Quantitative two-dimensional echocardiographic analysis of regional wall motion in patients with perioperative myocardial infarction

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ABSTRACT Regional left ventricular wall motion was evaluated by two-dimensional echocardiographic techniques with fixed- and floating-axis analytical algorithms in three groups of subjects: normal subjects (n = 15), patients undergoing uncomplicated coronary artery bypass graft surgery (CABG) (n = 10), and patients suffering perioperative myocardial infarction (n = 27). In patients undergoing uncomplicated CABG, fixed-axis analysis in the apical four-chamber view produced septal hypokinesis indistinguishable from the septal hypokinesis seen in patients with anterior myocardial infarction. In addition, fixed-axis analysis enhanced lateral wall motion so that patients with lateral myocardial infarction were classified as normal. Floating-axis analysis corrected these limitations by (1) producing regional left ventricular wall motion in the patients undergoing uncomplicated CABG, which was identical to that in normal subjects, and (2) producing regional left ventricular wall motion in patients with myocardial infarction that was hypokinetic in segments corresponding to the electrocardiographic area of involvement. In patients with new Q waves, fixed-axis analysis detected abnormalities of regional left ventricular wall motion in 24 of 34 (71%) electrocardiographically involved regions but also classified 44 of 100 segments in uncomplicated patients as abnormal. Floating-axis analysis detected regional left ventricular wall motion abnormalities in 30 of 34 patients (88%; p < .05 vs fixed-axis analysis) and only 15 of 100 segments in patients undergoing uncomplicated CABG were classified as abnormal (p < .001 vs fixed-axis analysis). We conclude that floating-axis analysis is a more accurate and clinically relevant method of evaluating regional left ventricular wall motion in patients undergoing CABG who suffer myocardial infarction as a perioperative complication.


SEVERAL studies have used noninvasive techniques to evaluate regional left ventricular function after acute myocardial infarction and have demonstrated that these analyses can provide valuable diagnostic and prognostic information.1-4 Similar studies have not been performed in patients undergoing coronary artery bypass graft surgery (CABG). This may be due in part to the difficulty in assessing postoperative regional function, particularly that of the interventricular septum, which has been found to be abnormal even when there is no other evidence of myocardial infarction.5-15 Recent work from this institution16 and others17, 18 has suggested that such abnormal motion of the interven-

tricular septum appears to be caused by exaggerated systolic anteromedial translation of the entire heart within the chest, as viewed from a fixed external frame of reference. We have demonstrated in patients with uncomplicated perioperative courses and normal septal function (as shown by preservation of systolic thickening of the interventricular septum) that the apparent deterioration in septal motion can be corrected by analyzing regional wall motion with an internal frame of reference or floating-axis analysis system.16 Floating-axis analysis not only corrects the abnormal motion of the septum but also normalizes the apparently enhanced motion of the lateral wall of the left ventricle concomitantly produced when a fixed external frame of reference is used.

These data, obtained from a homogeneous patient population with no evidence of perioperative myocardial dysfunction, implied that two-dimensional echocardiographic examination with a floating-axis system of analysis may represent a noninvasive method of
evaluating regional wall motion in patients undergoing CABG, which would provide data more consistent with their clinical course than methods dependent on a fixed-axis system. Floating-axis analysis is not without limitations, however. In nonsurgical patients imaged in the parasternal short-axis view, sensitivity for detection of the presence of a regional wall motion abnormality is good, but localization is suboptimal because regions that are hypokinetic tend to be normalized by floating analysis.19 We undertook this study to evaluate whether our observations in patients undergoing uncomplicated CABG could be extended to those who developed perioperative myocardial infarction.

Methods

Patient selection. We studied three groups of subjects. Group I consisted of 15 normal subjects, all with no evidence of cardiac disease by history or physical examination.

Group 2 (patients 1 to 10, table 1) consisted of 10 patients with normal preoperative wall motion as assessed by qualitative analysis of contrast ventriculograms; all underwent CABG without developing new Q waves. In addition, their clinical courses were uncomplicated, and peak creatinine kinase myocardial-specific bands (CK-MB), measured immediately after surgery and every 8 hr thereafter for a total of three samples, were all less than 90 IU/liter. At our institution, CK-MB levels of less than 90 IU/liter are only rarely associated with any detectable deterioration in regional or global left ventricular function when assessed by preoperative and postoperative quantitative two-dimensional echocardiography or radionuclide ventriculography.16 20

Group 3 was composed of 27 men selected over a 31 month period who developed evidence of perioperative myocardial infarction while undergoing CABG surgery. The sole selection criterion for this group was the development of new pathologic Q waves (at least 0.04 sec in duration and at least 25% of the height of the R wave) in two or more adjacent leads. We specified the localization of these Q waves to three separate regions: anterior, involving leads V1 to V2; inferior, involving leads 2, 3, and aVF; and lateral, involving leads 1, aVL, and/or V4 and V6. If Q waves appeared in more than one of these regions, then both regions were assumed to be affected. Preoperatively all patients had angina pectoris, six had histories of prior myocardial infarction, and seven had preoperative regional wall motion abnormalities as assessed by qualitative analysis of contrast ventriculograms (table 2).

Surgical technique. Myocardial preservation was achieved with systemic hypothermia and cold potassium cardioplegia. Left ventricular venting was performed through the right superior pulmonary vein. The pericardium was left open after surgery in all cases. Further details have been described in previous communications from this institution.16 21

Two-dimensional echocardiographic studies. The methods of image acquisition and quantitative analysis have been previously described16 and are described briefly here.

Image acquisition techniques. Two-dimensional echocardiographic studies were performed with a commercially available Hewlett Packard phased-array sector scanner (Model 77020A) with a 3.5 MHz transducer. The studies were performed at 5 to 12 days (mean 10) after surgery. Images adequate for detailed quantitative analysis usually could not be obtained before the fifth postoperative day. All patients were studied in the left lateral decubitus position.

Stop-frame end-diastolic and end-systolic images were displayed on a video monitor; end-diastole was defined as the peak of the R wave on the simultaneous electrocardiographic recording, and end-systole was defined as the smallest ventricular dimension during the last half of the T wave. Endocardial tracings were made on a transparent overlay placed on the monitor screen. All tracing was performed without knowledge of the electrocardiographic data.

Quantitative image analysis. Quantitative analysis of regional wall motion was made from the apical four-chamber and two-chamber (with aorta) views as described by Feigenbaum.22 The method of analysis with an Irex Cardio 80 computer has been previously described16 for the apical four-chamber view and was applied similarly to the apical two-chamber view. For floating-axis analysis, the long axis of the left ventricle was defined from internal points of reference for each diastolic and systolic image. The long axis was identified from the midpoint of the axis to the midpoint of the mitral valve for the apical four-chamber view and from the apex to the junction of the mitral and aortic valve in the apical two-chamber view. The midpoint of the long axis for each end-diastolic and end-systolic image was determined by the computer. The end-systolic image was transposed (or floated) so that the long axes and midpoints of each image were exactly superimposed. From the midpoint of the long axis 24 radii were drawn to the systolic and diastolic outlines of the left ventricle. The percentage change in area or area shrinkage for each segment was then calculated (figure 1). For the fixed-axis analysis the radii were generated from the midpoint of the diastolic long axis without transposing the end-systolic image (figure 1).

For the apical four-chamber view the segments were defined as follows (figure 1): radii 4 to 7, basal septum; 8 to 10, apical septum; 11 to 14, apex; 15 to 17, apical lateral; 18 to 21, basal lateral. For the apical two-chamber view, the following segments were similarly defined: anterobasal, anteroapical, apex, inferoapical, and inferobasal. The mean percent area shrinkage was determined for each region. Hypokinesis was defined as mean percent area shrinkage more than 2 SDs below the normal mean. Echocardiographic segments considered within the infarct zone for determination of ability to detect abnormalities of regional motion were arbitrarily defined as follows: anterior myocardial infarction, septal segments in apical four-chamber view and anterior segments in apical two-chamber view; lateral myocardial infarction, lateral segments in apical four-chamber view; inferior myocardial infarction, inferior segments in apical two-chamber view.

### TABLE 1

**Clinical data for group 2 patients (uncomplicated CABG)**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Pre-op. ECG</th>
<th>Pre-op. RLVWMA (CABG)</th>
<th>No. of grafts</th>
<th>ECG location of infarction</th>
<th>Peak CK-MB (IU/liter)</th>
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<tbody>
<tr>
<td>1</td>
<td>NL</td>
<td>—</td>
<td>3</td>
<td></td>
<td>71</td>
</tr>
<tr>
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<td>2</td>
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<td>38</td>
</tr>
<tr>
<td>3</td>
<td>LVH</td>
<td>—</td>
<td>1</td>
<td></td>
<td>58</td>
</tr>
<tr>
<td>4</td>
<td>NL</td>
<td>—</td>
<td>1</td>
<td></td>
<td>85</td>
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<td>NL</td>
<td>—</td>
<td>2</td>
<td></td>
<td>66</td>
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</tbody>
</table>

RLVWMA = regional left ventricular wall motion abnormality; NL = normal; LVH = left ventricular hypertrophy.
TABLE 2
Clinical data for group 3 patients (perioperative myocardial infarction)

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Pre-op. ECG</th>
<th>Pre-op. grafts (CABG)</th>
<th>No. of grafts RLWVMA</th>
<th>ECG location of infarction</th>
<th>Peak CK-MB (IU/liter)</th>
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<tbody>
<tr>
<td>11</td>
<td>LAHB</td>
<td>—</td>
<td>3</td>
<td>AMI</td>
<td>456</td>
</tr>
<tr>
<td>12</td>
<td>NL</td>
<td>—</td>
<td>2</td>
<td>AMI</td>
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</tr>
<tr>
<td>13</td>
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<td>3</td>
<td>AMI</td>
<td>286</td>
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<tr>
<td>14</td>
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<td>—</td>
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<td>AMI</td>
<td>139</td>
</tr>
<tr>
<td>15</td>
<td>IMI Inferior</td>
<td>—</td>
<td>3</td>
<td>AMI</td>
<td>128</td>
</tr>
<tr>
<td>16</td>
<td>NL</td>
<td>—</td>
<td>2</td>
<td>AMI</td>
<td>184</td>
</tr>
<tr>
<td>17</td>
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<td>—</td>
<td>2</td>
<td>AMI</td>
<td>516</td>
</tr>
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<td>18</td>
<td>NL</td>
<td>—</td>
<td>3</td>
<td>AMI</td>
<td>103</td>
</tr>
<tr>
<td>19</td>
<td>NL</td>
<td>—</td>
<td>3</td>
<td>AMI</td>
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<td>20</td>
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<td>—</td>
<td>2</td>
<td>AMI</td>
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<td>AMI</td>
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<td>—</td>
<td>3</td>
<td>AMI</td>
<td>N/A</td>
</tr>
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<td>25</td>
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<td>—</td>
<td>3</td>
<td>AMI</td>
<td>N/A</td>
</tr>
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<td>26</td>
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<td>—</td>
<td>3</td>
<td>AMI</td>
<td>103</td>
</tr>
<tr>
<td>27</td>
<td>NL</td>
<td>—</td>
<td>2</td>
<td>AMI</td>
<td>68</td>
</tr>
<tr>
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<td>3</td>
<td>AMI</td>
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<td>29</td>
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<td>LMI</td>
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<td>NL</td>
<td>—</td>
<td>5</td>
<td>LMI</td>
<td>181</td>
</tr>
<tr>
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<td>NL</td>
<td>—</td>
<td>6</td>
<td>LMI</td>
<td></td>
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<tr>
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<td>LMI</td>
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<td>1</td>
<td>IMI</td>
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<td>IMI</td>
<td>155</td>
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<td>35</td>
<td>NL</td>
<td>—</td>
<td>2</td>
<td>IMI</td>
<td>33</td>
</tr>
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<td>36</td>
<td>LAHB</td>
<td>—</td>
<td>3</td>
<td>IMI</td>
<td>42</td>
</tr>
<tr>
<td>37</td>
<td>NL</td>
<td>—</td>
<td>3</td>
<td>IMI</td>
<td>1176</td>
</tr>
</tbody>
</table>

NL = normal; LAHB = left anterior hemiblock; RLWVMA = regional left ventricular wall motion abnormality; AMI = anterior myocardial infarction; LMI = lateral myocardial infarction; IMI = inferior myocardial infarction; N/A = not available but greater than 100 IU/liter.

Results

Patients. Apical four-chamber views adequate for quantitative analysis were obtained from all 15 group 1 subjects. Eleven of these subjects (73%) had adequate apical two-chamber views.

For group 2, operative procedure and peak CK-MB levels are listed in table 1. Adequate apical four- and two-chamber views were obtained from all 10 of these patients.

For group 3, the procedure, electrocardiographic location of the infarction, and peak CM-MB levels are listed in table 2. Seventeen infarctions involved anterior leads, eight involved lateral leads, and nine involved inferior leads (five patients had infarctions involving two regions and one had all three regions involved). Twenty-five of the 27 patients developed Q waves on the first or second postoperative day, and 22 of the 25 had peak CK-MB levels greater than 100 IU/liter. Apical four-chamber views adequate for quantitative analysis were obtained from 26 of the 27 patients (96%) and adequate apical two-chamber views in 20 (74%).

Apical four-chamber view. In both groups of patients undergoing CABG (groups 2 and 3), septal endocardial motion was significantly less and lateral motion significantly greater when analyzed with a fixed-axis system than with the floating-axis system (table 3). This was not true for the normal subjects and is compatible with the previously described postoperative systolic anteromedial translation of the ventricle in the apical four-chamber plane.

Septal segments. Figure 2 shows the data points for septal segments in groups 1 and 2 and in the 16 group 3 patients with new Q waves in anterior leads. With fixed-axis analysis endocardial motion of both septal segments in group 2 patients was significantly less than normal (p < .01) and 85% of segments were classified as hypokinetic. Septal motion in the group 3 patients with anterior infarction was also significantly less than normal (p < .01) but was not different than that in group 2.

In contrast, when the floating-axis system was used, group 2 patients displayed septal endocardial motion that was indistinguishable from that of group 1 subjects, while the endocardial motion of the group 3 study. For the apical two-chamber view, intraobserver and interobserver variability were tested in 10 of the subjects randomly selected from this study. For all regions, correlation coefficients for intraobserver variability with fixed- and floating-axis systems varied from .88 to .97 and standard errors of the estimate (SEEs) were less than 6%. For interobserver variability, correlation coefficients varied from .88 to .95 and SEEs were less than 9%.

Statistically significant differences between fixed- and floating-axis analysis in the same group were determined with the t test for dependent means. Presence of a significant difference in segmental wall motion among the three groups was determined by analysis of variance, and if present, specific intergroup differences were then determined by the modification of Tukey's method for unequal sample sizes. In all figures data are plotted as the mean ± 1 SD.

Intraobserver and interobserver variability in the tracing of stopframe images and in the identification of the internal points of reference used to define the long axis of the left ventricle in the apical four-chamber view have been described in previous reports from our laboratory16, 23 and were not tested in this study.
patients with anterior myocardial infarction remained significantly depressed (p < .01 compared with groups 1 or 2).

Lateral segments. Figure 3 shows the data points for lateral segments in groups 1 and 2 and in the eight group 3 patients with new Q waves in lateral leads. With fixed-axis analysis endocardial motion of both lateral segments in group 2 was significantly greater than normal (p < .01) and 70% of segments were classified as hyperkinetic. In the group 3 patients with lateral myocardial infarction, endocardial motion was not significantly different from normal in either segment and only 12% of segments were classified as hypokinetic.

With floating-axis analysis endocardial motion of both lateral segments in group 2 was not significantly different from normal. In the group 3 patients with lateral myocardial infarction endocardial motion was significantly less than that of groups 1 and 2 (p < .01) and 94% of segments were hypokinetic.

Apical two-chamber view. In group 2 patients, anterior wall motion was slightly greater and inferior wall motion slightly less with floating-axis analysis than with fixed-axis analysis (table 4), but these trends did not achieve statistical significance. Thus results of fixed- and floating-axis analyses in the apical two-chamber view were more comparable than those in the apical four-chamber view.

**TABLE 3**

Mean percent area shrinkage of septum and lateral wall segments with fixed- and floating-axis analyses in the three study groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septum</td>
<td>Fd</td>
<td>Fl</td>
<td>Fd</td>
</tr>
<tr>
<td>Fd</td>
<td>51 ± 9%</td>
<td>0.5 ± 24.5%</td>
<td>0 ± 29%</td>
</tr>
<tr>
<td>Fl</td>
<td>53 ± 8%</td>
<td>53.5 ± 13%</td>
<td>30 ± 19%</td>
</tr>
<tr>
<td>Lateral</td>
<td>Fd</td>
<td>Fl</td>
<td>Fd</td>
</tr>
<tr>
<td>Fd</td>
<td>37 ± 7%</td>
<td>60 ± 14%</td>
<td>38 ± 12%</td>
</tr>
</tbody>
</table>
| Fl               | 40.5 ± 6% | 38 ± 6.5% & 24.5 ± 12% &

Fd = fixed-axis analysis; Fl = floating-axis analysis.

*Group 1, normal; group 2, patients undergoing uncomplicated CABG; group 3, patients with perioperative myocardial infarction.

Anterior segments. Figure 4 shows the data points for anterior segments in groups 1 and 2 and in the 14 group 3 patients imaged who developed new anterior Q waves. For the group 2 patients, endocardial motion of both anterior wall segments was not significantly different from normal with either fixed- or floating-axis analysis.

With fixed-axis analysis in the group 3 patients with anterior myocardial infarction, anterior wall motion was less marked than in both groups 1 and 2, but the trend did not achieve statistical significance in the anterobasal region (p = .10 vs group 1). With the floating-axis analysis, however, endocardial motion in both anterior segments was significantly less than that in group 1 or 2. However, both systems identified a comparable number of abnormal segments in the group 3 patients with anterior infarction; 13 of 28 with fixed-axis analysis and 14 of 28 with floating-axis analysis were hypokinetic. Ninety percent of segments were classified similarly with fixed- and floating-axis systems.

Inferior segments. Figure 5 shows the data points for inferior segments in groups 1 and 2 and in the eight group 3 patients imaged with new inferior Q waves. In group 2, wall motion of both inferior segments was not significantly different from normal with either system of analysis. For the group 3 patients with inferior infarction, endocardial motion of both inferior segments was significantly less than that in the other groups by either system of analysis. Ten of 16 segments with fixed-axis analysis and 11 of 16 with floating-axis analysis were classified as abnormal. Interestingly, four of the segments classified as normal were in patients 35 and 36, whose CK-MB activities were within the normal range (33 and 42 IU/liter).

Wall motion in the electrocardiographically uninvolved segments (i.e., anterior wall in patients with inferior infarction and inferior wall in patients with anterior infarction) was not significantly different from normal with either fixed- or floating-axis analysis.

Apex. Figure 6 shows the data points for groups 1
and 2 and for the 16 group 3 patients imaged in the apical four-chamber view and the 14 patients imaged in the apical two-chamber view with new anterior Q waves.

In group 2, apical endocardial motion was significantly different from normal with the floating-axis analysis in the apical four-chamber view (p < .05). Despite this statistically significant difference, only one of the 10 segments was classified as hypokinetic.

Analysis of the apex with either system in the apical four- or two-chamber view differentiated the group 3 patients with anterior infarction from patients in groups 1 and 2. In those patients with both apical four- and two-chamber views, agreement between the two views was 85% with fixed-axis analysis and 92% with floating-axis analysis.

**Overall.** There were 100 segments available for analysis in the 10 group 2 patients. With the floating-axis system, six segments were classified as hypokinetic (mean of 3% below the 2 SD limit) and nine were...
hyperkinetic (mean of 6% above the 2 SD limit). With fixed analysis, 21 segments were hypokinetic (mean of 29% below 2 SD) and 23 segments were hyperkinetic (mean of 14% above 2 SD). Floating-axis analysis classified significantly more segments as normal (p < .001 vs fixed-axis analysis).

Of the 34 regions in which new Q waves developed, 30 (88%) contained at least one abnormal segment when wall motion was analyzed with a floating-axis system. With fixed analysis, 24 of 34 (71%) electrocardiographically involved regions contained an abnormal segment. Floating-axis analysis was again superior (p < .05). This difference is primarily due to the inability of the fixed-axis system to identify patients with lateral myocardial infarction.

**Discussion**

Noninvasive evaluation of regional ventricular function is generally based on analysis of endocardial motion from a fixed external frame of reference (fixed-axis system). In most situations, motion of the whole heart is minimal and therefore regional endocardial motion does indeed represent regional function. In the postoperative CABG patient, however, we and others have shown that there is marked anterolateral translation of the heart, which when viewed with a fixed-axis system causes an apparent deterioration in septal endocardial motion and an apparent improvement in lateral endocardial motion. Thus regional endocardial motion no longer accurately reflects regional myocardial function, and paradoxical septal motion occurs despite normal systolic function (as assessed by systolic thickening) and preserved global ejection fraction (as assessed by radionuclide ventriculography).

These analytical problems are avoided in patients undergoing uncomplicated CABG by use of an internal frame of reference (floating-axis) system of analysis, which corrects for systolic translation and thus provides a potential method for noninvasively analyzing postoperative regional function.

In this study we further assessed the ability of fixed- and floating-axis systems to identify and correctly localize new regional wall motion abnormalities in patients who suffered perioperative myocardial infarction.

In the apical four-chamber view, fixed-axis analysis of patients who underwent uncomplicated CABG (group 2) resulted in marked septal hypokinesis that could not be differentiated from the septal hypokinesis seen in the group 3 patients with anterior myocardial infarction. In addition, it produced lateral wall hyper-

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**TABLE 4**

Mean percent area shrinkage of anterior and inferior wall segments with fixed- and floating-axis analyses in the three study groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior</td>
<td>Fd 56 ± 10.5%</td>
<td>57 ± 20.5%</td>
<td>40 ± 27%</td>
</tr>
<tr>
<td></td>
<td>Fl 55.5 ± 6%</td>
<td>63 ± 9%</td>
<td>40 ± 28%</td>
</tr>
<tr>
<td>Inferior</td>
<td>Fd 45 ± 9%</td>
<td>52 ± 13%</td>
<td>28 ± 27%</td>
</tr>
<tr>
<td></td>
<td>Fl 46.5 ± 7.5%</td>
<td>48 ± 11%</td>
<td>29.5 ± 26%</td>
</tr>
</tbody>
</table>

Fd = fixed-axis analysis; Fl = floating-axis analysis.

p = NS for all Fl vs Fd.

*Group 1, normal; group 2, patients undergoing uncomplicated CABG; group 3, patients with perioperative myocardial infarction.

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**FIGURE 4.** Anterior endocardial motion. Fixed- and floating-axis analyses of anterobasal and anterocapital wall motion as seen in the apical two-chamber view in groups 1 and 2 and in group 3 patients with new Q waves in anterior leads (Anterior Periop MI). Other abbreviations as in figure 2.
kinesis in group 2 patients and a "normalization" of regional wall motion in most lateral segments of the group 3 patients with lateral perioperative infarctions. For postoperative CABG patients these data raise serious questions as to the validity of any analysis of septal and lateral endocardial motion that depends on a fixed external frame of reference.

The floating-axis system functioned significantly better than the fixed-axis system in the apical four-chamber view in both groups of patients undergoing CABG. By removal of the effects of systolic translation, the apparent hypokinesis of the septal segments and hyperkinesis of the lateral segments in group 2 patients were completely corrected. In addition, the "normalization" of lateral segments in the group 3 patients with lateral myocardial infarctions was corrected with the floating-axis system.

Systolic translation of the heart appears to be minimal in the apical two-chamber plane and thus fixed-axis analysis in the apical two-chamber view coincides more closely with results of floating-axis analysis.

Overall, the floating-axis system performed well. In both the apical four- and two-chamber views, wall motion in segments involved by infarction was significantly less than that in groups 1 and 2. Thirty of 34 regions (88%) involved by infarction contained at least one abnormal segment.

How, then, are these results reconciled with pre-
vious work showing that floating-axis analysis tends to normalize regional wall motion abnormalities. First, the prior study was performed on nonsurgical patients in whom systolic translation of the ventricle is minimal. In addition, the parasternal short-axis view was used and floating-axis analysis requires floating a centroid or center of mass that is defined on the basis of the contour of the ventricle at end-diastole and end-systole. Examinations in the apical four- and two-chamber views, on the other hand, involve floating the long axis, which is defined by relatively consistent internal landmarks: the midpoint of the mitral valve, the mitral-aortic valve junction, and the apparent apex. Of these, the apparent apex is the most subjective, but this is true in contrast ventriculography as well and in this study did not appear to significantly limit our results.

In the past, other objections have been raised to the use of floating-axis systems. Ingels et al. implanted myocardial markers and then compared the motion of these to that seen with a fixed external frame of reference and internal frame of reference systems of analysis of contrast ventriculograms. They concluded that the least error was seen with the fixed external frame of reference. However, Clayton et al. used vector analysis of a spherical model of the ventricle and demonstrated that if any translation were introduced into the analysis, the fixed external reference system would no longer be necessarily superior. Given the marked systolic translation in the apical four-chamber plane, we believe any theoretical advantage to a fixed-axis analysis is certainly lost in the postoperative patient.

The problem of regional wall motion analysis in postoperative patients might better be approached by analyzing systolic thickening, since the effects of systolic translation on such an analysis would be negligible. In addition, analysis of systolic thickening appears to be superior to analysis of endocardial motion in experimental animals for both identification and quantification of myocardial infarction. We have found, however, that in the early postoperative period deterioration of parasternal short-axis images, possible secondary to mediastinal air, prevents quantitative analysis of thickening in a significant percentage of our patients. This deterioration is much less apparent in the apical view.

Limitations. Limitations can be divided into those related to this study and those related to the method. The major potential limitation of this study is our reliance on new pathologic Q waves as predictors of abnormal regional wall motion in the perioperative period. Although occasional reports have questioned the significance of postoperative Q waves, positive predictive accuracy in most contrast ventriculographic series has ranged from 73% to 100%. Combining new Q waves with elevation of serum CK-MB levels has increased positive predictive accuracy to nearly 100%.

Limitations of the method are largely those of two-dimensional echocardiographic techniques and relate to obtaining an image adequate for detailed quantitative analysis. As noted, inability to delineate endocardium or truncation of the chamber prevented analysis in 12% of the views. This problem is not unique to postoperative patients, since this figure is similar to that obtained in our preoperative patients and to that obtained by Quinones et al.

Another major limitation to the approach described here involves analysis of the apex. As can be seen from figure 6, normal apical area shrinkage is low, making differentiation of normal and abnormal motion difficult. This problem is not limited to two-dimensional echocardiography, however, since it has been described recently in a contrast ventriculographic study by Sheehan et al., who found that motion at the apex needed to be nearly dyskinetic to be classified as abnormal. In addition, venting through the left ventricular apex, although not usually done for CABG, might complicate analysis by creating a regional abnormality.

A potential limitation in the application of this approach relates to the frequent postoperative pericardial effusion. If large, an effusion can cause the scan plane to change dramatically from diastole to systole, thus confusing the interpretation of endocardial motion. Fortunately, large effusions are not common (less than 10%), and if they do not cause marked changes in scan plane, the floating-axis system should correct for systolic translation caused by the effusion.

Summary. In summary, this study supports our previous finding that use of a fixed external frame of reference in analyses of regional wall motion in CABG patients in the apical four-chamber view is prone to a systematic error. Floating-axis analysis corrects for this and is the more appropriate method for noninvasive assessment of regional wall motion in the postoperative patient. Quantitative two-dimensional echocardiography may therefore provide a means of examining important questions concerning the significance of perioperative myocardial infarction.

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