Interventricular Septal Motion in Patients with Proximal and Distal Left Anterior Descending Coronary Artery Lesions

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SUMMARY In order to evaluate the ability of the echocardiogram to detect and localize left main or left anterior descending (LAD) coronary artery lesions, 43 patients were studied. The systolic excursion of the left side of the septum and the ratio of posterior wall to septal excursion were measured. Seventeen patients had no LAD lesions; all had systolic septal excursion of 3 mm or greater. Twelve patients with septal excursion of 2 mm or less all had left main or LAD lesions, but 14 other patients with LAD lesions had septal excursion of 3 mm or greater. Nine of 16 patients with LAD lesions proximal to the first septal branch had reduced or absent septal excursion, as did three of ten with LAD lesions distal to the first septal branch. In a setting of coronary artery disease reduced or absent septal motion on echocardiography suggests involvement of the left main or left anterior descending coronary. However, the technique is relatively insensitive, with 54% of the LAD patients having normal septal motion.

ECHOCARDIOGRAPHY IS USEFUL in the evaluation of cardiac wall motion. When one or more areas of the left ventricle are dyskinetic, the ultrasound beam may traverse such areas and demonstrate abnormal wall motion. This usually occurs in a setting of acute myocardial ischemia. Angiographic studies have confirmed that reduced motion of the posterior cardiac wall seen on echocardiography is associated with obstructive lesions of the circumflex or right coronary arteries, while left anterior descending (LAD) obstruction affects interventricular septal motion. Joffe et al. have suggested that abnormalities of septal and posterior wall motion may be specific indicators of obstruction proximal to the first septal branch of the LAD. Since patients with significant coronary artery obstructive lesions of the left main and proximal left anterior descending coronary arteries may constitute a group with an especially poor prognosis, the identification of such potentially high-risk individuals using noninvasive echocardiographic techniques may have great clinical importance.

The purpose of this study was to evaluate the ability of the echocardiogram to detect abnormalities of interventricular septal motion in patients with obstructions at various points in the left anterior descending coronary artery.

Methods

Eight hundred twenty-eight patients underwent diagnostic cardiac catheterization and coronary arteriography at the University of Iowa Hospitals and Iowa City Veterans Administration Hospital during the calendar year 1975. Of these patients, approximately 100 had M-mode echocardiography performed within 48 hours of catheterization for various clinical indications (evaluation for mitral prolapse, left ventricular function, etc.). All patients with congenital heart disease, IHSS, left bundle branch block, valvular insufficiency and other conditions known to affect echocardiographic septal motion were then excluded. Of the remaining 65 patients, 43 were considered, after initial review, to have echocardiograms of a quality sufficient to permit satisfactory measurement of both the interventricular septum and left ventricular posterior wall. The coronary arteriograms were of a quality sufficient to permit the accurate assessment of the presence or absence of any coronary arterial lesions, but particularly those involving the left main and left anterior descending coronary arteries. Of the forty-three patients selected there were twenty-six male and seventeen female. The age range was 30 to 75. All had chest pain considered to be probably or possibly angina. No patient was considered to be in congestive heart failure.

The echocardiograms were performed using an Echoline 20 ultrasonoscope with a 2.25 MHz transducer focussed at 7.5 cm and a Honeywell 1856 fiberoptic strip chart recorder. All patients were studied in the supine or partial left lateral decubitus positions. The transducer was placed at the left sternal border in the "standard interspace" — that is, the interspace which permitted recording of the anterior mitral leaflet while the transducer was held perpendicular to the chest wall, with no superior or inferior angulation. Echocardiographic M-mode sweeps from the aortic root to the posterior papillary muscle were performed. The motion of both the interventricular septum and posterior left ventricular wall was measured at the point in the sweep where the beam was directed just inferior to the mitral valve echo. Only tracings showing continuous left septal echoes, obtained as indicated, were considered acceptable for measurement. An illustrative echocardiogram is shown in figure 1. The septal excursion, in mm, was measured from the left side of the septum at the R wave of the ECG to its maximal excursion posteriorly. Posterior wall excursion was also measured in mm from the position of the posterior endocardium at the R wave of the ECG to the point of the maximum anterior motion of the endocardium. We also derived a ratio of posterior wall to septal excursion by dividing the posterior wall excursion by the septal excursion; using the criteria of Joffe et al., a ratio of 2.5 or greater was considered abnormal. Echocardiographic measurements were made without knowledge of the results of coronary angiography.

The coronary angiographic studies were performed using the percutaneous femoral technique. All patients underwent selective coronary arteriography with contrast injections in
FIGURE 1. Illustrative echocardiogram from a patient with left main coronary artery stenosis. Septal excursion (SE) was measured along the left side of the septum from its position at the R wave of the ECG to its point of maximum posterior motion. Posterior wall excursion (PWE) was measured at the R wave to its point of maximum anterior motion.

Each coronary artery in LAO, PA and RAO positions. They also underwent left ventriculography in the 30° right anterior oblique (RAO) position. The cineangiograms and coronary arteriograms were interpreted without knowledge of the echocardiograms. Luminal narrowing equal to or greater than 70% was considered abnormal. Particular note was made as to whether LAD lesions were proximal or distal to the first septal branch. Lesions involving the ostium of the first septal branch were treated as proximal lesions. Also, stenosis of the left main coronary artery was considered to be a proximal LAD lesion for purposes of this study. Statistical comparisons were made by use of an unpaired t-test.

Results

Coronary Arteriography

Seventeen patients had no LAD coronary lesions. Of these, 11 patients had entirely normal coronary arteries, while six had lesions in the right or circumflex coronary artery. Twenty-six patients had lesions either proximal to the first septal branch of the left anterior descending coronary artery (16 patients) or distal to that branch (10 patients). Of the 16 patients who had lesions proximal to the first septal branch, three had left main coronary lesions and 13 had LAD lesions.

Echocardiography: Septal Excursion

The echocardiographic septal excursion of each patient is shown in figure 2. All the patients with either no coronary disease or coronary disease not involving the LAD had septal excursion equal to or greater than 3 mm; none had 2 mm or less. Of the group with LAD disease, 12 of 26 (46%) had septal excursions of 2 mm or less. Of these LAD disease patients, 9 of 16 (56%) with proximal LAD lesions had 2 mm or less of septal excursion; 3 of 10 (30%) with distal LAD lesions similarly had 2 mm or less septal excursion. Of the three patients with left main coronary lesions two had less than 2 mm septal excursion. The third had 4 mm septal excursion. The difference in incidence of 2 mm or less septal excursion between these two groups of LAD lesion patients was not significant.

Echocardiography: Posterior Wall Excursion/Septal Excursion Ratio

The excursion ratio of each patient is shown in figure 3. Of the 17 patients with no LAD lesions, 13 (76%) had a normal ratio of less than 2.5, while four (24%) had an abnormal ratio equal to or greater than 2.5. Of the 26 patients with LAD disease, 15 (58%) had an abnormal ratio. Of this latter group, 12 of the 16 (75%) proximal LAD lesion patients had abnormal ratios, while three of the ten (30%) with distal LAD disease similarly had abnormally high ratios.

Electrocardiograms

Seven patients had electrocardiographic evidence of prior anterior myocardial infarction. Three had normal septal
forces on ECG but Q waves in the mid and lateral precordial leads. All three had LAD coronary lesions; two were proximal, with echocardiographic septal excursions of 1 and 3 mm, while the third, with distal LAD disease, had an echocardiographic septal excursion of 10 mm. All four of the patients with septal Q waves on ECG had proximal LAD coronary lesions, but nevertheless the septal motion was 10, 6 and 4 mm in three of these patients.

**Discussion**

This investigation demonstrates that reduced or absent systolic septal motion, in a setting of coronary artery disease, suggests involvement of the left anterior descending coronary artery. None of our 17 patients with normal LAD coronary arteries had a septal excursion of less than 3 mm, while almost half of the patients with obstructive LAD lesions had septal excursions of 0–2 mm. These results are in agreement with the study of Jacobs et al. who similarly found septal excursion of 3 to 8 mm in 10 patients without coronary disease, and septal excursion of less than 3 mm in 30 of 38 patients with LAD lesions.

On the other hand, normal interventricular septal motion may be seen in the presence of major obstructive lesions in the LAD, even if such lesions are located proximal to the first septal branch. We found normal septal excursion in 14 of 26 patients despite LAD lesions; seven of these had proximal lesions. Similarly, Jacobs et al. found normal septal motion in eight of 38 patients with LAD lesions. Presumably, the areas of septum traversed by the beam are not ischemic or fibrotic at the time of the examination, despite the presence of an obstructive arterial lesion. If myocardial ischemia is created in such patients by either increasing myocardial oxygen requirements (via exercise or other stress) or decreasing perfusion (via increasing coronary obstruction) septal motion may then become abnormal, “unmasking” the coronary lesion. Acute changes in echocardiographic septal and posterior wall motion in such settings have been demonstrated experimentally and clinically.

Can the echocardiogram predict the presence of proximal LAD lesions? Abnormal interventricular septal motion was encountered in both proximal and distal LAD disease, and although it was more common in the patients with proximal lesions, the difference between the two groups was not statistically significant. Joffe et al. used a ratio of posterior wall to septal excursion to classify coronary patients; this ratio would be made abnormally high by reduced septal excursion, increased posterior wall excursion (compensatory hyperactivity), or both. Although the majority (12 of 16) of patients with proximal LAD disease were indeed classified as abnormal using 2.5 as a normal ratio, three of ten patients with distal LAD lesions were also classified as abnormal; Joffe et al. themselves found this in two of 22 patients with distal LAD lesions. Moreover, four of the patients with no LAD disease at all would be classified as abnormal by this criterion. In addition, considering the small absolute values of the wall motion measurements made, any measurement errors may be magnified by utilizing a ratio.

Why should some patients with distal LAD disease display abnormal motion of the interventricular septum on echocardiography? Several explanations are possible. The coronary arteriogram provides only inferential information on myocardial perfusion; it is possible that the area of the septum traversed by the ultrasound beam may be ischemic despite a LAD lesion classified as distal by our criteria. This may be particularly likely if the lesion is immediately distal to the first septal branch and/or if the first septal artery is a small vessel supplying only a small area of the septum; this appeared to be the case in some of our patients. In addition, the currently accepted practice of assessing septal motion at the level of the chordae tendineae, below the mitral valve, results in the ultrasound beam traversing an area of the septum located toward the ventricular apex and more likely to be supplied by the distal LAD. But even if the beam is traversing an area of normally perfused septum, its motion may be altered by the presence of adjacent ischemic myocardium; experimental studies using echocardiography and epicardial strain gauges have demonstrated this.

All of the seven patients with ECG evidence of anterior or anteroseptal myocardial infarction had angiographic evidence of LAD disease. Only one of these patients, however, had abnormal septal motion by echocardiography. This suggests that myocardial fibrosis adequate to produce Q waves on ECG is not necessarily sufficient to prevent the septum from contracting normally. This may be a manifestation of the patchy nature of the myocardial fibrosis which occurs postinfarction.

In assessing septal motion echocardiographically, caution should be exercised in interpreting recordings from patients who have conditions known to affect septal motion independently of LAD lesions: left bundle branch block, right ventricular volume overload, valvular incompetence and others. Such patients were specifically excluded from this study.

We conclude that echocardiographically reduced or absent systolic motion of the interventricular septum in the patients with coronary artery disease and no other cause for abnormal septal motion suggests a left anterior descending coronary obstructive lesion. However, such abnormal motion was seen with lesions both proximal and distal to the first septal branch. Thus, it appears unlikely that the echocardiogram can predict the exact location of an LAD lesion with sufficient accuracy to be clinically useful.

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Comparison of Echocardiographic and Necropsy Measurements of Ventricular Wall Thicknesses in Patients with and without Disproportionate Septal Thickening

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SUMMARY. In several patients with asymmetric septal hypertrophy (ASH) diagnosed by echocardiography (septal-free wall thickness ratios \( \geq 1.3 \)), we have discovered marked discrepancies between the echocardiographic and necropsy measurements of wall thicknesses that led to uncertainty regarding the actual cardiac diagnosis. To resolve these apparent incongruities, the echocardiograms and hearts of 17 patients with cardiac disease were studied.

Six of nine patients with abnormal septal-free wall ratios \( \geq 1.3 \) during life had septal-free wall ratios that were not diagnostic of disproportionate septal thickening at necropsy. Such discrepancies may be explained as follows: 1) echocardiographic measurements during life were made in diastole (as per convention), but measurements at necropsy were made in hearts that appeared to have been in the systolic phase of the cardiac cycle; 2) the left ventricular free wall thickens considerably more than the ventricular septum in systole, as determined by echocardiography. This latter phenomenon resulted in septal-free wall ratios in systole that were consistently smaller than those in diastole. Furthermore, septal-free wall ratios obtained at necropsy corresponded most closely to those obtained by echocardiography in systole.

THE CHARACTERISTIC ANATOMIC FEATURE OF PATIENTS with asymmetric septal hypertrophy (ASH) is disproportionate thickening of the ventricular septum with respect to the posterobasal left ventricular free wall.\(^1-7\) This finding in patients with ASH usually can be demonstrated by either echocardiography\(^4\) or necropsy.\(^6,7\) The assessment of ventricular wall thicknesses by these two techniques have been shown to agree closely.\(^8\) However, recently we have observed a few patients with echocardiographically determined disproportionate septal thickening in whom the septal to posterobasal left ventricular wall thickness ratios, as determined at necropsy, were not diagnostic of disproportionate septal thickening. To determine better the factors responsible for such occasional disparities, we have measured ventricular wall thicknesses during life by echocardiography and, in the same patients, at necropsy. These studies were performed in nine patients with disproportionate septal thickening and eight other patients with either concentric thickening or normal wall dimensions.

Selection and Analysis of Patients

Initially, echocardiograms and heart specimens were analyzed in 36 patients evaluated at the National Heart, Lung and Blood Institute between 1972 and 1976 for cardiac disease. Nineteen patients were excluded from the study because either 1) the echocardiogram was not obtained relatively close to the time of death (13 patients) or 2) the echocardiogram (three patients) or necropsy specimen (three patients) was unsuitable for reliable measurement of ventricular wall thicknesses. The remaining 17 patients comprise the study group that was divided into two subgroups.

The first subgroup included eight patients in whom the ventricular septal to posterobasal left ventricular wall thickness ratio (determined by echocardiography in diastole) was \( < 1.3 \). These patients ranged in age from 25 to 66 years (mean 54); six were women and two were men. Included in this group were seven patients with acquired valvular disease (three with mitral valve disease, three with combined aortic and mitral valve disease and one with aortic valve disease);
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