Two-dimensional Echocardiographic Recognition of Myocardial Injury in Man: Comparison with Postmortem Studies

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SUMMARY To assess the accuracy of phased-array two-dimensional echocardiography in detecting, localizing and quantifying myocardial injury in man, the relationship of two-dimensional echocardiographic wall motion abnormalities to morphologic evidence of myocardial infarction was evaluated in 20 autopsied patients. Comparisons were made between independent two-dimensional echocardiographic readings of left ventricular segmental wall motion and morphologic evidence of myocardial injury. Of 15 infarcts, 14 were detected by regional akinesis, dyskinesia or hypokinesia. The relationship between abnormal segmental wall motion and morphologic evidence of myocardial necrosis or fibrosis was significant. Seventy-nine of 88 (90%) of infarcted segments showed abnormal wall motion, although 38 of 82 (46%) of morphologically normal segments also demonstrated wall motion abnormalities. Fifty-eight of 65 segments that showed regional akinesis or dyskinesia were transmurally infarcted. Twenty-five of 38 pathologically normal segments seen by two-dimensional echocardiography as akinetic or dyskinetic were adjacent to scar. Hypokinesia was non-specific (31 segments normal, 21 subendocardial infarction). Normal wall motion excluded transmural infarction (0 of 46 segments), but in one patient was associated with subendocardial injury (nine/42 segments).

We assessed circumferential extent of regional akinesis or dyskinesia in blind fashion with a light-pen system, expressed as a percentage of end-diastolic circumference, and compared this with the corresponding coronary section of the left ventricle examined pathologically. By linear regression, extent of two-dimensional echocardiographic akinesis or dyskinesia and extent of left ventricular circumference demonstrating morphologic evidence of transmural infarction correlated well: pathologic percent circumference infarcted = 1.14 (two-dimensional echocardiographic percent circumference akinetic/dyskinetic) - 14.48; r = 0.90. Wall motion abnormalities therefore overestimated the amount of myocardial circumference infarcted, possibly because of the proximity of morphologically normal segments to scar or because segments adjacent to the lesions were reversibly ischemic. The results suggest that two-dimensional echocardiographic evidence of regional wall motion abnormality is sensitive in detecting and localizing segmental pathologic myocardial lesions, but overestimates their extent.

THE ASSOCIATION of left ventricular regional wall motion abnormalities with pathologically proved myocardial infarction is well established in man using contrast ventriculographic techniques.1, 2 Real-time two-dimensional echocardiography holds promise for the noninvasive evaluation of patients with ischemic heart disease. Studies from this laboratory suggest that two-dimensional echocardiography permits detection of acute alterations in cardiac topography by serial examination of patients with acute myocardial infarction to make the antemortem diagnosis of regional myocardial dilatation and wall thinning.3 Wall motion abnormalities on two-dimensional echocardiography correlate remarkably well with angio- graphic asynergy.4 M-mode5 and sonomicroscopic6 techniques have demonstrated a clear correlation of regional wall motion abnormalities with pathologically proved myocardial infarction in the experimental animal. However, the extent to which such wall motion abnormalities on two-dimensional echocardiography predict the presence and extent of infarcted tissue in man is not known. The most direct approach to evaluating the ability of two-dimensional echocardiography to quantify injury or scar in man is to use postmortem studies as the standard for comparison.

In this report we address the questions: (1) Can real-time two-dimensional echocardiography accurately detect and localize myocardial infarction in man, using postmortem findings as the standard for comparison? (2) Can two-dimensional echocardiography provide a quantitative estimate of the circumferential extent of myocardial infarction? (3) What is the pathologic significance of regional wall motion abnormalities detected by two-dimensional echocardiography? To answer these questions, we compared two-dimensional echocardiograms with postmortem findings in 20 patients.

Methods

Patients

Of 800 patients studied by two-dimensional echocardiography at the Johns Hopkins Hospital, 20 came...
to postmortem examination and formed the study group. No patient was excluded from the study on the basis of the quality of the echocardiogram. However, three patients were excluded from quantitative circumferential analysis (vide infra) because of such technical limitations. The characteristics of the patient population are listed in figure 1. Of the 20 autopsied patients, 11 had coronary artery disease, four had valve disease, two had primary myocardial disease and three had normal hearts. Fifteen patients had old or recent myocardial infarction. Four had small lesions (two transmural, two subendocardial) involving less than 20% of the left ventricular mass. Three of these four had infarcts involving less than 5% of the myocardium. Eleven patients had large transmural lesions that involved more than 25% of the left ventricle. Sixteen of the 20 patients died within 14 days of echocardiographic examination. Of the four patients who died 24–75 days after echocardiographic examination, two experienced sudden arrhythmic deaths and two died acutely at surgery. Mean time from echocardiographic examination to death in this population was 12 ± 5 (±SD) days. No patient showed clinical evidence of new myocardial injury between the time of echocardiographic study and death.

Pathologic Studies

At the time of autopsy the heart was removed from its attachments after in situ inspection. The heart was weighed and inspected after removing as much postmortem clot as possible. The coronary artery ostia were examined and an appropriate size of polyethylene tubing for cannulation was selected (Clay Adams, Intramedic, PE 190–240). Cannulas were inserted in the coronary ostia, and a modified Schlesinger injection mass7 that contained barium gelatin and either Monastral red or Monastral blue pigment was used for the left and right coronary arteries, respectively. One milliliter of formaldehyde was added to the 14 ml of medium, stirred and poured into the appropriate syringe barrel. Air pressure was created in the syringes and monitored by a pressure gauge. Injection pressure was gradually increased to 100–150 mm Hg and maintained for 20–30 minutes. After completing the injection, the ligatures were loosened, the cannulas pulled out and the ligatures quickly tightened.

The specimen was immersed in 15% formalin and the chambers were distended with formalin through cannulas at a level of 20–40 cm H2O pressure through the tubes. This fixed the heart in a state approximating that during life. After overnight fixation, the cannulas were removed and the heart was washed. Photographs were taken of the heart in the anterior and posterior aspects. Stereoscopic radiographs were taken of the intact heart and its transverse (breadloaf) sections were prepared corresponding to the two-dimensional echocardiographic views to be discussed (vide infra). The heart was examined, the coronary arteries were transected at 5-mm (or closer) intervals and compared with the radiographs, and portions of the heart and coronary arteries were taken for histologic examination, using hematoxylin-eosin and Verhoeff/van Giesen elastic stains. Coronary artery stenosis was considered significant if greater than 75% luminal diameter.

The state of the myocardium was determined by gross examination and light microscopy, with specific reference to the echocardiographic sectors. Within the designated regions the presence, age and width of myocardial wall involvement by necrosis and fibrosis were determined. A quantification of the extent of myocardial damage by necrosis was then made.8 With

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**Figure 1.** Characteristics of the patient population. Of 800 patients studied by two-dimensional echocardiography, 20 underwent autopsy and formed the study group. Fifteen of these patients had myocardial infarctions. Three of four small infarcts and 11 of 11 large infarcts were detected by two-dimensional echocardiography (see text). TM = transmural; SE = subendocardial.
the heart sectioned transversely at approximately 1-
cm intervals from apex to base, tracings of the area
and circumference of the infarcted ventricular wall
were made by planimetry for each slice. Both cut sur-
faces were planimetered, and if a discrepancy existed,
an average value was taken. Histologic sections were
prepared from each slice and the extent of the infarct
was confirmed by microscopy. Extent of the infarct
was expressed both in terms of percentage of total cir-
cumference and degree of wall thickness. For the
latter, segments were scored as follows: 3 — no infar-
tion; 2 — subendocardial infarction; and 1 — trans-
mural or near-transmural infarction (75–100% of the
ventricular wall thickness).

Echocardiographic Studies

Two-dimensional echocardiograms (Varian 3000
phased-array ultrasonograph) of the 20 autopsied pa-
tients were analyzed independent of both clinical
history and pathologic findings. Segmental wall mo-
tion analysis was performed according to the diagram
in figure 2. When technically feasible, three cross-
sectional views were obtained. Several segments are
present in more than one section (fig. 2).

To match each echocardiographic cross-sectional
view to its postmortem equivalent, care was taken that
each view traversed one of the following sets of inter-
nal landmarks: mitral valve tips, papillary muscle tips,
the first appearance of the ventricular cavity at the
apex of the heart. Each postmortem heart was cut
carefully at these levels to optimize the correspon-
dence between echocardiographic views and patho-
logic specimens.

Analysis of Data

Two-dimensional echocardiograms were analyzed
visually by two independent observers, and the
segments described above were each given a numerical
score, based on the degree of regional wall motion ab-
normality: 3 — normal segmental wall motion; 2 —
regional hypokinesis; and 1 — akinesis or dyskinesis.

For the independent two-dimensional echocardi-
ographic scores described above, discrepancy analysis
was carried out. Discrepancies were classified as either
major (observers differing by a numerical score of
more than 1) or minor (differing by a score of 1). Of
170 echocardiographic segments analyzed, we found
19 (11%) minor and five (3%) major discrepancies.
These discrepancies were resolved by consensus and a
final score was recorded.

We also assessed circumferential extent of regional
akinesis/dyskinesis by two-dimensional echocardiog-
raphy, expressed as a percentage of end-diastolic cir-
cumference, and compared it with the corresponding
cross section of the left ventricle examined patho-
logically (fig. 3). We measured endocardial circum-
cumference with a light-pen system (Varian Instruments)
interfaced with a videotape recorder (International
Video Corp.). By means of this system, circum-
ferential extent of regional akinesis or dyskinesis,
expressed as a percentage of the end-diastolic cir-
cumference, was determined at the mitral valve,
papillary muscle tips and the apical levels in the 11
patients with transmural infarctions, when such deter-
minations were technically feasible.

The method of calculating endocardial circum-
ferential extent of akinesis or dyskinesis is shown in
figure 4.

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**Figure 2. Format for segmental wall motion analysis of two-dimensional echocardiograms. On the left is
the standard sagittal view, divided into five anatomic segments. When technically feasible, three cross-
sectional views were obtained. Segments of anterolateral and posteroinferior walls are referred to as 4A, 5A
and 4P, 5P, respectively; 4A and 5A not readily visible on sagittal view.**
Statistical Analysis

To disprove the null hypothesis that there is no significant relationship between echocardiographic wall motion abnormalities and the pathologic state of the myocardium, both in terms of detection and localization of infarcted regions, we used the chi-square statistic with contingency format. We also used a 3 × 3 contingency table format when analyzing the relationship between the degree of wall motion abnormality seen echocardiographically and the degree of infarct thickness noted pathologically. To disprove the null hypothesis that there is no significant relationship between the percentage of abnormally contracting circumference and the percentage of endocardial circumference involved by myocardial infarction, circumferences determined by the two techniques were compared by using two-variable, least-squares, linear regression analysis.

Results

Detection of Myocardial Infarction

Fifteen of the twenty autopsied patients had old or recent myocardial infarction (fig. 1). Eleven patients had large transmural lesions that involved more than 25% of the left ventricle. Of the 15 myocardial infarctions, 14 were detected by regional wall motion abnormalities of akinesis, dyskinesis or severe hypokinesis. Three of the four small myocardial infarctions were detected. Two of these had been clinically silent, undetected before death. All of the 11 large infarctions were detected by two-dimensional echocardiography. The one infarct not detected clinically or by two-dimensional echocardiography was a healed circumferential subendocardial infarction involving less than 5% of the left ventricular myocardium in a 19-year-old man with remote valve surgery. There was a highly significant relationship between echocardiographic regional wall motion abnormalities and the presence of myocardial infarction (p = 0.0003, Fisher's exact test).

Figure 3. Light-pen method of assessing circumferential extent of regional akinesis/dyskinesis by two-dimensional echocardiography. (A) Standard cross-sectional echocardiographic view of the left ventricle (here at the level of the papillary muscles) without light-pen contour of the endocardial circumference. (B) Same view with such contour.

Figure 4. Method of calculating endocardial circumferential extent of akinesis or dyskinesis. Superimposing stop-frame end-diastolic and end-systolic circumferences, extent of circumference showing either absent or dyskinetic motion (A) is calculated as a percent of the entire end-diastolic circumference comprising \( A + B \). \( B \) represents that portion of circumference contracting normally. %ACC (percent abnormally contracting circumference) = \( A \), the abnormally moving segment length, \( \times \) \( \frac{A}{A+B} \times 100 \).
Echocardiographic Localization of Infarcted Segments

We assessed the accuracy of two-dimensional echocardiography in localizing infarcted myocardial segments by using the segmental wall motion analysis described above. In the 20 patients studied, 170 segments were technically adequate for visual analysis. Table 1 displays these segments in contingency table format, comparing for each the presence or absence of abnormal regional wall motion (top axis) with the presence or absence of pathologically established myocardial infarction (side axis). The relationship between abnormal segmental wall motion on two-dimensional echocardiography and postmortem findings was again highly significant. Thus, infarct was almost always associated with regional wall motion abnormalities, which were present in 90% of the pathologically infarcted segments. The nine segments that were infarcted but showed normal wall motion were all from the patient who had a thin circumferential subendocardial infarction after remote valve surgery and that involved less than 5% of the left ventricular mass. On the other hand, though infarct usually meant regional wall motion abnormality, regional wall motion abnormality signified myocardial infarction far less frequently. Thirty-eight (46%) of the morphologically normal segments demonstrated segmental wall motion abnormalities. Twenty-five of these segments (66%) were directly adjacent to scar, while 13 (34%) were not. Thus, although two-dimensional echocardiographic evidence of regional wall motion abnormality appears sensitive in localizing scar, it may overestimate its extent. This may in part be the result of chronic ischemia or the direct proximity to scar in the majority of the spuriously abnormal echocardiographic areas.

Relationship of Segmental Wall Motion Abnormalities to Infarct Thickness

Using the segmental analysis described above, the relationship of echocardiographic segmental wall motion abnormalities to infarct thickness was assessed. To do this, the echocardiographic segments were subjected to a × 3 contingency analysis, comparing for each the presence of normal, hypokinetic and akinetic or dyskinetic wall motion with histologically normal muscle, subendocardial or transmural infarction. Table 2 shows this analysis. Examining the data from the standpoint of pathologic findings, to which the listed percentages refer, all transmurally infarcted segments were akinetic or dyskinetic. Subendocardially infarcted segments demonstrated hypokinesis 50% of the time, more often than either normal or absent wall motion. Nonetheless, nine (21%) of subendocardially infarcted segments moved normally and 12 (29%) were akinetic or dyskinetic. Fifty-four percent of the morphologically normal segments showed normal echocardiographic motion, but revealed varying degrees of abnormal wall motion in a substantial percentage of cases (table 2). Hypokinesis was the most frequent spurious finding in these pathologically normal segments; akinosis or dyskinesis was far less frequent. It should be emphasized again that 66% of the pathologically normal segments with abnormal wall motion were directly adjacent to scar and 34% were not.

Echocardiographically, 46 (71%) of the 65 akinetic or dyskinetic segments (table 2) were transmurally infarcted. Hypokinesis could not distinguish between subendocardial infarction and uninjured myocardium (31 and 21 segments, respectively), but never signified transmural infarction. Normal wall motion (table 2) signified normal myocardium in 44 segments and subendocardial infarction in only nine segments), but excluded transmural infarction. As seen by the chi-square value, the relationship between infarct thickness and the degree of wall motion abnormality on two-dimensional echocardiography is significant. The degree of wall motion abnormality usually corresponds to the degree of thickness of the infarct.

Circumferential Extent of Myocardial Infarction

To determine the accuracy of two-dimensional echocardiography in quantifying the circumferential

<table>
<thead>
<tr>
<th>Postmortem findings</th>
<th>Two-dimensional echo segmental wall motion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Normal (54%) Hypokinesis (38%) Akinesis/dyskinesis (8%)</td>
</tr>
<tr>
<td>Infarction</td>
<td>9 (21%) 21 (50%) 12 (29%)</td>
</tr>
<tr>
<td>Transmural MI‡</td>
<td>0 (0%) 0 (0%) 46 (100%)</td>
</tr>
</tbody>
</table>

**df = 4, χ² = 115.4, p < 0.0001 (Yates’ continuity correction).**

*Twenty-five segments were adjacent to scar; 13 were not.

Numbers refer to individual segments.

†Five segments were adjacent to scar; two were not.

‡Percentages refer to postmortem findings.

§All < 33% of the transmural thickness of the left ventricle.

†All ≥ 75% of the transmural thickness of the left ventricle.

Abbreviation: MI = myocardial infarction.
extent of myocardial infarction, we examined the relationship of the extent of the left ventricular circumference that demonstrated the segmental wall motion abnormalities of akinesis or dyskinesis to the percent of transmurally infarcted left ventricular circumference by postmortem examination (fig. 5). In the 11 patients with postmortem evidence of transmural infarction, 13–100% of the left ventricular circumference was involved. One hundred percent circumferential involvement was, in all cases, localized to the apex. By linear regression analysis, the extent of two-dimensional echocardiographic segmental wall motion abnormalities, expressed as a percentage of the end-diastolic circumference, correlates well with the percentage of left ventricular circumference demonstrating morphologic infarction (r = 0.90). The regression equation is: Path = 1.14 Echo − 14.48, where path = percent circumference of infarcted myocardium (postmortem studies), and echo = percent circumference of segmental wall motion abnormalities (two-dimensional echocardiography). Echocardiographic wall motion abnormalities thus tended to exceed, by approximately 14%, the amount of myocardial circumference involved by injury.

Six more patients without transmural infarcts had echocardiograms that were technically adequate for digitization. This provided an additional 16 data points, all at the origin (0,0). When these six patients are included in the regression analysis, with the origin thus heavily weighted, the equation is: Path = 0.88 Echo + 0.18; r = 0.94, SEE = 8.87.

Coronary Arteriography of Abnormally Moving Noninfarcted Segments

Examining the relationship between the abnormally moving noninfarcted segments and postmortem coronary angiography, 26 of these 37 segments (70%) were in the distribution of a coronary artery with greater than 75% stenosis. Of the seven akinetic or dyskinetic segments that were not infarcted, six were in the distribution of a significantly stenosed coronary vessel.

Discussion

We have examined the correlation of real-time two-dimensional echocardiography with postmortem studies. To assess the accuracy of phased-array two-dimensional echocardiographic determinations of myocardial lesions, we compared two-dimensional echocardiographic readings of left ventricular regional wall motion with morphologic evidence of myocardial injury in 20 autopsied patients. We found a significant relationship between echocardiographic regional wall motion abnormalities and myocardial infarction. Regional akinesis or dyskinesis usually signified transmural infarction. Hypokinesis was non-specific, being unable to differentiate between subendocardial infarction and pathologically normal segments. Normal wall motion excluded transmural infarction, but occasionally was associated with subendocardial injury. To assess the ability of two-dimensional echocardiography to quantify myocardial injury in man, we examined the relationship of circumferential echocardiographic evidence of akinesis or dyskinesis to morphologic evidence of myocardial infarction on a section-by-section basis. Extent of akinesis or dyskinesis by two-dimensional echocardiography and the extent of left ventricular circumference that showed morphologic evidence of transmural infarction correlated well. Two-dimensional echocardiography moderately overestimated the actual amount of myocardial circumference infarcted.

Both M-mode and sonomicrometric techniques have demonstrated that regional wall motion abnormalities correlate well with pathologically demonstrated myocardial infarction in the experimental animal. Few studies have examined the ability of echocardiography to localize or quantify ischemia or scar in the human using postmortem studies as the standard for comparison. Studies that have compared two-dimensional echocardiographic findings with postmortem studies have been carried out only in a few patients. Using M-mode echocardiography, Rasmussen et al. detected scar tissue; Heikkila and
Nieminen detected asynergy in acute myocardial infarction in patients, and confirmed the validity of such evaluations of segmental ventricular function in two patients who underwent necropsy. The site of asynergic left ventricular wall abnormalities also correlated well with the electrocardiographic prediction of the site of the acute myocardial infarction, as was the case in a recent two-dimensional echocardiographic study by Heger et al. In this latter report, data on four autopsied patients suggested that two-dimensional echocardiography could detect infarcted tissue by the presence of asynergy.

By examining the data from the echocardiographic standpoint we assessed the pathologic significance of wall motion abnormalities. All transmurally infarcted segments visible on two-dimensional echocardiography were akinetic or dyskinetic; normal wall motion thus excluded transmural infarction. Hence, akinesis appeared to be a sensitive indicator of transmural infarction, but not specific. Eight percent of the akinetic or dyskinetic segments were histologically normal, and 29% were subendocardially infarcted. Hypokinesis was still less specific, being relatively evenly distributed between normal myocardium (38%) and subendocardially infarcted myocardium (50%). No hypokinetic segment was transmurally infarcted, however. Thus, in this patient population, transmural infarction was always associated with akinesis or dyskinesis, which in turn was moderately, though not completely, predictive of transmural injury. These findings are consistent with those of Hutchins et al. in their study of the relationship of left ventriculography to postmortem findings. In the present study, nine segments that moved normally by two-dimensional echocardiography were nonetheless subendocardially infarcted on pathologic examination. All nine of these segments were from a 19-year-old man with a circumferential subendocardial infarction after remote valve surgery. Two-dimensional echocardiography may therefore not recognize subendocardial injury if it involves the entire left ventricular circumference.

Using segmental wall motion analysis, we examined the relationship of echocardiographic wall motion abnormalities to infarct thickness and found that this relationship was significant (table 2). These results support investigations by Theroux et al., who found a high correlation between percentage of scar and the reduction in percent shortening using sonomicrometers to measure regional ventricular function in dogs. The human studies of Ideker et al. are also consistent with our findings. In their angiographic investigation, mean fibrosis increased with increasing severity of quantitatively determined angiographic asynergy.

In the present study two-dimensional echocardiography appeared to overestimate the amount of scar, for both the extent of wall motion abnormality, measured on a segment-by-segment basis, and for the circumferential analysis (fig. 5). There are at least two possible explanations for this overestimation. First, the majority of hypokinetic and akinetic/dyskinetic segments that were pathologically noninfarcted were adjacent to scar. Experimentally, myocardium immediately adjacent to injured myocardium, though itself not injured, demonstrates hypokinesis or dyskinesis. Kerber et al. found that despite preservation of normal myocardial perfusion in nonischemic areas, wall motion abnormalities are nevertheless evident. These changes may be the result of transient undetected ischemia in the segments struck by the ultrasound beam, or possibly a passive change in the motion of normally perfused areas by the severe motion abnormalities of the adjacent ischemic myocardium. These, and similar observations by Wyatt et al., are consistent with the present study. Second, of the 37 abnormally moving segments pathologically without infarct, 70% (including six of seven akinetic or dyskinetic segments) were in the distribution of an occluded coronary artery and presumably were within the anatomic risk region. These segments may have been reversibly ischemic. The possibility of reversible ischemia (as opposed to infarction) accounting for such wall motion abnormalities is supported by several studies showing that resting asynergy may be improved by interventions such as nitroglycerin, epi-nephrine or postextrasystolic potentiation.

In this study we could not determine absolute infarct size, as technical difficulties would have precluded evaluation of all segments in all patients. Indeed, circumferential analysis was done on at most three cross sections per patient. However, an estimation of the circumferential extent of infarction in a given cross-section was possible. Nevertheless, the accuracy of estimating circumferential extent of infarction is limited by the large standard error of the estimate (fig. 5). Because two-dimensional echocardiography can, under most circumstances, visualize a large portion of the left ventricle at any point, it seems well suited to the study of the relation of wall motion to regional pathologic changes. That it is possible to correlate, to the approximations reported herein, segmental or circumferential regional wall motion abnormalities with the degree of scar provides encouragement for further attempts at infarct size evaluation with this noninvasive technique. Improvements in the quality of ultrasonic instrumentation should eliminate some of the current imaging problems. Further methodologic refinements should also be expected by application of computer-aided techniques to the evaluation of wall motion abnormalities to allow quantification of regional wall motion and thickening. The latter, determined more readily by echocardiography than by angiography or scintigraphy, may be more sensitive than endocardial motion in the detection of myocardial injury.

We conclude the following about the accuracy of real-time two-dimensional echocardiographic determination of myocardial lesions: (1) Circumferential extent of akinesis/dyskinesis and circumferential extent of scar correlate closely; (2) normal segmental wall motion excludes transmural infarction, but is occasionally associated with subendocardial injury; (3) hypokinesis is nonspecific, and is seen in either subendocardial infarction or histologically normal myo-
cardium; (4) regional akinesis or dyskinesis usually signifies, but is not entirely predictive of, transmural infarction; and (5) circumferential and regional scar are overestimated, because most pathologically normal segments seen by echocardiography as akinetic or dyskinetic are either adjacent to scar or in the distribution of a critically stenosed coronary artery. Such segments may, therefore, be reversibly ischemic.

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