Immediate Diagnosis of Acute Myocardial Infarction by Two-dimensional Echocardiography

RICHARD S. HOROWITZ, M.D., JOEL MORGANROTH, M.D., CONNIE PARROTTO,
CHIN C. CHEN, M.D., JOSEPH SOFFER, M.D., AND FERREL J. PAULETTO, M.D.

SUMMARY To define the role of portable two-dimensional echocardiography (2-D echo) in the immediate diagnosis of acute chest pain syndrome, 80 consecutive patients were studied. Adequate 2-D echo studies were obtained in 65 (81%). Thirty-three patients had clinical evidence of transmural or nontransmural acute myocardial infarction (AMI), 18 of whom had nondiagnostic initial ECGs. Thirty-two did not have a clinical AMI. Thirty-one of the 33 (94%) patients with clinical AMI had regional wall motion abnormalities on the initial 2-D echo; the other two had uncomplicated nontransmural AMIs, diagnosed only by ECG in one and by ECG and moderate elevation of CK-MB isoenzyme in the other.

Twenty-seven of the 32 patients without clinical AMI had normal regional wall motion on the initial 2-D echo and none had a complication (severe arrhythmia, recurrent pain, heart failure or death) during the hospital course. Conversely, 10 of the 36 patients with initial 2-D echo regional wall motion abnormalities had a complication (p < 0.05). Thus, in patients with acute chest pain syndrome, an initial 2-D echo that shows no regional wall motion abnormality suggests that such patients will not develop an AMI or clinical complication during the hospital course.

An initial 2-D echo with regional wall motion abnormality identifies a high-risk group of patients who are likely to have AMI and important cardiac complications and may, therefore, benefit from admission to an intensive care unit.

PATIENTS with suspected acute myocardial infarction are usually admitted to the hospital's intensive care unit, where sophisticated monitoring equipment and highly trained personnel are used and traditional serial studies are obtained to establish a definite diagnosis. Unfortunately, diagnostic tests for acute myocardial infarction at the time of admission are limited. If the standard 12-lead ECG shows a classic pattern of myocardial infarction, then an immediately confirmed diagnosis is made, but often, the time lag in developing the characteristic ECG changes significantly limits this procedure as a screening test.1 Creatine kinase (CK) determination, by including the new B-subunit radioimmunoassay method,2 has excellent specificity and sensitivity in diagnosing a myocardial infarction, but usually requires several hours for its determination. Radionuclide cardiac imaging to determine myocardial viability using thallium-201 has been useful in the immediate diagnosis of myocardial infarction, but its limited specificity3,4 the need for expensive equipment and radiisotope reservoirs, and the small but definite radiation exposure5 limit its usefulness.

Increasing interest has focused on two-dimensional echocardiography for detecting acute myocardial infarction.6,7 This technique is a reliable method for defining the presence and location of regional wall motion abnormalities associated with acute myocardial infarction, which correlate well with pathologic data and with the site of infarction on the ECG. The purpose of this study was to investigate the role of two-dimensional echocardiography in the immediate diagnosis of acute myocardial infarction in patients who present with acute chest pain to the emergency room.

Materials and Methods

The study population consisted of 80 consecutive patients with a symptom complex of prolonged chest discomfort occurring within 12 hours of admission to an intensive care unit. Patients with valvular heart disease, previous myocardial infarction, cardiomyopathy or significant pericardial effusion were excluded by clinical history, physical examination or echocardiography. M-mode and two-dimensional echocardiograms were obtained within 8 hours of admission. Fifty-four males and 26 females, ages 32–85 years, were studied. Myocardial infarction was diagnosed by a typical history of chest pain along with either characteristic electrocardiographic changes or a rise in serum CK-MB. ECGs were performed at least once daily and CK-MB was measured every 8 hours for the first 24 hours and then daily for at least 2 days. CK-MB was performed by the cellulose acetate electrophoretic method. A transmural myocardial infarction was diagnosed by the development of new Q waves ≥ 0.04 second in more than one lead or an R:S ratio > 1 in lead V1 or V2 with at least abnormal repolarization in the inferior leads. A nontransmural myocardial infarction was diagnosed by an elevation of CK-MB enzyme without necessitating important ECG changes or classic evolution of deep symmetric T-wave inversions. The infarction was considered inferior if the Q waves or symmetrically inverted T waves occurred in leads 2, 3, or aV3; anterior if in leads V1–V5; and lateral if in leads 1, aV4, or V6–V8. The clinical course was monitored in the intensive care unit and thereafter. A cardiac complication was

From the Departments of Medicine and Research, Lankenau Hospital, and the Department of Medicine, Jefferson Medical College of the Thomas Jefferson University, Philadelphia, Pennsylvania.

Dr. Horowitz's present address: Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania 19104.

Address for correspondence: Joel Morganroth, M.D., Cardiovascular Division, Lankenau Hospital, Lancaster Avenue and City Line Avenue, Philadelphia, Pennsylvania 19151.

Received October 7, 1980; revision accepted April 30, 1981.


323
defined by the presence of a severe arrhythmia (complete atroventricular block, ventricular tachycardia or ventricular fibrillation); recurrent severe chest pain with evidence of myocardial necrosis by ECG or CK-MB; left ventricular pump failure confirmed by right-heart catheterization or pulmonary edema; or sudden cardiac death.

Echocardiography was performed using a commercially available, wide-angle, three-element rotary mechanical sector scanner (ATL Mark III, Advanced Technology Laboratory) with a 3.5-MHz transducer. The left ventricular long-axis, multiple short-axis and apical views of the left ventricle were imaged. Two-dimensional echocardiograms were recorded on a Sanyo (model 7100) 1/2-inch videotape recorder. Individual frames selected for the figures were photographed using Polaroid film from the videotape recording. There was significant degradation of the stop-frame images because only one of the two fields could be visualized during stop-frame imaging.

For purposes of analysis, left ventricular long-axis, short-axis at the mitral valve and papillary muscle level, and four-chamber views were all reviewed blindly without knowledge of the patient's course or other clinical data by three independent observers. A regional wall motion abnormality was defined by decreased contraction of a region of endocardium in comparison to the remainder of the endocardium visualized. Location of a regional wall motion abnormality was assessed by the above schema, separating the left ventricle on the standard two-dimensional views into the ventricular septum, anterior, posterolateral, posterior and apical regions (fig. 1). An echocardiographic study was considered technically difficult if all endocardial segments were not visualized well enough to assess the presence or absence of abnormal wall motion or if at least more than 50% of the endocardium was not visualized in any region. If any of the observers disagreed, the studies were reviewed and agreement of at least two reviewers was required.

Statistical Analysis
The sensitivity and specificity of the presence of regional wall motion abnormalities on the initial echocardiogram signifying a clinical myocardial infarction were calculated by the formulas:

\[
\text{% sensitivity} = \frac{\text{true positives}}{\text{true positives + false negatives}} \times 100
\]

and

\[
\text{% specificity} = \frac{\text{true negatives}}{\text{true negatives + false positives}} \times 100.
\]

A chi-square statistical analysis was used in the between group comparison.

Results
Echocardiographic studies sufficient for analysis of regional wall motion were recorded in 65 of the 80 study patients (81%). Of the 15 patients excluded because of technically inadequate echocardiograms, eight had a clinical myocardial infarction. Among the 65 final study patients, 33 (mean age 61 ± 16 years, 79% males) had a clinical acute myocardial infarction and 32 (mean age 61 ± 12 years, 56% males) did not. Nineteen of those with myocardial infarction sustained a transmural and 14 a nontransmural infarction. The location of the transmural myocardial infarctions was inferior in 12 patients and anterior in seven. Nontransmural myocardial infarctions were inferior in three patients, anterior in three, lateral in four, and the site was not determined by ECG in four.

Two-dimensional Echocardiographic Findings (fig. 2)
Patients with Clinical Myocardial Infarction
Of the 33 patients who suffered a clinical myocardial infarction, a regional wall motion abnormality was detected by echocardiography in 31 (94%). A regional wall motion abnormality was evident in 19 of 19 (100%) with a transmural myocardial infarction and in 12 of 14 (86%) with a nontransmural infarct.

![Figure 1. The cardiac tomographic two-dimensional echocardiographic planes used in this study. (A) Left ventricular long-axis view. (B) Apical four-chamber cardiac view. (C) Short-axis view of the left ventricle at the level of the mitral valve orifice (mvo). (D) Short-axis left ventricular view at the level of the papillary muscles (p). Segment 1 represents the ventricular septum, segment 2 the anterior left ventricular wall, segment 3 the posterolateral wall, segment 4 the posterior wall and segment 5 the cardiac apex.](http://circ.ahajournals.org/lookup/doi/10.1161/01.CIR.65.2.324)
80 patients admitted with chest pain

15 technically difficult

36 abnormal RWM on echo

31 clinical MI

29 normal RWM on echo

5** no clinical MI

2* clinical (subendocard.) MI

3/3 had CAD on angiography

10 cardiac complications

27 no clinical complications

no clinical complications

FIGURE 2. The echocardiographic and clinical results in each patient group. RWM = regional wall motion; CAD = coronary artery disease; MI = myocardial infarction; Echo = two-dimensional echocardiography; subendocard. = subendocardial.

Figure 3 is a short-axis view of the left ventricle at the papillary muscle level of a patient who sustained a clinical inferior transmural myocardial infarction, and it shows an inferior regional wall motion abnormality. One of the two patients with false-negative echocardiograms had typical chest pain and evolved symmetrically inverted anterior T-wave inversions, but did not have an elevation in CK-MB isoenzyme; the other had lateral symmetric T-wave inversions with a modest elevation in CK-MB isoenzyme (table 1).

The site of a regional wall motion abnormality directly correlated with the ECG site of myocardial infarction in 17 of 19 of those cases with a transmural myocardial infarction and nine of 14 of those with nontransmural myocardial infarction. Those cases of disagreement involved a more extensive area of abnormal regional wall motion on echocardiography than that suggested by the ECG.

Despite the evolution of a clinical myocardial infarction and an abnormal echocardiogram on admission, the initial ECG was considered nondiagnostic in 18 of 33 patients. The initial CK-MB isoenzyme level in this group was not elevated in 16 of 33 patients. Ten cardiovascular complications occurred in these 33 patients with clinical myocardial infarctions. Two patients had left-heart failure, two had left-heart failure and severe arrhythmia, three had left-heart failure and recurrent chest pain, two had

FIGURE 3. Two-dimensional echocardiographic short-axis left ventricular views of a patient with acute chest pain syndrome demonstrating (left) end-diastole and (right) end-systole. The schematic is a light-pen outline of the endocardial surface in systole and in diastole superimposed and demonstrates a posterior (inferior) regional wall motion abnormality (akinesis).
TABLE 1. Discordant Echocardiographic and Clinical Diagnoses

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Peak CK level*</th>
<th>ECG</th>
<th>2-D echo</th>
<th>Clinical course</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regional Wall Motion Abnormality on Two-dimensional Echocardiogram But No Clinical Myocardial Infarction (False Positive)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>54</td>
<td>M</td>
<td>47</td>
<td>Normal</td>
<td>Posterior hypokinesis</td>
<td>Uncomplicated; normal ventriculography, 3-vessel CAD, CABG</td>
</tr>
<tr>
<td>2</td>
<td>44</td>
<td>M</td>
<td>41</td>
<td>Normal</td>
<td>Posterior and septal hypokinesis</td>
<td>Uncomplicated; normal ventriculography, 99% LAD lesion, CABG</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>M</td>
<td>46</td>
<td>Left anterior hemiblock; otherwise normal</td>
<td>Septal hypokinesis</td>
<td>Uncomplicated; ventriculography: anterior apical hypokinesis, 60% LAD lesion</td>
</tr>
<tr>
<td>4</td>
<td>53</td>
<td>M</td>
<td>25</td>
<td>NS ST-T-wave abn</td>
<td>Anterior hypokinesis</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>5</td>
<td>71</td>
<td>M</td>
<td>83</td>
<td>NS ST-T-Wave abn</td>
<td>Apical hypokinesis</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>Clinical myocardial infarction but normal regional wall motion abnormalities on 2-D echo</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>61</td>
<td>M</td>
<td>44</td>
<td>Symmetric anterior T-wave inversions</td>
<td>Normal</td>
<td>Uncomplicated</td>
</tr>
<tr>
<td>2</td>
<td>78</td>
<td>M</td>
<td>214MB+</td>
<td>Symmetric lateral T-wave inversions</td>
<td>Normal</td>
<td>Uncomplicated</td>
</tr>
</tbody>
</table>

Abbreviations: CAD = coronary artery disease; LAD = left anterior descending CAD; CABG = coronary artery bypass graft surgery; NS ST-T-wave abn = nonspecific ST-T-wave abnormalities; MB+ = positive MB band on cellular acetate.

*Normal = 0–100 U for males.

recurrent chest pain and one patient suffered sudden death on the sixth hospital day. Both patients with clinical nontransmural infarction whose echocardiograms revealed no abnormal regional wall motion abnormalities had an uncomplicated clinical course. Patients Without a Clinical Myocardial Infarction

Of the 32 patients who did not evolve a clinical myocardial infarction, 27 (84%) had no regional wall motion abnormality demonstrated on their initial echocardiogram. Figure 4 is a short-axis view at the

![CARDIAC SILHOUETTE](http://circ.ahajournals.org/)

**FIGURE 4.** (top) Cardiac silhouette demonstrating the aorta (AO) and pulmonary artery (PA) and the tomographic slice that represents the short-axis view of the left ventricle at the level of the papillary muscles (p). The ventricular septum (vs) anterior (ant.), posterolateral (post. lat.), and posterior (post.) segments are identified. (bottom) The end-diastolic (diast.) and end-systolic (syst.) echocardiograms are shown in the bottom two panels. During systole, all regions of the endocardium move toward the center of the left ventricle, demonstrating no regional wall motion abnormality in this patient with chest pain syndrome.
papillary muscle level of the left ventricle in which normal symmetric ventricular contraction without regional wall motion abnormality was evident. Five of the 32 patients without clinical myocardial infarction had regional wall motion abnormalities, and thus were considered to be false positives. All of the 27 patients without regional wall motion abnormalities by echocardiography had an entirely uncomplicated hospital course. Seven of these 27 had cardiac catheterization at the discretion of their attending cardiologist and four were shown to have severe coronary artery disease. The ventriculograms in all seven patients were normal. Of the five patients without a clinical myocardial infarction but with regional wall motion abnormalities on echocardiography, three had further invasive evaluation, and all three had coronary artery disease; two required coronary artery bypass graft surgery (table 1).

Patients Without Regional Wall Motion Abnormality on the Initial Two-dimensional Echocardiogram

Of the 29 patients with normal regional wall motion on the initial echocardiogram, including 27 who did not suffer myocardial infarction and two with nontransmural myocardial infarctions, none had serious cardiac complications. Cardiac catheterization was performed in seven patients, three of whom later required coronary artery bypass graft surgery.

Patients with Regional Wall Motion Abnormality on the Initial Two-dimensional Echocardiogram

Of the 36 patients with regional wall motion abnormalities on the initial echocardiogram, 31 had a documented clinical myocardial infarction, 10 of whom had serious cardiovascular complications ($p < 0.05$ compared with the patients with no regional wall motion abnormalities).

M-mode Echocardiography

The initial M-mode echocardiogram in the patients without clinical myocardial infarction was normal in all 32 cases, including the five with false-positive two-dimensional echocardiographic studies. Among the 33 cases with a clinical myocardial infarction, only 10 (30%) had abnormal wall motion, whereas 23 had no wall motion abnormality on M-mode echocardiography. This contrasts with the 31 of 33 patients who had a regional wall motion abnormality on two-dimensional echocardiography.

Interobserver Variability

In 56 of the 65 study patients, the three echocardiographers were in complete agreement in their assessments of regional wall motion. In the other nine cases (13%), two of the three observers were in agreement.

Discussion

The commonly used criteria for the clinically diagnosis of acute myocardial infarction as defined by the World Health Organization include chest pain, classic ECG abnormalities and elevation of serum cardiac enzymes. If the initial ECG is diagnostic for myocardial infarction, an immediate diagnosis is made. However, diagnostic ECG abnormalities on admission are frequently not evident. In this study, 54% of our patients who had a clinical myocardial infarction demonstrated only nonspecific repolarization changes on their initial ECG. Also, other cardiac conditions can both mimic infarction (e.g., central nervous system events and cardiomyopathy) and mask infarction (e.g., left bundle branch block). An initial negative ECG has only a 0.77 predictive value in ruling out myocardial infarction.

CK enzyme determination, especially the more specific isoenzyme CK-MB, although occasionally increased in other disease states, is relatively sensitive and specific for diagnosing acute myocardial infarction. A new radioimmunoassay determination of the B subunit of the CK-MB isoenzyme demonstrated almost 100% sensitivity and specificity in diagnosing acute myocardial infarction, but the assay takes more than 24 hours to complete. The physiologic time delay for elevation of serum CK-MB isoenzyme after infarction, the time necessary to perform the laboratory determination, and the decreased sensitivity in small subendocardial infarctions have all contributed to the realization that the CK determination is not useful for the immediate diagnosis of myocardial infarction.

Investigation has also been directed toward the use of myocardial scintigraphy to diagnose acute myocardial infarction. The infarcted region is seen as a negative scintigraphic image on a thallium scan and a positive image on a technetium pyrophosphate scan. Each of these studies may provide useful information. Thallium scanning for myocardial viability showed a defect in 82% of 200 selected patients with acute myocardial infarction. Of the patients with small infarctions as defined by cardiac enzyme criteria, only 57% had an abnormal thallium scan. All scans done within 6 hours from the onset of chest pain were abnormal but the sensitivity fell rapidly when the patients were studied more than 6 hours after the onset of symptoms. In a prospective study of 203 patients admitted to the intensive care unit with suspected myocardial infarction, an abnormal thallium scan had a 61% diagnostic accuracy in detecting acute myocardial infarction.

Infarct scintigraphy using technetium pyrophosphate cannot be practically used to diagnose acute myocardial infarction immediately at the time of admission because more than 24 hours are required before an abnormal scan can be obtained. This technique is also limited by poor sensitivity and specificity. Acute radionuclide scintigraphy requires more expensive and less mobile equipment than two-dimensional echocardiography. The problems of small but finite radiation exposure and the difficult availability of appropriate quantities of isotope also limits the widespread use of scintigraphy.

Two-dimensional echocardiography has recently gained popularity as a noninvasive diagnostic aid in the evaluation of various forms of heart disease. Although M-mode echocardiography is valuable in detecting wall motion changes related to ischemia, and occasionally in predicting the clinical course of
patients after myocardial infarction, two-dimensional echocardiography, with its expanded view in real-time of the left ventricle, has enhanced our ability to evaluate wall motion changes caused by coronary artery disease.17, 18

Experimental studies have documented regional wall motion asynergy after coronary ligation, which occurs even before classic electrocardiographic changes evolve.19 Reversible exercise-induced two-dimensional echocardiographic wall motion abnormalities secondary to ischemia have also been demonstrated in patients with angiographically documented coronary artery disease supplying the appropriate region of myocardium.19 The value of two-dimensional echocardiography for detecting wall motion abnormalities in patients with transmural myocardial infarction, with good correlation between the region of asynergy on two-dimensional echocardiography and both the ECG and the pathologic location of the myocardial infarction, is established.6, 9 In fact, two-dimensional echocardiography improves on the ECG in localizing the region of acute myocardial infarction.7 The extent and nature of regional asynergy detected by cross-sectional echocardiography correlates with hemodynamic evaluation of left ventricular function.8 This noninvasive procedure has also been used to identify a subgroup of acute myocardial infarction patients with regional cardiac dilatation, which was associated with an increase in mortality.6

The results of this study substantiate the ability of two-dimensional echocardiography to immediately diagnose acute myocardial infarction in patients who have not sustained a prior myocardial infarction. This technique is also effective as a screening tool to identify high- and low-risk subsets of patients.

Ninety-four percent of patients with a clinically documented transmural or nontransmural myocardial infarction had an immediate regional wall motion abnormality on the two-dimensional echocardiogram. One hundred percent of the patients with clinical transmural myocardial infarction were predicted. The two patients with a false-negative echocardiographic study had nontransmural myocardial infarction, but neither had complications during their hospital course. Of the 32 patients who did not have a clinical myocardial infarction, 27 had no regional wall motion abnormality on the initial echocardiogram. Three of the five patients with false-positive echocardiograms on subsequent angiography demonstrated significant coronary artery disease and two underwent coronary artery bypass graft surgery for recurrent ischemia. In addition, 31 of the 36 patients with an abnormal echocardiogram on admission experienced a clinical myocardial infarction, 10 of whom had a complicated course. On the other hand, none of the patients with normal regional wall motion on admission had a complicated course. Thus, two-dimensional echocardiography permits an immediate diagnosis of acute myocardial infarction with a high predictability and also identifies the high-risk patients most likely to have a complicated course. These results might be useful in the proper triage of patients presenting with acute chest pain syndrome.

Patients who did not evolve a myocardial infarction and experienced no significant cardiovascular complications were only followed during their initial hospitalization. This subgroup of patients may, in fact, have significant cardiovascular morbidity, comparable to that in patients who suffer a myocardial infarction but survive the hospital course.20

The technical difficulties of obtaining echocardiograms of high enough quality for easy interpretation of wall motion changes in all patients is a drawback to this technique. Eighteen percent (15 of 80) of our echocardiograms were technically difficult, a rate that compares favorably with other studies.7 In obtaining the short-axis views of the left ventricle, these images must be obtained from a plane parallel to the long axis to avoid false-positive results.

Patients with previous myocardial infarction were not included in this study and are more difficult to assess because regional asynergy from a prior myocardial infarction may persist. A limitation of this study is that we relied on history or electrocardiographic evidence to detect prior infarction, which is not always unequivocal. This makes a decision about a new regional wall motion abnormality difficult without a previous echocardiogram for comparison. Also, this study relied on a subjective interpretation of echocardiographic data because no system was available to quantitate wall motion changes. A normal wall motion pattern (fig. 4) was defined when all endocardial regions moved toward the center of the left ventricular cavity equally. More precise evaluation of wall motion will be possible with a standardized quantitative computer approval to its measurement.

The tremendously increasing cost of health care, fueled considerably by the expense of new technology and sophisticated personnel training, has led to a reexamination of appropriate means of using limited resources.4 Such studies have attempted to identify low-risk patients admitted to the intensive care unit who may need less or no time in the intensive care unit, and thereby reduce hospital costs.21-23 The use of two-dimensional echocardiography in immediately diagnosing a myocardial infarction and in establishing a low-risk group unlikely to have cardiac complications might have a significant impact upon this problem.

References
2-D ECHO IN AMI/Horowitz et al.

Heart J 98: 752, 1979
20. Schroeder JS, Lamb IH, Hu M: Do patients in whom myocardial infarction has been ruled out have a better prognosis after hospitalization than those surviving infarction? N Engl J Med 303: 1, 1980
Immediate diagnosis of acute myocardial infarction by two-dimensional echocardiography.

R S Horowitz, J Morganroth, C Parrotto, C C Chen, J Soffer and F J Pauletto

Circulation. 1982;65:323-329
doi: 10.1161/01.CIR.65.2.323

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/65/2/323

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the
Editorial Office. Once the online version of the published article for which permission is being requested is
located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/