Apex two dimensional echocardiography
Alternative approach to quantification of acute myocardial infarction

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SUMMARY Apex echocardiography has been chosen as an approach to detect and quantify acute myocardial infarction because the usual parasternal acoustic windows are often occluded. Fifty-three patients were studied, all within 12 hours after the onset of symptoms of their first myocardial infarction. Three apical long axis views were obtained, that is the two and four chamber views, and the right anterior oblique equivalent or three chamber view. Satisfactory echocardiograms were obtained in 48 patients (91%). The individual apical views were divided into equal segments and the area of asynergy was estimated in each view.

Left ventricular asynergy was present in all 48 patients. In 46 patients a positive correlation between the electrocardiogram and the echocardiogram was obtained, as far as infarct localisation was concerned. The estimated asynergic area correlated well with the peak value of the isoenzyme of creatine kinase (CK MB).

Apex echocardiography is a reliable alternative method of detecting and quantifying myocardial infarction soon after the onset of symptoms.

The immediate and long term prognosis of patients with an acute myocardial infarction depends largely on the site and extent of the infarcted myocardium.[1-3] It is of clinical significance, therefore, to be able to obtain reliable data regarding these features early after the onset of symptoms.

Various non-invasive techniques to determine infarct size have been developed recently,[4-6] of which echocardiography appears particularly useful since it can be performed at the bedside, involves no risk for the patient, is reproducible, and is capable of visualising the entire left ventricular wall.[7] In animal[8-10] and human[11-13] studies the regional wall motion abnormalities that occur with myocardial infarction have been visualised on two dimensional echocardiography and used to quantify the necrotic area. The success rate of obtaining adequate echocardiographic results in patients with an acute myocardial infarction, however, is limited[11 14] since the parasternal acoustic windows are often closed because of chronic lung disease.

It is for this reason that we used the site of the cardiac apex for the two dimensional echocardiographic localisation and quantification of regional wall motion abnormalities in patients with an acute myocardial infarction.

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Subjects and methods

Fifty-three consecutive patients (45 men; eight women) with a first myocardial infarction were studied. Their average age was 57 years (range 36 to 82). Acute myocardial infarction was documented by a typical clinical history, diagnostic Q waves on the electrocardiogram, and elevation of the isoenzyme of creatine kinase (CK MB).

Echocardiographic examination was performed in all patients within 12 hours after the onset of symptoms. Serial CK MB determinations were performed on admission and repeated every four hours thereafter until the peak value was reached.[15] A routine 12 lead electrocardiogram was obtained on admission and repeated every 12 hours during the first three days in hospital. The electrocardiographic infarct localisation was determined according to the criteria of the New York Heart Association.[16]

TWO DIMENSIONAL ECHOCARDIOGRAPHY

All echocardiographic studies were performed using a commercial mechanical sector scanner (Eko-Sector 1, Smith Kline Instruments) with an 82° sector arc and a 2·25 MHz transducer. The images obtained were stored on videotape for analysis by two observers.

All patients were studied in the left lateral decubitus position. The transducer was placed at the point of maximal impulse of the cardiac apex and the sector was directed towards the left atrium (Fig. 1). The apical four chamber view was first obtained showing the inlet part of the ventricular septum together with the apex and posterolateral wall of the left ventricle.[17–19] By rotating the transducer approximately 60 degrees clockwise a three chamber view was obtained comparable with the right anterior oblique equivalent. This view shows the outlet part of the ventricular septum, the apex, and the posterior wall of the left ventricle. A two chamber view was obtained by rotating the transducer approximately 60 degrees counterclockwise. This view shows the anterolateral and inferior wall of the left ventricle and the apex.[20]

ECHOCARDIOGRAPHIC ANALYSIS

Left ventricular wall motion of each study was analysed for the presence and extent of asynergy by two investigators who had no prior knowledge of the clinical status of the patient. The results were compared and occasional discrepancies were resolved by consensus. Precomparison interobserver variation for extent of asynergy was low (r=0·95). Asynergy was defined as hypokinesia, akinesia, or dyskinesia, as judged by the overall visual impression after reviewing each study in real time, slow motion, and stop frame formats.

Asynergy was termed "anterior" if it occurred in the apical, septal, or anterolateral regions and "inferior" if it occurred in the posterior, posterolateral, or inferior regions of the left ventricle. The basic principles used to estimate infarct size are shown schematically in Fig. 2. Each apical long axis view was divided into equal segments, and the estimated asynergic area was expressed as a percentage of the total number of segments.

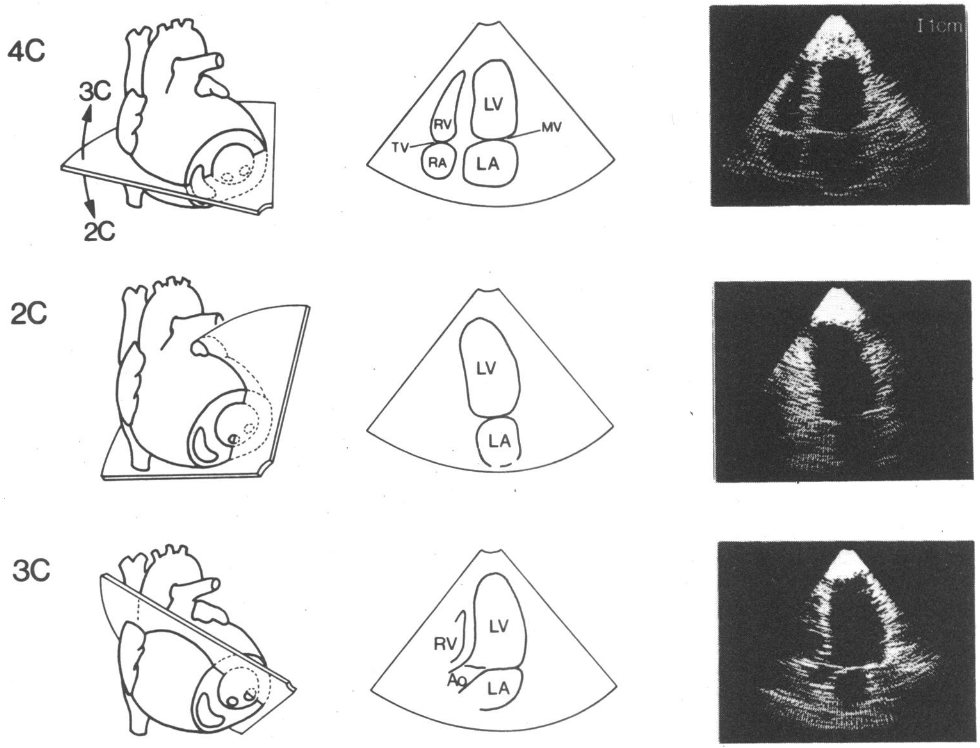

Fig. 1 *Diagrams of the three different planes of transsection, together with corresponding stop frames. Ao, aorta; LA, left atrium; LV, left ventricle; MV, mitral valve; RV, right ventricle; TV, tricuspid valve; 4C, four chamber view; 2C, two chamber view; 3C, three chamber view.*

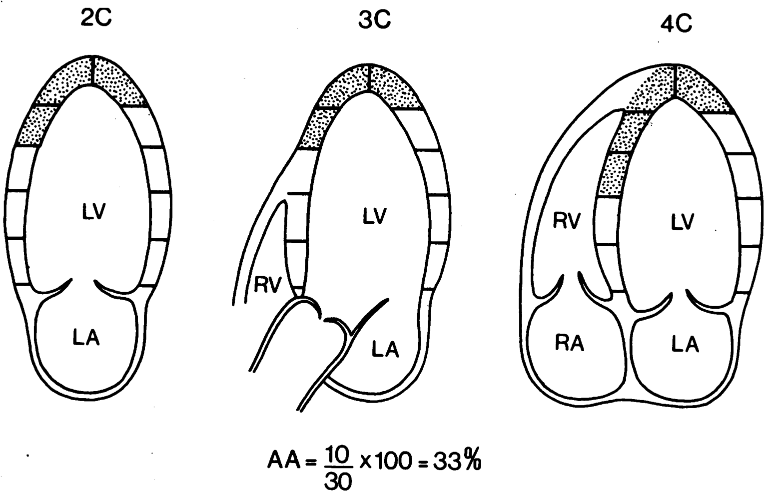

Fig. 2 *Schematic diagram showing the three apical long axis views. The left ventricular free wall and septum is divided into equal segments. The asynergic area (AA) of the left ventricular wall (dotted segments) is expressed as a percentage of the total number of segments (30). In this example a total infarct size of 33% is obtained. Abbreviations as in Fig. 1.*

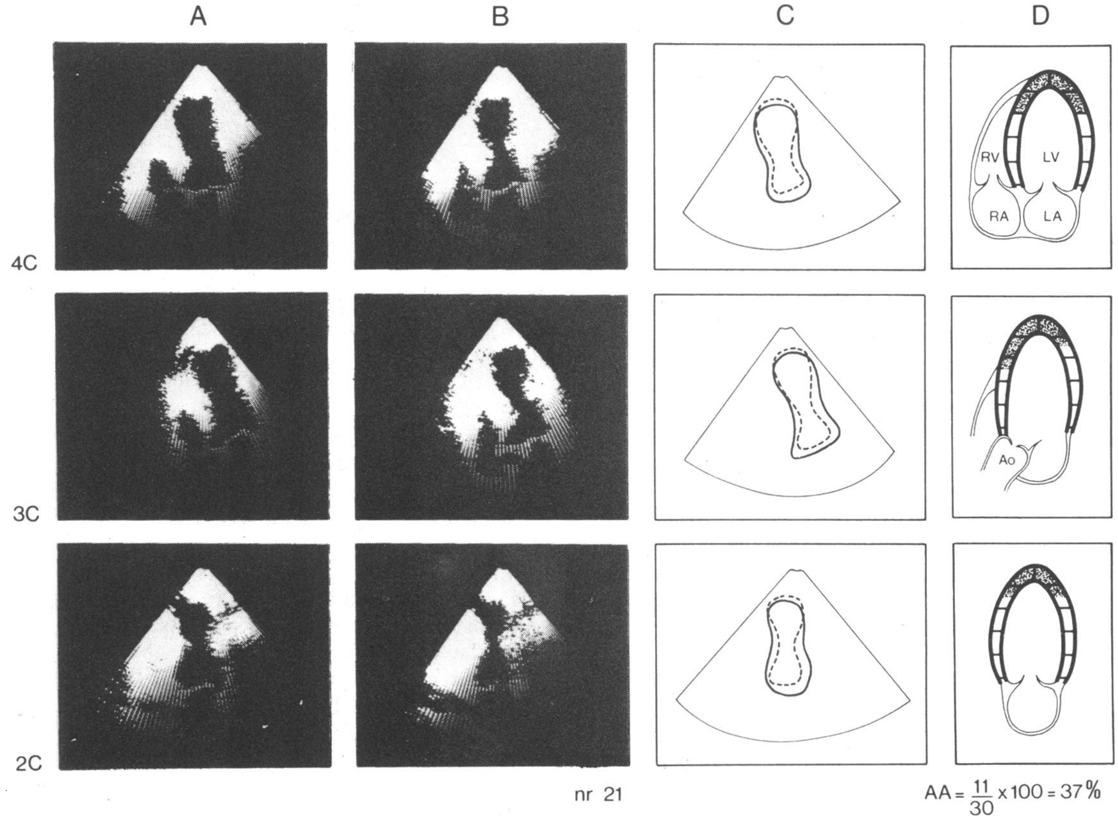

Fig. 3 *The panels A and B show the end-diastolic and end-systolic stop frames of each of the three long axis apical views in a patient with an acute anterior infarction, eight hours after onset of symptoms. Panel C shows the diastolic (solid line) and systolic (dotted line) outlines of the left ventricle. There is extensive asynergy. Note also the abnormal diastolic configuration of the left ventricle. In panel D the dotted segments represent the visually estimated asynergic area (AA) of the left ventricular wall, calculated at 37%. Abbreviations as in Fig. 1.*

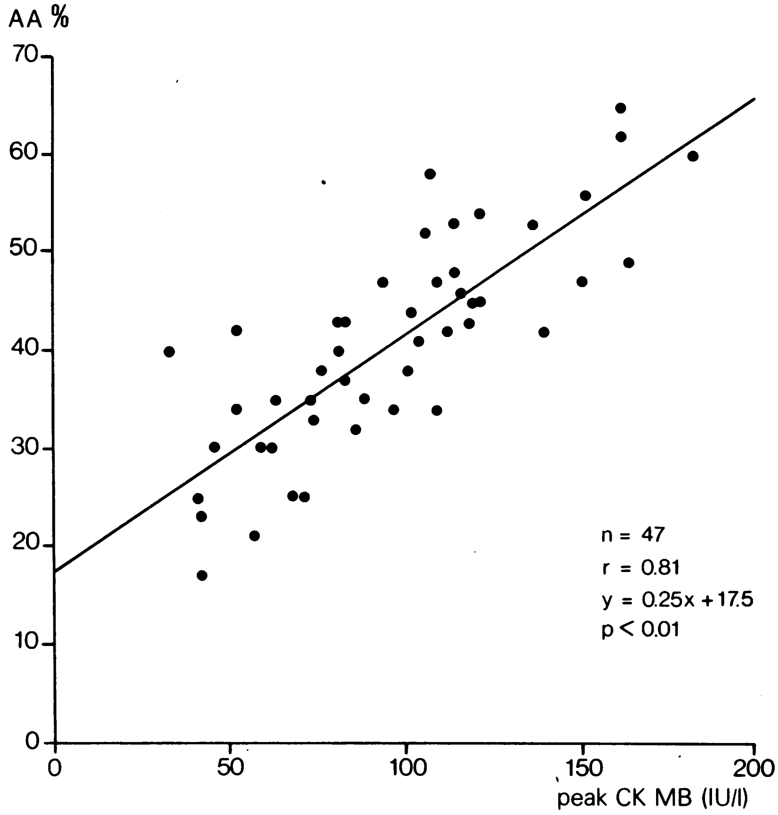

Fig. 4 *Diagram of the correlation between the peak value of CK MB and the echocardiographically estimated infarct size (AA). Abbreviation as in Fig. 2.*

Results

In 48 (91%) of the 53 patients (40 men; eight women) a complete and adequate two dimensional echocardiographic study was obtained (Fig. 3). Left ventricular asynergy was present in each of the 48 patients.

The unsatisfactory results in the five patients were caused by the left lung overlying the anterolateral left ventricular wall in the two chamber view.

LOCALISATION OF MYOCARDIAL INFARCT

The electrocardiographic site of infarction was anterior in 24 patients, inferior in 18, and anteroinferior in six patients. The echocardiographic site of infarction was the same in 46 of the 48 patients.

Discrepancy between the electrocardiogram and echocardiogram was present in two patients. One patient had an isolated inferior wall infarction on the electrocardiogram while the echocardiogram disclosed additional involvement of the ventricular septum. The other patient had an isolated anterior wall infarction on the electrocardiogram, but the echocardiogram showed asynergy of the apico-septal wall as well as asynergy of the posterior wall.

QUANTIFICATION OF INFARCT SIZE

Using the three cross-sections, infarct sizes could be quantified in each of the 48 patients. A correlation with the peak CK MB could be obtained in 47 patients, since one patient died within 18 hours after onset of symptoms. The correlation between both variables is shown in Fig. 4.

POST-MORTEM CORRELATION

During the period of this study only one patient died. This patient had extensive asynergy of the apical, septal, and anterolateral wall on the echocardiogram. The diastolic configuration, moreover, showed a distinct outward bulging of the affected area (Fig. 5A).

Necropsy showed an extensive transmural

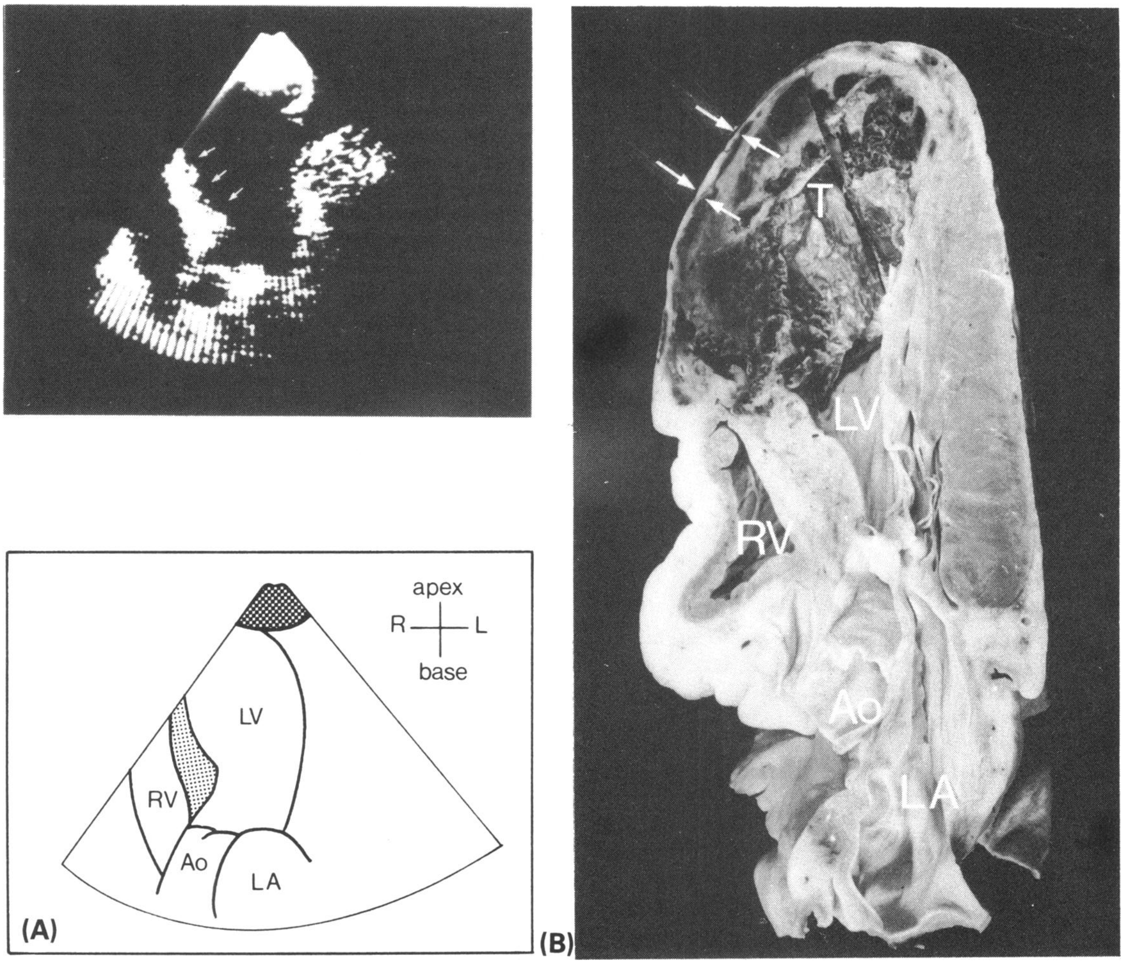

Fig. 5 *(A) End-diastolic three chamber view in a patient with an acute anterior infarction, who died 18 hours after admission. Note the obvious thinning of the septum (arrows) and distortion of the shape of the left ventricle. At the time of this study no intracavitary thrombus was identified. (B) A cross-section through the heart of the same patient comparable to the apical three chamber view. There is an extensive anteroseptal infarction with extreme wall thinning (arrows). Part of the left ventricular cavity is filled with thrombus (T).*

anteroseptal myocardial infarction of recent onset, which also extended onto the apex and the lateral free wall. An apical aneurysm was present (Fig. 5B). Rupture of the free wall had occurred leading to tamponade as the ultimate cause of death.

Quantification of the infarct size was performed using similar three cross-sections as used echocardiographically. Gross inspection thus disclosed a visually estimated infarct size of approximately 45%.

Discussion

Echocardiography is accepted as a reliable method of detecting wall motion abnormalities in patients with an acute myocardial infarction.[21–23] Unfortunately, the parasternal acoustic windows are often occluded in patients with coronary artery disease because of accompanying pulmonary disease. On the other hand, the site of the cardiac apex is often readily amenable to ultrasound penetration. In many patients with myocardial infarcts, particularly those with anterior wall infarction, the apex of the left ventricle is also involved[24 25] and, hence, because of apical dilatation, contributes to the size of this acoustic window. With this approach adequate echocardiographic images were obtained in 91% of the patients. This percentage is in accordance with that of reports in which apical long axis views were applied to determine left ventricular volumes,[20 26 27] but is better than the figures reported when only parasternal acoustic windows were used.[11 14]

CORRELATION BETWEEN SITE OF ASYNERGY AND ELECTROCARDIOGRAM

The abnormalities in wall motion were almost totally restricted to the region that correlated with the electrocardiographic infarct site. This finding can be explained by patient selection, since all of the patients were having their first myocardial infarction. A discrepancy was present in only two of the 48 patients. The electrocardiogram of one of these showed an isolated inferior infarction, whereas the echocardiogram disclosed additional asynergy of the adjacent septum. There is still uncertainty as to whether or not the septum was truly infarcted.

Clinical and experimental studies have shown that segments of the left ventricle adjacent to a region of infarction may exhibit asynergy and yet be normal morphologically.[28–30] On the other hand, inferior infarction may well extend into the interventricular septum without producing characteristic electrocardiographic changes.[25 31]

The echocardiogram of the second patient disclosed asynergy of the apex and septum, in keeping with the electrocardiogram, but in addition showed asynergy of a non-adjacent segment of the posterior wall. The posterior area of the left ventricle is difficult to evaluate electrocardiographically[32] and this may explain the discrepancy.

QUANTIFICATION OF INFARCT AREA

As previously outlined, we have taken asynergy as an indicator for myocardial infarction, though we realise that this is not necessarily always the case. At present, however, in judging infarct size this particular feature is the most reliable echocardiographic variable. We showed a linear correlation between infarct size judged from the echocardiograms and that based on the peak values of CK MB, indicating the reliability of the method employed. This conclusion is in keeping with the observation that wall motion abnormality correlates with enzymatic estimates of infarct size.[33]

It is important, furthermore, that the three cross-sections do not cover all of the ventricular wall. Despite this restriction, asynergy was encountered in all patients. This could be explained by the fact that the lowest value of the peak CK MB in our population was 33 IU/l (normal: 4 IU/l or less), so that the infarcts were probably all sufficiently large to be encountered in at least one of the echocardiographic sections. The asynergic area in 47 patients ranged from 17 to 66%. Some of these values are likely to be exaggerated since a true loss of myocardium of more than 40% is usually fatal.[1] Moreover, it is well known from experimental and clinical studies that abnormalities in wall motion extend beyond the region of actual infarction.[13 34] It is of interest, in this regard, that the only patient in this study who died had an estimated infarct size on echocardiographic grounds of approximately 55%, while the necropsy disclosed an infarct of approximately 45%.

From a practical point of view it thus appears that the usual percentages of myocardial loss quoted to predict prognosis, which are all related to the total left ventricular myocardial mass, need an adjustment when based on this particular echocardiographic approach. In our hands this alternative method provides a reliable option in the quantification of infarct size.

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