Two-dimensional Echocardiographic Analysis of Segmental Left Ventricular Wall Motion Before and After Coronary Artery Bypass Surgery

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SUMMARY Twenty patients with coronary artery disease were studied with two-dimensional echocardiography the day before saphenous vein bypass graft surgery. Serial studies were obtained 7.4 ± 2.5 (± SD) and 43.4 ± 13.1 days postoperatively to qualitatively assess the effect of bypass surgery on regional wall motion. Changes in segmental wall motion were assessed semiquantitatively by assigning a segmental wall motion score to each of nine echocardiographically defined segments. Preoperatively, 18% of the segments moved abnormally. The mean overall segment wall motion score did not change significantly, as shown by comparing the postoperative studies with the preoperative study. However, there was a significant worsening in septal motion (apical and basal) and a significant improvement in posterior wall motion (apical and basal) after bypass surgery. Anterior and lateral wall motion were not significantly changed. Nonseptal segments that were normal preoperatively usually remained normal; abnormal nonseptal segments usually improved or were unchanged by surgery. The motion of septal segments, however, generally worsened postoperatively whether they were normal or abnormal preoperatively. We conclude that segmental wall motion assessed by two-dimensional echocardiography may improve after revascularization surgery, but the interventricular septum shows impaired motion. This effect of coronary artery bypass surgery on wall motion is better demonstrated relatively late after operation than early in the postoperative course, as has been done in some previous studies.

STUDIES on the effect of coronary artery bypass graft surgery on left ventricular wall motion have yielded conflicting results. Some investigators have noted postoperative improvement in overall ventricular function, regional wall motion, or ejection fraction after surgery, but others have detected no change or a deterioration in these variables in significant numbers of patients. Two-dimensional echocardiography permits assessment of segmental left ventricular wall motion serially and without risk to the patients, although it currently provides less quantitative information than other techniques.

As part of our ongoing evaluation of patients with coronary artery disease using two-dimensional echocardiography, we assessed segmental left ventricular wall motion in patients immediately before and twice after coronary artery bypass graft surgery. Our goals were to use multiple two-dimensional echocardiograms to analyze segmental motion over the entire ventricle; to observe the overall results of revascularization by comparing the effects of surgery on both normal and abnormal (preoperative) segments; and to assess changes in regional wall motion occurring 1–6 weeks postoperatively.

Methods

Patient Population

Twenty patients admitted to the University Cardiovascular Surgery Service were studied. Pertinent clinical and anatomic data are summarized in table 1. No patient had suffered a myocardial infarction within 1 month before operation. Patients with unstable angina and documented or suspected coronary artery spasm were excluded. All patients had a history of typical angina pectoris refractory to medical therapy. Obstructive coronary lesions compromising 50% or more of the luminal diameter were identified angiographically. The patients had an average of 2.8 ± 0.7 (± SD) lesions, and the mean percent decrease in luminal diameter was 85.9 ± 14.5%.

The basic operative technique used low-flow, low-pressure cardiopulmonary bypass and modest (30–31°C) systemic hypothermia. Left ventricular venting was avoided, but decompression of the left heart was accomplished during the ischemic arrest interval by a small (4F) pulmonary artery sump vent in some patients. Reversed saphenous vein was used exclusively; "jump grafts" were used only for left anterior descending–diagonal combinations. Six patients required right coronary endarterectomies. All distal anastomoses were constructed during one period of aortic cross-clamping, during which the myocardium was protected by continuous profound (4°C) topical hypothermia supplemented by a single 500-ml bolus of cold (4°C) hyperkalemic ([K+] = 30 mEq/l) cardioplegic solution. The average anoxic interval was 50 ± 17 minutes. All proximal anastomoses were performed during cardiopulmonary bypass during resuscitation of the empty, beating heart and rewarming. An average of 3.0 ± 1.0 grafts were placed. The average cardiopulmonary bypass time was 89 ± 28 minutes. The pericardium was left open in all patients.

Sodium nitroprusside was used liberally in the early postoperative period to maintain mean arterial pressure below 80 mm Hg. There were no operative deaths.
Perioperative myocardial infarction was assessed by the appearance of new pathologic Q waves on serial ECGs. The overall incidences of perioperative myocardial infarction and operative mortality during calendar year 1979 in 437 patients undergoing isolated coronary artery bypass grafting at Stanford Medical Center were 2.8% and 0.7%, respectively.

Data Acquisition

All 20 patients underwent two-dimensional echocardiography on the day before coronary artery bypass surgery. Each patient gave informed consent in accordance with the requirements of the Committee on the Use of Human Subjects in Research at Stanford University Medical Center. Early postoperative echocardiograms were obtained an average of 7.4 days after surgery (range 5–17 days), when the patients were ambulatory and ready for discharge; 16 of the 20 patients returned for a “late” postoperative clinic appointment and a second postoperative echocardiogram. The average interval between surgery and the late postoperative study was 43 days (range 21–86 days). Clinical information on the four patients who failed to return for echocardiography at 1 month was obtained from the patient’s private physician, but the remainder were evaluated in the cardiovascular surgery clinic. Eighteen patients were asymptomatic at the time of follow-up. Patient 5 continued to have

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Mean ± SD 62.7 ± 7.9 2.8 ± 0.7 85.9 ± 14.5 3.0 ± 1.0 7.4 ± 2.5 43.4 ± 13.1

*Percent maximal decrease in luminal diameter.

Abbreviations: CX = circumflex coronary artery; 2-D echo = two-dimensional echocardiography; Diag = diagonal branch; Inter = intermedius coronary artery; LAD = left anterior descending coronary artery; LMCA = left main coronary artery; OM = obtuse marginal branch; PDA = posterior descending artery; RCA = right coronary artery.

severe congestive heart failure; patient 14 continued to have chest pain postoperatively that was suspected to be angina.

Echocardiographic Methods

The two-dimensional echocardiograms were obtained with a Toshiba ultrasonoscope (SSH-10A Ultrasonolayergraph) with a 2.25-MHz transducer. Patients were examined supine or in variable degrees of left lateral decubitus position to optimize image quality. Sector scans were recorded on videotape for subsequent analysis. A simultaneous audio channel was used to record a description of the patient position to minimize differences over the three examinations. Each echocardiographic study included standard views from parasternal, apical and subcostal transducer positions.13

For purposes of segmental wall motion analysis, the left ventricle was divided into nine segments (fig. 1). These segments were labeled anatomically but conform with the terminology used for angiographic ventricular wall segments in the Collaborative Study in Coronary Artery Surgery.14 Most segments were identified from more than one transducer position. The nine echocardiographic segments were based on subdivision of the ventricle into three craniocaudal regions: the base, extending from the aortic annulus and atrioventricular ring to just above the papillary muscles; the midportion, including the papillary muscles; and the apex, distal to the base of each papillary muscle. Four ventricular walls were identified and each was divided into basal and midportion segments. Thus, the anterior wall contained the anterobasal and anterolateral segments; the posterior wall contained the posterobasal and diaphragmatic segments; the lateral wall contained the superolateral and inferolateral segments; and the

Figure 1. Left ventricular wall segment classification. (A) Parasternal long-axis view (above) showing the segments defined for this study. Lower drawings show segments at the mitral valve level (left) in the basal portion of the heart and at the papillary muscle level (right) in the more apical portion of the heart from parasternal short-axis views. (B) Apical long-axis and four-chamber views of the heart showing the segments defined for this study.
medial wall (septum) contained the basal septal and apical-septal segments.

A segment was considered suitable for wall motion analysis if 75% of its entire endocardial outline was visible continuously throughout systole and diastole. Ventricular wall motion was classified as normal, hypokinetic, akinetic or dyskinetic according to normal usage as applied to contrast angiography. As with qualitative angiographic analysis, motion in a given patient was judged hypokinetic both relative to motion of other segments considered normal in the same patient and relative to prior experience of the observers. For each patient, an average index of regional wall motion was calculated for each study by assigning a numerical score to each segment analyzed. A score of 2 was assigned for normal motion, 1 for hypokinesis, 0 for akinesis and −1 for dyskinesis. Thus, a patient with normal motion in each of nine segments would be assigned a total score of 18 and an average index of 2.0. All studies were reviewed by at least two observers without knowledge of the electrocardiographic or angiographic findings and the final interpretation of motion was made by consensus. Grading of the motion of the basal segments of the intersected walls was facilitated by the use of M-mode tracings obtained by guiding an M-mode cursor through the appropriate segment while observing the real-time two-dimensional image from the parasternal or subcostal transducer location.

The paired t test was used to compare the indexes of regional wall motion between the preoperative and either the early or late postoperative study. A two-tailed p value \( \leq 0.05 \) was considered significant.

**Results**

**Number of Segments Suitable for Analysis**

A direct segment-by-segment comparison of wall motion was obtained for 157 of the 180 (88%) possible segments between the preoperative study and early postoperative (early) study in 20 patients. In the 16 patients who had a late postoperative (late) study, 129 of a possible 144 segments (90%) were suitable for analysis.

**Effect of Bypass Surgery on Segmental Wall Motion**

These results are summarized in table 2 and figures 2 and 3.

**Overall**

At the early study, qualitative analysis showed that compared with the preoperative study, 12 segments (8%) were improved, 102 segments (65%) were un-

### Table 2A. Quantitative Motion Analysis of All Segments

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Values are mean ± sd.

* p < 0.05.
† p < 0.01.
‡ p < 0.005.
§ p < 0.001.

Abbreviations: Early = early postoperative study; Late = late postoperative study; Pre = preoperative study; SWM = segmental wall motion.
### Table 2B. Quantitative Motion Analysis of Preoperatively Normal and Abnormal Segments

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<tr>
<td></td>
<td>Inferior</td>
<td>14</td>
<td>2.00 ± 0.00</td>
<td>14</td>
<td>2.00 ± 0.00</td>
<td>13</td>
<td>2.00 ± 0.00</td>
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<tr>
<td></td>
<td>Both</td>
<td>32</td>
<td>2.00 ± 0.00</td>
<td>30</td>
<td>2.00 ± 0.00</td>
<td>28</td>
<td>2.00 ± 0.00</td>
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<td>2.00 ± 0.00</td>
<td>14</td>
<td>1.64 ± 0.84</td>
<td>11</td>
<td>2.00 ± 0.00</td>
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<td>13</td>
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<td>13</td>
<td>1.77 ± 0.60</td>
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<tr>
<td></td>
<td>Both</td>
<td>28</td>
<td>2.00 ± 0.00</td>
<td>27</td>
<td>1.70 ± 0.72</td>
<td>21</td>
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<td>Total</td>
<td>141</td>
<td>2.00 ± 0.00</td>
<td>133</td>
<td>1.59 ± 0.71§</td>
<td>113</td>
<td>1.68 ± 0.71§</td>
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<tr>
<td></td>
<td>Septum</td>
<td>8</td>
<td>0.63 ± 0.74</td>
<td>7</td>
<td>0.29 ± 0.49</td>
<td>6</td>
<td>0.50 ± 1.05</td>
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<td>Anterior</td>
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<td>0.38 ± 0.74</td>
<td>8</td>
<td>0.50 ± 0.76</td>
<td>4</td>
<td>1.25 ± 0.96</td>
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<td>Lateral</td>
<td>4</td>
<td>0.75 ± 0.50</td>
<td>3</td>
<td>1.33 ± 1.15</td>
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<tr>
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<td>Posterior</td>
<td>11</td>
<td>0.45 ± 0.82</td>
<td>11</td>
<td>1.45 ± 0.82*</td>
<td>8</td>
<td>1.88 ± 0.35†</td>
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<tr>
<td></td>
<td>Total</td>
<td>31</td>
<td>0.52 ± 0.72</td>
<td>29</td>
<td>0.90 ± 0.90</td>
<td>20</td>
<td>1.35 ± 0.93‡</td>
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</table>

Values are mean ± SD.

* p < 0.05.

† p < 0.01.

‡ p < 0.005.

§ p < 0.001.

Abbreviations: See table 2A.

changed, and 43 segments (27%) were worse. At the late study, 13 segments (10%) were improved, 92 segments (71%) were unchanged, and 24 segments (19%) were worse compared with the preoperative study. There was a significant quantitative change in the segmental contraction score in several instances (table 2).

If only the nonseptal segments are considered, 11 (9%) were improved, 91 (76%) were unchanged and 17 (14%) were worse at the early study compared with the preoperative study (figs. 4 and 5). Similarly, when the late study was compared with the preoperative study, 12 segments (12%) were improved, 79 (80%) were unchanged and eight (8%) were worse. If only the septal segments are considered, one segment (2%)
Abnormal Preoperative Changes

The worse at 0.52 ± 0.01) and study, preoperative abnormal motion before index when the wall motion index increased from 0.72 to 1.02 when the two posterior segments were considered together or in combination with the two lateral segments, there was a significant improvement in wall motion index postoperatively (both early and late). Specifically, when the 14 abnormal posterior and lateral wall segments were considered together, the average wall motion index increased from 0.5 ± 0.76 at the preoperative study, to 1.43 ± 0.85 at the early (p < 0.01) and to 1.90 ± 0.32 at the late study (p < 0.005). When all the abnormal segments were analyzed, the average wall motion index increased from 0.52 ± 0.72 to 0.90 ± 0.90 (p < 0.05) at the early study and to 1.35 ± 0.93 (p < 0.005) at the late study.

Normal Preoperative Segments

Of the 157 segments available for comparison between the preoperative study and the early study, 128 (82%) were normal preoperatively (table 2). The qualitative changes at the early and late studies are shown in figure 2. None of the segments could improve at the early study because all were normal originally. When these changes were analyzed quantitatively there was a significant (p < 0.001) decline in the mean wall motion index, from 2.00 ± 0.00 at the preoperative study to 1.60 ± 0.70 at the early study and to 1.67 ± 0.72 at the late study. There was no significant change between the early study and the late study.

Figure 4 shows the qualitative changes when the two septal segments were excluded from the analysis. Quantitatively, there was still a significant (p < 0.01) decline in mean segment score, but the mean score for the septal segments was 0.90 ± 0.83 at the early study and 0.96 ± 1.02 at the late study, whereas for all the other segments together, it was 1.82 ± 0.48 at the early study and 1.89 ± 0.35 at the late study.

Wall Motion Changes as a Function of Anatomic Location

Analysis of regional wall motion for each anatomic location showed that the effect of bypass surgery was not uniform throughout the ventricle. Abnormal wall motion was improved more frequently in the inferior
and lateral walls. Nine of 10 segments (90%) that were contracting abnormally (seven hypokinetic, one akinetic and two dyskinetic) preoperatively were improved at the late study. All nine segments then showed normal contraction. The tenth remained hypokinetic. In the inferior and lateral segments, preoperative normal wall motion was preserved in all 47 segments available for evaluation.

In the anterior and apical segments, of the eight segments with abnormal contraction preoperatively (four hypokinetic, three akinetic and one dyskinetic), two were improved at the early study and three of four available for late study were improved. Anterior and apical segments that were normal preoperatively remained normal in 30 of 38 instances (79%).

By contrast, of the eight septal segments with abnormal preoperative wall motion (six hypokinetic, one akinetic and one dyskinetic), only one of six (17%) available for late analysis was improved. Furthermore, of the 25 segments that were originally normal, 15 (60%) were abnormal at the late study. The mean septal wall motion index of the preoperatively normal segments decreased from 2.00 ± 0.00 to 0.96 ± 1.02. Overall septal motion index (normal and abnormal segments) decreased from 1.73 ± 0.64 at the preoperative study to 0.82 ± 0.86 at the early study (p < 0.001) and was not significantly different at the late study.

**Incidence of Perioperative Myocardial Infarction**

Perioperative myocardial infarction was diagnosed in one patient and suspected in two others. In patient 7, new Q waves indicative of inferior infarction were accompanied by deterioration in apical septal motion (from normal to severe hypokinesis), although the posterobasal, posteroapical and inferolateral wall motion improved at the time of early echocardiographic study. Patient 10 showed electrocardiographic signs of possible posterolateral injury and deterioration in posterolateral segment wall motion. Patient 12 also showed ECG signs of possible inferior infarction postoperatively. His lateral and posterobasal segments at the early and late studies were unchanged compared with the preoperative study; but his posteroapical segment was not well seen preoperatively, was hypokinetic at the early study, and was normal at the late study. This pattern is consistent with resolving myocardial injury. His posteroapical segment was normal angiographically before surgery.

**Discussion**

Coronary artery bypass graft surgery is effective in relieving angina pectoris. It is generally believed, although less well documented, that ventricular function also may be improved after operations in many patients. The impact of myocardial revascularization on regional ventricular wall motion has been studied, but the results of these studies are conflicting. Little change or deterioration in regional function has been described by some authors, whereas others have reported improvement. In some cases, resting wall motion was unchanged but improvement was seen with exercise or during pacing stress. The extent to which these conflicting results may be due to differences in the imaging technique (e.g., contrast angiography) and method of wall motion analysis is uncertain. The timing of the postoperative evaluation and vein graft patency rate may also be important determinants of regional wall motion. Finally, most investigators have limited their analysis of wall motion to a small number of ventricular wall segments that are readily accessible to the technique applied.

We attempted to extend these earlier findings by systematically dividing the left ventricle into nine segments that are distinguishable by two-dimensional echocardiography and correspond anatomically to each of the angiographic segments accessible to biplane angiography. A precise schema for classification of ventricular wall segments using echocardiography has not been agreed upon, and several similar classifications have been described. Our method of wall motion analysis is qualitative and depends upon identification of the endocardium throughout the cardiac cycle, as described by Kisslo et al. Performing both
an early and late postoperative examination has enabled us to evaluate the effects of surgery as a function of time.

We observed a high incidence of new wall motion abnormalities at the time of the early postoperative study. Ninety percent of the patients had at least one segment in which wall motion deteriorated; overall, approximately 30% of the segments were worse. Similar results have been reported by other investigators; however, in these analyses, the effect of operation has been considered on a global basis, incorporating all wall segments. In our patient population, only 18% of the segments showed wall motion abnormalities preoperatively. Since the remaining normal segments may only remain unchanged or deteriorate, the number of segments capable of manifesting improved wall motion is substantially lower than the total available for analysis.

Postoperative total occlusion of native vessels proximal to a patent graft or of vein grafts has been suggested as a cause of perioperative myocardial infarction and new postoperative wall motion abnormalities. In our study, one patient suffered an unequivocal perioperative myocardial infarction and two had suggestive ECG changes. However, we are able to correlate the location of the damage by ECG with an area of new (or worse) wall motion abnormality in only one segment in each of the patients with equivocal changes. All three patients with ECG changes in inferior or leads had grafts to the distal right coronary artery or the posterior descending coronary artery. None of our patients underwent postoperative angiography. Thus, the graft patency rate in this selected group is unknown. It is reasonable to expect that the graft patency rate would equal or exceed the 75% rate reported for similar patients from Stanford in 1973. Grafs were placed into the distribution of each major vessel with a 50% or greater stenosis in an attempt to completely revascularize the left ventricle (table 1). The occurrence of perioperative infarction in our patients may be due to graft closure, but that is an unlikely explanation for the observed frequency of new wall motion abnormalities.

We were not so surprised by the apparent deterioration in normal preoperative septal or other segmental motion in some subjects as we were specially interested to note clearly improved motion after revascularization in most segments that had abnormal preoperative motion. This represents a small series with abnormal resting preoperative motion, but for logistical reasons we did not seek or identify only stress-induced abnormal motion or attempt to improve resting motion abnormalities by provocative tests in these patients. Nevertheless, 41% of the abnormal preoperative segments were improved 1 week after surgery and 65% showed improvement when assessed late postoperatively. The mean wall motion score for abnormal preoperative segments increased from 0.52 ± 0.72 to 1.35 ± 0.93 (p < 0.005) at the late study.

Although 37 of 128 preoperative segments (29%) showed new abnormal motion early postoperatively, 16 of 28 segments (57%) studied late returned to normal. Thus, 1–2 weeks after surgery is a suboptimal time to evaluate the effects of bypass surgery on wall motion. This may be due to ongoing dynamic changes in (1) myocardial edema and compliance caused by the anoxic interval and cardiopulmonary bypass during surgery; and (2) the pleural-myocardial-pericardial spatial and tension relationships caused by pericardiotomy and variable amounts of residual intrapericardial thrombus or fluid. Substantial additional improvement in regional wall motion may be observed at least over the first postoperative month. Similarly, ejection fraction decreases early after operation and then increases later in the postoperative period.

Postoperative echocardiographic abnormalities of septal motion have been observed frequently after valve replacement and coronary bypass surgery. The mechanism of this abnormality has been debated. Both pericardiotomy and occult septal myocardial damage have been proposed as the cause of this phenomenon. Akins et al. implicated cardiopulmonary bypass or the technique of myocardial preservation by showing preserved or improved septal motion without using cardiopulmonary bypass in patients undergoing isolated bypass grafts to the left anterior descending or right coronary artery. In our studies, postoperative deterioration in wall motion was observed in approximately one-fifth of the segments analyzed at 6 weeks. In the majority of these patients, the new wall motion abnormality occurred in septal segments. Thus, it is important to assess the effects of surgery by specific anatomic regions. In our analysis, the inferior and lateral segments improved most frequently after surgery. At 1 month, eight of nine inferior and all three lateral segments that were abnormal preoperatively showed improved wall motion. Normal wall motion preoperatively was restored in all of the inferior and lateral segments at 1 month. Overall improved or preserved wall motion was observed for the anterior and apical segments as well, albeit less frequently. In contrast, 15 of 25 normal septal segments deteriorated at the late study and only one of six abnormal segments improved. Although our criteria for excluding perioperative septal infarction were less rigorous than the criteria of other investigators, it seems unlikely that this factor could account for the high frequency of septal abnormalities in our patients.

Bulkley and Hutchins reported a high incidence of major regional myocardial contraction band necrosis in the distribution of patent grafts that may be due to reperfusion injury but is not detected by electrocardiography or enzymes.

**Limitations**

Our results are based on qualitative observations in a small series of patients, and the number of segments with resting wall motion abnormalities preoperatively was limited. By evaluating only resting left ventricular segmental wall motion, two-dimensional echocardiography may fail to identify reversible, ischemia-related wall motion abnormalities that may potentially be im-
proved by revascularization. Thus, we might have underestimated the percentage of segments that actually improved. Inclusion of more patients with more severe and diffuse wall motion abnormalities might strengthen or alter our observations. Good agreement has been reported for wall motion analysis by two-dimensional echocardiography and contrast angiography, although we recognize the limitations of arbitrarily subdividing the ventricle into nine segments when sharp demarcation between segments may not be completely accurate. Using each subject as his own control minimizes variability of identifying wall segments and evaluating their respective motion status. Our data show significant improvement during the first 6 weeks postoperatively in wall motion in segments with abnormal motion preoperatively. However, we have not assessed the duration of these changes. When we observed deterioration in wall motion postoperatively, we have not attempted to assess related early graft or native coronary occlusion or perioperative complications other than infarction.

We conclude that bypass grafting frequently improves regional ventricular wall motion in segments with resting abnormal preoperative motion. Segments with normal motion preoperatively usually remain normal. However, only 12% of the preoperatively abnormal septal segments improved after operation, and 58% of the preoperatively normal segments were abnormal at the late evaluation. We cannot explain this observation. Segmental wall motion was generally better at the late study than at the early study, which suggests that the final effects of surgery may not be evident until after the patient is discharged from the hospital.

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