Cross-sectional Echocardiographic Analysis of the Extent of Left Ventricular Asynergy in Acute Myocardial Infarction

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SUMMARY Cross-sectional echocardiography was used to study left ventricular wall motion in 44 patients with myocardial infarction, and the extent of observed asynergy was correlated with left ventricular function. Echocardiographic studies were performed in short and long axes of the ventricle and nine segments were identified for analysis. Wall motion in each segment was classified as normal, hyperkinetic, hypokinetic, akinetic or dyskinetic. Based on this analysis a wall motion index was derived as an overall assessment of left ventricular asynergy. Left ventricular function was measured by clinical and hemodynamic parameters to note the presence of pulmonary congestion or peripheral hypoperfusion or both.

Segmental asynergy was detected in all patients with acute myocardial infarction. Patients with uncomplicated infarction had a wall motion index of 3.2 ± 2.4, which was significantly less than that in patients with pulmonary congestion (9.7 ± 3.1, p < 0.001) or with both pulmonary congestion and hypoperfusion (10.6 ± 4.8, p < 0.001).

In nine patients with acute ventricular septal defect or acute mitral regurgitation, wall motion index was 6.7 ± 1.9, significantly less than with other complicated infarcts (p < 0.001) but greater than with uncomplicated infarcts (p < 0.005). Wall motion index also discriminated complicated from uncomplicated infarction when death was used as the end point.

Cross-sectional echocardiography provides a method of measuring the extent of left ventricular asynergy during acute myocardial infarction that correlates well with hemodynamic parameters of left ventricular function.

LEFT VENTRICULAR pump failure manifest by congestive heart failure or cardiogenic shock remains the principal cause of in-hospital mortality after acute myocardial infarction. Clinical and pathologic studies have shown that the degree of left ventricular power failure is determined by the amount of myocardium involved in the infarction process. According to a great deal of recent investigation is directed toward quantitating infarct area. It is hoped that these techniques may then be used to evaluate methods of limiting infarct size and preserving jeopardized myocardium. Acute alterations in the pattern of myocardial contraction are an early and sensitive indicator of myocardial ischemia. Thus, a method to monitor the location and extent of abnormal myocardial contraction might be used to determine infarct area and on this basis to predict left ventricular function during acute infarction.

Echocardiography is a valuable, noninvasive method for evaluating left ventricular wall motion. M-mode echocardiographic studies have shown the ability of this technique to detect abnormal wall motion in experimental models of ischemia as well as in acute and chronic myocardial infarction in man. Clinical studies have further shown that M-mode echocardiography can record and identify abnormal wall motion in multiple regions of the left ventricle and on this basis predict prognosis in patients with acute infarction. Cross-sectional echocardiography expands the capability of the M-mode technique by providing a wider field of vision and adding spatial orientation to the ultrasonic examination. An earlier report from this laboratory described the ability of cross-sectional echocardiography to detect regional asynergy in patients with acute myocardial infarction, with good correlation between location of abnormal wall motion and both electrocardiographic and pathologic estimates of infarct location. Additional experimental studies have supported the role of cross-sectional echocardiography as a means of detecting and quantitating regional asynergy and have substantiated the relationship of the extent of the area of asynergy to underlying infarct area. In this study, we extend these observations by examining the relationship between the extent and degree of abnormal left ventricular wall motion determined by cross-sectional echocardiography and various clinical and hemodynamic parameters of left ventricular function in patients with acute myocardial infarction.

Materials and Methods

Patient Population

The study population consisted of 44 consecutive patients admitted to the coronary care unit at the Indiana University Hospital with a diagnosis of acute transmural myocardial infarction. There were 31 males and 13 females, ages 33–86 years. The diagnosis
of acute infarction was documented in each case by a
typical history of chest pain, serial ECG changes and
serial elevation in serum enzymes. The electrocar-
diographic criteria for diagnosis of infarction were
the appearance of new Q waves of greater than 0.04 sec-
donor a tall R wave in V1 with R/S > 1 when accompa-
nied by changes in other leads. Two patients had
ECG documentation of a prior myocardial infarction
in addition to acute infarction. These patients are not
a typical sample of acute myocardial infarction but
were drawn from a referral hospital source, and in-
cluded many patients with complicated infarctions.

Cross-sectional Echocardiography

Cross-sectional echocardiographic studies were
performed in each patient with either a mechanical
sector scanner, developed in conjunction with the
Fortune-Fry Research Laboratories at Indiana
University School of Medicine, or a commercially
available sector scanner (Eko-Sector I, Smith Kline
Instruments). These systems consist of a modified
Ekoline 20A echograph with pulse-repetition rate of
approximately 4 kHz/sec. The scanner probe con-
tained a 2.25-MHz transducer that was mechanically
driven through a 30° or 82° sector at a rate of 30
cycles/sec. This operating mode yielded an image rate
of 30 frames (60 fields)/sec, with a resultant line den-
sity of approximately 60 lines per field.

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

Cross-sectional images of the left ventricular en-
docardial surface were obtained by recording short-
and long-axis scans through basal, midventricular and
apical regions of the left ventricle. The basal region by
definition extended from the insertion of the interven-
tricular septum into the aortic root anteriorly and the
atrioventricular ring posteriorly to a level just above
the papillary muscles. This scan area included both
anterior and posterior mitral leaflets and associated
chordal structures. The midventricular region in-
cluded the area of the papillary muscles and the apical
region extended from the most distal extent of the
papillary muscles to the tip of the cardiac apex. To
record the base of the heart, the transducer was placed
on the anterior chest wall directly above the anterior
mitral leaflet. The scan plane was initially aligned
parallel to the ventricular long axis with the midpor-
tion of the sector at the free edge of the mitral leaflet.
Appropriate rotation of the plane was defined by
determining the point at which maximal ventricular
diameter was recorded at both margins of the sector.
Orientation in the midportion of the ventricle was then
assured by angling the scan plane from the medial to
lateral ventricular walls and recording the position at
which the maximum diameter was observed. Having
recorded the long axis at this level the scan plane was
rotated 90° to record a short axis. Again, appropriate
short-axis orientation was assured by recording the
image with the least horizontal or vertical obliquity.
To record the midventricular region the transducer
was either angled toward the cardiac apex or placed
lower on the anterior chest wall. The method of plane
orientation was similar to that used at the base. When
recording the cardiac apex, the transducer was placed
directly over the apical impulse. Careful attention was
paid to ensure that the entire circumference of the ven-
tricle was recorded at the basal and papillary muscle
levels, as well as the cardiac apex in long axis.

For purposes of analysis, the ventricle was divided
into nine segments identified by their location on the
cross-sectional echocardiographic image (fig. 1). Both
the basal and midventricular regions were divided into
four segments — anterior, medial, posterior and
lateral. The apical region was taken as the ninth seg-
ment. Studies were considered adequate for analysis
only when each of these nine segments was included in
the scans. Examples of cross-sectional echocardiograms
from patients with acute myocardial infarction are given in figures 2 and 3.

Each segment was evaluated for the presence and
degree of asynergy. Wall motion of each segment was
characterized as normal, hypokinetic, akinetic,
dyskinetic or hyperkinetic based upon a visual assess-
ment. A segment was characterized by the type of wall
motion present in 50% or more of the endocardial sur-
face of that segment. All cross-sectional studies were
examined independently by two observers without
knowledge of electrocardiographic or clinical data. The results of these two observations were then com-

![Diagram](https://via.placeholder.com/150)

**Figure 1.** Diagram showing the method used to identify nine myocardial segments from composite analysis of long-
and short-axis echocardiographic studies. MV = mitral valve.
pared and where discrepancies arose, these studies were reviewed and agreement was obtained.

To grade the severity of segmental asynergy, each segment was assigned a numerical score based upon the type of wall motion noted. These scores were assigned as follows: hyperkinesis, -1; normal, 0; hypokinesis, 1; akinesis, 2; and dyskinesis, 3. The wall motion index — an overall evaluation of left ventricular asynergy — was then obtained by summing the scores for each of the nine segments. Thus, a normal left ventricle would have a wall motion index of 0, while the theoretically maximal wall motion index would be 27 if all segments were dyskinetic.

Assessment of Left Ventricular Function

Clinical and hemodynamic parameters of left ventricular function were obtained by bedside evaluation, chest x-ray and pulmonary artery catheterization. On the basis of criteria proposed by Forrester and Swan, each patient was classified into a clinical-hemodynamic subset. Subset I included patients with no evidence of pulmonary congestion or peripheral hypoperfusion. Subset II indicated the presence of isolated pulmonary congestion. Subset III included patients with isolated peripheral hypoperfusion and subset IV included both pulmonary congestion and peripheral hypoperfusion. For purposes of discussion, patients without evidence of pulmonary congestion or peripheral hypoperfusion were considered to have uncomplicated myocardial infarction, while patients with either or both of these findings were considered to have complicated myocardial infarction. Hemodynamic intervention was performed when indicated by the overall status of each patient. Classification was determined by hemodynamic data in 15 patients and clinical data in 22 patients.

Four patients had infarctions complicated by an acute ventricular septal defect. This diagnosis was based on clinical signs of precordial thrill and pansystolic murmur and confirmed by recording an increase in oxygen saturation from right atrium to pulmonary artery, indicating a left-to-right shunt at the ventricular level. The diagnosis was also confirmed in each case by cardiac catheterization and cineangiography.

Five patients had evidence of significant, acute mitral regurgitation manifest by a new apical systolic murmur accompanied by heart failure with bedside catheterization showing large, early "V" waves in the pulmonary wedge tracing. Three of these five patients underwent cardiac catheterization with cineangiography, which confirmed the diagnosis.

Fixed analysis of the data was performed using the t test and a standard table of probability. Null hypotheses were rejected if their statistical probability was less than 5%.

Results

Cross-sectional echocardiographic examination disclosed evidence of regional asynergy in all 44 patients.
originally studied. Complete recordings of the entire left ventricular endocardial surface could not be obtained in seven patients, who were excluded from further analysis. Wall motion was adequately recorded in all nine segments of the ventricle in 37 patients, who form the subsequent study population.

We analyzed wall motion in 333 segments from 37 patients (table 1). One hundred eighty-one of 333 segments had normal wall motion, while 10 segments were hyperkinetic. One hundred forty-two segments had various degrees of diminished motion (i.e., asynergy). Of these 142 asynergic segments, hypokinesis was present in 40 (12%), akinesis in 48 (14%), and dyskinesis in 54 (16%).

To determine if cross-sectional echocardiographic evaluation of segmental asynergy correlated with left ventricular function, the wall motion index for each patient was compared with the clinical-hemodynamic classification and the presence of uncomplicated or complicated myocardial infarction (fig. 4). In 13 patients with uncomplicated myocardial infaracts, defined as absence of pulmonary congestion and peripheral hypoperfusion, the mean wall motion index was 3.2 ± 2.4. Complicated myocardial infarction was associated with wall motion index significantly greater than that for uncomplicated infarction. The 12 patients with isolated pulmonary congestion had a mean wall motion index of 9.7 ± 3.1 (p < 0.001), while the 10 patients with both pulmonary congestion and peripheral hypoperfusion had a mean wall motion index of 10.6 ± 4.8 (p < 0.001). In two patients with isolated peripheral hypoperfusion, the wall motion index was 8 and 17 (mean 12.5), which was also significantly greater than for uncomplicated infarction (p < 0.001).

Nine patients had an acute ventricular septal defect or acute mitral regurgitation complicating their infarction. This was manifest in each case by heart failure or shock (fig. 4). These nine patients had a mean wall motion index of 6.7 ± 1.9, which was significantly less than the wall motion index of other patients with complicated infarction (p < 0.001), but significantly greater than that found in uncomplicated infarction (p < 0.005). Patients with these acute mechanical defects resulting in physiologic afterload reduction of the ventricle, therefore, had significantly less asynergy by echocardiography than other patients with apparently comparable degrees of left ventricular dysfunction.

Comparing patients with complicated infarcts but without physiologic afterload reduction of the ventricle (fig. 5), 10 patients with isolated pulmonary con-

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**Table 1. Segmental Wall Motion Classification in 333 Segments**

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<tr>
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<th>Normal</th>
<th>Hyperkinesis</th>
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**Figure 4.** Comparison of echocardiographically derived wall motion index with clinical and hemodynamic parameters of left ventricular function in 37 study patients. Uncomplicated infarction (subset I) was clearly differentiated from complicated infarction (subsets II, III and IV) by echocardiographically derived wall motion index. VSD = acute ventricular septal defect; MR = acute mitral regurgitation.

**Figure 5.** Comparisons as in figure 4, after exclusion of patients with acute ventricular septal defect or mitral regurgitation. Patients with different degrees of left ventricular dysfunction were identified by cross-sectional echocardiographic wall motion index.
gestion had a mean wall motion index of 10.7 ± 4.7, significantly less than that in patients with both pulmonary congestion and peripheral hyperperfusion (15.8 ± 5.2, \( p < 0.025 \)). The wall motion index in both groups remains significantly greater than that in patients with uncomplicated infarction. Thus, a progressive increase in echocardiographic measure of asynergy was associated with increasing severity of left ventricular dysfunction.

Nine patients died during their acute hospital course (fig. 4). Six patients died after emergency cardiac surgery for ventricular septal defect repair or mitral valve replacement, and three died of progressive heart failure and cardiogenic shock. The wall motion index of patients who died, 9.2 ± 4.8, was similar to that in survivors with complicated infarcts, but significantly greater than that in patients with uncomplicated infarction (\( p < 0.001 \)).

**Discussion**

Cross-sectional echocardiography was used in this study to locate and quantitate abnormally contracting myocardium associated with acute myocardial infarction. Using this technique we successfully detected segmental asynergy in all patients with acute infarction. Besides detecting localized asynergy, we were able to record the entire left ventricular endocardial surface in 37 of 44 patients (84%) and analyze the location and extent of segmental dysfunction. Cross-sectional echocardiography improved detection and localization of segmental asynergy because it provides a wider field of vision with spatial orientation of the ultrasonic image and permits direct imaging of the entire left ventricle, including the cardiac apex, a region frequently involved in coronary disease.

From the cross-sectional echocardiographic recordings we identified nine segments of left ventricular endocardial surface. In each segment, wall motion was analyzed and classified as dyskinetic, akinetic, hypokinetic, normal or hyperkinetic. This ability to define qualitative characteristics of segmental wall motion has clinical importance. In experimental models, progressive changes in wall motion from normal to dyskinetic occur in response to increasing degrees of myocardial ischemia.\(^{19, 20}\) In clinicopathologic studies, histologic grade of myocardial fibrosis has been correlated with wall motion pattern in corresponding segments of the ventricle.\(^{21, 22}\) These studies suggest that different categories of segmental wall motion observed by cross-sectional echocardiography may reflect differences in degree of injury or dysfunction of the particular myocardial segment.

Based upon analysis and classification of wall motion in each segment, we calculated a wall motion index to represent a measure of overall left ventricular function. This wall motion index correlated well with clinical and hemodynamic parameters of left ventricular function. The measure of severity and extent of asynergy was directly related to the occurrence of heart failure, cardiogenic shock or death and clearly discriminated complicated from uncomplicated infarctions.

Before the advent of cross-sectional imaging techniques, application of echocardiography to acute myocardial infarction has been of limited value because of problems with the narrow field of vision provided by the M-mode echocardiographic beam. Although echocardiographic indexes of left ventricular function, such as internal diameter and mitral valve motion, have correlated with presence of congestive heart failure during acute infarction, these findings are relatively nonspecific because they provide only indirect evidence of the extent of segmental asynergy. Nieminen and Helkkila\(^ {14, 15}\) expanded the capabilities of M-mode echocardiographic techniques by using multiple transducer positions. They recorded asynergy in multiple segments of the ventricle and reported the extent of asynergy correlated with left ventricular function. Preliminary cross-sectional echocardiographic studies in acute infarction have also shown correlation between regional asynergy and clinical parameters of left ventricular function.\(^ {23, 24}\)

From examination of one cross-sectional plane of the left ventricle, changes in topography of the endocardium, with expansion and thinning of the infarct zone, have been recorded and correlated with clinical evidence of left ventricular dysfunction.\(^ {25}\) Our observation further supports these findings that the extent and severity of segmental asynergy correlate with clinical and hemodynamic parameters of left ventricular function. Echocardiographic methods that image segmental wall motion directly, locate and define asynergy and assess wall motion in infarcted and noninfarcted regions of the ventricle offer potential value in assessing patients with acute myocardial infarction.

Pathologic studies have established the concept that heart failure and cardiogenic shock after myocardial infarction are directly related to the quantity of infarcted myocardium found at postmortem examination.\(^ {1, 2}\) In the present study, we found the extent of segmental asynergy correlated well with hemodynamic function during acute myocardial infarction. Despite these clinical relationships, the extent of segmental asynergy measured by cross-sectional echocardiography may not provide a direct measure of infarct size as measured by pathologic examination. Our echocardiographic method analyzed endocardial wall motion, but indexes of left ventricular mass such as wall thickness were not measured. In experimental studies the echocardiographic measure of asynergy slightly overestimated the pathologic measure of infarct mass.\(^ {17}\) Cineangiographic studies, which also analyze endocardial wall motion, have established that segmental asynergy may not always be accompanied by histologic evidence of infarction.\(^ {25}\) This suggests that other factors, such as ischemia, may affect wall motion and produce asynergy in some segments. When serial echocardiographic studies have been performed after acute myocardial infarction, improvement of wall motion in some segments has been noted, again suggesting that a reversible process was responsible.
for the production of asynergy. Therefore, the cross-sectional echocardiographic measure of segmental asynergy is not a measure of the size of irreversibly infarcted myocardium, but rather provides a functional index of wall motion produced by the acute infarction.

In patients with acute ventricular septal defects or acute mitral regurgitation, analysis of segmental asynergy suggested better ventricular function than that measured by hemodynamic criteria. This finding suggests a potential limitation in correlating wall motion changes with hemodynamic indexes of left ventricular function. Wall motion amplitude is sensitive to changes in preload and afterload, being increased in conditions of volume overload and after pharmacologic reduction in afterload. Acute ventricular septal defect and mitral regurgitation may produce both volume overloading and physiologic unloading of the left ventricle. Both of these factors may result in exaggerated segmental wall motion. However, these patients have clinical evidence of heart failure or cardiogenic shock and hemodynamic evidence of pulmonary congestion and peripheral hypoperfusion. Therefore, analysis of segmental wall motion may not always correlate with the clinical or hemodynamic indexes of left ventricular function in patients with mechanical defects.

Cross-sectional echocardiography offers important potential in evaluating patients with acute myocardial infarction. The extent and nature of segmental asynergy detected by cross-sectional echocardiography correlates with hemodynamic evaluation of left ventricular function. Exceptions to this finding include patients with acute mechanical defects, where wall motion appears exaggerated relative to hemodynamic measurements. In addition, because wall motion changes did not correlate with pathologic or enzymatic parameters of infarct size, cross-sectional echocardiographic analysis of segmental asynergy was not a measure of infarct size, but rather provided a functional index of left ventricular wall motion during acute myocardial infarction.

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