Estimation of Myocardial Involvement in Patients with Acute Myocardial Infarction by Two-dimensional Echocardiography

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SUMMARY To determine whether real-time two-dimensional echocardiography (2-D echo) can estimate the extent of myocardial involvement in patients with acute myocardial infarction (MI), regional wall motion on serial short-axis 2-D echo recordings was analyzed and the summed scores were compared with estimates of infarct involvement by thallium-201 reperfusion (Tl) and technetium-99m stannous pyrophosphate (99mTc-PYP) scintigraphy.

Thirty-two consecutive male patients admitted with their first MI were studied; 10 patients had anterior, 16 had inferior, and six had subendocardial MIs. Two patients were technically unsuitable for 2-D echo studies. Twenty patients had TI scintigrams and 29 had 99mTc-PYP scintigrams. Summed 2-D echo scores correlated closely with estimates of infarct involvement by Tl (r = 0.87) and with estimates of infarct size by 99mTc-PYP (r = 0.74). The location of MI by 2-D echo agreed with the electrocardiographic location in 26 of 29 patients; discrepancies occurred in one inferior and two subendocardial MIs. Predischarge 2-D echo failed to identify extension of transmural infarction. However, two patients whose subendocardial MIs progressed to transmural MIs were identified.

This study shows that 2-D echo is a valid method for the early estimation of the extent of myocardial involvement in patients with acute MI, especially transmural MIs. In particular, 2-D echo correlates closely with Tl reperfusion scintigraphy because both detect areas of ischemia and infarction.

THE AMOUNT of myocardium damaged by a myocardial infarction is a major determinant of the likelihood of pump failure and of serious arrhythmias after infarction.1–3 The involved area of muscle usually includes a zone of jeopardized ischemic myocardium that varies in size. Preventing the progression of this jeopardized tissue to infarction may limit the eventual size of the myocardial infarction.4–6 In the last few years, much attention has been given to developing techniques capable of accurately characterizing the site, extent and evolution of infarcted and ischemic myocardial injury. Methods such as precordial electrocardiographic mapping,7 serum creatine kinase release,8,9 myocardial scintigraphy10–12 and computerized tomography13 have been used singly and in combination to characterize and quantify the amount of jeopardized myocardium. Efforts continue to define ideal methods for characterizing and quantifying myocardial infarction for determining prognosis, subsequent extension and response to therapy.

Recently, two-dimensional echocardiography has been shown to correlate significantly with other estimates of infarct size in animals and to identify the site of infarction in patients.14–19 We undertook the present study to determine whether two-dimensional echocardiography can estimate the extent of myocardial involvement in patients with acute myocardial infarction. Comparisons were made with the surface 12-lead ECG, thallium-201 and technetium-99m stannous pyrophosphate (99mTc-PYP) scintigraphy.

Materials and Methods

Patients

Consecutive male patients admitted to the coronary care unit from June 1978 to February 1979 with their first myocardial infarction were studied. ECGs and serum enzymes were obtained on admission and for 5 consecutive days. Acute myocardial infarction was documented by a typical clinical history with the development of significant Q waves (transmural infarction) or ST-segment depression and/or T-wave inversion (subendocardial infarction) on the ECG, associated with elevation of at least two of the three serum enzymes (creatine kinase, glutamic oxaloacetic transaminase and lactic dehydrogenase) to above twice the limit of normal. Electrocardiographic location of infarction to the anterior, inferior and lateral walls was made according to the Minnesota code.20 Electrocardiographic assessment of involvement of the true posterior wall was made by the criteria of Perloff.21 Patients who had hospital documentation or electrocardiographic evidence of a previous myocardial infarction were excluded.

Thirty-two male patients were studied; 10 had anterior, 16 had inferior and six had subendocardial infarctions. The patients were 35–70 years old (mean...
55 years). Pulmonary rales or third heart sounds (Killip class II) were present in four patients. Myocardial infarct extension was diagnosed in nine patients. Extension was defined as recurrent chest pain associated with further electrocardiographic changes and reelevation of myocardial enzymes.

Echocardiograms

Initial two-dimensional echocardiographic studies were obtained 2–4 days after admission. Predischarge studies were obtained 10–18 days after admission, within 48 hours of the patient's hospital discharge. Echocardiograms were obtained with a wide-angle, phased-array ultrasonograph (Varian V3000, Palo Alto, California) using a 2.25-MHz transducer. Images were recorded on videotape for subsequent analysis. All patients were studied in the recumbent or shallow right anterior oblique position.

The transducer was always placed perpendicular to the chest wall, and the short axis of the left ventricle was scanned until its image had a circular or almost circular configuration, closely approximating the true short axis. Serial scans were obtained by moving the transducer head inferolaterally on the chest wall to avoid extreme transducer angulation that would produce an oval rather than a circular image of the left ventricle and render interpretation of wall asynery unreliable.

Figure 1 shows the four short-axis views of the left ventricle used for the analysis of wall motion, previously identified as positions IV, Va, Vb and VI by Kisslo et al. Thus, echocardiographic scans were recorded at the level of maximal motion of the mitral valve leaflets (position IV), the tips of the papillary muscles (position Va), the bodies of the papillary muscles (position Vb) and through the ventricular apex (position VI). These views permit the analysis of the anterolateral, posterolateral, apical, septal and inferior regions of the left ventricular wall.

Ventricular wall motion was graded in each of the wall regions in each view by a modification of the terminology of Herman and Gorlin: 0 = normal, 1 = hypokinesia, 2 = akinesia, 3 = dyskinesia. The graded scores of all regions in each echocardiographic view were totaled and the scores obtained from all views were summed for each patient. This total, expressed as a summed segment score, was the echocardiographic estimate of the myocardium involved in the infarction of each patient. Figures 2 and 3 are examples of echocardiograms from patients with acute anterior and inferolateral myocardial infarctions.

Echocardiographic recordings were analyzed by two authors without prior knowledge of the identity or clinical status of the patient. Interobserver variation was minimal; correlations between numbers of regions involved and summed abnormal wall motion scores by each observer were $r = 0.97$ and $r = 0.95$, respectively. When discrepancies occurred, blind evaluation by a third observer was obtained. Persistent discrepancies were resolved by consensus.

To determine the validity of the method, 10 patients who underwent cardiac catheterization for chest pain and who had normal contrast ventriculography and coronary arteriography had echocardiographic studies performed within 48 hours of catheterization. The echocardiographic summed segment scores were zero for each patient.

Myocardial Scintigraphy

Serial thallium-201 myocardial scintigraphy at rest was performed within 48 hours of admission in 20 patients, as previously described in our laboratory. Areas of decreased or absent myocardial perfusion

![Schematic diagram showing the location of the five segments of left ventricular wall identified on the two-dimensional echocardiographic studies in serial short-axis scan positions (adapted from Kisslo et al.). Positions IV, Va, Vb and VI represent the ventricular wall in short-axis views recorded as the level of maximal motion of the mitral valve leaflets, the tips of the papillary muscles, the bodies of the papillary muscles and the ventricular apex, respectively.](http://circ.ahajournals.org/doi/abs/10.1161/01.cir.31.4.1249?journalCode=circ)
were identified on the reperfusion scintigram. Their extent was estimated as a radian or angle (to within 15°) of the total left ventricle, in the manner of Henning et al.20

In 29 patients, 99mTc-PYP scintigraphy was performed 2–4 days after admission, and the intensity of pyrophosphate uptake was classified as described by Parkey et al.26 Images were obtained in the anterior, 45° left anterior oblique and left lateral positions. The scintigram showing the largest area of pyrophosphate uptake was selected and the area was determined by planimetry, as described by Willerson et al.20 Figure 4 shows the scintigraphic studies of the patient shown in figure 3 with an inferolateral infarction.

Scintigraphic images were read independently in random sequence by two of the authors without prior knowledge of the patient at the time of the study. When discrepancies occurred, the measurements were remade at random and persistent discrepancies were averaged.

Data Analysis

Summed scores of abnormal wall motion obtained from the initial echocardiograms of each patient were compared with the defect on the thallium-201 reperfusion scintigram and the area of uptake on the 99mTc-PYP scintigram. Linear regressions and correlation coefficients were obtained for each comparison.

Results

Echocardiographic–Scintigraphic Correlations

The echocardiographic estimates of myocardial involvement and the scintigraphic estimates of infarct size are listed in table 1. A significant linear correlation was found between summed echocardiographic segment scores from the initial studies and thallium-201 reperfusion scintigraphic defects (r = 0.87) (fig. 5). A significant linear correlation between the same summed echocardiographic scores and 99mTc-PYP
scintigraphic image area was also obtained \( (r = 0.74) \) (fig. 6). The anterolateral segment in at least one echocardiographic scan position could not be visualized in three patients. Because all three patients had inferior wall infarctions, alterations in the correlation of the echocardiographic and scintigraphic data are unlikely.

**Location of Infarction**

The initial echocardiographic location agreed with the electrocardiographic location of the myocardial infarction in 26 of 29 patients in whom comparisons could be made (table 1). Suitable echocardiographic studies could not be obtained in two patients and one (patient 29) had a left bundle branch block morphology. Two of the three patients (nos. 2 and 4) in whom discrepancies were present sustained subendocardial infarctions and no wall motion abnormalities were identified by echocardiogram; the third, patient 22, whose inferior myocardial infarction on the ECG was confirmed by both thallium-201 and 99mTc-PYP scintigraphy, had abnormal anterolateral and apical wall motion identified by echocardiography.

Discrepancies between the initial echocardiographic and thallium-201 scintigraphic location of myocardial infarction were present in three of 29 patients, including patient 22. Patient 4, who had a subendocardial infarction, had no echocardiographically detectable wall motion abnormalities. Patient 23, whose perfusion defects on thallium-201 scintigraphy were located on the posterolateral wall, had abnormal anterolateral wall motion detected by echocardiogram.

Discrepancies between the initial echocardiographic

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**Figure 4.** Serial thallium-201 (201TI) and technetium-99m stannous pyrophosphate (Tc-PYP) scintigrams from the patient shown in figure 3 with an acute inferolateral myocardial infarction.

**Figure 5.** Comparison between echocardiographically estimated summed segment scores and thallium-201 reperfusion scintigraphic image size in 19 patients.

**Figure 6.** Comparison between echocardiographically estimated summed segment scores and technetium-99m stannous pyrophosphate scintigraphic image size in 21 patients with transmural myocardial infarction (MI).
### Table 1. Clinical, Scintigraphic and Echocardiographic Data

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Upper limit of normal laboratory values: CK = 160 IU/l, GOT = 30 IU/l, LDH = 220 IU/l.

*Patients with myocardial infarct extension.

Abbreviations: CK = creatine kinase; GOT = glutamic oxaloacetic transaminase; LDH = lactic dehydrogenase; TI-201 = thallium-201 reperfusion scintigram; PYP = technetium-99m stannous pyrophosphate scintigram; SEMI = subendocardial infarction; A = anterior; I = inferior; L = lateral; P = posterior; S = septal; Ap = apical; LBBB = left bundle branch block.
and \(^{99m}\text{Tc-PYP}\) scintigraphic location of myocardial infarction were present in six of 28 patients, including patient 22. Patients 2 and 4, who had subendocardial infarctions, had no wall motion abnormalities identified by echocardiogram. Patient 1 had a negative \(^{99m}\text{Tc-PYP}\) scintigram, but anterolateral and apical wall motion were abnormal by echocardiogram. Patients 24 and 28, whose infarct location by ECG and echocardiogram agreed, had \(^{99m}\text{Tc-PYP}\) scintigraphic location of the infarct at a different site.

Comparison of Initial and Predischarge Echocardiograms

The summed echocardiographic scores for the 25 patients who had predischarge echocardiograms are shown in table 1. Comparative data from 24 patients who had both initial and predischarge studies are shown in figure 7. Although the initial and predischarge studies varied widely, mean values tended to be lower in predischarge studies. No significant differences were found between the summed segment scores on initial and predischarge studies in patients with anterior (9.4 ± 1.0 vs 8.3 ± 8), inferior (4.8 ± 0.5 vs 3.7 ± 0.7) and subendocardial (2.0 ± 0.9 vs 3.2 ± 1.2) infarctions.

Myocardial Infarct Extensions

Nine patients (28%) with myocardial infarct extension are identified in table 1. Comparative initial and predischarge echocardiographic data from eight of the nine patients are shown in figure 7. Infarct extensions in four patients with anterior and two patients with inferior infarctions were judged to have occurred in the same location as the initial infarction. Predischarge echocardiographic studies in these patients failed to show any further wall motion abnormalities in either the initial segments involved or other segments. In patients 2 and 31, subendocardial infarction progressed to transmural infarction. In both cases, the summed segment score on the predischarge study was increased.

Adequacy of Echocardiograms

Two patients could not be evaluated echocardiographically owing to chronic obstructive lung disease. In patients 2, 7 and 16 the initial echocardiographic studies were incomplete because the anterolateral segment in at least one scan position was not visualized. Patient 2 had a subendocardial infarction, whereas the other two patients had inferior myocardial infarctions.

Three of the 25 patients who had predischarge echocardiograms had incomplete studies. The anterolateral segment in at least one position was not visualized in patients 7, 15 and 19. The summed echocardiographic scores in the predischarge study of patients 7 and 19 may have been affected by the inadequate study; these patients had abnormal wall motion on their initial studies in segments not seen on subsequent studies. The other patient had an inferior infarction.

Discussion

The results of this study show that two-dimensional echocardiography is a valid method for estimating the extent of myocardial involvement in patients early after acute myocardial infarction. The accuracy with which the echocardiogram located the infarction site concurred with previous reports. Extension of myocardial infarction was not detected in this series of patients, except in two patients whose infarctions progressed from subendocardial to transmural.

The value of real-time two-dimensional echocardiography in visualizing abnormal left ventricular wall motion has been examined previously in patients with chronic ischemic heart disease. Weyman et al.\(^{27}\) confirmed the presence and location of an angiographically proved left ventricular aneurysm in 31 patients. Kisslo et al.\(^{28}\) compared echocardiographic and angiographic techniques in 105 patients with segmental wall motion abnormalities. The echocardiogram was able to identify wall motion characteristics in 87% of the wall regions examined.

![Figure 7. Summed echocardiographic segment scores from initial (1) and predischarge (2) echocardiograms in 24 patients. Patients with infarct extension are identified by open circles. ANT = anterior infarction; INF = inferior infarction; SEMI = subendocardial infarction.](http://circ.ahajournals.org/)}
More recently, two-dimensional echocardiographic studies of patients with acute myocardial infarctions have shown that the technique can accurately locate the site of infarction, although sites of old infarcts identified by ECG were not localized as accurately as those of new infarctions.16-19 Myocardial infarct expansion detected by echocardiogram has been shown to correlate with in-hospital mortality.28 Our studies have confirmed that two-dimensional echocardiographic studies can accurately locate the site of a myocardial infarction and in particular that of a transmural infarction.

The use of 99mTc-PYP and thallium-201 scintigraphic techniques in the estimation of infarct size has been well documented in both animal and human studies.10-12, 29-31 Human studies in our laboratory have shown that the thallium-201 reperfusion scintigram obtained several hours after the initial study is a more accurate estimate of infarct size than the initial study.25 For this reason, echocardiographic estimates were compared with thallium-201 reperfusion scintigrams in our present study.

Animal studies have shown a close correlation between echocardiographically detected segmental wall motion abnormalities and experimentally induced infarct size estimated by 99mTc-PYP scintigraphy.17, 18 Our studies extend these observations to patients by showing that two-dimensional echocardiographic estimates of myocardial involvement in myocardial infarction correlate closely with scintigraphic estimates of involved myocardium. The better correlation of the echocardiographic estimates with thallium-201 scintigraphy than with 99mTc-PYP scintigraphy in our study is probably due to the difference in the respective mechanisms of isotope concentration in the heart. 99mTc-PYP accumulates primarily in irreversibly damaged myocardium,32 whereas thallium-201 concentrates in normal myocardium and closely reflects regional myocardial perfusion.33 Consequently, acutely infarcted myocardium, scarring from old infarction and areas of myocardial ischemia are represented as perfusion defects by thallium-201 scintigraphy. The echocardiogram can identify and assess wall motion abnormalities produced by both ischemia and infarction.34, 35 Thus, one would anticipate a better correlation between two techniques that identify areas of ischemia as well as infarction.

Another possible reason for the better correlation between echocardiographic and thallium-201 estimates is the larger number of inferior wall infarcts in our patient group. Estimations of infarct size by 99mTc-PYP scintigraphy have proved more accurate with anterior and lateral wall myocardial infarctions than with inferior wall infarctions.10, 12

Nine patients (28%) had myocardial infarction extension recognized by further clinical, electrocardiographic and enzymatic changes. This may be an underestimate of the true number of patients with extension of infarction.36-38 Only patients 2 and 31, whose infarctions progressed from subendocardial to transmural, had an increase in their summed echocardiographic scores on their predischarge study. If the area of ventricular wall involved in an infarct extension were confined to the original echocardiographic segment, these findings would be anticipated. In our study, the identification of infarct extension by real-time two-dimensional echocardiography was relatively insensitive. However, further patient studies with more frequent echocardiographic examinations are necessary to confirm or refute this observation.

The determination of whether patients in our study were suffering their first myocardial infarction was done as carefully as possible. Previous infarctions causing abnormal ventricular wall motion may have escaped detection by conventional clinical and electrocardiographic means. However, inclusion of such patients would not have improved and may well have worsened the correlations between echocardiographic and scintigraphic estimates of acute infarct involvement.

The use of results obtained by subjective evaluation in a comparative statistical analysis should be viewed with caution. Nevertheless, the correlations between the subjectively evaluated echocardiographic scores and both scintigraphic estimates of infarct size were statistically significant (p < 0.05).

In conclusion, real-time two-dimensional echocardiography appears to be a valid method of estimating the area of myocardial involvement in patients soon after their first myocardial infarction. Further, this technique promises to be useful in evaluating interventions to limit infarct size in patients. This role has already been defined in animal experiments where alterations in experimentally induced infarct size by nitroglycerin and phentylephrine have been accurately quantified.17, 18

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