>
<td>LAD, LCx</td>
<td>RM 0.61</td>
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<tr>
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<td>JM 0.54</td>
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<td>0.50</td>
<td>AL, D</td>
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| Mean | 0.43 | Mean $\# = 2.1$ | Mean | 0.61 | Mean $\# = 1.7$ |

Abbreviations: AMI = anterior myocardial infarction; IMI = inferior myocardial infarction; angio EF = angiographic ejection fraction; AB = anterobasal; AL = anterolateral; A = apex; D = diaphragmatic; RCA = right coronary artery; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery.

### Specificity in Identifying Normal Contraction

Sixteen of 18 group 2 patients (89%) had all 16 octants (both mitral valve and papillary muscle levels) and all their apical segments fall within the prior established range of normal contraction. The two patients with 2-D echo defects had a total of three defects, all detected in short-axis sections. In one patient it was limited to a single octant at the mitral level; another had two octants abnormal, again at the mitral valve level (fig. 3). Thus, of 288 octants within this group, 285 were within established normal limits, as were all 18 apical segments. The mean contraction for each region in these subjects compared with the normal range is shown in figure 4.

**Figure 1.** Plots of regional contraction (mean ± SEM) as measured by the fixed-axis octant-area method, for the patients in group 1 with anterior myocardial infarction vs those of the normal volunteers. The data from the mitral valve (MV) level short-axis section is plotted at the left, the papillary muscle (PM) short-axis section at the right, and the apex contraction data at the far right. S = septal; PS = posteroseptal; P = posterior; PL = posterolateral; L = lateral; AL = anterolateral; A = anterior; AS = anteroseptal.
Alternative Methods of Analysis

Table 2 is a list of the overall predictive accuracy of fixed- and floating-axis systems when different methods are used to detect patients with and without localized LV contraction abnormalities. There were no significant differences in accuracy between fixed- and floating-axis system approaches. Hemiaxis and perimeter measurements were consistently less accurate than the best area methods of analysis \( p < 0.05 \).

However, the floating-axis system failed to localize contraction defects in the same manner as the fixed-axis system. The floating system does not separate the...
defects of anterior from inferior infarction as clearly as the fixed-axis system (fig. 5).

**Discussion**

Two-dimensional echocardiography and angiography are not perfectly analogous descriptors of LV geometry. The former is a tomographic procedure and the latter is a silhouetting technique. Angiography displays image boundaries orthogonal to the field of view, thus maximizing ventricular dimensions perceived by the viewer. This finding has been documented in studies that show that 2-D echo linear measurements are consistently smaller than their angiographic counterparts. Angiography is also limited in that it cannot account for segments of the ventricle that do not fall on the silhouette perimeter. Despite these limitations, angiography does provide an objective basis for ascertaining the presence or absence of wall motion abnormalities, particularly when it is supported by ancillary information. Considering concomitant ECG data in this study, angiography has served as an important indicator of regional wall function. Compared with angiography, 2-D echo provides more extensive sampling of the ventricle around its entire circumference at multiple sectional levels and in several different projections. Two-dimensional echocardiography allows measurement of the extent of endocardial wall motion as reported herein, and also can provide assessment of regional wall thickening and echo density.

The criteria for selection of patients for inclusion in either group reflects the lack of one generally accepted standard for defining the presence, location and extent of myocardial infarction. Combined electrocardiographic and angiographic criteria have been used to provide strong evidence of permanent regional myocardial damage. However, this selection process has an important impact on our study results. To evaluate new methods and the relative accuracy of the different analytic techniques it was necessary to have a very clearly defined patient population. The selection criteria were designed to provide unequivocal evidence of the presence or absence of infarction, so patients with relatively large infarcts were identified. Thus, the sensitivity of the present methods to detect smaller, and in particular nontransmural, infarcts is untested. Further work is needed to establish how small an infarct can be detected by our analytic methods. Nevertheless, the sensitivity and localizing ability of the area-based fixed reference system make it more desirable than the alternative methods. Ingels et al. reported similar success when examining left ventricular angiograms by a fixed external axis system.

### Table 2. Sensitivity, Specificity, and Predictive Accuracy of Alternate Methods of Analysis

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<th>Fixed axis</th>
<th>Floating axis</th>
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<tr>
<td></td>
<td>Sens</td>
<td>Spec</td>
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<tr>
<td>Area octant</td>
<td>95%</td>
<td>89%</td>
</tr>
<tr>
<td>Area quadrant</td>
<td>95%</td>
<td>78%</td>
</tr>
<tr>
<td>Hemiaxis</td>
<td>88%</td>
<td>78%</td>
</tr>
<tr>
<td>Perimeter</td>
<td>90%</td>
<td>72%</td>
</tr>
</tbody>
</table>

*Values compared with fixed-axis area-octant accuracy:
  * p < 0.05.
  † p < 0.001.

Abbreviations: Sens = sensitivity; Spec = specificity; Ace = predictive accuracy.

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**FIGURE 5.** Localization of contraction defects by fixed-axis and floating-axis systems. Plots of regional contraction for the papillary muscle level short-axis sections for the groups of patients having anterior infarcts vs those with inferior infarcts, as measured using the fixed-axis (left) and floating-axis (right) methods.
Our approach is a pragmatic delineation of regional wall motion using multiple echocardiographic sections. The mitral valve and papillary muscle levels from the parasternal short-axis projection yield circumferential images at two reproducibly identifiable ventricular levels. It would be desirable to evaluate more LV cross sections, particularly the cardiac apex, in the same projection. The lack of intrinsic ventricular landmarks below the papillary muscles limits reproducibility of imaging the cardiac apex in the short-axis projection. Attempts at short-axis views of the apex will produce images whose contraction may vary from 50–100%, depending on how close the cross-sectional plane lies to the tip of the ventricle. Because of the sensitivity of the cardiac apex to ischemic heart disease, we used alternative views. We used apical rather than parasternal long-axis projections because they are easier to obtain and because the sectional plane can be guided using cardiac valves and chambers near the base of the heart as landmarks.

The results herein confirm observations made in the preceding paper, i.e., that the area method of analysis is superior to linear measurement for characterization of regional LV function. In particular, area analysis showed a higher sensitivity, specificity and accuracy compared with hemiaxis and segmental perimeter methods. This finding is consistent with the observations of others who have analyzed angiographic LV silhouettes by area and linear methods.9

Interestingly, fixed- and floating-axis systems had comparable predictive accuracy for detecting the presence of regional contraction abnormalities. A floating-axis system involves translation and rotation of the systolic ventricle to the putative spatial position of the heart in diastole. The system herein reorients the systolic ventricular image to the position of its diastolic center of mass, making the contraction pattern of the ventricle more symmetrical near the apex, where the heart swings most anteriorly during systole. In so doing, however, this system minimizes the differences between the infarct populations (fig. 5). Thus, while the floating system had equal accuracy in detecting the presence or absence of regional contraction defects, it was less adequate for localizing these defects.

Not all infarcts resulted in contraction defects localized strictly to the same area (fig. 3). While eight of nine anterior infarcts and all 11 of the inferior infarcts had at least one of 16 octants 2 standard deviations below normal, no single octant showed universally defective contraction in either subgroup. In addition, in the population subset studied, inferior infarcts frequently had contiguous involvement of the interventricular septum. Furthermore, both groups had unsuspected contralateral defects.13 This variability in localization of contraction defects has been observed in both 2-D echo8 and radionuclide ventriculographic14 studies.

Short-axis left ventricular 2-D echo images readily allow segmental subdivision into many parts. Large subdivisions may fail to detect small contraction defects; very small subdivisions have a likelihood of increased error as the resolution of the method is approached. From a practical standpoint, echocardiographic short-axis sections are readily conceptualized as quadrants, and previous qualitative studies have shown the usefulness of quadrant subdivision compared with angiographic measurements of regional contraction abnormality.8 Within the context of the present study, further subdivision of the ventricle into octants made no difference. This still must be tested with smaller defects (e.g., nontransmural infarcts) to examine whether they are more readily detected by an octant system.

Apical contraction can be evaluated by analogous application of a quantitative system to apical images. All 18 of our patients without remote infarction had normal apical contraction. In contrast, six of nine patients with anterior infarcts had apical contraction abnormalities, while similar defects were present in only two of 11 patients with inferior infarcts. The higher prevalence of apical contraction defects in anterior infarction coincides both with clinical experience and with the usual distribution of the left anterior descending coronary artery in the anterior interventricular groove enveloping the cardiac apex.

In conclusion, our data demonstrate that 2-D echo can be used quantitatively to identify both normal and abnormal regional LV function with a high degree of accuracy. Area measurements have better sensitivity and specificity than linear measurements. There do not appear to be important practical limitations to using a fixed-axis system. Indeed, in this study, a fixed-axis rather than floating-axis system localized segmental contraction defects more satisfactorily. Quadrant subdivision of short-axis LV cross sections is simple, practical and convenient for reporting regional function; however, accuracy does not deteriorate when octants are used.

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Repertive Responses to Single Ventricular Extrastimuli in Patients with Serious Ventricular Arrhythmias: Incidence and Clinical Significance

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SUMMARY Electrophysiologic studies were carried out in 85 patients with serious ventricular arrhythmias: 44 with recurrent sustained ventricular tachycardia (group A), 16 with recurrent nonsustained ventricular tachycardia (group B), and 25 with recent prehospital ventricular fibrillation not associated with acute myocardial infarction (group C). Programmed ventricular stimulation from the right ventricular apex included premature stimulation during normal sinus rhythm, atrial pacing, and ventricular pacing, as well as brief bursts of rapid ventricular pacing (RVP). A repetitive ventricular response (RVR) was defined as one or more nonstimulated premature ventricular depolarizations in response to a single paced premature ventricular depolarization during normal sinus rhythm or atrial pacing. RVRs were observed in seven of 44 (16%) group A patients, one of 16 (6%) group B patients, and three of 25 (12%) group C patients. In contrast, single and double premature ventricular stimuli during ventricular pacing and/or bursts of RVP resulted in the reproducible initiation of ventricular tachycardia in 40 of 44 (91%) group A patients, 10 of 16 (63%) group B patients, and 19 of 25 (76%) group C patients. We conclude that RVRs to single ventricular extrastimuli during normal sinus rhythm or atrial pacing are rare, and therefore are an insensitive index of susceptibility to serious ventricular arrhythmias in these patients.

THE INCIDENCE and clinical significance of repetitive ventricular responses (RVR) to single ventricular extrastimuli during normal sinus rhythm (NSR) or atrial pacing has stimulated considerable interest and debate.1,2 Recently, Greene and co-workers reported a high (＞85%) incidence of RVR in patients with a history of recurrent ventricular tachycardia (VT).1,2 Their findings suggested that the suppression of RVR might constitute a useful end point to guide the selection of long-term prophylactic antiarrhythmic drug therapy.2 The purpose of this study was to assess the incidence of RVR and to compare the sensitivity of this finding with that of electrically stimulated VT in three populations of patients with serious ventricular arrhythmias.

Methods

Electrophysiologic studies were carried out in 85 patients who were referred for the management of serious ventricular arrhythmias. Sixty-four males and 21 females, mean age 56 years (range 19–75 years), were studied. Sixty-three patients had coronary artery disease, 10 had valvular heart disease, and eight had primary myocardial disease. Four patients had no detectable evidence of structural heart disease. The patients were divided into three groups, based on the rhythm disturbance for which they were referred. Group A included 44 patients with a history of recurrent (two to 80) episodes of symptomatic sustained VT that required drug therapy or cardioversion for termination. VT was associated with loss of consciousness in 21 patients and with presyncope, congestive heart failure or angina pectoris in 23. Group B included 16 patients with recurrent episodes of symptomatic nonsustained VT (≥ 5 beats) that were associated with syncope in seven patients and presyncope or palpitations in nine patients. Group C

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