Detection of Left Ventricular Asynergy by Echocardiography

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Assisted by Mary Jo Black, Sonia Chang, and Charles L. Haine

SUMMARY

The purpose of this study was to determine if echocardiography could detect left ventricular asynergy. Forty-eight patients underwent selective coronary arteriography and cine-ventriculography for the evaluation of chest pain. Four patients were studied twice: three before and after myocardial revascularization and one before and after an intervening myocardial infarction. Echocardiographic M-mode scans were registered on a strip chart as the left ventricle was scanned with an ultrasonic beam from the aortic root to the region of the posterior papillary muscle approximately 18 hrs prior to the catheterization studies.

Ten of the forty-eight patients had no evidence of coronary artery disease. All ten patients had normal ventriculograms in right anterior oblique projection and their echocardiographic scans showed all areas of the left ventricular posterior wall endocardium to move anteriorly 0.9-1.4 cm (mean 1.2 cm) and all parts of the left side of the interventricular septum to move posteriorly 0.3-0.8 cm (mean 0.5 cm) during systole. The 38 patients with significant obstructive coronary artery disease had a total of 42 studies; 25 of these studies showed left ventricular asynergy on the ventriculogram taken in right anterior oblique. The echocardiograms associated with all but one of these studies demonstrating left ventricular asynergy had abnormal motion of some part of the interventricular septum and/or left ventricular posterior wall. Seventeen studies in patients with significant coronary artery disease did not exhibit left ventricular asynergy on the ventriculogram but eight of these studies were associated with distinctly abnormal echocardiograms.

None of the ten patients with significant coronary artery disease and normal echocardiograms had evidence of transmural infarction on their electrocardiograms. Echocardiographic abnormalities correlated with the anatomic area predicted by the myocardial infarction pattern on the electrocardiogram in 18 of 20 patients.

All patients demonstrating abnormal echographic interventricular septal motion had a significant obstructive lesion in the left anterior descending coronary artery. In the absence of significant involvement of the left anterior descending coronary artery, echographically recorded interventricular septal motion was invariably normal. On the other hand, eight patients had significant obstruction in their left anterior descending coronary artery and their echographic interventricular septal motion was normal.

The results of this correlative study indicate that M-mode echocardiographic scans can detect left ventricular asynergy and may possibly predict regional myocardial involvement in coronary artery disease.

Additional Indexing Words:

Echocardiography
Left ventricular asynergy
Ischemic heart disease

Ultrasound cardiology
Myocardial aneurysm
Angiocardiography
Coronary artery disease

Although paradoxical motion of left ventricular myocardium rendered acutely ischemic by coronary artery ligation in the dog was demonstrated in 1935, it has only been in recent years that local disturbances of configuration and temporal sequence of contraction (asynergy)2 have been the subject of major clinical and research interest in cardiology.3

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Circulation, Volume XLVIII, August 1973

263
Optimal definition of left ventricular asynergy is presently obtained through left ventricular cineangiography, a method requiring cardiac catheterization and the intracardiac injection of radioactive contrast material. Ventricular cineangiography carries an obligation expense, discomfort and risk for the patient. Echocardiography, by contrast, is a direct, sensitive and noninvasive method of recording motion of the interventricular septum and left ventricular posterior wall from a time display of reflected pulsed ultrasound. The purpose of this study was to determine the feasibility of detecting left ventricular asynergy by echocardiography.

**Method**

From the patients referred to Indiana University Medical Center for evaluation of chest pain and/or dyspnea, some were selected for further study by right and left heart catheterization, left ventricular cineangiography and selective coronary arteriography. They also had resting 12-lead electrocardiograms and M-mode echocardiograms within 12-24 hrs of their invasive studies. The availability of both high quality left ventricular cineangiograms and echocardiograms for each patient was the only criterion for inclusion in this study.

Sixty-three echocagrams were reviewed and 48, or 76%, were considered satisfactory for interpretation. This study group consisted of 36 men and 12 women. There were seven individuals between 30-39, 19 between 40-49, 18 between 50-59 and four between 60-65 years old. Four patients were studied twice: three were restudied three months following insertion of saphenous vein bypass grafts while a fourth had a second study three months following an acute myocardial infarction that occurred eight months after his initial study.

The angiographic studies were carried out under fluoroscopic television control with either a Phillips 5 inch or General Electric 6-9 inch image intensifier and were recorded on Kodak XX film at 60 frames/sec. An Enesco pressure injector delivered 30-50 cc of Hypaque for the left ventricular cineangiograms which were routinely done in 30 degrees of right anterior oblique (RAO) and occasionally also in 50-60 degrees of left anterior oblique (LAO). Coronary ateriograms were performed with 6-9 cc injections of Renografin-76 by the Judkins technique and recorded in multiple views.

The ultrasound studies were carried out with a 0.5 inch diameter, 2.25 megaHertz transducer focused at either 7.5 cm or 10.0 cm. The output of the echogram was displayed and recorded on a multichannel oscilloscopic recorder (Electronics for Medicine model DR-8). The patients were studied either in the horizontal position or in a semi-reclining posture with the trunk slightly elevated. All subjects were studied on their backs and the vast majority were studied in some degree of left lateral decubitus (left shoulder down) position as well. A soluble gel was applied to the chest in the third, fourth or fifth intercostal space just lateral to the left sternal margin in order to establish an airless contact between the transducer and the skin. Echocardiographic M-mode scans of the left ventricle between the aortic root and the posterior papillary muscle were carried out as has been described previously (fig. 1). Emphasis was placed on recording continuous echoes from the left side of the interventricular septum and the endocardium of the left ventricular posterior wall both in the “apical” region, at the level of the posterior papillary muscle and in the “standard” echocardiographic position where anterior and posterior leaflets of the mitral valve are simultaneously recorded within the left ventricular cavity. A standard limb lead, usually lead II, of the electrocardiogram was simultaneously recorded with the echograms.

Initially, the left ventricular cineangiograms and echocardiograms of patients were screened only for completeness and quality. Following this preselection, all of the left ventricular cineangiograms were reviewed by at least two of the authors utilizing a Tagarno 35 mm film projector. The left ventricular cavity of each ventricle was traced on a sheet of paper with frames representing end-systole superimposed upon frames representing end-diastole. Comments were also made on the temporal sequence and character of wall motion as the films were reviewed during rapid motion. All of the echocardiograms were also reviewed independently by at least two of the authors. Special attention was paid to the timing and extent of interventricular septal motion both in the “apical” and “standard” positions. Also, posterior wall amplitude was observed during scanning from the “standard” to the “apical” positions. All 12-lead resting electrocardiograms were interpreted by at least two of the investigators utilizing criteria for scalar electrocardiographic diagnosis of myocardial infarction as recently reviewed.

**Results**

Ten of the 48 patients in this study had normal selective coronary arteriograms and normal left ventricular cineangiograms in right anterior oblique projection. They had an average systolic posterior wall amplitude of 1.2 cm (range 0.9-1.4 cm) and interventricular septal excursions of 0.5 cm (range 0.3-0.8 cm). Figure 2 illustrates a left ventricular echogram from a normal subject.

Abnormal interventricular septal motion was classified as diminished (systolic posterior motion less than 0.3 cm), flat (no detectable motion) or paradoxical (anterior motion during systole). Exaggerated interventricular septal motion, greater than 0.8 cm posterior systolic excursion in the “standard” position, was felt most likely to represent a compensatory change due to loss of normal motion in some other area of the ventricle or possibly to a left ventricular volume overload. Therefore, exaggerated interventricular septal motion was not
characterized as abnormal per se but was the cause of a diligent search for areas of diminished motion elsewhere. Since the echo from the posterior left ventricular endocardium in normal subjects moves anteriorly 1.2 ± 0.2 cm during systole, anterior systolic movement less than 0.8 cm during systole was considered abnormally diminished. A reduction in posterior left ventricular endocardial amplitude to less than 0.8 cm was particularly significant if the systolic movement was within the normal range in one position, but upon scanning to another position the amplitude became abnormally diminished. As with exaggerated septal motion, posterior left ventricular endocardial systolic amplitude exceeding 1.6 cm in the “standard” position was felt possibly to represent a compensatory change due to inadequate myocardial contractility elsewhere. Diminished motion was therefore diligently sought, especially in the interventricular septum.

None of the patients in this study had an increased right ventricular dimension as measured from the right side of the interventricular septum to the echo from the anterior right ventricular wall. None of the patients had a left bundle branch block pattern on their electrocardiogram.

Thirty-eight patients had significant coronary artery disease. Four patients had single vessel, 19 had double vessel, and 15 had triple vessel involvement. Forty-two comparative angiographic and echocardiographic studies were performed, and 32 demonstrated abnormally diminished echocardiographic wall motion. Of these 32 studies, 24 (76%) showed isolated flattened, or paradoxical interventricular septal motion, six (19%) exhibited abnormal excursion of both the interventricular septum and the posterior wall and only two (6%) had solely impaired posterior wall amplitude. Cineventriculography in the right anterior oblique position demonstrated left ventricular asynergy in 25 studies, which was considered marked in 17 instances (marked hypokinesis or akinesia of essentially the entire anterolateral wall in six, similar involvement of the mid anterior and inferior walls including the apex in five, anterolateral dyskinesia in two, akinesis of the entire inferoapical segment in two and one each with generalized hypokinesis or large basal

Figure 1
Echocardiographic M-mode scan of a normal heart with the ultrasonic beam moving from the base to the apex of the left ventricle. The “routine position” is that area of the left ventricular cavity between the left side of the interventricular septum (LS) and the posterior left ventricular endocardium (EN) whereby parts of the anterior (AMV) and posterior (PMV) mitral valve leaflets are still present. The “apical” position no longer has mitral leaflet echoes. Instead echoes are recorded from the posterior papillary muscle (PPM). In this position the left ventricular dimension is diminished and the amplitude of septal motion is usually increased. PLA = posterior left atrial wall, PC = posterior chordae.
inferior aneurysm). Eight ventriculograms demonstrated less extensive disease (isolated apical hypokinesis or akinesis in three, discreet mid inferior or anterior wall hypokinesis or akinesis in three, and individual examples of hypokinesis of both the mid inferior and anterior wall or asynchrony of the mid anterior wall).

Table 1 compares the left ventricular cineangiograms in right anterior oblique with the echocardiographic M-mode scan on the 38 patients with angiographically proven coronary artery disease. Of the 25 patients with abnormal left ventricular angiograms, 24 had abnormal echocardiograms. The only echocardiographic false negative occurred in a patient with asynchrony of the mid anterior wall and a total occlusion of the proximal left anterior descending coronary artery. On the other hand, eight patients with significant coronary artery disease demonstrated abnormal left ventricular echocardiograms but normal left ventricular angiograms in the RAO projection. All eight had obstructive lesions in the left anterior descending coronary artery.

There was no correlation between the echograms and the amount of asynchrony or akinesis in the ventriculograms. The echocardiogram was just as likely to detect asynchrony in a patient with only a mildly abnormal ventriculogram as in one with a markedly abnormal ventriculogram. This lack of correlation also meant that the echocardiogram could not predict the extent of involvement as demonstrated on the ventriculogram.

Thirty-four of the 38 patients with coronary artery disease had significant involvement of the left anterior descending coronary artery, including the four patients who were studied twice. Three of the latter four were restudied following attempted bypass revascularization of the left anterior descending artery, which in each instance was unsuccessful. Interventricular septal motion was normal preoperatively in two of these three patients and became distinctly abnormal postoperatively while in the third case septal motion was abnormally diminished preoperatively and became paradoxical postoperatively. Two of these patients had normal preoperative left ventriculograms but postoperatively were shown to have mid anterior wall hypokinesis.

Table 2 demonstrates the relationship between echocardiographically detected abnormal interventricular septal motion and obstruction of the left

<table>
<thead>
<tr>
<th>Echocardiogram</th>
<th>Left ventricular cineangiogram</th>
<th>Normal</th>
<th>Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal patients without CAD</td>
<td>10</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Patients with CAD</td>
<td>9</td>
<td>1*</td>
<td></td>
</tr>
<tr>
<td>Abnormal – patients with CAD</td>
<td>8</td>
<td>24</td>
<td></td>
</tr>
</tbody>
</table>

$X^2 = 24.05, \ P < 0.005.$

*Asynchrony was the only abnormality noted on the ventriculogram.

<table>
<thead>
<tr>
<th>Septal echo pattern</th>
<th>Degree of obstruction in the left anterior descending coronary artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Less than 75%</td>
</tr>
<tr>
<td>Abnormal</td>
<td>Greater than 75%</td>
</tr>
</tbody>
</table>

$X^2 = 7.51, \ P < 0.01.$

*Only one of these patients had a transmural myocardial infarct on his resting electrocardiogram.

Circulation, Volume XLVIII, August 1973
Table 3

<table>
<thead>
<tr>
<th>Echo pattern</th>
<th>Electrocardiographic pattern</th>
<th>Transmural Infarction</th>
<th>Abnormal Echocardiographic Patterns of Motion in Patients with Coronary Artery Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>10</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>12</td>
<td>20</td>
<td></td>
</tr>
</tbody>
</table>

$X^2 = 9.57, P < 0.01$. Corrected for continuity.

anterior descending coronary artery. No patient with an essentially normal left anterior descending coronary artery had abnormal interventricular septal motion (no false positives) while 30 of 38 studies involving patients with significant obstruction had abnormal septal echo motion. Among these 30 patients are the eight who had significant coronary artery disease and abnormal echograms but normal left ventriculograms.

Table 3 compares abnormalities in the resting electrocardiogram with the anatomically related portion of the echocardiographic left ventricular scan. There were no false negatives (transmural myocardial infarction and a normal echocardiogram). In 18 of 20 patients with transmural myocardial infarction the electrocardiographic location of the infarction correlated with the area of the ventricle which moved abnormally on the echocardiogram. One exception was a patient with an electrocardiographic inferior wall myocardial infarction plus a concomitant 90% obstructive lesion in the proximal left anterior descending coronary artery. The echocardiogram demonstrated abnormal septal motion. Another patient had an anterior myocardial infarction pattern on his electrocardiogram but exhibited exaggerated echocardiographic septal motion. This case was complicated by the presence of significant mitral insufficiency.

Figures 3 through 7 provide representative angiograms and echocardiograms from the patients.
with left ventricular asynergy. Figure 3 contains recordings from a patient with a large inferobasal myocardial aneurysm and associated mitral insufficiency. Figures 4 and 5 are from a patient with obstruction of the left anterior descending coronary artery. Interestingly enough only the left anterior oblique angiogram showed the abnormal movement of the interventricular septum.

Patients with large akinetic or dyskinetic segments of their anterolateral walls on ventriculography had typical echocardiographic patterns. Superimposed outlines of the left ventricular cavity in diastole and in systole in both right anterior and left anterior oblique projections are provided in figure 6. Figure 7 shows two representative echocardiographic strips from the same patient whose angiograms are illustrated in figure 6.

Discussion

Utilizing contemporary echocardiographic techniques, the transducer is placed in either the third, fourth or fifth left intercostal space just lateral to the left sternal margin. In this position the ultrasonic beam passes through only the superior-anterior portion of the interventricular septum and the basal portion of the posterior left ventricular wall. Upon scanning to the "apical" area, the transducer is merely angled inferolaterally to the region of the posterior papillary muscle with the beam still only passing through the anterior portion of the interventricular septum and possibly part of the diaphragmatic left ventricular wall. Since the transducer is moved in an arc, the ultrasonic beam traverses more on the posterior wall than the septum; the anterolateral left ventricular wall and the apex are not directly visualized by this technique. Even though the echocardiogram does not examine large areas of the left ventricle, in this study the echocardiograms were abnormal in a very high percentage of the patients who demonstrated akinesis on the ventriculograms. One possible explanation might be that the akinetic areas were quite extensive. Even though the echocardiogram did not visualize the akinetic apex or lateral wall, there was some area of the ventricle which was transsected by the ultrasonic beam and moved abnormally. For example, obstruction to the left anterior descending coronary artery may have produced a large akinetic area in the apex as well as abnormal septal motion. On the angiogram the apical abnormality was most obvious whereas the echocardiogram noted the abnormal septal motion.

Another possible reason for the high percentage of abnormal echograms was that the majority of the patients in this study had significant obstruction of the left anterior descending artery. This vessel is the major source of myocardial blood supply to the left ventricular anterior wall, apex and anterior two-thirds of the interventricular septum.21, 22 Because the coronary atherosclerotic process tends to involve segmentally only the epicardial course of a coronary artery and involvement of the left anterior descending most frequently is proximal,23 the left ventricular anterior wall, apex and interventricular septum all tend to be included in the ischemic process when this vessel is afflicted. Alterations in the echocardiographic pattern of interventricular septal motion in patients with ischemic heart disease should reflect the state of the myocardium served by the left anterior descending coronary artery and indirectly, therefore, the status of this vessel.

Every patient with an abnormal echographic pattern of interventricular septal motion had a significant flow-restricting lesion in the left anterior

![Figure 5](image)

*Figure 5*  
Line drawings of the ventriculogram in the right anterior oblique (LVRAO) and the left anterior oblique (LVLAO) projections of the same patient whose echogram appears in figure 4. The RAO projection demonstrates only generalized decreased contractility. The LAO ventriculogram shows diminished septal motion and exaggerated posterior wall motion.

![Figure 6](image)

*Figure 6*  
Line drawings of the left ventriculograms in both obliques from a patient with asynergy of the entire anterior wall.

*Circulation, Volume XLVIII. August 1973*
ventricular asynergy. However, eight patients with such lesions had normal echocardiographic septal motion. Since completion of this study, echograms have been interpreted prospectively in order to predict left ventricular asynergy in patients with ischemic heart disease. These results have been comparable to those reported herein. One particularly instructive case had normal echographic interventricular septal motion at rest, but the pattern of motion became distinctly abnormal during angina induced by isometric exercise. Selective coronary arteriograms on this patient demonstrated a high grade obstructive lesion in the left anterior descending coronary artery.

The higher incidence of abnormalities in the echocardiograms than in the ventriculograms among the patients with angiographically proven coronary artery disease was an intriguing and somewhat surprising observation. There was actually only one patient with left ventricular asynergy detected by left ventricular cineangiography who had a normal echocardiogram. This patient had synchrony of the mid anterior wall of the left ventricle. On the other hand there were eight patients with normal left ventriculograms in right anterior oblique projection who had abnormal echocardiograms. These eight patients all had 75% obstructive lesions in their left anterior descending coronary arteries. It is impossible to prove that these patients truly had left ventricular asynergy since the available ventriculograms were normal; however all eight patients had significant obstruction to the left anterior descending artery which theoretically could produce septal asynergy. In addition the echographic findings in these eight patients were identical to the findings in those patients who were found to have proven asynergy on the ventriculograms. Furthermore these findings were not noted in any of the subjects without left anterior descending obstruction. Thus it is highly probable that these eight patients did have asynergy as recorded on the echogram but not on the ventriculogram.

There are many possible explanations for the normal ventriculograms and abnormal echograms in patients with left anterior descending coronary artery disease. First of all the echocardiogram and the right anterior oblique ventriculogram do not look at the same areas of the left ventricle. In the right anterior oblique ventriculogram the anterolateral and inferomedial walls of the ventricle are visualized. The echogram records the anterior portion of the interventricular septum, the true posterior wall and part of the diaphragmatic
borders of the ventricle. In one case (figs. 4–5) abnormal septal motion was not appreciated in the right anterior oblique ventriculogram but was noted in the left anterior oblique projection. The mid portion of the interventricular septum makes up one of the borders of the left anterior oblique ventriculogram and thus might correlate better with the echogram in some cases. Actually a lateral ventriculogram would probably correlate better with the echogram than would either oblique projection.

It is possible that the echocardiogram may actually be a more sensitive means of detecting asynergy. With an echocardiogram one can measure abnormal wall motion within a few millimeters. One also has the advantage of examining numerous cardiac cycles to make this judgment. With an angiographic ventriculogram the detection of asynergy may have many technical difficulties. At best one has two or three cardiac cycles to examine. There frequently may be a catheter-induced arrhythmia. There is also a subjective element in determining the quality of contraction. Another point which might work in favor of using echocardiography for detecting asynergy secondary to left anterior descending artery disease may be the particular area of the ventricle examined by the ultrasonic beam. The anterior third of the interventricular septum is probably supplied by the left anterior descending artery and is thus a very specific area of the ventricle with respect to this artery. The blood supply to the anterolateral wall and the apex, that portion usually seen with the RAO ventriculogram, could have a more variable blood supply.

The good correlation between the electrocardiogram and the echocardiogram with respect to localizing the area of ischemic damage also supports the claim that echocardiography can detect localized areas of asynergy. Significant Q waves on the electrocardiogram has long been equated with irreversible structural changes resulting from myocardial necrosis. Eighteen of 20 patients with electrocardiographic evidence of old transmural myocardial infarction had abnormal echograms in the anatomical area predicted by the infarct pattern. One exception was a patient with an inferior wall myocardial infarction and a 90% occlusive lesion in the left anterior descending coronary artery. The echogram showed the loss of interventricular septal motion. In addition there was a patient who had electrocardiographic evidence of an old transmural anterior wall myocardial infarction but had exaggerated rather than diminished septal motion. However he had significant mitral insufficiency, and a volume overload of the left ventricle has been known to produce excessive septal motion. The mechanism for the exaggerated interventricular septal motion in a patient with both a volume overload of the left ventricle and an old anterior wall infarction remains obscure, but the finding implies that at least some portion of the myocardium of the interventricular septum was preserved and permitted an appropriate response to the volume stress.

The most common echocardiographic abnormality found in this study was diminished or paradoxical interventricular septal motion. Other diseases have been reported to produce abnormal septal motion; so this finding in itself is not necessarily specific. However, there are some possible clues to help make the proper diagnosis. First of all none of the patients in the present report had a right ventricular dimension beyond the normal range; therefore, the paradoxical interventricular septal motion most likely has a pathogenesis different from that suggested for volume overload of the right ventricle. The abnormal interventricular septal motion recorded in the present report appears completely passive when compared with "active" or rapid movements during certain phases of the cardiac cycle in patients with a right ventricular volume overload. In addition, several patients' echograms (fig. 7) showed a thinning of the interventricular septum with an apparent increase in echo density of this structure, perhaps analogous to the fibrotic involvement of the mitral valve in mitral stenosis. Patients with left bundle branch block with or without coronary artery disease may also exhibit abnormal septal motion and must be considered when this echocardiographic finding is present. None of the patients in this study had a left bundle branch block.

In order to detect left ventricular asynergy with echocardiography the echocardiographer must examine as many different areas of the ventricle as possible. The patient whose echograms are in figure 7 had normal echographic interventricular septal motion when supine, but when he was turned into the left lateral position, the septal motion became paradoxical. The importance of M-mode scanning was demonstrated by several patients in whom the "standard" or "apical" echograms were vastly different. The presence of exaggerated wall motion was a helpful hint that asynergy existed and one
needed to search further to find the affected area of the ventricle.

This study is admittedly preliminary and the findings must be substantiated by others. In addition it must be remembered that the echocardiographic examination is frequently technically difficult in patients with coronary artery disease. We were able to obtain satisfactory echograms on only 76% of the patients initially studied. Our percentage should increase with experience and with improvements in technique and instrumentation, but for the time being the inability to record interpretable echograms on all patients with coronary artery disease remains a significant limitation. Despite these problems, the results indicate that in certain patients, especially those with obstruction to the left anterior descending coronary artery, echocardiography can detect regional left ventricular asynchrony with possibly a high degree of sensitiviy.

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