Identification of Transient and Persistent Segmental Wall Motion Abnormalities in Patients with Unstable Angina by Two-dimensional Echocardiography

J.V. Nixon, M.D., Charles N. Brown, M.D., and Thomas C. Smitherman, M.D.

SUMMARY To determine the value of real-time, two-dimensional echocardiography (2-D echo) in unstable angina, regional wall motion on serial short-axis 2-D echo recordings was analyzed and summed segment scores of abnormal motion were compared and classified according to each patient's clinical status 12 weeks after hospital discharge.

Nineteen male patients who fulfilled criteria for unstable angina and responded to medical therapy underwent 2-D echo study within 48 hours of admission and discharge. Of 11 patients with abnormal 2-D echo scores on admission, five patients had reduced scores and six patients had similar or increased scores at discharge. Six of eight patients who had scores of zero on admission had scores of zero at discharge. At follow-up, 11 patients had minimal or no angina pectoris (group 1), and eight patients had worsening angina or recurrent unstable angina (group 2). At discharge, 2-D echo studies showed that all group 1 patients had reduced or zero scores, while group 2 patients retained or increased their abnormal scores.

This study shows that in patients with unstable angina, both transient and persistent abnormalities can be identified by 2-D echo. Abnormal segmental wall motion was transient or absent in patients with a good outcome, and worsened or remained abnormal in patients with a poor outcome.

ORIGINAL CONCEPTS concerning the rapid recovery of regional myocardial functional derangement due to myocardial ischemia after the termination of ischemia have recently been revised. Brief interruptions of coronary flow produce alterations in regional function that persist after the return of myocardial blood flow to normal in conscious dogs. In humans, the demonstration of transient alterations in left ventricular function during myocardial ischemia has required either arterial pacing or exercise with ventriculography. Recently, regional left ventricular performance was assessed at cardiac catheterization during spontaneous angina pectoris. Little information is available, however, on serial changes in regional myocardial function caused by myocardial ischemia in humans. The pathophysiologic basis of the syndrome of unstable angina appears to be transient limitation of coronary blood flow. Thus, patients with unstable angina are ideal for the study of serial changes in segmental wall motion.

M-mode echocardiography identifies serial changes in global left ventricular function after unstable angina. In both animal and human studies, two-dimensional echocardiography has proved useful in accurately identifying the location and extent of an acute myocardial infarction and the existence of wall motion abnormalities in chronic ischemic heart disease. This technique may be useful in evaluating serial changes in wall motion abnormalities in patients with unstable angina. The object of this study was to identify the presence and duration of left ventricular wall motion abnormalities by two-dimensional echocardiography and to examine whether this technique can be used to determine the subsequent clinical course of patients with unstable angina.

Materials and Methods

Patient Selection

All patients who came to the hospital with a diagnosis of unstable angina were accepted if they met all the following previously used criteria: (1) The patient had pain or discomfort typical of myocardial ischemia occurring with either sudden onset of an unstable pattern or a recent deterioration in the pattern of angina within 1 month of admission. (2) There was no evidence of an acute myocardial infarction, documented by the absence of diagnostic QRS changes on the ECG, and the absence of an elevation of a least two of the three serum enzymes (creatine kinase, SGOT and LDH) to above twice the limit of normal. (3) There was objective evidence for ischemic heart disease, established by electrocardiographic evidence of myocardial ischemia (> 1 mm of horizontal or downsloping ST-segment depression sustained for 0.08 second beyond the J point), a positive maximal exercise test, documented disease by coronary arteriography, or a history of typical angina pectoris. (4) There were no apparent precipitating events or serious concurrent diseases.

Patients with a known or strongly suspected previous myocardial infarction were excluded from the study to eliminate as many patients as possible with persistent segmental wall motion abnormalities due to myocardial infarction. Only patients who fulfilled these criteria and who responded to medical...
therapy were considered for study. The response to medical therapy was adequate if the patient was pain-free or returned to the previous pattern of chronic stable angina pectoris during his hospitalization.

Nineteen male patients, ages 46–69 years (mean 57 years), fulfilled the criteria for study. Three other patients who fulfilled the criteria were subsequently excluded because their echocardiographic recordings were inadequate. All patients except patient 4 were receiving or were given long-acting nitrates (isosorbide nitrate) at admission. All but patients 6 and 11 were treated with propranolol. Two other patients were excluded because they had an acute myocardial infarction after the initial echocardiographic studies.

Follow-up clinical evaluation of all 19 patients was made 12 weeks after hospital discharge. Eleven patients had no further chest pain or only mild stable angina pectoris (New York Heart Association [NYHA] classes I and II). Eight patients had either an increase in frequency or severity of their stable angina pattern or had recurrent unstable angina (classes III and IV).

Echocardiograms

Initial two-dimensional echocardiograms were obtained after the patients were asymptomatic, within 48 hours of admission. Predischarge studies were obtained 7–10 days after admission, within 24 hours of the patient's hospital discharge. Studies were always performed during pain-free periods. Echocardiograms were obtained with a wide-angle, phased-array ultrasonograph (Varian V3000) using a 2.25-MHz transducer. Images were recorded on videotape for subsequent analysis. All patients were studied in the recumbent or shallow right anterior oblique position.

Our method of evaluating left ventricular segmental wall motion by summed segment score has been reported. The transducer was always placed perpendicular to the chest wall, and the short axis of the left ventricle was scanned until its image was circular or almost circular, closely approximating the short axis. Serial scans were obtained by moving the transducer head inferolaterally on the chest wall to avoid extreme transducer angulation that would produce an oval rather than a circular image of the left ventricle and render interpretation of wall asynergy unreliable.

The four short-axis views of the left ventricle used to analyze wall motion were identified as positions IV, Va, Vb, and VI by Kisslo et al (fig. 1). These views permit the analysis of the anterolateral, posterolateral, apical, septal and inferior regions of the left ventricular wall.

Ventricular wall motion was graded in each wall region in each view using a modification of the terminology of Herman and Gorlin: 0 = normal, 1 = hypokinesia, 2 = akinesia and 3 = dyskinesia. The graded scores of all regions in each echocardiographic view were totaled and the scores obtained from all views were summed for each patient. This summed segment score was the echocardiographic estimate of the myocardium involved in the ischemic process. Figures 2 and 3 show serial echocardiograms from patients with transient and persistent wall motion abnormalities.

Echocardiographic recordings were analyzed by two authors without knowledge of the identity or clinical status of the patient or, in the case of the predischarge echocardiographic study, the results of the initial study. Interobserver variation was minimal; correlations between numbers of regions involved and summed abnormal wall motion scores by each observer were r = 0.98 and r = 0.96, respectively. When discrepancies occurred, blind evaluation by a third observer was obtained. Persistent discrepancies were resolved by consensus.

This method was validated by echocardiographic study within 48 hours of catheterization in 10 patients.
who underwent cardiac catheterization for chest pain and had normal contrast ventriculography and coronary arteriography.\textsuperscript{15} The echocardiographic summed segment scores were zero for each patient.

Results

The echocardiographic locations and respective summed segment scores for each study are listed in table 1. Eight patients had summed scores of zero on their initial study. At their predischarge studies, six patients had scores of zero, but two (patients 7 and 15) had abnormal scores. The 11 other patients had abnormal summed echo scores on their initial study; at their predischarge study, scores had decreased in patients 1, 4, 5, 9 and 10, remained the same in patients 2, 17 and 19, and increased in patients 6, 13 and 16.

On the predischarge study, four patients had abnormal wall motion in a region that moved normally on the admission study. Patients 13 and 16 increased their previously abnormal score and patients 7 and 15 increased their previously zero score. Persistently abnormal scores on the discharge study were always caused by abnormal wall motion in the same segment or segments as on the initial study.

Follow-up data on the 19 patients obtained 12 weeks after hospital discharge are listed in table 1. Eleven patients had no further chest pain or only mild, infrequent angina pectoris (NYHA class I or II) and are classified as group 1. Eight patients had further episodes of severe, stable angina pectoris (NYHA class III) or further episodes of unstable angina (NYHA class IV) and are classified as group 2. The initial and predischarge summed echo scores of the patients in each group are shown in figure 4. The 11 patients in group 1 all had reduced or zero scores on their predischarge echocardiograms. Their mean score was reduced from 1.8 to 0.8. The eight patients in group 2 had either an identical abnormal score (patients 2, 17 and 19) or an increased abnormal score (five patients) ($\chi^2 = 7.816, p < 0.01$). The mean score of group 2 patients increased from 2.5 to 4.8.

All patients except patients 5 and 9 were taking oral propranolol, mean dose 196 mg/day, at the initial study. Eighteen patients were receiving isosorbide dinitrate, mean dose 72 mg/day. When patients in

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{Initial (A) and predischarge (B) short-axis, two-dimensional echocardiograms in position Vb from patient 5. The central line drawings show hypokinesia of the anterolateral and posterolateral segments (A) and normal contraction of all segments (B).}
\end{figure}
groups 1 and 2 were compared, neither the presence nor the dosage of antianginal therapy was statistically different.

**Discussion**

This study shows that serial two-dimensional echocardiographic studies detect transient and persistent left ventricular segmental wall motion abnormalities in patients with unstable angina who respond to medical therapy. The persistence or worsening of segmental dysfunction detected by this technique was associated with an unfavorable clinical outcome 3 months after hospital discharge. Conversely, an improvement in or absence of segmental dysfunction was associated with a favorable clinical outcome.

Echocardiography has been shown to be a suitable technique for detecting left ventricular wall motion abnormalities induced by myocardial ischemia. Both M-mode and two-dimensional echocardiographic techniques accurately identify left ventricular wall motion abnormalities in acute and chronic ischemic heart disease in humans. Kisslo et al. compared two-dimensional echocardiographic and angiographic techniques in 105 patients with segmental wall motion abnormalities. The echocardiogram identified wall motion characteristics in 87% of the segments that were abnormal by angiography. Two-dimensional echocardiographic studies of patients with acute myocardial infarctions can accurately locate the site of a transmural infarction and estimate the extent of myocardial involvement. Furthermore, serial echocardiographic studies after acute myocardial infarction identify a reduction in the extent of regional dysfunction caused by reduced peri-infarctional ischemia.

Substantial evidence now exists favoring reversible decreases in coronary blood flow leading to transient myocardial ischemia as the mechanism of unstable angina. Experimental studies of regional myocardial function have documented transient abnormalities of left ventricular wall motion after coronary arterial occlusion. Kerber et al. showed that cardiac wall motion abnormalities identified by reflected ultrasound persisted for at least 30 minutes after restoration of normal myocardial perfusion after 45 minutes of coronary occlusion. Heyndrickx et al. reported that interruptions in coronary blood flow of 5 and 15 minutes result in local myocardial dysfunction that lasts more than 3 hours.
Few studies of impaired regional myocardial function induced by transient ischemia in humans have been done.6-8, 27 Several investigators have demonstrated transient regional contraction abnormalities during myocardial ischemia induced by atrial pacing.6, 7, 27 After finding similar changes during exercise-induced ischemia, Sharma et al.28 reported similarities between wall motion changes due to myocardial ischemia induced by atrial pacing and by exercise. More recently, left ventricular performance was studied during spontaneous angina by serial ventriculography. New areas of abnormal wall motion or extension of existing areas of abnormal wall motion were found in all 14 patients. Sublingual nitroglycerin restored both global and regional ventricular function to its previous status in 10 patients. The initial echocardiographic examinations in our present study were obtained during a pain-free interval within 48 hours of the patient's admission. Each patient had angina on the day of hospitalization, and many continued to experience spontaneous pain after admission. The transient wall motion abnormalities identified with these initial studies can be interpreted as segmental dysfunction induced by earlier acute ischemia that had not yet resolved.

The causes for the persistent wall motion abnormalities are unclear. These persistent changes may represent continuing asymptomatic ischemia, as the echocardiographic evidence for persistent segmental dysfunction was restricted to patients who had an unfavorable clinical outcome. This possibility is consistent with reports that many patients with unstable angina progress to coronary occlusion within 4 months,21 and that wall motion abnormalities in patients with unstable angina may be reversed by coronary artery bypass surgery.29, 30 Further echo-
cardiographic studies in similar patients who have a resolution of segmental dysfunction at the predischarge study with time or bypass surgery will be necessary to confirm this hypothesis.

Other explanations for the persistent wall motion abnormalities seem less plausible. These abnormalities may represent small areas of new myocardial necrosis not recognizable by conventional clinical methods. Correlative studies with MB-CK activity curves are needed to determine the degree of sensitivity of two-dimensional echocardiography in detecting small amounts of myocardial necrosis. The persistent wall motion abnormalities in our patients may also represent old myocardial infarction. The screening out of patients who may have suffered a previous myocardial infarction was done as carefully as possible. Nevertheless, previous infarctions causing abnormal ventricular wall motion may have escaped detection by conventional clinical electrocardiographic and scintigraphic means.

Approximately one-third of patients with unstable angina develop subsequent episodes of unstable angina or intractable angina pectoris. Several clinical features have been implicated as predictors of an unfavorable clinical course. However, in patients who respond to medical therapy, predictors of the subsequent clinical course, including severity of angina, have only recently been sought. The increased cost of frequent rehospitalization may be alleviated somewhat by early coronary artery bypass surgery in patients with unstable angina. While surgical intervention soon after unstable angina is effective in decreasing the frequency and severity of angina, it has not reduced the incidence of sudden death and has increased the incidence of myocardial infarction. Thus, in patients who stabilize after unstable angina, any attempt to identify those likely to have an unfavorable clinical course appears warranted.

The demonstration of persistent wall motion abnormalities identified by two-dimensional echocardiography predicts an unfavorable clinical course after hospital discharge in these patients with unstable angina. The persistence of or an increase in the abnormal summed echocardiographic score significantly identified patients who progressed to NYHA class III or IV symptoms. Thus, serial two-dimensional echocardiography appears to be of value in identifying patients who require further evaluation by cardiac catheterization and coronary angiography.

Acknowledgment

The authors express their gratitude to Arvella Peters for technical assistance, to Larry Burden for administrative assistance, and to Lucy Pittman for secretarial help. The authors acknowledge the cooperation and support of the house officers and nurses of the Coronary Care Unit at the VA Medical Center, Dallas, Texas, in the performance of these studies.

References

2. Puri PS: Modification of experimental myocardial infarct size by cardiac drugs. Am J Cardiol 33: 521, 1974
24. Heikkila J, Tabakin BS, Hugenholtz PG: Quantification of...
Identification of transient and persistent segmental wall motion abnormalities in patients with unstable angina by two-dimensional echocardiography.

J V Nixon, C N Brown and T C Smitherman

Circulation. 1982;65:1497-1503
doi: 10.1161/01.CIR.65.7.1497

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1982 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/65/7/1497

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/