Cross-sectional Echocardiography in Acute Myocardial Infarction: Detection and Localization of Regional Left Ventricular Asynergy

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SUMMARY Left ventricular asynergy associated with acute myocardial infarction was evaluated by cross-sectional echocardiography. Patients with acute infarction were studied within 48 hours of admission, and a segmental analysis of left ventricular wall motion was performed using nine segments obtained by short- and long-axis recordings of the left ventricle. By this segmental approach, analysis of wall motion in the entire left ventricle was possible. Complete studies were recorded in 37 of 44 original patients. Segmental wall motion abnormalities were recorded and localized in each of the 37 study patients. Asynergy was detected in 142 segments, and 29 patients had multiple segment involvement. Asynergy was most common in the apical segments of the left ventricle, but the cross-sectional scan permitted detection of asynergy in all segments. Correlation between the ECG and the cross-sectional echocardiogram revealed that 19 of 20 patients with inferior infarction had asynergy in posterior segments, 14 of 14 patients with anterior infarction had asynergy in anterior segments, and three of three patients with anteroinferior infarction had asynergy both anterior and posterior segments. In addition, the location of segmental asynergy followed specific patterns for each ECG subgroup of infarction. In four patients with postmortem examination, 21 of 22 segments that had asynergy by cross-sectional echocardiography also had pathologic evidence of infarction. Therefore, the cross-sectional echocardiogram provides a reliable method for detecting the presence and location of regional asynergy associated with acute myocardial infarction.

TENNANT AND WIGGERS originally observed that acute coronary ligation resulted in a loss of normal myocardial contraction in the region supplied by the affected vessel. The initial localized decrease in contraction was quickly followed by systolic bulging or expansion of the ischemic muscle. Subsequently, the observation that abnormal left ventricular wall motion occurs in response to myocardial ischemia and infarction has been confirmed by a variety of techniques in both the experimental animal and man. M-mode echocardiography is a valuable and sensitive noninvasive method for recording left ventricular wall motion and evaluating multiple parameters of left ventricular function. Clinical studies have further demonstrated the feasibility of recording localized areas of left ventricular asynergy using the M-mode technique in patients with both acute and chronic myocardial ischemia. In experimental animals, a correspondence between infarct location and regional asynergy as well as a progressive decrease in the amplitude of wall motion in response to sequential decline in regional perfusion have been documented.

The more recent development of cross-sectional echocardiography adds spatial orientation to the echocardiographic display and permits not only the presence, but also the location and extent of regional asynergy to be determined. Initial studies in patients with chronic ischemic heart disease have demonstrated good correlation between the cross-sectional echocardiographic and cineangiographic detection of both left ventricular aneurysms and areas of regional asynergy. Preliminary observations have also suggested that areas of left ventricular asynergy can be observed in patients with acute myocardial infarction. Subsequent experimental studies have established a relationship between both the location and the extent of the region of asynergy and the underlying infarct area.

The traditional clinical method for determining acute myocardial infarction has been the 12-lead ECG. In a series of classic clinicopathologic studies, the presence and specific pattern of electrocardiographic Q-wave distribution has been correlated with the anatomic location of infarcted myocardium. These studies are the basis for localizing the site and extent of myocardial infarction from ECGs. The relationship of electrocardiographic Q waves to the location and extent of left ventricular asynergy in patients with acute myocardial infarction, however, remains to be defined. We undertook the present study in order to: 1) determine the capacity of cross-sectional echocardiography to demonstrate asynergy in patients with acute myocardial infarction, 2) compare the location and extent of asynergy with the corresponding Q-wave pattern on the standard 12-lead electrocardiogram, and 3) compare the location of asynergy with the presence or absence of infarcted myocardium when pathologic data were available.
Materials and Methods

Patients

Cross-sectional echocardiographic examinations of the left ventricle were performed in 44 consecutive patients admitted to the coronary care unit at the Indiana University Hospital with a diagnosis of acute, transmural myocardial infarction. Acute infarction was documented in each case by a typical history of chest pain, serial ECG changes, and serial elevation in serum enzymes. The electrocardiographic criteria for the diagnosis and localization of infarction were the appearance of new Q waves ≥0.04 second or a tall R wave in V<sub>1</sub> with R/S >1 when associated with changes in other leads. The 44 patients consisted of 31 males and 13 females (age range 33–86 years). This is a referred population and is not a typical sample of patients with acute myocardial infarction, as evidenced by a high incidence of cardiogenic shock, heart failure, mitral regurgitation, arrhythmia, and the presence of four cases of acute ventricular septal defect.

ECG and Postmortem Studies

Standard 12-lead ECGs were performed at the time of admission and at least once daily for the first 3 days of hospitalization. The electrocardiographic site of infarction was categorized into the following subgroups: 1) isolated inferior infarction — Q waves in leads II, III, and aV<sub>3</sub>; 2) inferoposterior infarction — Q waves in leads II, III, aV<sub>3</sub>, and tall, wide R waves in V<sub>1</sub>; 3) inferolateral infarction — Q waves in leads II, III, aV<sub>3</sub>, and V<sub>5</sub>–V<sub>6</sub>, and/or aV<sub>1</sub>; 4) anteroseptal infarction — Q waves in leads V<sub>1</sub>–V<sub>3</sub>; 5) anterolateral infarction — Q waves in leads V<sub>4</sub>–V<sub>5</sub>, 6) anteroinferior infarction — Q waves in leads II, III, and aV<sub>3</sub> and V<sub>6</sub>. There were no cases of isolated apical infarction, but the ECG was considered to indicate apical involvement when leads V<sub>4</sub>–V<sub>6</sub> exhibited abnormal Q waves.

Postmortem examinations were available in four of the patients. From the pathologic description the site of myocardial infarction was localized to the anterior, posterior, lateral, septal or apical regions of the left ventricle.

Cross-sectional Echocardiography

Cross-sectional echocardiographic studies were performed with either a mechanical sector scanner, developed in conjunction with the Fortune-Fry Research Laboratories at the Indiana University School of Medicine, or a commercially available sector scanner, EkoSector I (Smith Kline Instruments). These systems consisted of a modified Ekoline 20A echograph with a pulse repetition rate of approximately 4 MHz. The scanner probe contained a 2.25 MHz transducer that was mechanically driven through a 30° sector at a rate of 30 Hz/sec. This operating mode permitted data recording at 30 frames (60 fields)/sec, which yielded a line density of approximately 60 lines/field.

Cross-sectional studies were recorded on half-inch videotape using a Sanyo VTC-7100 cassette recorder. These images were then available for redisplay and evaluation in real-time, slow-motion or single-frame format. The individual frames were converted to hard copy using a standard Polaroid photographic system.

Cross-sectional imaging was also performed using an 82° sector arc; however, for purposes of wall motion analysis, the 30° arc was superior because the higher line density per field permitted more precise definition of endocardial motion. The images were displayed on an oscilloscope screen that provided crisp, well-defined images for each field, without the field-to-field persistence of television display.

All cross-sectional studies were performed with the patient in either the supine or 30° left lateral position. Cross-sectional recordings were initially made with the plane of the cross-sectional scan aligned parallel to the long axis of the left ventricle at the base of the heart. With the transducer in this position it was possible to record the basal segment of the interventricular septum and a corresponding but greater portion of the posterior left ventricular wall. This scan area included both the anterior and posterior mitral leaflets. The transducer was then tipped toward the cardiac apex and a more extensive area of the posterior left ventricular wall, including the papillary muscles, as well as a slightly more distal portion of the anterior septal segment could be recorded. The transducer was then moved down one interspace to record a more extensive area of the anterior and posterior wall in the midportion of the ventricle, again including the papillary muscles. Finally, the transducer was placed directly over the cardiac apex and a long-axis scan of the apex itself recorded.

In order to evaluate the short-axis configuration of the ventricle, the transducer was returned to the original basal position and rotated 90° to record a short-axis scan at the level of the mitral valve. From this position, the anterior and posterior as well as the medial and lateral segments of the left ventricle at the base of the heart were assessed. The transducer was again moved sequentially down the chest to record the left ventricle at the papillary muscle region and at the region of the cardiac apex. In each case, attention was paid to insure that the anterior, posterior, medial and lateral segments of the ventricle were recorded in a short-axis view at both the basal and papillary muscle levels, and that the cardiac apex was adequately recorded in a long-axis view. Studies were only considered adequate for analysis when each of these sections were included in the scans.

Segmental Analysis For Asynergy (figs 1 and 2)

For purposes of analysis the ventricle was divided into nine segments that corresponded to the method in which the scans were obtained. The ventricle was initially divided into three regions: a basal region, which extended from the insertion of the interventricular septum into the aortic root anteriorly and the atrioventricular ring posteriorly, to a level just above but not including the papillary muscles; a midventricular region, which included the area of the
All cross-sectional studies were examined independently by two observers without knowledge of the electrocardiographic findings. The results of these two observations were then compared. If there were discrepancies, the studies were reviewed and a consensus was obtained.

For purposes of data display the heart was considered as a series of three concentric rings, with four basal segments in the outer ring, the midportion of the ventricle in the middle ring, and the cardiac apex in the center (fig. 2). Thus, the ventricle is viewed as if from the apex toward the base, with the medial portion of the ventricle to the left and the lateral portion to the right. Since still-frame images are used for illustrations in this text, some important limitations of this method should be emphasized. The still-frame image results in loss of image integrity and clarity. In a real-time format, data are visually integrated and wall motion characteristics are readily appreciated. Furthermore, the still-frame represents only one field of data, or half of one frame, so there may be loss of target definition as well.

### Results

**Detection of Asynergy by Cross-sectional Echocardiography (table 1)**

Complete cross-sectional echocardiographic studies were obtained in 37 of the original 44 patients. Seven studies were rejected for analysis because of inadequate definition of wall motion in all nine segments.

Left ventricular asynergy, involving one or more segments, was detected in all 37 study patients with acute myocardial infarction. Asynergy was limited to one segment in eight patients, while 29 patients had 2–8 asynergic segments. For the entire group, asynergy was recorded in 142 of 333 segments available for analysis.

Table 1 lists the frequency with which asynergy was present in each of nine segments. There was relatively diffuse cardiac involvement, as asynergy was found in all segments. The most frequently involved segments were the anterior (#5), posterior (#7), medial (#8), and lateral (#6) segments at the midventricular level, and the apical segment (#9). The diffuse cardiac involvement reflects the heterogenous nature of infarct patterns in this study population and suggests that cross-sectional echocardiography can detect asynergy in all segments of the ventricle.

As an example of segmental asynergy, figures 3 and 4 are still-frame recordings of the left ventricle in long and short axes from a patient with an acute anterolateral myocardial infarction. Asynergy of the anterior wall is present and includes both basal and midventricular segments (segments 1 and 5).

**Correlation of Asynergy with ECG Infarct Location**

The comparison between infarct location predicted from the standard ECG and the corresponding asynergy recorded by cross-sectional echocardiography is shown in figures 5 and 6. The absolute
number of patients in whom a particular segment was asynergic is indicated numerically. The percentage of patients in each infarct subgroup with asynergy of a particular segment is then indicated by the shading of each box.

**Inferior Infarction (fig. 5)**

There were 20 patients in the category of inferior infarction that included ECG subgroups of isolated inferior, inferoposterior and inferolateral infarctions. Asynergy of the posterior segments (#3 and #7) was present in 19 of 20 patients. One patient with an inferolateral infarction did not have asynergy of the posterior segments. Thus, good correlation exists between ECG evidence of inferior infarction and asynergy in posterior segments of the cross-sectional echocardiogram. In addition, there were specific patterns of asynergy for each ECG subgroup of inferior infarction.

The ECG pattern of isolated inferior infarction was seen in 14 patients, four of whom had an acute ventricular septal defect. In the 10 patients with uncomplicated, isolated inferior infarction, asynergy was generally limited to the two posterior segments of the left ventricle (#3 and #7). One patient had ECG documentation of an old anterior infarction in addition to an acute isolated inferior infarction. This patient had asynergy in a relatively diffuse area of the ventricle, providing the only instance in these 10 cases where anterior, lateral or apical segments had asynergy. In the four cases with isolated inferior infarction complicated by an acute ventricular septal defect, asynergy was present in the posterior segments in all four, but each also had asynergy of the medial segments (#4 and #8). In each of these four cases the medial segment was dyskinetic, the normal septal curvature was markedly distorted, and the septum protruded well into the right ventricle.

The ECG subgroup of inferoposterior infarction was present in two patients. Both had asynergy in posterior and lateral (#2 and #6) segments.

There were four patients in the ECG subgroup of inferolateral infarction. In three of four cases, posterior and lateral segments at the basal level (#2 and #3), as well as the posterior segment at the midventricular level (#7), were asynergic. In addition, there was variable involvement of the apical two-thirds of the ventricle, as the apical segment (#9) and anterior, lateral and medial segments at the midventricular level (#5, #6 and #8) had asynergy in 50% of cases. Thus, the ECG subgroup of inferolateral infarction tended to have a more diffuse pattern of asynergy than the other subgroups of inferior wall infarction.

![Figure 3](http://circ.ahajournals.org/download/579_766_534.jpg)
Anterior Infarction (fig. 6)

There were 14 patients in the category of anterior wall infarction that included ECG subgroups of anteroseptal and anterolateral infarcts. Asynergy was present in the anterior wall segments (#1 and #5) in each of these 14 patients. Different patterns of asynergy for each ECG subgroup were also evident.

In five patients in the anteroseptal ECG subgroup, asynergy was most frequent in the anterior and medial segments and in the cardiac apex. The anterior segment at the midventricular level (#5) was abnormal in all five and in four of five the apical segment (#9) and the medial segment at the midventricular level (#8) were also abnormal. The anterobasal and medial-basal segments (#1 and #4) were frequently abnormal, but less so than the more distal segments of the ventricle.

A different pattern of asynergy was present in nine patients in the ECG subgroup of anterolateral infarction. All nine had asynergy in the apical segment (#9) and in the anterior segment at the midventricular level (#5). In seven of nine, the lateral segment at the midventricular level (#6) was asynergic. In addition,
the remainder of the ventricle was more heterogeneously involved than in the subgroup of anteroseptal infarction.

One patient with an acute anterolateral infarction had ECG evidence of a prior inferior infarction; however, the pattern of asynnergy did not differ from that seen in the other patients in the ECG subgroup.

Anteroinferior Infarction (fig. 6)

There were three patients with an ECG pattern of acute anterior and inferior myocardial infarction, and each had asynnergy in both anterior and posterior segments. Patients in this ECG subgroup had extensive areas of asynnergy, as the apical segment (#9), the entire midventricular portion of the ventricle (#5, #6, #7 and #8) and the lateral (#2) and posterior (#3) basal segments were abnormal in each case.

Postmortem Correlation (table 2)

Postmortem examinations were obtained in four patients. Table 2 is a comparison of the pathologic location of infarction with the corresponding segments that were asynergic by cross-sectional echocardiogram. In these four patients, 22 segments were identified as asynergic. In 21 of 22 segments, pathologic inspection identified corresponding areas of myocardial infarction. The only exception was an apical segment with asynnergy, but without evidence of infarction. Thus, in 21 of 22 segments, asynnergy by cross-sectional echocardiography correlated with pathologic myocardial infarction. In one instance a medial segment had normal wall motion, while at postmortem examination there was evidence of an old infarction in this region. No other segment identified as normal by cross-sectional echocardiography was found to have pathologic evidence of infarction.

Discussion

Acute myocardial infarction is a leading cause of cardiovascular morbidity and mortality. Pathologic and clinical evidence indicates that the quantity and location of infarcted or ischemic myocardium directly contribute to the morbidity and mortality associated with acute myocardial infarction.24-27 Since asynnergy of left ventricular wall motion serves as a marker for coronary artery disease, a method of detecting asynnergy in acute myocardial infarction may provide a powerful tool for studying this condition. This study was undertaken to test the capability of cross-sectional echocardiography to detect and localize left ventricular asynnergy associated with acute myocardial infarction.

Detection of Left Ventricular Asynnergy

The initial aim of this study was to determine the capacity of cross-sectional echocardiography to detect regional asynnergy. We found segmental wall motion abnormality in each patient with clinically documented acute myocardial infarction. This finding is consistent with prior echocardiographic techniques for evaluating wall motion after acute myocardial infarction.3, 28, 29 We used a semiquantitative method of analysis that permitted both the number and location of asynnergic segments to be determined. Thus, as few as one asynnergic segment was seen in a number of patients, while others had as many as eight asynergic segments. Most patients (29 of 37) had more than one segment with asynnergy, a finding consistent with the nature of the study population: most of the patients were referred for treatment and many had severe left ventricular dysfunction.

For the total study population, asynnergy was more common in segments at the midventricular level and cardiac apex. However, in cases with inferior infarction, asynnergy was often present in segments of the basal region, particularly the posterior segments, as opposed to cases with anterior infarction, which more often had asynnergy in apical regions. The finding of asynnergy at different levels of the ventricle for anterior and inferior infarction is expected from the pattern of coronary distribution, and agrees with clinicopathologic findings comparing ECG patterns with the location of infarction.23, 30

Correlation of Asynnergic Segments with the ECG Pattern

The 12-lead ECG is a traditional method for locating acute myocardial infarction. To assess the reliability of the cross-sectional echocardiographic location of asynnergy, we compared the ECG pattern of Q waves with the segmental location of asynnergy. We found that asynnergic segments in the anterior or posterior region of the ventricle corresponded to ECG evidence of anterior or inferior infarction. Within this

Table 2. Comparison of Pathologic Location of Infarction with Abnormal Wall Motion

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pathologic location of infarction</th>
<th>Abnormal segments by cross-sectional echocardiography</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>1</td>
<td>Posterior, lateral, apex</td>
<td>X</td>
</tr>
<tr>
<td>2</td>
<td>Septum, lateral, posterior</td>
<td>X</td>
</tr>
<tr>
<td>3</td>
<td>Posterior, lateral old infaret; anterior, septum, apex</td>
<td>X</td>
</tr>
<tr>
<td>4</td>
<td>Anterior, septum</td>
<td>X</td>
</tr>
</tbody>
</table>

Identification of segments as in figures 1 and 2. Abbreviations: ANT = anterior; LAT = lateral; POST = posterior; MED = medial.
general classification, the cross-sectional echocardiogram also identified specific patterns of asynergy in each of the ECG subgroups.

Asynergy in the ECG subgroup of isolated inferior infarction was limited to one or two posterior segments; in inferolateral infarction, asynergy was more extensive, including posterior, lateral and apical segments. These findings are consistent with clinical experience that inferolateral infarctions have a more serious prognosis than isolated inferior infarctions. Also, these findings agree with pathologic evaluations of Horan et al. and Savage et al. who found that pathologic evidence of inferior infarction was limited to the posterolateral regions, while inferolateral infarction usually involved a much larger area of the ventricle, including the cardiac apex.

Nearly all of the patients in the two subgroups of anterior wall myocardial infarction had segmental asynergy in the anterior and apical region, which agrees with clinicopathologic findings of Meyers et al. The subgroup of anterolateral infarction, however, had more extensive areas of asynergy than anteroseptal infarction. These findings also agree with the electrocardiographic and pathologic correlative studies of Savage et al. and Meyers et al. who found that the extent of pathologic involvement was greater in anterolateral than in anteroseptal infarction.

Cross-sectional echocardiography, then, was able to detect different patterns of segmental asynergy for each ECG subgroup. These findings are supported by pathologic studies and suggest that cross-sectional echocardiography provides an accurate method for evaluating regional asynergy.

Discrepancies Between ECG and Cross-sectional Echocardiography

In two patients with ECG evidence of inferolateral infarction, the cross-sectional echocardiogram disclosed asynergy in an unexpected segment of the distal anterior wall (segment 5). In both cases, the asynergic anterior segment was directly adjacent to the abnormal apical segment. Although this finding may represent an error in echographic interpretation, several other possibilities exist. Histopathologic and angiographic studies have shown that segments adjacent to a region of infarction may exhibit asynergy and yet be morphologically normal, while other segments distant from the region of infarction may exhibit asynergy because of ischemia produced by additional coronary obstructions. Although we do not feel the echocardiographic interpretation was in error, it is impossible to determine the reason for finding asynergy in regions that may not have been involved in the infarction process. Further elucidation of this point by studies of coronary anatomy and myocardial perfusion may explain this apparent discrepancy.

Another discrepancy between ECG and cross-sectional echocardiographic correlation occurred in four patients with an acute ventricular septal defect accompanying isolated inferior infarction, who were further distinguished from the other patients with isolated inferior infarction by having asynergy of the medial segments. Thus, infarction and perforation of the interventricular septum were accompanied by asynergy of the medial segment of the cross-sectional echocardiogram, although the ECG showed no evidence of septal infarction. These findings agree with pathologic reports by Meyers et al. and Savage et al. that inferior infarction may extend into the interventricular septum without producing characteristic ECG changes. The medial segment motion in each of our four cases was dyskinetic or paradoxical, and this particular pattern of asynergy may be a marker for an acutely perforated septum in the setting of acute inferior infarction.

Differentiation of Old and New Infarction

Two patients in this study had documented myocardial infarctions before the onset of acute infarction. Cross-sectional echocardiograms performed at the time of the second myocardial infarction disclosed asynergy in multiple segments. However, analysis of wall motion by cross-sectional echocardiography could not differentiate acutely from chronically infarcted regions. Methods using M-mode echocardiography to detect the effects of acute infarction on wall motion as well as scar due to old myocardial infarction have been described; however, these techniques were not applied to the present study. The future application of these techniques to the cross-sectional echocardiographic examination may provide a method for differentiating acute from chronic infarction.

Limitations

In seven of 44 patients with acute myocardial infarction, complete cross-sectional echocardiograms were not obtained because of difficulties in visualizing all nine segments required for analysis. However, partial studies were obtained in every case and regions of asynergy could be identified even when the entire ventricle was not adequately recorded. The base of the heart was most difficult to visualize, because patients with coronary artery disease often have a large chest diameter, which places the basal regions at a great distance from the transducer. Apical or epigastric views may improve recording, but were not routinely used for this study. Adequate studies for interpretation of wall motion in all nine segments of the ventricle were obtained in 37 of 44 patients, a success rate of 84%, suggesting that this technique is a practical method for evaluating patients with acute myocardial infarction.

In summary, using cross-sectional echocardiography, we could evaluate regional asynergy associated with acute myocardial infarction. The location of segmental asynergy corresponded to the ECG location of Q waves and to the pathologic location of infarction. The cross-sectional echocardiogram also detected segmental asynergy in regions where the ECG showed no evidence of infarction. Careful analysis of segmental wall motion by cross-sectional
echocardiography provides a method for assessing the location of segmental asynergy and for evaluating the function of the left ventricle in acute myocardial infarction.

References

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