Distribution of Coronary Artery Disease
Prediction by Echocardiography

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SUMMARY To assess the sensitivity of standard echocardiography in detecting ventricular motion abnormalities in patients with coronary artery disease (CAD) without prior myocardial infarction, 56 consecutive patients with a history of angina pectoris were studied during an angina-free period. In the 48 patients with adequate echocardiograms, the amplitude of septal and posterior wall motion in the high, mid, and low left ventricle was determined and used to predict prospectively in a blinded fashion the sites of angiographically-determined CAD. Twenty-eight of 35 patients (80%) with disease of the left anterior descending artery (LAD) had diminished intraventricular septal motion (P < 0.001) and 14 of 27 patients (52%) with disease of posterior vessels had diminished posterior wall motion on echocardiogram. When abnormalities of echocardiographic wall motion were compared with left ventriculography, the results were similar. Echocardiography may aid in predicting the presence and distribution of CAD, especially LAD disease.

THE PRODUCTION of myocardial septal and posterior wall motion abnormalities by acute myocardial infarction or angina pectoris is well documented.1, 2 These abnormalities typically occur in segmental zones. Previous studies in humans have found good correlation between disease of the left anterior descending artery and abnormal echocardiographic septal motion.3-5 Other studies have correlated acute inferior wall myocardial infarction with changes in echocardiographic posterior wall motion in humans.6-7 No studies have been performed to evaluate the predictive ability of echocardiographic wall motion abnormalities in an unselected group of patients with coronary artery disease (CAD) without evidence of prior myocardial infarction by history or diagnostic Q waves on electrocardiogram. The purpose of our study was to determine in patients with chronic angina without prior myocardial infarction if the anatomic sites of coronary artery disease would be predicted accurately by echocardiographic measurements of septal and posterior wall motion when studied during an angina-free interval.

Methods

From the patients referred to the Milton S. Hershey Medical Center for cardiac catheterization for the evaluation of chest pain without prior myocardial infarction and without left bundle branch block, 56 consecutive patients were studied by echocardiography immediately prior to undergoing selective coronary angiography and left ventricular biplane cineangiography. All patients were studied by one of us. All patients had a resting 12-lead electrocardiogram prior to echocardiography. Forty-eight echocardiograms (86%) were found to be satisfactory. The study group consisted of 42 men and six women with an age range of 39 to 66 years, a mean of 53 years. Following the determination of left ventricular hemodynamics, biplane (AP and lateral) left ventricular cineangiography was performed by injecting Renografin 76 at 20–30 ml/sec using a 9-inch image intensifier and filming at 64 frames/sec. Coronary arteriograms were performed by the Sones4 or Judkins5 techniques and injections were filmed in multiple views.

Echocardiographic studies were carried out with an Ekoline 20 echocardiograph using a 0.5 inch diameter 2.25 MHz transducer focused at 7.5 cm. The echocardiograms were recorded on a Honeywell 1856 strip chart recorder using light-sensitive Kodak 2022 paper. The patients were studied in a semi-recumbent or left lateral decubitus position. The transducer was placed in the standard position usually at the left fourth intercostal space lateral to the left sternal margin. An M-mode scan was then performed from the aortic root to the apex of the left ventricle. Paper speed on the strip chart recorder was set to emphasize septal and posterior wall motion. A standard electrocardiogram (lead II) was simultaneously recorded on the echocardiogram. On all echocardiograms suitable for interpretation, amplitude of septal and posterior wall motion was measured in three positions in the left ventricle: 1) immediately at the appearance of both the anterior and posterior valve leaflets; 2) at the point where the anterior and posterior mitral valve leaflets disappear; 3) deep in the left ventricle when the posterior papillary muscles were first seen (fig. 1). Examples of the types of abnormalities encountered are seen in figure 2. Diminution of septal motion was taken to be less than 0.3 cm2 and diminution of posterior wall motion less than 0.8 cm on echocardiogram.8 No attempt was made to evaluate septal motion higher in the left ventricle than at the level of the anterior and posterior mitral valve leaflets when the posterior wall was clearly seen.

The study was divided into two parts. First, we examined the ability to predict disease of vessels supplying the myocardium on the basis of diminished interven tricular septal motion or posterior wall motion on echocardiogram. We also compared the ability to predict wall motion abnormalities on ventriculogram with diminished interven tricular septal motion or posterior wall motion on echocardiogram. Coronary artery disease was considered significant if obstructions were greater than 70%. The right coronary artery was considered to supply the posteroinferior wall in right dominant distributions and the left circumflex coronary artery if the circulation was left dominant.9 There were 42 right dominant distributions, five left dominant distributions, and

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one balanced. In the ventriculogram, the anterior wall included the septum, anterolateral wall, and apex of the heart, while the posterior wall included the inferior and true posterior regions. The reference system used in this analysis was identical to the methods of Dodge et al. and Herman and Gorlin. Hypokinesis of a segment of the left ventricular wall was considered to be a reduction in the normal extent of contraction, and dyskinesis of a segment was considered to be paradoxical systolic expansion of a local portion of the left ventricle. All left ventricular cineangiograms and coronary arteriograms were independently reviewed by two of the investigators with good agreement. All the echocardiograms were reviewed in a similar fashion. Thus, the angiographic wall motion abnormalities and the sites of coronary artery disease were predicted in a blinded fashion. Statistical analysis of data was performed using the Chi square test without Yeates correction.

Results

No patients had either paradoxical septal motion or right ventricular enlargement on the echocardiogram. Patients with either left bundle branch block or prior myocardial infarction on electrocardiogram were excluded from the study. We defined true positives as those patients with disease of vessels (fig. 3) or ventriculographic evidence of hypokinesis (fig. 4) and diminution of the respective wall motion on the echocardiogram. False positives were defined as those patients with normal anatomy but diminished wall motion on echocardiogram. With true negatives, both the echocardiogram and the angiogram correctly determined that abnormalities were not present. False negatives were those patients with angiographic evidence of vessel or wall disease with normally moving echocardiographic wall motion.

I. Prediction of Coronary Artery Abnormalities by Echocardiogram

Of 35 patients with disease of the left anterior descending artery, 28 (80%) had diminished interventricular septal motion on echocardiogram. Of 13 patients with nonsignificant disease of the LAD, four (31%) had diminished interventricular septal motion (fig. 3A) (<0.001 for both populations). Of 27 patients with disease of vessels supplying the postero-inferior wall, 14 (52%) had diminished posterior wall motion on echocardiogram (fig. 3B). Of 21 patients with nonsignificant disease of the posterior wall vessels, seven (33%) had diminished posterior wall motion on echocardiogram (<0.1 for both populations). Of 19 patients with disease of vessels supplying both the anterior and posterior walls of the heart, nine (47%) had diminished septal and posterior wall motion together on echocardiogram (fig. 3C). Of nine patients with entirely normal vessels, only one (11%) had diminished septal and posterior wall motion on echocardiogram (<0.1 for both populations).

II. Prediction of Ventriculographic Abnormalities by Echocardiography

In the second part of our study, we compared echocardiographic abnormalities with the results of the ventriculogram to see if predictive correlations could be improved. Of 28 patients with anterior wall hypokinesis on ventriculogram, 22 (79%) had diminished septal motion on echocardiogram (fig. 4A). Of 19 patients with normal anterior wall motion on ventriculogram, 10 (53%) had diminished septal motion on echocardiogram (<0.05 for both populations). Of 37 patients with posterior wall hypokinesis on ventriculogram, 18 (49%) had diminished posterior wall motion on echocardiogram (fig. 4B). Of 11 patients with normal inferior wall motion on ventriculogram, only two (18%) had diminished ventricular posterior wall motion. Of 27 patients with anterior and posterior wall hypokinesis on ventriculogram, 10 (37%) had diminished anterior and posterior wall motion on echocardiogram (fig. 4C). Of nine patients with entirely normal ventriculograms, only one (11%) had diminished anterior and posterior wall motion on echocardiogram.

Discussion

This study reveals some surprising facts. The population studied was unique in that no patients had prior myocardial infarction by either Q waves on electrocardiogram or by history, and no patient had angina pectoris during the performance of the echocardiographic studies. In this light, comparisons between populations that are significant at the 10% level assume considerable importance. It is therefore interesting that in our study such a large number of patients showed evidence of diminished septal and posterior wall motion on the echocardiogram as well as hypokinesis on the ventriculogram. This could be interpreted to mean that the echocardiogram was surprisingly sensitive in detecting small areas of damage in the myocardium that may have occurred previously without historical or electrocardiographic evidence of myocardial infarction. This is the most likely explanation. An alternative explanation is that partially obstructed vessels cause diminished wall motion without actual myocardial infarction. It has been reported that patients with angina pectoris and no myocardial infarction during an angina-free interval had significant 37% reduction in isovolumic left ventricular contractility indices when compared with normal subjects. Raftery et al. described a series of patients without myocardial infarction and with occlusive coronary disease presenting as primary congestive cardiomyopathy. Cohn et al. describe a patient with no
history of myocardial infarction and occlusive coronary disease who had regional myocardial wall motion abnormalities. Nakhjavan et al. have shown in dogs that myocardial ischemia not resulting in gross myocardial infarction or fibrosis at autopsy leads to a decrease in contractility in the ischemic areas. Thus it is possible that diminished wall motion may occur without myocardial infarction or angina pectoris in chronically ischemic areas of the myocardium.

We found the echocardiogram to be a far more reliable predictor of anterior vessel disease (diminished septal motion) than posterior vessel disease (diminished posterior wall). There were only seven of 35 (20%) patients who had disease of the LAD that the echocardiogram did not detect (fig 3A). More interesting, however, were the four false positives, a figure that differs from Jacobs et al. who detected no patients with diminished interventricular septal motion who had normal LAD arteries. The reason for these false positives is difficult to explain. It may be that vessels other than the LAD supplied the area of septum that the echocardiogram evaluated (e.g., the posterior descending vessel).

Figure 2. Examples of echocardiograms showing the abnormalities found in this study. A) Diminished septal motion alone. B) Diminished posterior wall motion alone. C) Diminished septal and posterior wall motion together.

Figure 3. Comparing the predictability of respective coronary vessel disease on the basis of diminished echocardiographic septal motion (panel A), posterior wall motion (panel B), septal and posterior wall motion (panel C).
artery). Another possibility is that less extensive lesions (between 50% and 70%) may cause some degree of septal motion abnormalities. The false negatives are probably explained by the fact that the echo beam records only the superior or upper portion of the septum while the LAD artery supplies the septum, the anterior and lateral walls, and the apex of the heart. Thus, the echo beam would miss certain areas of the myocardium that the LAD artery supplies.

The predictive correlations of posterior wall vessels were not nearly so satisfactory. We only detected 52% of patients with anatomic vascular disease (fig. 3B); therefore, this is not a particularly sensitive method of predicting posterior-inferior wall coronary artery disease. Although Corya et al. found a high percentage of patients with acute inferior infarction to have echocardiographic posterior wall motion abnormalities, it would appear that chronic angina does not result in as great a number of detectable motion abnormalities. The reasons for the false positives may be similar to those discussed above. There is obviously a large area of the diaphragmatic surface of the heart not visualized by the echocardiographic beam. When we compared disease of vessels of both anterior and posterior wall of the myocardium with echocardiographically-determined diminished septal and posterior wall motion together, we found that only one patient with diminished septal and posterior wall motion on echocardiogram had entirely normal vessels.

In the second part of our study, our predictive correlations of the echocardiogram with ventriculography were not significantly improved (fig. 4). Of the 28 patients with anterior wall motion abnormalities on the ventriculogram, 22 (79%) had echocardiographic septal motion diminution (fig. 4A). Again, when posterior motion abnormalities on ventriculogram are correlated with posterior wall motion abnormalities on echocardiogram, it is obvious that a considerable number of patients with posterior wall motion abnormalities on ventriculogram are missed by the echocardiogram (fig. 4B). However, when septal and posterior motion abnormalities occur together on echocardiogram, it is seen that it is most unlikely there would be a normal ventriculogram. Again, it was surprising for us to note that a large number of patients with angina and no evidence of myocardial infarction had hypokinesia detectable by ventriculogram as well as by echocardiogram during an angina-free interval.

One aspect of our findings illustrates an important limitation of blande ventriculography in the analysis of the three-dimensional information that is necessary to fully quantitate the extent of ischemic left ventricular motion abnormalities. Although ventriculography is normally accepted as the reference with which noninvasive techniques are compared, it may not be such a reliable standard. In our study, there were ten false positives when septal motion abnormalities on the echocardiogram were employed to predict corresponding abnormal ventriculographic systolic motion (fig. 4A). It should be noted that five of these ten patients had disease of the LAD artery. Thus, the echocardiogram detected five cases of septal abnormalities with LAD disease that the ventriculogram reported normal. Jacobs et al. noted a similar incidence of this phenomenon. The reason may well be that neither the lateral nor the frontal projections of the ventriculogram visualize the septum wall; the echocardiogram, with its ability to detect small changes in septal motion may, in fact, be more sensitive. In
figure 5, we have attempted to graphically portray this limitation of the ventriculogram in detecting the extent of abnormal ischemic motion in the myocardium. Two three-dimensional left ventricular endocardial surfaces are shown, along with their ventriculographic biplane projections. In each model, the total area of ventricular dyssynergy is equal and indicated by the circled area. It can be seen that biplane ventriculography would reasonably quantify the extent of dyssynergy in the model to the left but would grossly underestimate it in the model to the right. It is clear that the ventriculogram looks at only a shadow of the circumferential rim of endocardium. When biplane projections are used, a second circumferential rim of information is added. It is important to note that a considerable amount of endocardium between these projections is not seen at all and that major assumptions regarding uniformity of myocardium between the two projections are necessary for the quantification of overall left ventricular pump function. These assumptions have a more major limitation if one uses ventriculography to quantitate regional motion abnormalities. Thus, in figure 5, the total area of dyssynergy cannot be estimated by ventriculography in either case. Therefore, it is possible and even probable that the echo beam may detect small areas of hypokinesis on the left ventricular endocardial surface not seen in the angiographic silhouette. We note that out of our 48 patients, 12 patients had an abnormal echocardiogram and normal ventriculogram.

The sagittal planes evaluated by the standard echocardiogram scan from apex to aorta are more than evaluated by the standard lateral projection of the ventriculogram, since on repeat scans, multiple sagittal planes are intersected, including the left anterior oblique projection. Had we also repeated ventriculography to evaluate the left anterior oblique projection, we might have increased the sensitivity of ventriculography in detecting regional motion abnormalities. However, multiple ventriculograms are not possible in most patients. Further developments in echocardiography that allow for three-dimensional analysis of motion should ultimately prove useful in evaluating the total area of dyssynergy. In the interim, the single piston transducer may still have considerable clinical utility in coronary artery disease. The importance of M-mode scanning to cover as much of the left ventricle as possible to evaluate the areas in the three positions diagrammed in figure 1 is stressed. Satisfactory echocardiograms can be obtained in over 80% of patients. The inability to obtain echo scans of diagnostic quality in some patients may prove to be the ultimate limitation in the study of patients with coronary artery disease. Even in its current form we consider echocardiography to be a useful noninvasive ancillary aid in the diagnostic workup of a patient with coronary artery disease.

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References

Hemodynamic Correlates of the Normal Aortic Valve Echogram
A Study of Sound, Flow, and Motion

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SUMMARY The aortic valve echogram was recorded in open chest dogs simultaneously with aortic flow, acceleration of flow, aortic and left ventricular pressures, and intracardiac phonocardiograms.

Comparison of echographic with hemodynamic data showed the following: The aortic valve started its opening with the onset of flow, at the same point that left ventricular pressure exceeded aortic pressure. Complete valve opening preceded peak aortic flow by an average of 43 msec and the cusps started to move toward closure while flow was still accelerating. Final closure of the valve was achieved at the time of zero flow and preceded the aortic second sound by 4-10 msec. The echographically determined "valve orifice area" correlated well with aortic stroke volume \( r = 0.94 \).

The intensity of the aortic first sound was related to peak acceleration of aortic flow. The intensity of the second sound was not related to the amplitude of cusp motion but correlated well with the aortic pressure at the time of closure and with peak flow deceleration.

Material and Methods

Fifteen dogs, 19-25 kg, were anesthetized with pentobarbital (30 mg/kg), the chest was opened at the mid-sternum and the heart exposed. Left ventricular and aortic pressures were measured with high-fidelity catheter-tipped transducers (Micro-Tip model PC-350, Millar Instruments). The left ventricular catheter was inserted through an apical puncture and access to the aorta was gained by retrograde passage via the right common carotid artery. Both catheters were adjusted for common zero and equal sensitivity, which was checked repeatedy throughout the experiments by momentarily positioning the aortic catheter in the left ventricle or by advancing the left ventricular catheter into the aorta above the valve. A short stiff catheter connected to Statham P23Db pressure transducer was introduced into the left atrium via a pulmonary vein for measurements of left atrial pressure.

An electromagnetic flow probe was placed around the ascending aorta just above its root and instantaneous aortic flow was recorded by a square wave electromagnetic flowmeter (Model 501, Carolina Medical Electronics). The fre-
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