Echocardiographic Analysis of Mitral Valve Motion After Acute Myocardial Infarction

By Gary A. Bergeron, M.D., Michael V. Cohen, M.D., Louis E. Teichholz, M.D., and Richard Gorlin, M.D.

SUMMARY
To examine the effects of acute myocardial infarction on mitral valve diastolic velocity, echocardiograms were performed in 18 patients on admission, daily in the Coronary Care Unit, and at 3-day intervals during the remainder of hospitalization. These patients were divided into three groups based on the time interval between onset of symptoms and initial echocardiogram. Five of six patients admitted within 5 hours of onset of myocardial infarction had a triphasic response of mitral valve diastolic velocity with a transient rise above initial values, followed by a fall to below initial values, and then a slow rise during recovery. Seven of eight patients admitted 1-2 days after onset of myocardial infarction had a biphasic response, i.e., a fall from initial values and then a slow rise. Four patients admitted later in the course of myocardial infarction had a monophasic response, i.e., low initial velocity followed by a slow recovery. We conclude that in patients with myocardial infarction the mitral valve diastolic velocity following myocardial infarction shows a triphasic response which may appear biphasic or monophasic depending on the interval between myocardial infarction and admission. The temporal pattern of mitral valve diastolic velocity changes may reflect the dynamic alterations of myocardial function and compliance that are occurring after acute myocardial infarction and during the recovery period.

Additional Indexing Words:
Myocardial function Papillary muscle dysfunction Mitral valve diastolic velocity Left ventricular compliance Catecholamine

ECHOCARDIOGRAPHY HAS BEEN used to examine mitral valve and ventricular wall motion in both normal and pathologic conditions. Several studies have demonstrated echocardiographic abnormalities in left ventricular wall motion during acute myocardial infarction. However, with standard echocardiographic techniques only small segments of the ventricular wall are visualized. In the presence of asymmetrical ventricular contraction with segmental wall injury, the motion of this posterior segment may not be representative of that of the whole ventricle. Therefore, we have applied diagnostic echocardiographic techniques to determine whether dynamic changes in mitral valve motion take place which reflect the changes in over-all left ventricular function that occur in acute myocardial infarction.

Material and Methods
Echocardiograms were recorded from a consecutive group of 26 patients who were admitted to the hospital with the initial diagnosis of possible acute myocardial infarction. Eight patients were excluded from the study, five because a small acoustic window precluded obtaining uniformly satisfactory serial echocardiograms of the mitral valve, and three because of lack of evidence for acute myocardial infarction. Admission of the remaining 18 patients did not always coincide with the first day of infarction; the probable time of infarction was dated from the onset of characteristic chest pain.

The patients varied in age from 43 to 70 years (mean 59 years). Sixteen patients were male and two were female. The diagnosis of infarction was established by the criteria of the World Health Organization. Localization of the infarction was determined electrocardiographically. Thirteen patients had transmural myocardial infarction: 8 anterior; 4 inferior; and 1 posterior. Five patients had nontransmural infarction as supported by serial ECG and enzyme changes. Seven patients had had at least one previous myocardial infarction. One patient with an anterior transmural infarction died in cardiogenic shock 10 days following admission. All patients were in normal sinus rhythm with the exception of one who had ventricular bigeminy on two occasions, and data collected at these times were not used in this analysis. Heart rate was determined from the average R-R interval of an
ECG recorded simultaneously with the echocardiogram. Additionally, patients were ausculted daily in the Coronary Care Unit by the same observer. To provide a control frame of reference concerning reproducibility, echocardiograms were performed over a 2–3 week interval in nine normal subjects.

Initial echocardiograms were recorded within 5 hours of admission to the Coronary Care Unit. Repeat studies were done at daily intervals in the Coronary Care Unit and at three to four day intervals after transfer until discharge from the hospital.

With the patients in the recumbent position, echocardiograms were recorded on Polaroid film using a Unirad ultrasonicoscope and a 2.25 MHz transducer. The ultrasound transducer was placed in the fourth or fifth left intercostal space close to the sternum with care to mark the transducer position for each patient during follow-up studies. To determine if myocardial infarction induces distinctive patterns of mitral valve motion, echocardiograms of the anterior leaflet of the mitral valve were analyzed for initial diastolic closing velocity (fig. 1, interval E-F) and amplitude (fig. 1, interval C-E). The velocity represented by the diastolic slope was expressed in mm/sec. The amplitude of the mitral valve echogram represents the entire antero-posterior excursion of the anterior leaflet of the mitral valve and was measured in mm. The data were analyzed for serial changes. The limits of normal for the diastolic slope and excursion were accepted as 80–170 mm/sec and 20–33 mm, respectively. 

All echocardiograms were examined by two observers and independent measurements of diastolic velocity were insignificantly different. All studies were performed with the informed consent of the patients.

Results

Patients who had sustained an acute myocardial infarction were divided into three groups based on the interval between clinical onset of symptoms and the first echocardiogram. Group A consisted of six patients admitted within five hours of the onset of symptoms. The average mitral valve diastolic velocity on admission of the group was 79 ± 6 mm/sec (table 1, fig. 2). In five of the six patients, this velocity demonstrated a transient rise, a subsequent fall and finally a slow increase. The mitral valve diastolic velocity at the time of discharge from the hospital was always greater than on admission. There was one patient in this group with clinically suspected papillary muscle dysfunction who did not demonstrate this pattern. Instead, the velocity continued to rise during his hospitalization (top left pattern, fig. 2).

Table 1

<table>
<thead>
<tr>
<th>Group</th>
<th>No. patients</th>
<th>Time interval following myocardial infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>≤ 5 hours (mm/sec)</td>
</tr>
<tr>
<td>A</td>
<td>6</td>
<td>79 ± 6.0</td>
</tr>
<tr>
<td>B</td>
<td>8</td>
<td>—</td>
</tr>
<tr>
<td>C</td>
<td>4</td>
<td>—</td>
</tr>
</tbody>
</table>

Circulation, Volume 51, January 1975
Group B comprised eight patients admitted 6 hours to 2 days after sustaining a myocardial infarction. Following admission, mitral valve diastolic velocity underwent a biphasic response with a fall in seven of the eight patients from an initial value averaging 91 ± 1.2 mm/sec and then a slow rise during the latter part of hospitalization (table 1, fig. 3). The one patient in group B who did not demonstrate this pattern developed an apical systolic murmur in the hospital and was thought to have papillary muscle dysfunction.

Group C consisted of four patients admitted late in the course of their myocardial infarction. Mitral valve diastolic velocity in this group demonstrated a monophasic response with an average initial value of 72 ± 5.0 mm/sec and a gradual increase during hospitalization (table 1, fig. 4).

Therefore, if the first echocardiogram on individual patients were recorded within a few hours following the onset of myocardial infarction, the changes in mitral valve diastolic velocity followed a triphasic response with time; this pattern appeared biphasic or monophasic depending on the interval between infarction and hospital admission (table 1, fig. 5).

In the majority of the 18 patients with proven myocardial infarction, mitral valve velocity appeared to be initially depressed (83 ± 6.7 mm/sec) and was significantly faster (105 ± 6.7 mm/sec) at the time of discharge from the hospital (fig. 6).
The variations in mitral valve velocity with time in a control group of nine males without known heart disease is seen in figure 7. No significant difference in the values in respect to time is seen in the group as a whole.

The extent of mitral valve excursion as well as its velocity was studied in all patients. In addition, the presence of a new apical systolic murmur was recorded. There was no significant change in mitral valve excursion from admission to discharge in any of the patients. Eight of the patients had new systolic murmurs noted in the Coronary Care Unit. However, only one of these had an abnormally rapid mitral valve velocity (180 mm/sec). Despite this one patient, there was no consistent correlation of increased mitral valve diastolic velocity and a new apical systolic murmur (table 2). The seven patients who had previously sustained a myocardial infarction were evenly distributed among the three groups. In addition, the daily heart rates and medications of every patient in each group were variable and there was no apparent correlation with mitral valve velocity.

Discussion

This study suggests that mitral valve diastolic velocity follows a characteristic triphasic pattern after acute myocardial infarction. In addition, an initial slow diastolic velocity with a subsequent increase prior to discharge from the hospital was noted (fig. 6). Serial echocardiographic analysis of posterior wall motion during the course of acute myocardial infarction demonstrates similar early depression and later recovery.2-4

Table 2

<table>
<thead>
<tr>
<th></th>
<th>Echocardiographic Abnormalities</th>
<th>Systolic Murmur</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Normal</td>
<td>No murmur (N = 10)</td>
</tr>
<tr>
<td>Mitral valve velocity (&gt; 170 mm/sec)</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Excursion (&gt; 33 mm)</td>
<td>0</td>
<td>3</td>
</tr>
</tbody>
</table>

Figure 6

Admission and discharge mitral valve velocities of 18 patients hospitalized with myocardial infarction. The open circles represent the mean mitral valve velocity on admission and discharge — the difference is significant (P < .05).

Figure 7

Temporal course of mitral valve diastolic velocity in nine normal subjects. The tracings around the periphery show the individual patterns. The central graph illustrates the grouped data. Horizontal bars indicate the time interval over which the mitral valve velocity is averaged. Vertical bars represent two standard errors of the mean.
There are many possible explanations for our observations. Factors influencing mitral valve motion are multiple and complex. Feigenbaum has stated that mitral valve motion is a function of mitral valve flow as well as the interrelationship between left atrial and left ventricular pressures. The left ventricular pressure-volume relationship is determined by ventricular geometry and compliance. Factors which alter these parameters would also be expected to affect mitral valve motion. Ventricular geometry and compliance are both altered in the acute phase of myocardial infarction. Therefore, one would anticipate changes in mitral valve diastolic velocity during the course of acute myocardial infarction.

A possible hypothesis to explain our findings might include the following. The early increase in the rate of mitral valve diastolic velocity noted in group A may in part be related to an increased compliance associated with acute aneurysmal bulging of the infarct. In addition, catecholamine release at this time may affect diastolic left ventricular mechanics and thus influence mitral valve motion. Pathologic changes seen in acute myocardial infarction begin with central necrosis, edema, and leucocytic infiltration in the infarcted area. This acute healing phase is likely to lead to increased stiffness of the infarcted area with a decrease in over-all ventricular compliance. The fall in mitral valve diastolic velocity seen in group A may be the result of a decreased compliance as well as gradual lessening of the catechol effect while contractility is still depressed. During the recuperative phase, mitral valve diastolic velocity increases in the majority of patients (fig. 6). Ventricular compliance most likely begins to slowly increase as the edema and leucocytic infiltration in the abnormal area subside. This hypothesis is illustrated in figure 8. In spite of the attractive nature of this hypothesis, there is at present a lack of experimental data in human beings to support it.

We found no apparent correlation between the presence of a new apical systolic murmur and magnitude of mitral valve diastolic velocity. Therefore, we are unable to confirm the conclusion that the echocardiographic analysis of mitral valve motion during acute myocardial infarction is a reliable method for documenting or following variations in papillary muscle dysfunction in acute myocardial infarction. This may be due, in part, to a difference in the patient populations between the two studies.

We have demonstrated in this group of patients that mitral valve diastolic velocity appears to follow a characteristic sequence from the onset of infarction to discharge from the hospital which is probably related to the dynamic alterations of myocardial function and compliance that are occurring at this time. Further investigation will be necessary to assess the clinical usefulness of these findings.

References

22. TALBURG VK, DE PASQUALE NP, BURCH GE: The echocardiogram in papillary muscle dysfunction. Am Heart J 83: 12, 1972
Echocardiographic analysis of mitral valve motion after acute myocardial infarction.

G A Bergeron, M V Cohen, L E Teichholz and R Gorlin

Circulation. 1975;51:82-87
doi: 10.1161/01.CIR.51.1.82

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1975 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/51/1/82

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/