Two-dimensional Echocardiographic Recognition of Ruptured Chordae Tendineae

GARY S. MINTZ, M.D., MORRIS N. KOTLER, M.D., BERNARD L. SEGAL, M.D., AND WAYNE R. PARRY

SUMMARY Real-time, phased-array, two-dimensional echocardiographic studies identified ruptured chordae tendineae in five patients; four patients had a flail mitral valve and one had flail mitral and tricuspid valves. The characteristic abnormality was a rapid systolic motion of the involved leaflet beyond the line of valve closure into the atrium. The maximal abnormal systolic motion was greatest at the tip of the leaflet with a loss of the normal coaptation point. By contrast, the two-dimensional echocardiographic feature of mitral valve prolapse is an abnormal systolic motion that is maximal in the body of the leaflet with intact leaflet coaptation. Thus, two-dimensional echocardiography can identify flail mitral and tricuspid valves and is useful in distinguishing ruptured chordae from valvular prolapse.

M-MODE ECHOCARDIOGRAPHY may be helpful in the diagnosis of mitral regurgitation secondary to ruptured chordae tendineae. Several investigators have described echocardiographic findings for the diagnosis of ruptured chordae tendineae. These include systolic intracavitary left atrial echoes, holosystolic mitral valve prolapse, systolic mitral leaflet fluttering, chaotic diastolic anterior motion of a flail posterior mitral valve leaflet, or coarse diastolic fluttering of a flail anterior mitral valve leaflet. Because of the limited number of patients reported in the literature, the sensitivity and specificity of these findings have not been conclusively determined. Thus, the preoperative diagnosis of a flail mitral valve is often based on hemodynamic and angiographic findings. Furthermore, criteria for the M-mode diagnosis of flail tricuspid leaflets have not been described.

Real-time, two-dimensional echocardiography can provide spatial information concerning intracardiac structures that cannot be obtained by M-mode echocardiography. The purpose of this report is twofold: 1) to demonstrate the ability of real-time, two-dimensional echocardiography to visualize flail mitral and tricuspid valves secondary to ruptured chordae tendineae and 2) to demonstrate that two-dimensional echocardiography may be more sensitive than M-mode echocardiography in diagnosing ruptured chordae tendineae.

Material and Methods

M-mode and two-dimensional echocardiograms were recorded in five patients in whom the surgical findings later confirmed the diagnosis of flail mitral or tricuspid valves. Four patients had flail mitral valves and one patient had flail mitral and tricuspid valves. Clinical data based on symptoms, physical findings, electrocardiograms, and chest X-rays were analyzed. The clinical, echocardiographic, angiographic, and anatomic data on these five patients form the basis of this report.

M-mode echocardiographic studies were performed in the supine or left lateral decubitus position utilizing a Smith Kline Ekoline 20A ultrasonoscope with a 2.25 MHz medium (7.5 cm) internally focused transducer. Permanent records were obtained with an Irex 101 Continutrace Recorder. Slow and rapid full M-mode echocardiographic sweeps from the aortic valve leaflets to below the level of the mitral valve leaflets were performed in each patient. Careful attention was paid to transducer position and gain setting to avoid missing small valvular or intra-atrial cavitory abnormalities. Large scale mitral valve echograms were recorded through several cardiac cycles to magnify the details of mitral valve motion. Similar attention was directed to assessing tricuspid valve motion.

Two-dimensional echocardiographic studies were performed using a Grumman Health Systems RT-400 phased-array sector scanner. Serial excitation of the 32 transducer elements produces an acoustic wave which is directed toward the target. The hand-held focusable linear array generates an 80° field in a circular sector format to produce tomographic images of the heart in real time. A digital computer controls the scan format. The images are displayed on a television screen and, via an optical link, permanent records of images are made on one-half inch video tape for later playback and analysis. A simultaneously recorded electrocardiogram provided a time reference within the cardiac cycle specifically to identify the mid and late diastolic and systolic frames shown in the illustrations. Illustrations presented in this manuscript were photographed from single
frame video tape images using a Tektronix camera and Polaroid film. Such illustrations suffer from the loss of visual integration of motion that normally accompanies real-time imaging and a degradation of image quality caused by photographing a single field of a complete video tape frame that normally consists of two interlaced fields. The long axis position was used to evaluate the integrity of the mitral valve. This view includes the aorta and the aortic valves, the left atrium, anterior and posterior mitral leaflets, interventricular septum, left ventricular cavity, and left ventricular posterior wall. The long axis is analogous to the typical M-mode echocardiographic sweep. The entire mitral valve was carefully examined by sweeping the two-dimensional echocardiographic plane laterally to medially. The tricuspid valve was evaluated using a transducer position through the long axis of the right atrium, tricuspid valve, and right ventricle.

Cardiac catheterization with ventricular angiography was performed in four of five patients. The degree of atrioventricular valve incompetence was judged qualitatively by established criteria on a scale of 1 to 4 (1 represents minimal regurgitation, 2 indicates more than minimal regurgitation without full atrial opacification, 3 indicates full atrial opacification after more than one systole, and 4 indicates full atrial opacification on the first systole).

All five patients underwent cardiac surgery. Four patients had a mitral valve replacement, and one patient had combined mitral and tricuspid valve replacements.

Table 1: Clinical Findings

<table>
<thead>
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<th>Physical Findings</th>
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<tr>
<td>Loud holosystolic crescendo dekrecendo murmurs</td>
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<td>SS</td>
<td>5/5</td>
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<tr>
<td>S4</td>
<td>2/5</td>
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<td>Accentuated pulmonic component of the second sound</td>
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Electrocardiogram

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<tbody>
<tr>
<td>Atrial fibrillation</td>
<td>1/5</td>
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<tr>
<td>ST-T changes</td>
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Chest X-ray

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<th>Cardiomegaly</th>
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<td>Pulmonary edema</td>
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<th>Findings</th>
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<td>Pulmonary edema</td>
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Results

All five patients (all males, ages 19-59) presented with signs and symptoms of progressive congestive heart failure. Three patients had no previous history of heart disease; one patient was known to have mitral valve prolapse. The physical, electrocardiographic, and X-ray findings are summarized in table 1.

M-mode Echocardiographic Findings

The M-mode echocardiographic data are listed in table 2. Systolic intraventricular left atrial echoes, systolic mitral valve fluttering, coarse diastolic anterior mitral leaflet fluttering, chaotic anterior diastolic posterior mitral leaflet motion, or systolic mitral valve prolapse were tabulated. No one finding was present in all patients, and in one patient none of these findings were present. In addition, one patient had an exaggerated left atrial posterior wall motion of 1.4 cm (normal 1.0 cm). All patients had vigorous septal and left ventricular posterior wall motions. Example echograms from one patient with a flail anterior mitral valve (fig. 1) and from another with a flail posterior mitral valve leaflet (fig. 2) are shown.

In the one patient who also had a flail tricuspid valve, the diastolic right ventricular dimension (1.8 cm) was normal. Septal motion was also normal. The M-mode echogram of the tricuspid valve only showed excessive systolic tricuspid valve leaflet separation (fig. 3).

Two-Dimensional Echocardiographic Findings

Two patients were found to have a flail anterior mitral valve leaflet, and three patients were found to have a flail posterior mitral valve leaflet. One patient with a flail anterior mitral leaflet was also found to have flail anterior and posterior tricuspid valve leaflets.

In the two patients with a flail anterior mitral valve leaflet, the two-dimensional echocardiogram image in the longitudinal plane showed superior and posterior systolic motion of the anterior leaflet to a point beyond the line of the mitral valve closure. The tip of the anterior leaflet bent perpendicular to the body and projected into the left atrium. The abnormal motion was maximal at the leaflet's tip. These abnormalities are demonstrated in figure 4.

Table 2: The Echocardiographic, Angiographic, and Pathological Findings

<table>
<thead>
<tr>
<th>Patient</th>
<th>M-mode echocardiographic findings</th>
<th>Diastole</th>
<th>2-D echo findings</th>
<th>Angiographic findings</th>
<th>Surgical/pathological findings</th>
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<tr>
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<td>LA echoes</td>
<td>MV flutter</td>
<td>MV prolapse</td>
<td>Chaotic PML motion</td>
<td>Coarse AML flutter</td>
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Abbreviations: LA = left atrium; MV = mitral valve; PML = posterior mitral leaflet; AML = anterior mitral leaflet; ATL + = anterior tricuspid leaflet; PTL = posterior tricuspid leaflet; CT = chordae tendineae; MR = mitral regurgitation, TR = tricuspid regurgitation.
In the three patients with a flail posterior mitral valve leaflet, the two-dimensional echocardiographic image in the longitudinal plane showed systolic motion of the posterior leaflet through a 180° arc into the left atrium (fig. 5). The abnormal motion was again maximal at the tip of the leaflets.

In the one patient with flail anterior and posterior tricuspid valve leaflets, the two-dimensional echocardiographic image showed systolic motion of both leaflets through 180° arcs into the right atrium (fig. 6). The motion of the tricuspid valve was maximal at the tip of the leaflets.

Cardiac Catheterization Results

In the four patients with a flail valve, left ventriculography showed severe (4+) mitral regurgitation. In the one patient who also had a flail tricuspid valve, right ventriculography showed moderate (3+) tricuspid insufficiency.

Surgical and Pathological Findings

The surgical and pathological findings are shown in table 2. Two patients had flail anterior mitral leaflets (one from spontaneous rupture and one from bacterial endocarditis)
and three patients had flail posterior mitral valve leaflets (all spontaneous rupture). One patient with a flail anterior mitral leaflet secondary to *Staphylococcal aureus* endocarditis also had flail anterior and posterior tricuspid valve leaflets as part of the pathological process.

**FIGURE 3.** An M-mode echogram from a patient with flail anterior (ATL) and posterior (PTL) tricuspid leaflets. The echocardiogram shows excessive systolic separation of the two leaflets. PA = pulmonic area, MF = medium frequency.

**FIGURE 4.** Two-dimensional echograms from a patient with a flail anterior mitral leaflet (patient 3 in table 2). Each single frame is accompanied by a labeled, idealized schematic diagram. Panel A is recorded in mid-diastole. The anterior (aml) and posterior (pml) mitral leaflets open into the left ventricle (LV). The aortic valve is closed. Panel B is recorded at end-diastole. The mitral leaflets are moving toward the line of mitral valve closure. Panel C is recorded during systole. The tip of the aml has bent perpendicular to the body of the aml; it has moved beyond the line of mitral closure into the left atrium (LA). The abnormal motion is maximum at the tip of the leaflet. The normal point of coaptation of the aml and pml is lost. AO = aorta.

**Discussion**

Although acute severe mitral regurgitation secondary to a flail mitral valve is not a common occurrence, it requires aggressive surgical management. Thus, the diagnosis must be made as accurately and as expeditiously as possible. Numerous M-mode echocardiographic findings in patients with flail mitral valves have been described.\(^1^6\) The intracavitary left atrial echoes presumably originate from the ruptured chordae or the flail valve that has prolapsed into the left atrium during systole. The high frequency systolic mitral valve fluttering is attributed to the high velocity regurgitant jet flowing across the freely moving leaflet margin that is held perpendicular to the flow by the remaining intact chordae.\(^2\) By contrast, coarse diastolic anterior leaflet fluttering is explained by the lower velocity antegrade diastolic flow that runs parallel to the untethered mitral leaflet edge analogous to a sail flapping in the wind.\(^3\)

Some of these findings may be seen in other cardiac abnormalities. Moving intracavitary left atrial echoes may be seen in atrial clot or myxoma.\(^11\) Holosystolic prolapse is one of the presentations of the mitral valve prolapse syndrome.\(^8\) Systolic mitral valve fluttering has been seen in patients with mitral prolapse and bacterial endocarditis;\(^12\) the possible coexistence of a flail leaflet in that series was not clarified. Anterior mitral leaflet fluttering in diastole may be seen in aortic insufficiency;\(^13\) although usually fine, this fluttering may occasionally have coarse characteristics.\(^14\) Coarse diastolic anterior leaflet fluttering may be seen in atrial flutter or fibrillation (dysrhythmias that may occur in patients with mitral regurgitation, as was seen in one of our patients).\(^14\) Generally, however, this type of fluttering is very...
FIGURE 5. Two-dimensional echograms from a patient with a flail posterior mitral leaflet (patient 1 in table 2). Panel A is recorded in mid-diastole. The anterior (aml) and posterior (pml) mitral leaflets open into the left ventricle (LV). The aortic valve is closed. Panel B is recorded at end-diastole. The mitral leaflets are moving toward the line of mitral valve closure. The aortic valve is still closed. Panel C is recorded during systole. The pml has moved past the line of mitral closure into the left atrium (LA). The normal point of coaptation of the aml and the pml is lost. The aortic valve is open.

FIGURE 6. Two-dimensional echograms from a patient (patient 4 in table 2) with flail anterior (atl) and posterior (ptl) tricuspid leaflets. Panel A is recorded in mid-diastole. The atl and ptl open into the right ventricle (RV). Panel B is recorded at end diastole. The atl and ptl are in the normal line of tricuspid valve closure. Panel C is recorded during systole. Both the atl and ptl have moved beyond the line of tricuspid valve closure into the right atrium (RA). The maximum abnormal motion is at the tip of each leaflet.
Coarse and rhythmic. In addition, diastolic fluttering has been reported to occur in left-to-right shunts in patients, for example, following balloon atrial septostomy. The diagnosis of flail mitral valves by the M-mode technique must be made by inference since the actual flailing is rarely seen. Furthermore, each of the findings may be mimicked by other pathologic states. In our five patients with surgically proven flail mitral valves secondary to ruptured chordae tendineae, no one finding was absolutely sensitive for this diagnosis. In fact, one patient had a normal M-mode mitral valve echogram. In the one patient with concomitant flail tricuspid valves, the M-mode echogram did not provide conclusively diagnostic information. Indeed, there was no evidence of right ventricular volume overload.

By contrast, real-time, two-dimensional echocardiography allowed direct visualization of the flail mitral valve leaflet in each of the five patients. The flail mitral valve moved past the line of mitral valve closure into the left atrium. Because the abnormal motion was greatest at the leaflet's tip, the normal systolic coaption point of the anterior and posterior leaflets was lost. These findings have not been reported in other diseases of the mitral valve. Furthermore, this sequence of events was clearly different from that of valvular prolapse. The two-dimensional criteria that Gilbert et al. have established for mitral valve prolapse include 1) excessive posterior closure of the coaptation point of the mitral leaflets, 2) an accentuation of the angle formed in systole by the posterior aortic wall and the anterior mitral valve, 3) systolic curling of the posterior mitral ring on its adjacent myocardium, and 4) superior arching of the anterior and/or posterior mitral leaflets above the mitral ring. The coaptation point remained intact, and the maximal abnormal motion occurred in the body of the leaflets. An example of mitral valve prolapse is shown in figure 7.

The two-dimensional echocardiographic picture of the flail anterior and posterior tricuspid valves resembled that recorded by Kisslo in another case of a flail tricuspid valve. In that case the anterior leaflet was seen to move through a 180° arc into the right atrium during systole. Abnormal valve motion is variable depending on the number of chordae that are involved.

Technique is also important. With the transducer in the long axis of the left ventricle, it is essential to rock the transducer laterally to medially to visualize the maximal abnormal mitral leaflet motion. Similarly, with the transducer in the long axis of the right ventricle, it also is essential to rock the transducer to visualize the maximal abnormal tricuspid leaflet motion.

In this small study of five patients, real-time, two-dimensional echocardiography proved to be more sensitive than M-mode echocardiography for the diagnosis of flail atrioventricular valves. Although the absolute sensitivity and specificity of two-dimensional echocardiography remain to be determined, this technique should provide the clinician with a rapid, easy, and reliable noninvasive tool for the diagnosis of ruptured chordae tendineae.

**Figure 7.** Two-dimensional echograms from a patient with mitral valve prolapse. Panel A is recorded in mid-diastole. The anterior (aml) and posterior (pml) mitral leaflets open into the left ventricle (LV). Panel B is recorded at end-diastole. The aml and pml are in the normal line of mitral valve closure. Panel C is recorded during systole. The aml and pml have arched into the left atrium (LA) beyond the line of mitral valve closure. The point of mitral valve leaflet coaptation is intact. The abnormal motion is maximum in the body of the leaflets.
Prevalence and Characteristics of Disproportionate Ventricular Septal Thickening in Patients with Coronary Artery Disease

BARRY J. MARON, M.D., DANIEL D. SAVAGE, M.D., PH.D., CHESTER E. CLARK, M.D., WALTER L. HENRY, M.D., ZEEV VLODAVER, M.D., JESSE E. EDWARDS, M.D., AND STEPHEN E. EPSTEIN, M.D.

SUMMARY Echocardiographic or necropsy studies were performed in 151 patients with coronary artery disease. Prevalence of disproportionate septal thickening (septal to free wall ratio ≥ 1.3) was 11%. An abnormally increased septal-to-wall ratio in these patients had two principal etiologies. First, it was a manifestation of genetically transmitted hypertrophic cardiomyopathy, as evidenced by disproportionate septal thickening in first degree relatives. Second, it was due to disproportionate septal thickening which did not appear to be a manifestation of genetically transmitted hypertrophic cardiomyopathy. This latter conclusion was suggested by negative echocardiographic studies in some families of patients with both coronary artery disease and disproportionate septal thickening. In addition, numerous disorganized cardiac muscle cells, characteristically present in patients with genetically transmitted hypertrophic cardiomyopathy, were absent from the septum of all patients with disproportionate septal thickening studied at necropsy. Although the mechanism responsible for this secondary type of disproportionate septal thickening is unknown, our results indicate that the presence of disproportionate septal thickening in a patient with coronary artery disease does not, per se, indicate the coexistence of genetically transmitted hypertrophic cardiomyopathy.

ASYMMETRIC SEPTAL HYPERTROPHY (ASH) or hypertrophic cardiomyopathy, is a genetically transmitted disease of cardiac muscle that is characterized by disproportionate thickening of the ventricular septum with respect to the posterobasal left ventricular wall. In previous studies we have determined that disproportionate septal thickening also may occur in some patients with acquired or congenital heart disease, and is usually secondary to the patient's particular hemodynamic state rather than a primary manifestation of an etiologically separate cardiac disorder (i.e., genetically transmitted hypertrophic cardiomyopathy).

Clinically evident hypertrophic cardiomyopathy with left ventricular outflow obstruction may occur in patients with coronary artery disease, although the prevalence of this association is unknown. Furthermore, it is not known whether the disproportionate septal thickening in such patients is merely secondary to coronary artery disease or represents a coexisting primary cardiomyopathy. The latter may be the major cause of symptoms that otherwise would be attributable to the underlying coronary artery disease. Hence, the present study was undertaken to evaluate: 1) the prevalence of disproportionate septal thickening in patients with coronary artery disease and 2) the frequency with which disproportionate septal thickening in such patients is a manifestation of genetically transmitted hypertrophic cardiomyopathy.
Two-dimensional echocardiographic recognition of ruptured chordae tendineae.
G S Mintz, M N Kotler, B L Segal and W R Parry

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