Esophageal Echocardiographic Left Ventricular Anterolateral Wall Motion in Normal Subjects and Patients with Coronary Artery Disease

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SUMMARY Esophageal echocardiography was developed for recording left ventricular anterolateral wall (LVAW) echocardiograms and was applied clinically to 14 normal subjects and 21 patients with coronary artery disease. LVAW echocardiograms were obtained satisfactorily in 11 of 14 normal subjects (75%) and 20 of 21 patients (95%) with coronary artery disease. LVAW echocardiograms were obtained by conventional anterior echocardiography in eight of 21 patients (38%) with coronary artery disease. In 11 normal subjects, LVAW excursion averaged 10.8 ± 1.7 mm (range 8–13 mm); mean systolic velocity ranged from 28–41 mm/sec (mean 34.3 ± 5.2 mm/sec); and diastolic wall thickness ranged from 9–12.5 mm (mean 11.2 ± 0.7 mm). In 20 patients with coronary artery disease, LVAW motion obtained by esophageal echocardiography was classified into five groups according to the excursion, and the findings were in good agreement (80%) with those obtained by left ventriculography. Classification of LVAW motion by conventional echocardiography agreed with that of left ventriculography in only three of eight patients, although all eight patients had abnormal LVAW motion by the conventional method. In all patients except one, whose LVAW echocardiograms were obtained by conventional echocardiography, excursion was much less than that obtained by esophageal echocardiography. We conclude that the projection of an ultrasonic beam from the intraesophageal transducer is a better approach for accurate measurement of LVAW motion.

ABNORMAL left ventricular wall motion occurs in some patients with severe coronary artery disease. The left ventricular anterior wall (LVAW) and interventricular septum (IVS) are supplied by the left anterior descending coronary artery (LAD), and generally, motion of those walls is impaired if severe stenosis of this artery causes ischemia.

Echocardiography is one of the most useful noninvasive procedures for evaluating left ventricular regional wall motion. In patients with severe obstruction of the LAD, abnormal IVS motion may be seen by "conventional echocardiography" by placing the transducer along the left sternal border. However, this does not indicate that LAD lesions always lead to abnormality of the IVS motion. Jacobs et al., Gordon and Kerber, and Kolibash et al. have documented that about half of their patients with severe lesions of the LAD had normal echocardiographic IVS motion.

Corya et al. also measured LVAW motion echocardiographically, and found that abnormal LVAW motion is closely correlated to the presence of an LAD lesion. However, an LVAW echogram is sometimes difficult to obtain in patients with coronary artery disease. Especially in the presence of severe obesity or chronic obstructive lung disease, the LVAW echogram cannot easily be obtained through a conventional technique.

To obtain an LVAW echocardiogram more easily and dependably, we developed the esophageal echocardiograph, and clinical application of this technique has provided new data. In this study we evaluated the relationship between LVAW motion measured by esophageal echocardiography and that measured by conventional echocardiography and examined the reliability of the esophageal technique in measuring LVAW motion by comparing it to left ventriculographic findings.

Methods

Patients

Thirty-five patients who underwent esophageal echocardiography in Yamaguchi University Hospital and Saiseikai Shimonoseki Hospital from September 1976 to September 1977 were studied. Twenty-one of these patients were found to have coronary artery disease. Each patient in this group was evaluated clinically and entered in the study only if the findings included two of four major criteria for the diagnosis of myocardial infarction or angina pectoris: classic history, serial enzyme changes, infarction pattern on the ECG, or angina attack with typical electrocardiographic change. This group included five women and 16 men, ages 32–75 years: 11 with anteroseptal myocardial infarction, five with inferior myocardial infarction, one with posterior myocardial infarction and four with angina pectoris without a history of myocardial infarction. The other 14 patients, five women

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and nine men, ages 27–71 years, were included in the control group. This group consisted of five patients with cardiac neurosis, two with chronic obstructive lung disease, one with paroxysmal atrial tachycardia, one with mild essential hypertension and five healthy volunteers. Coronary artery disease and other cardiac conditions that could cause abnormal LVAW motion were precluded, based on history, physical findings, ECG, chest x-ray, phonocardiogram and conventional echocardiogram.

Cardiac catheterization was performed in the 21 patients with coronary artery disease and in six of the 14 control subjects.

Echocardiograms

All patients were examined immediately before the cardiac catheterization studies with a commercial echocardiograph (Aloka Model SSD-80) using a 2.25-MHz, 10-mm nonfocused transducer. Echocardiograms were recorded on a Fukuda ECO-125A strip-chart recorder simultaneously with the lead II ECG and phonocardiogram. Conventional echocardiography was performed in the supine or in the left lateral decubitus position using previously described techniques.4

Esophageal echocardiography was performed using a 3.0-MHz, 6-mm nonfocused transducer (Aloka Industries, Ltd.) attached to the tip of a gastrocamera (Model V, Olympus Camera Co. Ltd.) (fig. 1). The transducer may be angled sufficiently at a point about 5 cm from the tip and can be rotated easily at the proper level. The echocardiograph and the recorder for esophageal echocardiography were the same as those for conventional echocardiography.

FIGURE 1. The esophageal echocardiographic transducer. The transducer is attached to the tip of a gastrocamera.

Before the study, the pharyngeal region was anesthetized locally with 2% viscous xylocaine. The procedure for inserting the esophageal transducer was similar to that for inserting the gastrocamera. The transducer was passed through the throat and placed at about a 35-cm depth from the mouthpiece. The patient was supine. The transducer, at this level, was situated slightly laterally and faced anteriorly, and the aortic valve usually could be identified (fig. 2). With further counterclockwise advancement of the transducer, the visible area of the right ventricular cavity between the IVS and the right ventricular anterior wall was gradually narrowed and finally disappeared. The procedure confirmed the continuity of the ventricular septum with the LVAW, which could be identified more clearly with slight counterclockwise rotation

FIGURE 2. Representations of the procedure for recording an M-mode scan of the left ventricular (LV) long axis by esophageal echocardiography: (A) Cardiac frontal view; (B) cardiac horizontal view; (C) esophageal echocardiographic scan (Eso Echo) of the LV long axis in a normal patient. Echoes from the aorta are shown on the right side of the lower panel (labeled (1), white arrow) and LV apical echoes are shown on the left (labeled (2), black arrow). Ao = aorta; P = pulmonary artery; RV = right ventricle; POST = posterior site of the chest; ANT = anterior site of the chest; LVPW = LV posterior wall; LVAW = LV anterior wall; PPM = posterior papillary muscle; AML = anterior mitral leaflet; IVS = interventricular septum.
The LVAW echo was identified with this procedure.

Three measurements were performed using LVAW echograms: systolic wall excursion, which was the amplitude of the inward motion of the endocardium from the beginning of the inward motion (usually the end of isometric contraction) to the time of the end-systole; mean systolic velocity, calculated by dividing the wall excursion by the ventricular ejection time; and diastolic wall thickness at the beginning of P wave in lead II. Systolic LVAW motion in the 20 patients with coronary artery disease was classified into five groups according to the values of the systolic wall excursion: hyperkinetic — excursion ≥ 14 mm; normokinetic — 8–13 mm; hypokinetic — 3–7 mm; akinetic — ≤ 2 mm; and dyskinetic — outward motion in systole. The normal upper and lower ranges (14 and 8 mm) of the excursion of the LVAW were assumed to be the values of the mean ± 2 SD in the control group.

**Left Ventriculography and Coronary Arteriography**

Left ventriculography was performed by contrast injection using a Thomson 6- or 9-inch image intensifier and Arritechno (R35–90) 35-mm cinecamer in 30° right anterior oblique position, filming at 48 frames/sec. Ventriculograms were analyzed with a Tagano 35-mm film projector. The outlines of the ventricle at end-diastole and at end-systole were traced from projected images of the cinefilm and superimposed on the same paper. The spine and ribs were used as the fixed points for the tracing. End-diastole was taken from the frame at the R wave in the simultaneously recorded ECG. End-systole was taken from the last frame that showed uniform inward motion of the wall. A reduction in the normal systolic excursion of LVAW was defined to be hypokinesis, and a paradoxical systolic wall expansion to be dyskinesis. Selective coronary arteriography was performed by the Sones technique, and injections were filmed in multiple views.

Conventional and esophageal echocardiograms, ventriculograms and coronary arteriograms were reviewed and analyzed independently by four specialists without clinical information.

**Results**

With the esophageal echocardiographic technique, the LVAW echogram was recorded satisfactorily in 31 of the 35 patients (11 of the 14 normal subjects and 20 of the 21 patients with coronary artery disease). With the conventional echocardiographic technique, LVAW echograms were recorded in only eight of the 21 patients with coronary artery disease (table 1).

**Esophageal Echocardiographic LVAW Motion in the Control Group**

A representative example is shown in figure 3. In this case, the wall excursion, the mean systolic velocity and the diastolic wall thickness were 9 mm, 35 mm/sec and 10 mm, respectively. The mean values of the wall excursion and the mean systolic velocity of the LVAW in the control group were 10.8 ± 1.7 mm (range 8–13 mm) and 34.3 ± 5.2 mm/sec (range 28–41 mm/sec), respectively. The mean value of the diastolic wall thickness was 11.2 ± 0.7 mm (range 9–12.5 mm).

**Esophageal Echocardiographic LVAW Motion in Patients with Coronary Artery Disease**

The systolic LVAW motion in the 20 patients with coronary artery disease was classified into five groups according to the values of the systolic wall excursion as described in the methods section. Figure 4 shows examples of LVAW motion in each group. Table 1 is a comparison of LVAW motion detected by esophageal echocardiography, conventional echocardiography and left ventriculography. Correlation between esophageal echocardiograms and left ventriculograms is shown in table 2. In 16 of 20 patients, the evaluation of LVAW motion by esophageal echocardiography coincided with that in left ventriculography. With conventional echocardiography, the classification of LVAW motion agreed with that of left ventriculography in only three of eight patients, although all eight had abnormal LVAW motion by the conventional method. In all patients except one (case 4 in table 1), excursion of LVAW was much less with conventional echocardiography than that obtained by esophageal echocardiography.

Figure 5 is a comparison of LVAW motion measured by conventional echocardiography with that by esophageal echocardiography in a 63-year-old man with three-vessel disease (case 12 in table 1). Left ventriculography proves hypokinetic motion of the LVAW, and the finding is in good agreement with that of esophageal echocardiography. However, with conventional echocardiography (apex approach), the LVAW echocardiogram shows slightly outward motion during systole. Thus, in this case, the abnormal
motion of the LVAW is overestimated by the conventional approach.

Figure 6 shows a conventional echocardiogram of the septum and posterior wall (the left side of the upper panel), esophageal echocardiogram (the center of the upper panel), left coronary arteriogram and left ventriculograms in a 52-year-old man with anteroseptal myocardial infarction (case 7 in table 1). The IVS echocardiogram demonstrates normal excursion and the normal systolic thickening. However, the esophageal echocardiogram reveals akinesis of LVAW motion (2 mm in excursion), decreased mean systolic velocity (10 mm/sec), and a thin LVAW (5 mm). These findings correspond well to the left ventriculographic findings. The left coronary arteriogram shows severe stenosis of the LAD distal to the first septal branch and a severe lesion of the left circumflex artery.

In one of the 21 patients with coronary artery disease, dyskinetic LVAW motion was seen with esophageal echocardiography (fig. 7; case 17 in table 1). This 56-year-old man had an old inferior myocardial infarction and angina pectoris at rest. The LVAW echo obtained by esophageal echocardiography after sublingual nitroglycerin shows a slightly outward motion during atrial contraction, followed by further outward movement. This increased outward motion is believed to occur during left ventricular isovolumetric contraction. Thereafter, a slight inward motion occurs. This paradoxical wall motion disappeared rapidly immediately after the second heart sound in the simultaneously recorded phonocardiogram. The left ventriculograms reveal a dyskinesia extending widely from the anterobasal to the apical portion, and show an irregular endocardial surface of the anterolateral wall compared with that of the posterobasal and inferior walls during the entire cardiac cycle. A mural thrombus might be adhering to the LVAW in this case, but this patient died suddenly and an autopsy was not performed. The coronary arteriograms show severe obstruction of three main coronary arteries, and the myocardium was found to be supplied by many collateral vessels.

Discussion

Left ventriculography has provided numerous data on ventricular wall motion in coronary artery disease. On the other hand, echocardiography is one of the most valuable noninvasive techniques for evaluating regional wall dynamics. The location of abnormal echo motion generally corresponds to the area distal to the severe stenosis of the coronary artery. However, it is sometimes difficult to assess the entire segment of the left ventricle with conventional echocardiography. Heikkinen and Nieminen attempted to take an echocardiogram of the entire left ventricle in acute myocardial infarction and demonstrated good correlation of the sites of infarction between the echocardiographic and the electrocardio-
TABLE 1. Comparison of Left Ventricular Anterior Wall Motions Obtained by Left Ventriculography with Those by Esophageal and Conventional Echocardiography

<table>
<thead>
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<th>Pt (years)</th>
<th>Sex</th>
<th>Diagnosis</th>
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<th>Ant. echo</th>
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<th>% internal area reduction</th>
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<td></td>
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</table>

Abbreviations: Eso. echo = esophageal echocardiography; Ant. echo = conventional echocardiography; LVG = left ventriculography; CAG = coronary arteriography; MI = myocardial infarction; A = akinetic; H = hypokinetic; N = normokinetic; D = dyskinetic; NO = not obtained; LVAW = left ventricular anterior wall; LAD = left anterior descending coronary artery; CX = circumflex coronary artery; LMC = left main coronary artery.

Graphic studies. Corya et al.4 recorded LVAW echoes in 50 of 54 patients with coronary artery disease using conventional echocardiography with the transducer on the chest wall. In their study, LVAW echoes that moved toward the transducer or the absence of wall motion during ejection were considered abnormal, and movement away from the transducer during ejection was interpreted as normal. The correlation of the LVAW echocardiogram with the left ventriculogram was examined, with agreement found in only 66% of the cases. One explanation for this poor correlation was that the areas examined were not always the same.

In our study, LVAW echo motion that was inter-

TABLE 2. Comparison of Left Ventricular Anterior Wall Motion on Esophageal Echocardiography and Left Ventriculography

<table>
<thead>
<tr>
<th>LVAW eso. echo</th>
<th>Hyper- and normokinetic</th>
<th>Hypokinetic</th>
<th>Akinetic</th>
<th>Dyskinetic</th>
<th>Total</th>
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<td>—</td>
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<td>6*</td>
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<td>Dyskinetic</td>
<td>—</td>
<td></td>
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<td>1</td>
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<tr>
<td>Total</td>
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<td>8</td>
<td>6</td>
<td>1</td>
<td>20</td>
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</table>

*Agreement of wall motion obtained by the two methods.

Abbreviations: LVAW = left ventricular anterior wall; eso. echo = esophageal echocardiogram.
Figure 5. Comparison of the left ventricular anterior wall (LVAW) motion obtained by conventional echocardiography (apex approach) and that by esophageal echocardiography in a patient with inferior myocardial infarction (case 12 in table 1). In the conventional echocardiogram, the LVAW shows slightly outward motion in systole. However, in esophageal echocardiogram (Eso Echo), the LVAW shows hypokinetic inward motion, which corresponds well to the left ventriculographic finding. LVPW = left ventricular posterior wall; PCG = phonocardiogram.
Figure 7. Dyskinetic left ventricular anterior wall (LVAW) motion obtained by esophageal echocardiography in a patient with an old inferior myocardial infarction and angina pectoris (case 17 in table 1). Left ventriculograms show a dyskinetic LVAW with an extensive mural thrombus, and coronary arteriograms show severe obstruction of the main coronary arteries. The white spots and dotted lines in the left ventriculogram indicate simultaneously recorded ECG and phonocardiogram, respectively. PML = posterior mitral leaflet; AML = anterior mitral leaflet; IVS = interventricular septum; LCA = left coronary arteriogram; RCA = right coronary arteriogram.

Interpreted as normal by Corya's definition was further classified as hyperkinetic, normokinetic or hypokinetic, while abnormal LVAW motion as defined by Corya was classified as akinetic or dyskinetic. Satisfactory LVAW echoes were recorded by esophageal echocardiography in 20 of 21 patients (95%) with coronary artery disease. In 16 of these 20 (80%), the classification of LVAW motion by esophageal echocardiography coincided well with that by left ventriculography. LVAW echoes recorded by conventional echocardiography were satisfactory in only eight of the 21 patients (38%). All eight of these patients had abnormal LVAW motion on the conventional echocardiogram, but the LVAW motion classification by conventional echocardiography was compatible with that obtained by left ventriculography in only three patients (37%). Moreover, LVAW echoes were recorded by conventional echocardiography showed less systolic excursion compared with that shown in those recorded by esophageal echocardiography. Previous investigators^{16, 18, 22} have pointed out that the anterior shift of the whole heart occurring during systole may cause underestimation of the systolic excursion of the LVAW. Leighton et al.\textsuperscript{22} used left ventriculography and documented that systolic excursion of the LVAW did not differ greatly from that of the left ventricular posterior wall. Our data are consistent with this observation and suggest that in the normal heart, excursion of the LVAW shown by esophageal echocardiography would be similar to that of the left ventricular posterior wall obtained by conventional echocardiography. The esophageal transducer probably could move along with the whole heart motion in each cardiac cycle, which would minimize the artifact of underestimation of LVAW motion. Thus,

Figure 6. Conventional echocardiogram, esophageal echocardiogram, left coronary arteriogram (LCAG) and left ventriculograms in a patient with anteroseptal myocardial infarction (case 7 in table 1). The interventricular septum (IVS) demonstrates normal excursion and normal systolic wall thickening. However, the esophageal echo shows akinetic left ventricular anterior (LVAW) motion of 2 mm in excursion, which corresponds well to left ventriculographic findings. The thin LVAW and its increased echo density may suggest myocardial fibrosis of this region. The coronary arteriogram reveals severe stenosis of the left anterior descending artery distal to the major septal branch and also shows severe lesion of the left circumflex artery. LAO = left anterior oblique projection; RAO = right anterior oblique projection; LVPW = left ventricular posterior wall.
our results emphasize that the projection of an ultrasonic beam from the intrasophageal transducer would be a better approach for accurate evaluation of LVAW motion compared with the conventional approach. The esophageal transducer should be very useful in examining numerous segments that are inaccessible to transducers applied to the chest, particularly in the presence of obesity or emphysema, which makes penetration by the ultrasonic waves difficult. If one is familiar with the technique of the gastrocamera, esophageal echocardiography can be performed very easily in about 15 minutes without complications.

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References

15. Herman MV, Gorlin R: Implication of left ventricular asynergy. Am J Cardiol 23: 538, 1969
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