Computer analysis of cross sectional echocardiogram for quantitative evaluation of left ventricular asynergy in myocardial infarction

JUNICHI FUJII, HITOSHI SAWADA, TADANORI AIZAWA, KAZUZO KATO, MORIO ONOE, YOSHINORI KUNO

From the Cardiovascular Institute, Minato-ku, Tokyo, and the Institute of Industrial Science, University of Tokyo, Japan

SUMMARY Left ventricular asynergy in myocardial infarction was assessed quantitatively by computer analysis of the cross sectional echocardiogram.

Short axis cross sectional images of the left ventricle at the levels of the mitral valve, papillary muscle, and apex were recorded by a phased array sector scanner in 30 patients with healed myocardial infarction and 15 normal controls. Endocardial and epicardial short axis images of the left ventricle were transferred from video tape to a minicomputer through the interface circuits, then digitised and processed automatically by a minicomputer. Automatic edge detection of the endocardial and epicardial wall was performed by applying sequential steps including smoothing, second derivative technique, dynamic thresholding, and approximation of boundaries by a spline curve. To quantify regional wall motion, the short axis cross sectional left ventricular wall of each level was divided into eight octants with eight axes at 45° angles from the initial standard axis which was constructed from the geometric centre of the end diastolic left ventricular cavity to the posterior end of the right side of the interventricular septum. Segmental hemiaxis, segmental area, segmental wall thickness, and those changes during cardiac cycle were measured and calculated in each segment automatically by a computer. Regional contractility of the left ventricle was evaluated by percentage systolic changes of the segmental hemiaxis, area, and wall thickness.

These values were significantly reduced in the infarcted left ventricular wall as defined by left ventriculography and electrocardiography. Moreover, percentage hemiaxis changes obtained by quantitative left ventriculography described by Herman and colleagues correlated well with those using our analytical method of cross sectional echocardiography in the corresponding segments.

The geometric centre of the left ventricular cavity determined by the computer moved slightly towards the anterior wall during systole in normal subjects, possibly reflecting the anterior swinging motion of the heart. The geometric centre of the left ventricular cavity in myocardial infarction moved towards the infarcted wall, showing that the floating reference system was inferior to the fixed reference system for the quantification of abnormal wall motion in myocardial infarction.

In conclusion, a computer analysis of the short axis cross sectional echocardiogram of the left ventricle using the fixed reference system has shown its ability to evaluate left ventricular contraction abnormalities, especially systolic wall thickening, which is relatively free of arbitrary interpretation of the wall motion caused by the anterior swinging motion of the heart.

It is well known that left ventricular wall motion abnormalities are present in patients with myocardial infarction, and the nature and extent of the abnormalities are important determinants of left ventricular function.

Left ventricular cineangiography has long been used to evaluate left ventricular contraction abnormalities but the procedure is not without risk, especially in severe left ventricular dysfunction or acute myocardial infarction, and repeat studies are not often performed.
Cross sectional echocardiography is non-invasive, may be repeated frequently, and offers a method of quantitative analysis of wall motion abnormalities. A few approaches have been reported, but as yet no standard quantitative technique has been established for the regional analysis of cross sectional echocardiographic images.

This study was designed to assess a computerised method of analysis of cross sectional echocardiograms in patients with myocardial infarction with particular reference to the quantitative analysis of regional wall motion abnormalities.

**Patients and method**

Thirty patients with healed transmural myocardial infarction and 15 normal subjects were studied. There were 41 men and four women, aged 23 to 69 (mean age 52) years (Table 1). The sites of infarction were anteroseptal in 15 and inferoposterior in 15, all of which were confirmed by diagnostic electrocardiographic findings, left ventriculography, and coronary angiography. Abnormal wall motion was observed in at least two of segments 1, 2, 3, 6 of AHA classification in anteroseptal infarction and in at least two of segments 4, 5, 7 in inferoposterior infarction. Abnormal wall motion was defined qualitatively according to the terminology of Herman et al. by two cineangiographers. Left ventricular cineangiography and coronary angiography were performed by a standard technique and those results are shown in Table 1.

Short axis cross sectional images of the left ventricle at the levels of the mitral valve, papillary muscle, and
apex were recorded at end diastole and end systole by using a phased array sector scanner (ALOKA SSD-800) and video recorder (Sony), simultaneously with the electrocardiogram and phonocardiogram (Fig. 1). Technically adequate images for cross sectional echocardiographic measurement were obtained in about 70% of patients with myocardial infarction by angling or moving the transducer. The onset of the QRS complex and aortic component of the second heart sound were used as markers of end diastole and end systole, respectively. Recordings of cross sectional echocardiograms were performed within 24 hours of the time of left ventriculography.

The short axis cross sectional images of the left ventricle at each level at end diastole and end systole were transferred from video tape to a minicomputer through the interface circuits using a Video Motion Analyzer (Sony), then digitised, and processed automatically by a minicomputer. Each digitised image consisted of 256 x 256 pixels of 256 values. A minicomputer (HP 2100, HP 2112) executed the principal programs and image processing.

**IMAGE PROCESSING TECHNIQUE**

Automatic edge detection of the endocardial and epicardial wall was performed by applying sequential steps as shown in Fig 2. Smoothing was performed by replacing each pixel with average grey level values of the neighbouring 5 x 5 pixels (procedure 2 in Fig. 2). The rationale of edge detection depended on the assumption that the abrupt changes of the grey level occurred at the boundary and the points with the maximum gradient value are boundaries of the left ventricle. A portion of the edge detection is shown in Fig 2. Digitalisation of the video signal was followed by smoothing (procedure 2), digitisation of echo edge by logical product of the above processes (procedure 5), extraction of echo region by extracting brighter region than echo edge (procedure 6), extraction of candidates for the points on the boundaries (procedure 7), extraction of points making up convex figure (procedure 8), and approximation of outlines by a spline curve (procedure 9). The digits of variables were calculated (procedure 10).

**Fig. 1** End diastolic and end systolic short axis images of the left ventricle at the levels of the mitral valve (MV), papillary muscle (PM), and apex (AP). Numbers in the tracings show the location of AHA seven segments used in left ventriculography.6

**Fig. 2** Flow chart of the computer image processing of the cross sectional echocardiogram.
ventricular wall. The digitised image was subdivided into 256 small square regions with a side of 16 pixels. Then, differences in the sum of grey level values between upper and lower sides and between right and left sides of a subdivided square region were measured in each square region. The presence of more than a 64 grey level difference among either of the two values led to the decision that the edge exists in its square region and the threshold of grey level for the detection of pixels on the edge was determined as average grey level values of the two sides with a larger grey level difference (procedure 3). On the other hand, the smoothed edge was also subdivided into 49 square regions with a side of 64 pixels, and the grey level threshold for detecting echo signals was determined by an automatic threshold selection method based on the discriminant and least squares criteria (procedure 4). The details of this method were reported by Ohtsu. Among edges determined by procedure 3, those that satisfy the threshold determined by procedure 4 were detected as echo edges (procedure 5). Then, the echo region was extracted by detecting brighter pixels than echo edges determined as mentioned above (procedure 6). From many candidate points on endocardial and epicardial outlines, only the points that satisfied the following criteria were selected as the most likely candidates for points on the boundaries of the left ventricular wall (procedure 7). The first one was an outline which was generally smooth without any abrupt deviations of curvature. The second one consisted of outlines that did not cross. Best fit contours of endocardial and epicardial edges were drawn by fitting a spline through these points (procedure 9), after extraction of points making up the convex figure (procedure 8). Protruding echoes of trabeculae or papillary muscle were excluded in this procedure. Fig. 3 shows the digitised image of an end diastolic short axis view of the left ventricle at papillary muscle level. Fig. 4 shows the edge determined by sequential steps of smoothing, edge extraction, decision of grey level threshold for echo signals, and detection of edge which satisfy this threshold. Extraction of candidate points on endocardial and epicardial outlines was performed after procedure 6 (Fig. 5). Fig. 6 demonstrates best fit contours of endocardial and epicardial edges selected using a spline fitting technique after procedure 8.

To quantify regional wall motion, short axis cross sectional images of the left ventricle were divided automatically into eight octants with eight axes at 45° angles from the initial standard axis which was constructed from the geometric centre of the end diastolic left ventricular chamber to the posterior end of the right side of the interventricular septum (fixed axis method) (Fig. 7). End diastolic and end systolic segmental hemiaxis (Dd, Ds), segmental area (Ad, As), segmental wall thickness (Thd, Ths), and changes

---

Fig. 3 Digitisation of the end diastolic short axis image of the left ventricle at the papillary muscle level and its display on cathode ray tube.

Fig. 4 Edges determined by sequential steps of smoothing, edge detection, decision of grey level threshold for echo signals, and detection of the edge that satisfies the threshold for echo signals.

Fig. 5 Extraction of candidates for points on endocardial and epicardial outlines performed after procedure 6 in Fig. 2.
Computer analysis of cross sectional echo in infarction

during the cardiac cycle were measured and calculated by the computer in each of septal (S1, S2), anterior (A1, A2), lateral (L1, L2), and posterior (P1, P2) segments (Fig. 7). Regional contractility of the left ventricular wall was evaluated by systolic percentage changes of segmental hemiaxis, segmental area, and segmental wall thickness, measured as \((\text{Dd}-\text{Ds})/\text{Dd}\), \((\text{Ad}-\text{As})/\text{Ad}\), and \((\text{Ths}-\text{Thd})/\text{Thd}\) in each octants.

Regional contractility of the left ventricular wall was also quantified from 35 mm right anterior oblique cineangiograms using the hemiaxis approach of Herman et al.\(^7\) (Fig. 8). Percentage hemiaxis changes from the left ventriculogram were compared with those from the cross sectional echocardiogram in the corresponding segments.

Results

**Quantitative Analysis of Left Ventricular Segmental Wall Motion by Computer Analysis of Cross Sectional Echocardiogram**

**Regional wall motion in normal subjects**

The mean ± one standard deviation (SD) of \((\text{Dd}-\text{Ds})/\text{Dd}\), \((\text{Ad}-\text{As})/\text{Ad}\), and \((\text{Ths}-\text{Thd})/\text{Thd}\) values in eight octants at three levels in 15 normal subjects are shown in Table 2. \((\text{Dd}-\text{Ds})/\text{Dd}\) and \((\text{Ad}-\text{As})/\text{Ad}\) values in the anteroseptal wall (S1 S2 A1 A2) at the mitral valve and papillary muscle levels tended to be lower than those in the posterolateral wall (L1 L2 P1 P2). These values in apex level, however, showed wide variations with large standard deviations. On the other hand \((\text{Ths}-\text{Thd})/\text{Thd}\) values did not show any tendency to be lower in the anteroseptal wall than in the posterolateral wall, in contrast with \((\text{Dd}-\text{Ds})/\text{Dd}\) and \((\text{Ad}-\text{As})/\text{Ad}\) values. Measured values in the apex level, however, showed considerably wide variations (Table 2) and were excluded from this study.

The mean value ± 2 SD was considered to be within the normal range in each measurement except in the apex level, as shown with two solid lines in Figs. 9 and 10.

**Regional wall motion in myocardial infarction**

Fig. 9 shows \((\text{Dd}-\text{Ds})/\text{Dd}\), \((\text{Ad}-\text{As})/\text{Ad}\), and \((\text{Ths}-\text{Thd})/\text{Thd}\) values of eight octants at three levels in patients with anteroseptal infarction. \((\text{Dd}-\text{Ds})/\text{Dd}\) and \((\text{Ad}-\text{As})/\text{Ad}\) values were considerably decreased in each level compared with normal subjects, while \((\text{Ths}-\text{Thd})/\text{Thd}\) values were wide variations even in the apex level.
(Ad-As)/Ad values in S2 A1 of the mitral valve section and S1 S2 A1 of the papillary muscle section, and (Ths-Thd)/Thd values in S2 A1 of the mitral valve section and S2 A1 A2 of the papillary muscle section were significantly lower than those in normal subjects.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Segments</th>
</tr>
</thead>
<tbody>
<tr>
<td>MV section</td>
<td>S1</td>
</tr>
<tr>
<td>(Dd-Ds)/Dd</td>
<td>0.21±0.06</td>
</tr>
<tr>
<td>(Ad-As)/Ad</td>
<td>0.38±0.09</td>
</tr>
<tr>
<td>(Ths-Thd)/Thd</td>
<td>0.40±0.18</td>
</tr>
</tbody>
</table>

PM section
| (Dd-Ds)/Dd | 0.30±0.05 | 0.25±0.05 | 0.27±0.08 | 0.31±0.05 | 0.37±0.08 | 0.41±0.08 | 0.41±0.08 | 0.35±0.08 |
| (Ad-As)/Ad | 0.51±0.07 | 0.43±0.08 | 0.47±0.09 | 0.53±0.07 | 0.60±0.08 | 0.63±0.10 | 0.64±0.10 | 0.56±0.08 |
| (Ths-Thd)/Thd | 0.33±0.13 | 0.23±0.09 | 0.26±0.10 | 0.26±0.10 | 0.32±0.12 | 0.29±0.11 | 0.37±0.13 | 0.32±0.14 |

AP section
| (Dd-Ds)/Dd | 0.34±0.08 | 0.30±0.08 | 0.33±0.08 | 0.45±0.12 | 0.42±0.08 | 0.40±0.07 | 0.37±0.08 | 0.37±0.06 |
| (Ad-As)/Ad | 0.56±0.10 | 0.50±0.11 | 0.55±0.09 | 0.67±0.13 | 0.65±0.08 | 0.64±0.07 | 0.62±0.07 | 0.60±0.07 |
| (Ths-Thd)/Thd | 0.38±0.25 | 0.24±0.19 | 0.23±0.12 | 0.29±0.21 | 0.09±0.16 | 0.28±0.26 | 0.20±0.12 | 0.20±0.19 |

On the other hand in inferoposterior infarction, (Dd-Ds)/Dd, (Ad-As)/Ad, and (Ths-Thd)/Thd values in L2 P1 P2 of the mitral valve section and L2 P1 P2 S1 of the papillary muscle section were significantly lower than those in normal subjects, as shown in Fig. 10. These values were significantly reduced in the segments nearly corresponding to the infarcted areas.
Computer analysis of cross sectional echo in infarction

documented by cineventriculography, coronary angiography, and electrocardiography. Some of the measured values in infarcted segments were negative, showing the outward movements (bulged out) of the infarcted wall.

Comparison between (Dd-Ds)/Dd and percentage hemiaxis changes by right anterior oblique cineventriculography

Fig. 11 shows the relation between (Rd-Rs)/Rd and (Dd-Ds)/Dd in the corresponding segments. R1 corresponds to A1-2 (mitral valve), R2 to A1-2 (papillary muscle), R3 to A1-2 (apex), R4 to P1-2 (apex), R5 to P1-2 (papillary muscle), and R6 to P1-2 (mitral valve), respectively (Fig. 7 and 8). (Rd-Rs)/Rd values correlated well with (Dd-Ds)/Dd values in the corresponding segments, with correlation coefficients of 0.87–0.97 (p<0.001).

Motion of geometric centre of left ventricular chamber during cardiac cycle in normal subjects and myocardial infarction

Figs. 12–14 show the motion of the geometric centre of the left ventricular cavity during systole in normal subjects and those with myocardial infarction. The X axis is the initial reference axis of the left ventricular short axis image, the Y axis is the axis intersecting the X axis at right angles, and the intersecting point is the position of the geometric centre at end diastole. Therefore, the first quadrant (A1, A2) corresponds to the anterior area, the second quadrant (L1, L2) to the lateral area, the third quadrant (P1, P2) to the posterior area, the fourth quadrant (S1, S2) to the septal area, respectively. Each dot in the figures shows the position of the geometric centre at end systole.

In normal subjects, the geometric centre of the left ventricular cavity at three levels moved only 0 to 4 (mean 2) mm towards the anteroseptal wall from end diastole to end systole (Fig. 12). In comparison, in myocardial infarction there was a movement towards the infarcted wall. In anteroseptal infarction, the movement was 2 to 8 (mean 4) mm to the anteroseptal wall which is significantly larger than normal (Fig. 13). On the other hand in patients with inferoposterior infarction, the movement was 2 to 9 (mean 5) mm towards the inferoposterior wall, contrasting with normal and anteroseptal infarction (Fig. 14).

![Figure 11](http://heart.bmj.com/BrHeartJ:Firstpublishedasa10.1136/hrt.51.2.139on1February1984.Downloadedfromhttp://heart.bmj.com/onJuly14,2022byguest.Protectedbycopyright.)

Fig. 11 Relations between (Rd-Rs)/Rd and (Dd-Ds)/Dd in 15 normal subjects (open circle), 15 patients with anteroseptal infarction (closed circle), and 15 patients with inferoposterior infarction (closed triangle) in the corresponding segments. Regression line (solid line) and identity line (dotted line) are shown.
Discussion

Quantitative analysis of regional wall motion provides a useful method for assessing the severity and extent of myocardial ischaemia and infarction, determining a prognosis and evaluating the effects of interventions including exercise and pacing.

Left ventricular cineangiography detects wall motion abnormalities and several approaches for quantitative analysis of angiographic images have also been reported. Angiography, however, is not only an invasive method, but also can display only image boundaries orthogonal to the field of view. In contrast, cross sectional echocardiography provides a more extensive image of the left ventricle around its entire circumference at multiple sectional levels non-invasively and can easily determine myocardial thickness.

Changes in segmental hemiaxis, segmental area, and segmental wall thickness in each octant corresponding to the infarcted area documented by left ventricular cineangiography and electrocardiography were significantly lower than normal values and fell more than two standard deviations below the mean values established in 15 normal subjects.

Measured values of (Dd-Ds)/Dd and (Ad-As)/Ad by our analytical method are lower in anteroseptal segments than in posterolateral ones in normal subjects when the fixed reference point of the geometric centre of end diastolic left ventricular cavity is used, reflecting anterior swinging motion of the whole heart during systole. This swinging motion of the entire heart during systole is also confirmed by the fact that the geometric centre of the left ventricular cavity shifts a little towards the anteroseptal wall during systole. Nevertheless, the regional contractility of the left ventricular wall can be evaluated by comparing these values reflecting the net systolic motion of the heart with the normal range obtained in normal subjects. Moreover, (Rd-Rs)/Rd obtained by a generally ac-

Fig. 12 Motion of the geometric centre of left ventricular chamber during systole in 15 normal subjects. X axis is the initial reference axis. Y axis is the axis intersecting the X axis at a 90° angle. The intersecting point (0) is the position of the geometric centre at end diastole. Closed circles show the position of the geometric centre at end systole. The first quadrant corresponds to the anterior area (A1, A2), the second one to the lateral area (L1, L2), the third one to the posterior area (P1, P2), the fourth one to the septal area (S1, S2).

Fig. 13 Motion of the geometric centre of the left ventricular cavity during systole in 15 patients with anteroseptal infarction.
accepted method of quantitative left ventriculography correlated well with (Dd-Ds)/Dd in the corresponding segments, though no gold standard has been established for the regional analysis of left ventriculographic images. Therefore, our analytical method using cross sectional echocardiography and a fixed reference system is considered to be available and useful in a clinical setting.

In addition, we should emphasise the findings about depressed wall thickening in patients with myocardial infarction. Measurements of systolic wall thickening are relatively free of arbitrary interpretation caused by using a fixed reference system to measure wall motion, and echocardiography is the only technique by which measurement of wall thickness can be obtained.

The gravity centre shifts towards the infarcted wall during systole in patients with myocardial infarction, showing that the floating axis method is inferior to the fixed axis method for the quantification of abnormal regional motion in myocardial infarction.

There are still some unsolved problems. First, we should record cross sectional echocardiograms with an appropriate gain setting for detecting the edge close to the anatomical endocardial and epicardial edge. Reproducibility of this computer method to measure segmental hemiasis, area, and wall thickness of the short axis cross sectional echocardiogram recorded with the best gain setting was excellent. When two successive beats in 20 patients in this study group were measured, variabilities of measured values by our computer method were within 7.4%. Second, the inside of the endocardial and epicardial echoes was traced automatically by the computer, implying that the trailing edge of the echoes along the anterior part of the ventricle and the inner border of the echoes in the lateral walls were traced. Therefore, the cavity area detected by this method probably underestimates that based on a truly anatomical boundary. The echo pulse width and beam width of our instrument are, however, considerably small. Moreover, the degree of underestimation is considered to be almost the same in each patient. Third, we applied a fixed axis method for quantification of regional contractility of the left ventricle. Validity of the fixed axis method is still controversial. Theoretically, measurement of systolic wall thickening is relatively free of arbitrary interpretation caused by using a fixed reference system to measure wall motion, and echocardiography is the only technique by which measurement of wall thickness can be made. Much better resolution of the instrument should be required, to measure the left ventricular wall thickness accurately because systolic changes in thickness are relatively small. Fourthly, the measured values in the apical segments were variable with a large standard deviation. Because the apex is a small portion of a deformed chamber with a large motion, one cannot accurately measure apical segments by our analytical method.

Fifthly, the amount of time required for analysing a single cardiac cycle by our method was about 30 minutes. Real time computerisation of the cross sectional echocardiogram will be necessary.

The use of computers for the analysis of echocardiographic images can be possible and very useful to quantify the regional contractility of the left ventricle in a clinical setting. The computer can analyse large amounts of data very quickly, subdivide images to any extent desired, measure areas, and determine the centre of gravity very easily, and perform analyses with excellent reproducibility.

In conclusion, this method has shown that left ventricular contraction abnormalities and changes in the geometric centre of the left ventricular chamber in myocardial infarction can be accurately determined.
The method is non-invasive and can be repeated safely at frequent intervals so that the course of left ventricular disease can be followed.

References

4 Parisi AF, Moynihan PF, Folland ED, Feldman CL. Quantitative detection of regional left ventricular contraction abnormalities by two-dimensional echocardiography.

Fujii, Sawada, Aizawa, Kato, Onoe, Kuno
