Correlation between echocardiographic endocardial surface mapping of abnormal wall motion and pathologic infarct size in autopsied hearts

GERARD T. WILKINS, M.B., Ch.B., JAMES F. SOUTHERN, M.D., Ph.D., CHRISTOPHER Y. CHOONG, M.B. B. CHIR., Ph.D., JAMES D. THOMAS, M.D., JOHN T. FALLON, M.D., Ph.D., DAVID E. GUYER, M.D., AND ARTHUR E. WEYMAN, M.D.

ABSTRACT We previously developed a cross-sectional echocardiographic technique for quantitatively mapping the endocardial surface of the left ventricle and on which regions of abnormal wall motion can be superimposed in their correct spatial distribution. This endocardial mapping technique (EMT) provides a measure of the left ventricular endocardial surface area (ESA in cm²), the area of abnormal wall motion (AWM in cm²), and the overall percent dysfunction (%AWM) as a measure of the functional “infarct size.” To test this approach, we compared the EMT measurements with the actual endocardial surface area (in cm²) and pathologic infarct size (both percent infarct by volume and percent endocardial surface overlying infarct) measured at later autopsy in 20 adults (14 men, six women) ranging in age from 47 to 76 years (mean 64 ± 9.6 years). The median interval from echocardiographic study to death was 19 days (range 1 to 269 days). Patients were divided into two groups based on the age of their infracts at the time of death: (1) recent (infarct age < 14 days; mean age 5.3 ± 4.6 days) and (2) old (infarct age > 6 months; mean age 3.6 ± 3 years). When the left ventricular endocardial surface area at autopsy was compared with the EMT-derived ESA, a close correlation was found (EMT area = 1.17 × autopsy area + 20.4; r = .94, p = .0001), with the systematic difference in the measurements accounted for by systolic arrest, loss of distending pressure, and specimen shrinkage. The echocardiographic measure of infarct size (%AWM) correlated well with the autopsy percent infarction by volume (%AWM = 1.1 × infarct volume + 5.5; r = .82, p = .0001). Similarly, a good correlation was found for the percent abnormal wall motion and the autopsy percent endocardial surface area overlying infarction (%AWM = 0.89 × infarct area - 0.9; r = .89, p = .0001). When the data were examined in relation to the age of the myocardial infarct, the echocardiographic %AWM appeared to overestimate the autopsy infarct size (by percent infarct volume) in the recent infarct group (n = 6), and underestimate the extent in the old infarct group (n = 13). The findings suggest that the EMT will provide a useful quantitative measure of left ventricular endocardial surface area and the extent of ischemic/infarct-related dysfunction.


WITH INCREASING INTEREST in myocardial salvage after presumed acute coronary occlusion, an accurate, repeatable measure of the amount of left ventricle rendered dysfunctional by the ischemic process is needed.¹ Ideally, such a method should be quantitatively accurate, noninvasive, and easily repeatable so that comparisons through time and between subjects can be made. Any acceptable functional measure of “infarct size” should also demonstrate reasonable agreement with the extent of pathologic change. We previously developed a quantitative echocardiographic method for mapping the left ventricular endocardial surface on which regions of abnormal wall motion can be superimposed and measured.² ³ Although this method has been experimentally validated in a canine preparation of infarction, the relationship of the mapped area of dysfunction to the pathologic infarct size in humans has not been examined.

Methods

Study group. The study group was drawn from a larger consecutive series of patients who were autopsied at the Mas-
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sachusetts General Hospital as part of an ongoing coronary artery disease research protocol (Ischemic SCOR). From 1984 to 1987, 90 patients were studied in this series. On review, it was found that a cross-sectional echocardiographic study had been performed for a clinical indication in 39 of these patients within the year before death. Nineteen of these patients were excluded from further correlative study either because they suffered an additional definite myocardial infarction (diagnosed clinically) between the time of echocardiography and autopsy (n = 17) or because they had an unsuitable echocardiogram (n = 2). In the latter cases, the echocardiographic images were technically inadequate such that the endocardial border could not be visualized in all five imaging planes necessary to produce a wall motion map. Most of those patients (13 of 17) who sustained additional infarcts after the time of echocardiography did so in relation to some procedure such as cardiac surgery or percutaneous transluminal angioplasty.

The study group therefore consisted of 20 patients (14 men, six women) ranging in age from 47 to 76 years (mean age 69 ± 9.6 years). Each patient had at least one myocardial infarction documented clinically before echocardiographic study and death, and all had evidence of coronary artery disease at autopsy. In those patients (table 1) in whom coronary artery surgery was performed before death, no evidence of a further episode of infarction related to the procedure was found at autopsy. The study patients were divided into two categories: (1) patients dying within 2 weeks of their acute myocardial infarction (i.e.,

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>Intervals between myocardial infarction, echocardiography, and death, with cause of death</th>
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<tr>
<td>Patient</td>
<td>Interval</td>
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</tr>
<tr>
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<td>2</td>
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Intervals are given between myocardial infarction and death (MI = death), and echocardiographic study and death (echo = death).

GI = gastrointestinal; CABG = coronary artery bypass grafting; CHF = congestive heart failure; VT = ventricular tachycardia; SLE = systemic lupus erythematosus; VF = ventricular fibrillation; CVA = cerebrovascular accident; Ca = carcinoma; COPD = chronic obstructive pulmonary disease; Rupt PM = rupture of papillary muscle with acute infarction; ATN = acute renal tubular necrosis.

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Results in each study patient</th>
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<tbody>
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<td>Patient</td>
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Mean 156 116 43 36 50
SD 99 56 27 21 27

Individual results for each study patient, including classification of the age of the myocardial infarct in the time of death (R = recent, less than 14 days; O = old, greater than 6 months).

ESAmap = endocardial surface area (in cm²) from the echocardiographic mapping technique; ESAut = endocardial surface area measured at autopsy (in cm²); %AWM = percent abnormal wall motion from the echocardiographic mapping technique; %volume = percent myocardial infarction by volume at autopsy examination; %area = percent endocardial surface area overlying infarction at autopsy.

Six patients were placed in the recent infarction group. The cause of death is listed in table 1 for each patient along with the interval between infarction and death and between echocardiography and death.

Six patients were placed in the recent infarction group. The interval between infarction and death for this group ranged from 1 to 13 days (mean interval 5.3 ± 4.6 days). Similarly, the interval between echocardiography and death was short, ranging from 1 to 4 days (mean interval 1.7 ± 1.2 days). Most patients in this group (n = 5) died of cardiogenic shock and one died of a fatal cerebrovascular event after coronary artery surgery.

Thirteen patients were placed in the old infarction group. The interval between infarction and death for this group ranged from 6 months to 12 years (mean interval 3.6 ± 3 years). The interval between echocardiographic study and death was longer in this group, ranging from 1 to 269 days (median 41, mean 69 ± 88 days).

One patient could not be placed in the recent or old infarction group (No. 19; tables 1 and 2). This patient, with two previous percutaneous transluminal coronary angioplasties for angina, died 7 months after a presumptive clinical myocardial infarction diagnosed on the basis of prolonged chest pain and persistent ST-T wave changes on the electrocardiogram (ECG). On admission several days later, cardiac enzyme levels were normal. The echocardiographic study performed 68 days before death from

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metastatic colon carcinoma was normal. At autopsy no evidence of infarction (scar) could be found, although evidence of pulmonary embolism (pulmonary infarction) of similar age to the presumed myocardial infarct was evident.

Cross-sectional echocardiography

Data acquisition. All patients underwent cross-sectional echocardiographic studies performed using either an ATL mk300, ATL mk600 mechanical sector scanner or Hewlett-Packard 77020A phased-array scanner. All studies were recorded on 1/2 inch VHS tape. Patients were examined supine, generally in the left lateral decubitus position, where five standard echocardiographic imaging planes were obtained. From the parasternal transducer position, short-axis views were recorded at the level of the mitral valve, the midportion of the papillary muscles, and at the apex. Care was taken that the views were perpendicular to the long axis of the heart and without distortion due to incorrect angulation or rotation. The transducer was then moved to the cardiac apex where the apical four-chamber and apical two-chamber views were recorded. In each case, care was taken to maximize the long-axis length of the left ventricle. The orientation and intersection of these left ventricular imaging planes is displayed diagrammatically in figure 1, A. Images were considered suitable for analysis when they were correctly aligned and the endocardial border could be seen around the whole perimeter of the view throughout the cardiac cycle.

Data analysis. Suitable diastolic images from these five planes were then used to derive cardiac dimensional and wall motion measurements for the endocardial mapping technique (EMT). This technique has been previously reported by Guyer et al. The process of data measurement and map construction proceeded as follows.

**FIGURE 1.** A, Relative positions of echocardiographic imaging planes shown as they intersect the left ventricle. Ap 4CH = apical four-chamber plane; Ap 2CH = apical two-chamber plane; S_{a-MV} = mitral valve short-axis plane; S_{a-PM} = papillary muscle short-axis plane; S_{a-APEX} = apical short-axis plane. B, The procedure for plotting an endocardial map from echocardiographic data. A schematic diagram of the left ventricular endocardial surface has been quadrisected by two perpendicular apical imaging planes. The transverse lines represent the positions at which short-axis planes would intersect the endocardial surface. S_{A-L-L-S} values represent the endocardial apex-to-base segment lengths. C, A representative endocardial map resulting from flattening of the four quadrants of the endocardial surface, as shown in B. The ventricular long-axis dimension is used as the map quadrant midline (M) value. Short-axis data are then plotted at their correct positions (shown) along the quadrant midlines. The apex-to-base segment lengths are then fitted as smooth curves between the ends of the short-axis arms and the apex points of each quadrant. S_{A} = anterior wall endocardial segment length; S_{I} = inferior wall endocardial segment length; S_{L} = lateral wall endocardial segment length; S_{S} = septal endocardial segment length; M = map quadrant midline dimension; C_{MV} = endocardial short-axis circumference at the mitral valve; C_{PM} = circumference at midpapillary muscle valve; C_{AP} = circumference at apical level. (Illustration adapted from Guyer et al., with permission).
Correctly aligned short-axis views from the mitral valve plane, midpapillary muscle plane, and the cardiac apex were transferred, in turn, to the videodisk as before. At end-diastole, the circumference of the endocardial surface was measured. At the mitral and apical levels the endocardial surface was followed exactly. However, at the midpapillary muscle level a smooth curve was continued through the bases of the papillary muscles. In each short-axis view, the region of abnormal wall motion, if present, was identified and the length of this segment recorded. A segment length was then measured from the start point of abnormal wall motion to an endocardial landmark within each plane. At the mitral valve level the landmark was taken as the posteromedial commissure of the mitral valve, at the midpapillary muscle level it was the midpoint of the posteromedial papillary muscle, and at the apical level it was the point on the endocardial surface opposite the posterior interventricular groove.

For each of the five planes, all dimensions were determined from three separate frames, and the results for each dimension were averaged to obtain a single value.

This procedure produced two basic data sets: (1) Endocardial dimensions of the left ventricle. (2) The endocardial extent, as a segment length, of abnormal wall motion in each view and the position on the endocardial surface of the abnormally moving region relative to an internal landmark.

CONSTRUCTION OF ENDOCARDIAL SURFACE MAPS FROM ECHOCARDIOGRAPHIC MEASUREMENTS. These data were entered into an algorithm we have recently developed for producing planar maps of the left ventricular surface. This mapping technique has been shown to be quantitatively accurate in a series of excised canine ventricles and further validated mathematically. \(^2\) \(^3\) The endocardial surface mapping technique considers the left ventricular endocardial surface to be a thin membrane that, when quadrisection by apex-to-base cuts oriented like the apical echocardiographic imaging planes, yields four sections that can be laid flat with a minimum of distortion to form a map (figure 1, B). This is analogous to the cartographers procedure for making a planar map of the terrestrial globe (Cartesian projection). To actually construct endocardial maps from the echocardiographic measurements, an experimentally validated algorithm was used. The algorithm first puts the midline dimension of each map quadrant, \(M\), on each of the four axes of a standard cartesian coordinate system, as shown in figure 1, C. The central ventricular LAX is taken as the initial value for this \(M\) dimension in the first iteration of the mapping procedure. Next, straight line segments equal in length to one-quarter of the endocardial circumferences (from the short-axis views) were plotted on each of the quadrant midlines at predefined positions along the axes. The algorithm assumes that the apical imaging plane lies one-sixth of the way from the apex to the base of the heart, the midpapillary muscle plane is half the way, and the mitral valve plane five-sixths of the way from the apex to base in an undistorted heart. In addition, it assumes that the endocardial circumference at the base of the heart is essentially the same as the circumference measured at the mitral valve level. These straight line segments, plotted at the appropriate positions along and bisected by the quadrant midlines, form the skeleton for the endocardial maps, as shown in figure 1, C. End points of the short-axis arms and the apex points of each quadrant are then connected by a parabolic arc-fitting program.

These curves (\(SL_{a,b,L}\), figure 1, B), which have been added diagrammatically to the map skeleton in figure 3, B, represent the endocardial apex-to-base segments of the ventricle. Their computed lengths were then compared with the corresponding segment lengths actually measured from the echocardiographic images in the two apical projections. If the map endocardial segment lengths were within a present tolerance of the echocardiographic values, the map was considered complete. If not, however, the \(M\) dimension of the map was adjusted and the entire mapping procedure repeated iteratively with new \(M\) values until the computed and the measured segment lengths fell within the accepted tolerance. In its present form, the algorithm adjusts \(M\) by increasing or decreasing its value in succeeding iterations by the difference between measured (echocardiographic) and map endocardial segment lengths:

\[
M_{i+1} = M_i + (SL\text{ echo } SL[i])
\]

where the index \([i]\) denotes the dimension value from the \(i\)th map iteration, and where \(M_1 = LAX\).

With this technique of adjusting the midline dimension, \(M\), the map endocardial segment lengths rapidly converged to the echocardiographically measured values. It was never necessary for the mapping algorithm to perform more than two iterations to meet the tolerance specification, which was set at 5%. Once the maps were completed, the endocardial surface area of the left ventricle was calculated as the sum of the surface area of the four map quadrants.

MAPPING REGIONAL DYSSYNERGY. As noted above, areas of abnormal endocardial excursion were measured and defined in relation to internal cardiac landmarks. These measurements were then transferred directly onto the endocardial surface map. The end points of the abnormal segments were then connected by straight line segments by the mapping algorithm and the enclosed area was taken to represent the region of abnormal wall motion. The percentage of the endocardial surface area that moved abnormally was calculated as the ratio:

\[
\frac{\text{Abnormally moving area}}{\text{Total endocardial area}} \times 100
\]

An example of the output of the mapping algorithm with input dimensions and derived areas is shown in figure 2. The analysis

**FIGURE 2.** Representative echocardiographic endocardial surface map from patient 11 (tables 1 and 2). The four quadrants are displayed and the region of abnormal wall motion is mapped in its correct spatial distribution (shaded area). The orientation of the septal, anterior, lateral, and inferior apex-to-base axes are displayed. The numeric data in the upper corner include the initial map input dimensions (abbreviations as in figure 3), the calculated map endocardial surface area (ESA, in cm²), the map of abnormal wall motion (AWM, in cm²), and the percent abnormality (%AWM).
sequence therefore provided: cardiac dimensions derived from five standard imaging planes, a measure of total endocardial surface area of the left ventricle, a measure of the total endocardial surface area overlying the area of abnormal wall motion (if present), and the percent of the endocardial surface moving abnormally as a measure of the functional infarct size.

**Cardiac pathology.** Each patient underwent a detailed cardiac pathologic examination. Autopsies were performed at a mean of 13 ± 5 hr after death (range 5 to 20 hr). At the time of autopsy, the heart was removed from its attachments and weighed and adherent clots were removed. The coronary ostia were cannulated and perfused under pressure with barium gelatin, with the use of monastral blue or monastra red for the left and right coronary arteries, respectively. The heart chambers were distended with rubber gloves and immersed in 5% formalin solution for fixation over 48 hr. This resulted in a uniform distending pressure of approximately 10 mm Hg. Subsequently the intact hearts were x-rayed and then sectioned transversely at approximately 1 cm intervals along their long axes (figure 3, A). Heart weights increased less than 3% after injection and fixation.

The state of the entire myocardium was evaluated by gross and selected microscopic examination. Gross areas of fresh infarction were identified by edema, congestion, interstitial hemorrhage, and marginal resorption zones. Areas of previous infarction were identified by the presence of fibrous scar and myocardial thinning. A complete cross section through the region of maximal infarct or scar was examined histologically. Other areas of questionable infarct border were also examined histologically to better define the extent of infarct or scarring with a Masson trichrome stain. Interobserver variation for infarct area was 5%. The surface area of each slice was then traced and the exact distribution of new myocardial infarction and old scar was shaded on the outline. This process was repeated for each slice from apex to base. The remaining basal slice, still connected to the other cardiac structures, was also traced as above and its height was measured. The extent of anatomic myocardial infarction was then calculated in two ways for each heart obtained at autopsy.

**Percent myocardial infarction by volume.** The total volume of each slice was calculated as follows. The surface area of the slice was calculated by electronic planimetry of each tracing and the volume was calculated by multiplying this slice area by its height. The total tissue volume was then calculated as the sum of all the slices (including the base). The volume of infarcted tissue was obtained in a similar manner—by planimetering the infarcted surface area and multiplying by the slice height (figure 3, B). The percent infarction was then calculated as:

\[
\text{Percent infarction} = \left( \frac{\text{Infarct volume}}{\text{Total volume}} \right) \times 100
\]

**Percent endocardial surface area overlying infarction.** The total endocardial surface area was calculated as follows. The endocardial circumference of each slice was measured by electronically tracing the endocardial surface border. The endocardial surface area for each slice was then calculated by multiplying each circumference by the individual height of each slice (figure 3, B). The total autopsy endocardial surface area of each heart was then calculated as the sum of surface areas for each slice (including the base). This measurement was later used in a direct comparison of the surface area at autopsy vs the echocardiographic surface area (vide below). The area overlying regions of myocardial infarct was derived in a similar way. The margins of anatomic/histologic infarction were marked on the tracing of each slice. A line was then drawn from these points (passing through an imaginary left ventricular center point) so that it crossed the endocardial surface area perpendicularly. The segment length(s) so defined were measured and the surface area of each slice was calculated by multiplying by the height of each individual slice. The area overlying infarction for the entire ventricle was then calculated as the sum of areas for each slice (including the base). The percent surface area overlying infarction was calculated as:

\[
\text{Percent surface area overlying infarction} = \left( \frac{\text{Endocardial surface area overlying infarction}}{\text{Total endocardial surface area}} \right) \times 100
\]

**Statistical analysis.** Since the pathologic and echocardiographic methods used both produced a measure of percent ventricle affected, we correlated these measures directly by least squares linear regression. The direct comparison of echocardiographic endocardial surface area (cm²) and that at autopsy (cm²) was also performed by linear regression. Values for the means of variables are given ± 1 SD. Interobserver variability was calculated as the mean of the unsigned differences (as a percent of the overall mean) between two sets of measurements performed by two experienced observers. Intraobserver variability was calculated in a similar

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**FIGURE 3.** A, Schematic diagram showing how the formalin-fixed left ventricle was sliced transversely for quantification of the infarct size and the pathologic endocardial surface area. B, Methods of determination of the pathologic extent of infarction by calculating the percent fraction of the endocardial surface overlying infarction and the percent fraction of the myocardial volume infarcted. (Illustration reprinted from Guler et al., with permission.)
manner with measurements performed by the same observer on the same cases separated by 24 hr.

**Results**

Individual results derived from the echocardiographic and autopsy examinations are given in table 2.

The mean endocardial surface area by the echocardiographic EMT was 156 ± 69 cm² and ranged from 96 to 426 cm². The mean area of abnormal wall motion was 73 ± 69 cm² (range 0 to 309 cm²). The mean percent abnormal wall motion was 43 ± 27% and ranged from 0% to 86%.

From the autopsy examination, the mean left ventricular endocardial surface area was 116 ± 56 cm² and ranged from 64 to 320 cm². The mean percent myocardial infarction by tissue volume was 36 ± 21% and ranged from 0% to 69%. The mean percent endocardial surface area overlying infarction from the autopsy hearts was 50 ± 27% and ranged from 0% to 100%.

Interobserver and intraobserver variabilities for the EMT-derived endocardial surface area and percent abnormal wall motion were calculated in five randomly selected cases. Mean percent intraobserver variability for endocardial surface area was 6 ± 3% and that for percent abnormal wall motion 4.4 ± 4%. Similarly, mean percent intraobserver variability was 5 ± 3% for endocardial surface area and 4 ± 3% for percent abnormal wall motion.

**Comparison of the echocardiographic endocardial surface area with that at autopsy.** A direct comparison of the endocardial surface area (in cm²) measured by the echocardiographic EMT and that measured at autopsy in the 20 patients demonstrated a close correlation (EMT area = 1.17 × autopsy area + 20.2; r = .94, p = .0001, SD of regression = 25) (figure 4). Of note, the correlation remained strong with the removal of the data from the largest aneurysmal heart from this comparison (r = .72, p = .001).

**Comparison of percent abnormal wall motion with the percent infarction by volume determined at autopsy.** A comparison of the EMT-derived percent abnormal wall motion with the percent myocardial infarction by volume at autopsy demonstrated a close relationship (percent abnormal wall motion = 1.1 × infarct volume + 5.5; r = .82, p = .0001, SD of regression = 15.8, n = 20) (figure 5).

When the data points above and below the regression line were examined, it was noted that the six patients with recent myocardial infarction (denoted by circles, figure 5), tended to cluster above the line. When a correlation was sought with data from those six patients with recent myocardial infarction only, the slope increased from 1.1 to 1.25 and the intercept moved from 5.5 to 13. Conversely, when infarct volume and percent abnormal wall motion were compared in the remaining 13 patients with old infarction (figure 5), the slope fell from 1.1 to 0.93.

**Comparison of percent abnormal wall motion with the percent endocardial surface area overlying infarction determined at autopsy.** When the EMT percent abnormal...
When data from the six patients in the recent infarct group (figure 6) were analyzed separately, the resulting regression equation was similar to that for the whole group. Similarly, comparison of surface overlying infarction with percent abnormal wall motion in those 13 patients with old infarcts (figure 6) produced a
Discussion

This echocardiographic-autopsy study had a dual purpose: (1) to validate the use of the endocardial surface mapping technique in man, and (2) to investigate the size of the echocardiographic functional infarct relative to the pathologic infarct.

Although this algorithm has been previously validated\(^2\), \(^3\) in computer simulations and in canine experiments, it was designed for use in clinical studies and therefore its further validation in human hearts was essential. It should be noted that other approaches to mapping the endocardial surface area of the ventricle have been reported by ourselves\(^5\), \(^6\) and others,\(^7\)-\(^12\) but only one has quantitated an endocardial area\(^6\) and none have previously been validated in a human autopsy series.

The technique of endocardial surface mapping used in this investigation is based on a quantitative reconstruction of the left ventricular endocardial surface from multiple intersecting echocardiographic planes. The area of abnormal wall motion is superimposed onto this three-dimensional surface, and thus the accuracy of the percent dysfunction is dependent on the quantitative accuracy of the overall surface area. The comparison between mapped endocardial surface area and autopsy area is therefore pivotal to this study. The endocardial surface area measured by the echocardiographic map showed an excellent agreement with the endocardial surface area directly measured at autopsy (\(r = .94\), \(p = .0001\)). As expected, the correlation (figure 4) demonstrated a significant deviation from the line of identity (map endocardial surface area = 1.17 \(\times\) autopsy area + 20.2).

This difference can be viewed as an overestimation by the echocardiographic method or an underestimation of the true surface area of the beating heart by the autopsy measurements. Several factors could lead to systematic underestimation of the dimensions in vivo by the autopsy measurements: (1) The majority of human hearts probably arrest in systole, while the echocardiographic maps were obtained in diastole. Indeed, diastolic arrest is only accomplished experimentally by the infusion of potassium chloride.\(^13\) (2) After death there is a loss of the normal physiological distending pressure in the left ventricle. (3) Fixation of hearts obtained at autopsy leads to significant shrinkage.\(^14\) These factors alone probably account for the consistent difference in the two sets of measurements. A systematic overestimation by the echocardiographic method could result from: (1) Inaccurate tracing of the endocardial borders from the left ventricular images, or incorrect plane location. However, it should be expected that such errors would be random, resulting in both over- and underestimations, rather than consistent overestimation. (2) The algorithm, in the process of fitting the two-dimensional measurements into a three-dimensional planar surface, may lead to a systematic overestimation. This did not appear to occur in extensive theoretical testing of a range of ventricular shapes and geometries.\(^2\)

When the extent of abnormal wall motion was derived by superimposing the regions of dysfunction onto this planar map and then comparison with the pathologic extent of infarction, a strong relationship was demonstrated. This correlation held whether the percent abnormal wall motion was compared with the percent volume of myocardial infarction (\(r = .82\), \(p = .0001\)) or to the percent surface area overlying myocardial infarction (\(r = .88\), \(p = .0001\)). The results of this investigation therefore agree closely with the original experimental validation of this technique in a canine preparation.\(^2\), \(^3\)

A direct comparison with the percent volume infarcted demonstrates a slope near 1 (1.1) and a small positive intercept (5.5), suggesting a small but consistent overestimation of the extent of pathologic infarction by the echocardiographic method (figure 5). An imperfect comparison of the functional expression of a myocardial infarction and its underlying histology should be expected due to the complex interrelationship of loading conditions, sympathetic tone, and transmural extent of necrosis.\(^15\) Nonetheless, the overestimation demonstrated here is consistent with results from previous experimental echocardiographic\(^5\), \(^15\), \(^16\) and sonomicrometer studies,\(^17\) and with human echocardiographic\(^18\) and angiographic studies.\(^19\) This phenomenon may be related to either borderzone ischemic dysfunction without infarction or to mechanical tethering at the margins of the histologically infarcted zone.\(^20\), \(^21\) In part it may also reflect the conceptual tendency, in the determination of infarct size measured by volume, to consideration of the infarcted myocardium as a single zone of maximal thickness, rather than a more common patchy, inhomogeneous region with transmural and subendocardial components. Conversely, the correlation of extent surface overlying infarction from the autopsy examinations with the echocardiographic extent of abnormal wall motion suggested a small underestimation by the mapping method. A similar result was found experimentally for this measure of autopsy infarct extent,\(^3\) and it appears
to result from the increased weighting given to the percent of histologic infarction by extensive subendocardial necrosis. In this regard, the slope of the correlation (figure 6) is significantly influenced by data from patient 1 (table 1), in whom a moderately sized infarct by volume (51.8%), with a small transmural and complete subendocardial component, was represented as 100% by area overlying infarction.

Another possible cause for a difference between echocardiographic and pathologic data might be the change in infarct size (expansion and remodelling) that can occur during transition from the acute to the chronic stage of evolving infarction. However, since this was not a natural history study in which patients were examined during acute infarction and followed to death, but rather a study of separate groups of patients with acute (studied within hours or days of death) and chronic segmental dysfunction (first studied ≥6 months after infarction), significant ventricular expansion or remodelling between the time of echocardiographic study and death would not have been expected.

Previous echocardiographic-autopsy studies in man have dealt with a nonquantitative segmental or single-plane comparison, both of which are limited in their ability to sum the percent dysfunction for the entire ventricle. Heger et al. demonstrated an agreement of infarct area with the segment(s) of abnormal wall motion in four autopsied patients. Weiss et al., in the only other study of this type, examined 15 postinfection hearts by a semiquantitative five segment approach. They found a satisfactory agreement with respect to site and an apparent overestimation of the pathologic extent of infarction by the five segment echocardiographic model. In an attempt to provide a more quantitative measure, they also assessed the circumferential extent of regional akinesis/dyskinesis in a limited number of short-axis planes (24 cross sections in 11 patients) from patients with suitable echocardiographic studies. Thus, although the method precluded an echocardiographic measure of the infarct size, they were only able to quantitatively examine this relationship in selected planes. Their data also suggests an overestimation by the echocardiographic method (although the slope alone suggests an underestimation, the majority of points in the lower range fell below the line of identity).

The experimental literature, however, suggests that this overestimation is related to the age of the infarct. Gillam et al. demonstrated that the extent of abnormal wall motion, measured at a single midventricular plane in the canine ventricle, overestimated the histochec-
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