Incomplete Mitral Leaflet Closure in Patients with Papillary Muscle Dysfunction

ROBERT W. GODLEY, M.D., L. SAMUEL WANN, M.D., EDWIN W. ROGERS, M.D.,
HARVEY FEIGENBAUM, AND ARTHUR E. WEYMAN, M.D.

SUMMARY Clinical acceptance of an association between papillary muscle dysfunction and mitral regurgitation is widespread, despite the lack of objective support. To evaluate a possible association, we performed echocardiographic examinations on 22 patients with prior myocardial infarction and clinical evidence of papillary muscle dysfunction, 40 patients with prior myocardial infarction and no clinical evidence of papillary muscle dysfunction, and 20 normal subjects. There was a unique pattern of incomplete mitral leaflet closure in a high percentage (91%) of infarct patients with mitral regurgitation. In these patients, one or both leaflets were effectively arrested within the cavity of the left ventricle during ventricular systole. Dyskinetic wall motion in the region immediately surrounding one of the papillary muscles was present in 23 of 24 patients (96%) with demonstrated incomplete closure. This study provides the first objective evidence that de novo mitral regurgitation in patients with prior myocardial infarction is due to dyskinesis involving the left ventricular myocardium beneath one of the papillary muscles, producing increased tension on the mitral leaflets and preventing normal closure.

MITRAL INSUFFICIENCY has been recognized for many years to result from disorders of the mitral leaflets, chordae tendineae, annulus and related structures. In 1963, Burch and colleagues directed attention to the possible etiologic role of papillary muscle dysfunction in producing mitral valve incompetence. They suggested two mechanisms by which this might occur. First, ischemia or fibrosis of a papillary muscle might prevent normal contraction. This would weaken systemic support for the valve leaflets and result in leaflet evasion or prolapse into the left atrium. Alternatively, ischemia or infarction of the left ventricular myocardium at the base of a papillary muscle might produce dyskinesis in this region. This would pull the papillary muscle away from the valve orifice, increasing tension on the leaflets and preventing complete closure.

Angiographic studies have shown the occurrence of mitral valve prolapse in association with ischemic disease involving a papillary muscle, supporting the occurrence of the first of these two phenomena. Angiography is poorly suited to demonstrate displacement of a leaflet downward into the cavity of the ventricle. As a result, the alternative mechanism, leaflet arrest with resultant malapposition and valvular incompetence, has not been shown to occur.

Cross-sectional echocardiography is a noninvasive method of assessing mitral leaflet motion through the cardiac cycle in multiple tomographic planes. As such, it should be for examining the patterns of mitral leaflet motion in patients with clinical papillary muscle dysfunction. The purposes of this study were (1) to determine whether abnormal leaflet motion or closure patterns could be observed by the cross-sectional echocardiogram in patients with clinical evidence of papillary muscle dysfunction, and (2) if abnormal motion did occur, how it related to the mechanisms proposed for leaflet malapposition with papillary muscle dysfunction.

Materials and Methods

Cross-sectional echocardiographic studies of the mitral valve and left ventricle were performed in 22 patients with prior myocardial infarction and clinical evidence of papillary muscle dysfunction (group 1), 40 patients with prior myocardial infarction and no clinical evidence of papillary muscle dysfunction (group 2), and 20 normal subjects (group 3). The study group represents consecutive patients with the diagnosis of myocardial infarction referred for a two-dimensional echocardiogram. The diagnosis of papillary muscle dysfunction was based on the occurrence of a new systolic murmur consistent with mitral regurgitation together with historical and electrocardiographic evidence of myocardial infarction temporally related to the onset of the murmur. The diagnosis of myocardial infarction was based on a typical clinical history and electrocardiographic evidence of transmural infarct. The location of the infarct was based on standard electrocardiographic criteria. Group 1, patients with evidence of both myocardial infarction and papillary muscle dysfunction, consisted of 20 males and two females, ages 46–80 years (mean 62 years). Four patients had anteroseptal infarcts, six had anteroseptal and inferoseptal infarcts, and 12 had inferoposterior infarcts. Three of these 22 patients had cardiac catheterization, and in each case mitral regurgitation was demonstrated. The mean left ventricular internal dimension was 5.5 cm in group 1.
Group 2, patients with myocardial infarction and no clinical evidence of papillary muscle dysfunction, included 36 males and four females, ages 42–84 years (mean 60 years). Twenty-three patients had anteroapical infarcts, seven had anteroseptal infarcts and 10 had inferoposterior infarcts. Nineteen of these 40 patients had cardiac catheterization, and in no case was mitral regurgitation demonstrated. The mean left ventricular internal dimension was 5.4 cm.

Group 3, the normal subjects, contained 14 males and six females, ages 28–52 years (mean 39 years). These subjects had no historical or clinical evidence of heart disease and had not undergone cardiac catheterization.

No patient had physical or historical evidence of mitral regurgitation before infarction, nor did any patient have a history of rheumatic heart disease or mitral valve prolapse syndrome.

All cross-sectional studies were performed using a commercially available mechanical sector scanner (Smith-Kline Instrument, Eko-Sector 1). This device consisted of a modified Echoline 20A echograph with a pulse repetition rate of 4 kHz. Two types of scanner probes were used during the course of this study: an oscillating 30° probe and an 82° rotary probe. The 30° probe contained a 2.25-MHz transducer focused at 5 cm and swept at 30 cycles (60 fields/sec.) The 82° rotary probe contained four 2.25-MHz transducers focused at 5 cm and rotated at 60 fields/sec. The sweep rate of both systems resulted in 54 lines of information per field. Two consecutive fields could be simultaneously displayed, generating a total of 108 lines per frame at 30 frames/sec. Cross-sectional images were recorded via direct ultrasonic recordings on a Sanyo VTC 7100 videotape recorder. This recording format introduced little image persistence, and hence limited persistence artifact, and improved leaflet definition. The cross-sectional images could be viewed during the examination or subsequently from the videotape in real-time, slow-motion, or single-frame formats. Individual frames were converted to hard copy using a standard Polaroid photographic system.

The cross-sectional studies were performed with patients in either the supine or 30° left lateral position. Each patient was examined in the standard parasternal long- and short-axis views and the apical four-chamber view. Only patients in whom all views were successfully obtained were included in the study. Two infarct patients in whom satisfactory recordings were not obtained in one of the three views were not included in the final data.

The mitral valve echocardiogram was analyzed in each of these views to determine the point of leaflet coaptation and the peak systolic position of the bodies of the mitral leaflets relative to the plane of the atrioventricular ring. The coaptation point was defined as the point at which any portion of one mitral leaflet first apposed the other during systolic closure. Incomplete closure was defined as failure of one or both of the leaflets to reach the plane of the atrioventricular ring at the point of its peak superior systolic movement. The leaflets were considered to prolapse if a portion of one or both of the mitral valve leaflets everted into the left atrium, crossing the plane of the atrioventricular ring, during systole.

Left ventricular wall motion was analyzed using multiple imaging planes, including the left ventricular long-axis with the transducer on both parasternal and apical locations, and multiple short-axis scans from the atrioventricular ring to the smallest short-axis scan that could be obtained proximal to the cardiac apex. Wall motion in each plane was categorized as hyperkinetic, normal, hypokinetic or dyskinetic.

Results

Analysis of the pattern of mitral leaflet motion and systolic leaflet position revealed no characteristic abnormality in either the long- or short-axis views. In the apical four-chamber view, however, one or both of the mitral valve leaflets remained partially within the left ventricular cavity during systole in 23 of the 82 patients. Mitral leaflet motion in these cases appeared to be arrested before the expected plane of valve closure. This incomplete closure occurred in 20 of the 22 infarct patients with papillary muscle dysfunction and in three of the 40 infarct patients without papillary muscle dysfunction, but was not observed in any of the normal subjects. Figure 1 is an example of the normal pattern of mitral leaflet closure in the four-chamber view. At end-systole, the leaflets lie in the plane of the atrioventricular ring. Figure 2 is an example of mitral leaflet closure from a patient with incomplete mitral leaflet closure. In this case, during systole, the leaflets remain partially within the left ventricular cavity and fail to reach the atrioventricular ring, resulting in incomplete systolic closure of the mitral valve. Figure 3 summarizes the relationship of incomplete mitral valve leaflet closure and the presence or absence of papillary muscle dysfunction in the patients with myocardial infarction (groups 1 and 2).

To determine the relationship of abnormal mitral leaflet closure to myocardial dysfunction, the pattern of left ventricular wall motion in the region surrounding the papillary muscles was then analyzed in each case. Of the 62 patients with myocardial infarction, 24 had dyskinetic, five had hypokinetic and 33 had normal left ventricular wall motion. Of the 24 patients with dyskinetic wall motion, 23 had incomplete mitral valve leaflet closure and one had normal closure. Of the 38 patients with hypokinetic or normal wall motion, none had incomplete mitral valve leaflet closure. Figure 4 shows the relationship between incomplete mitral valve leaflet closure, clinical papillary muscle dysfunction and abnormal wall motion at the base of a papillary muscle in the study population. Twenty of the 22 patients with papillary muscle dysfunction had both incomplete mitral leaflet closure and dyskinesis in the area of one papillary muscle. Thirteen had involvement of the posteromedial papillary muscle and seven had involvement of the anterolateral papillary muscle. In two patients, clinical evidence of papillary muscle dysfunction was not accompanied by abnor-
Figure 1. Still frame in the four-chamber view taken at end-systole from a normal patient and the corresponding schematic diagram. In systole, the mitral valve leaflets lie within the plane of the atrioventricular ring. LV = left ventricle; LA = left atrium; RV = right ventricle; RA = right atrium; AML = anterior mitral leaflet; PML = posterior mitral leaflet.

Figure 2. Still frame in the four-chamber view taken at end-systole from a patient with incomplete mitral leaflet closure and the corresponding schematic diagram. At end-systole, the anterior mitral leaflet (AML) fails to reach the atrioventricular ring, suggesting incomplete systolic closure of the mitral valve. LV = left ventricle; LA = left atrium; RV = right ventricle; RA = right atrium; PML = posterior mitral leaflet.

Figure 3. The relationship of mitral valve leaflet closure to the presence or absence of clinical papillary muscle dysfunction in the patients with prior myocardial infarction.

In contrast, 32 of the 40 patients with myocardial infarction and no clinical evidence of papillary muscle dysfunction (group 2) had normal wall motion in the area of both papillary muscles, five had hypokinesis and three had dyskinesis in the region of a single papillary muscle. The three patients with dyskinesis were also the three with incomplete mitral leaflet closure but without clinical evidence of mitral regurgitation. In each of these three patients, the dyskinetic wall motion involved the posteromedial papillary muscle.

None of the normal subjects (group 3) had either abnormal mitral leaflet closure or abnormal wall motion.

The anterior mitral valve leaflet was involved in every case in which there was abnormal mitral leaflet closure. Associated abnormal posterior leaflet closure
was observed in 12% of the cases. Abnormal leaflet closure occurred in only 8% of the infarct patients without mitral regurgitation and was not seen at all in the normal group.

No patient in any group had mitral valve prolapse.

**Discussion**

Although there is widespread clinical acceptance of the association between papillary muscle dysfunction and mitral regurgitation proposed by Burch and colleagues,4,5 little objective evidence supports either its occurrence or defines its relative frequency. This paucity of data has reflected the lack of a method to evaluate both systolic mitral leaflet motion and papillary muscle and subjacent ventricular myocardial function. Cross-sectional echocardiography, however, provides a means for visualizing the structural relationships and motion patterns of both regions non-invasively. In the present study, therefore, the cross-sectional technique was used to determine if abnormal mitral leaflet motion could be visualized in patients with mitral regurgitation developing de novo in the setting of ischemic heart disease and if abnormal leaflet apposition did occur, its relationship to papillary muscle and left ventricular function.

These echocardiographic studies revealed a unique pattern of abnormal mitral leaflet closure in 91% of infarct patients with mitral regurgitation. This abnormal leaflet closure pattern was characterized by failure of one or both mitral leaflets to reach a normal peak superior or cephalad position relative to the plane of the atrioventricular ring during systole. The leaflets were thus effectively arrested within the cavity of the left ventricle, disrupting the normal relationship of the mitral leaflets to each other and the valve ring and producing a pattern suggesting incomplete mitral valve closure. The anterior mitral leaflet failed to reach the appropriate closure plane in every case in which an abnormal systolic pattern was observed. Associated abnormal posterior leaflet closure occurred in only 12% of cases. In contrast, abnormal leaflet closure was rarely observed in infarct patients without mitral regurgitation (8%) and was not seen at all in the normal group. These data, therefore, lend strong anatomic support to one of the patterns of leaflet malapposition, incomplete leaflet closure, originally suggested to result from papillary muscle dysfunction.

The second proposed pattern of abnormal leaflet apposition, mitral valve prolapse, was not observed in any of the patients. This is in contrast to angiographic studies in which mitral prolapse in patients with the syndrome of papillary muscle dysfunction was found.6,7 The difference in these results may reflect our exclusion of patients with clinical or echocardiographic evidence of prolapse before their ischemic event. The relatively high reported incidence of mitral prolapse in the random population suggests that prolapse and ischemic disease should occur together frequently by mere chance association. Any large, random study of patients with ischemic heart disease, therefore, should include a significant number of patients with associated mitral prolapse. When patients with recognized or suspected prolapse before the infarct were excluded, however, no new instances of prolapse were noted after infarction. These data, therefore, suggest that mitral prolapse develops rarely, if at all, as a direct result of papillary muscle dysfunction alone and that studies demonstrating mitral prolapse in patients with ischemic disease may merely reflect the random association of two relatively common clinical phenomena. Prolapse may appear in patients with chordal or papillary muscle rupture; however, its cause in these cases is clearly different from that with papillary muscle dysfunction alone.

The second integral component of the syndrome of papillary muscle dysfunction is failure of the papillary muscles themselves to contract normally as a result of ischemia or infarction and/or loss of normal structural support for the papillary muscles due to dyskinesis of the left ventricular myocardium at their base. The contractile function of the papillary muscles cannot be assessed by means of cross-sectional echocardiography. Regional left ventricular wall motion at the base of the papillary muscles, however, can be reliably assessed when good target visualization is achieved.8 Experimental data suggest that loss of papillary muscle function alone, without dyskinesis of the subjacent left ventricular wall, is not sufficient to produce abnormal leaflet closure. Rider et al.9 and Tsakaris et al.10 have shown that destruction of a single papillary muscle without abnormal motion of the underlying left ventricular wall did not result in abnormal mitral leaflet closure.10,11 These investigators
suggested that abnormal leaflet closure occurred only if there were both infarcted papillary muscle and abnormal wall motion of the region surrounding the damaged papillary muscle. This obligatory association of papillary muscle dysfunction and abnormal wall motion suggests that echocardiographic evaluation of wall motion alone should be adequate to assess the myocardial–papillary muscle component of this syndrome.

Abnormal wall motion in the region immediately surrounding one of the papillary muscles was observed in 29 of the 62 infarct patients. The abnormal motion was classified as dyskinetic in 24 and hypokinetic in five. None of the patients with normal or hypokinetic motion showed abnormal leaflet closure. Leaflet closure was judged to be abnormal, however, in 23 of the 24 patients with dyskinesis at the base of one of the papillary muscles. Although the left ventricle in the region of the posteromedial muscle was primarily affected (16 of 23 cases), the association of dyskinetic wall motion and abnormal leaflet closure appeared independent of the papillary muscle involved.

Figures 5 and 6 illustrate how abnormal leaflet closure might occur with equal frequency regardless of whether the dyskinesis involves the anterolateral or posteromedial papillary muscle and why it should always affect the anterior mitral leaflet. Each papillary muscle gives off chordal attachments to both the anterior and posterior mitral leaflets equally. These chordae attach to both the free edges and the body of the leaflets. Presumably, the chordal length is such that tension is normally distributed equally during systole. Although designated anatomically as the posteromedial and anterolateral papillary muscles, echocardiographically both papillary muscles lie in the posterior half of the left ventricle with a line joining their tips running parallel to the coaptation line of the mitral leaflets. When there is dyskinesis at the base of either of these muscles, therefore, the papillary muscle will be displaced posteriorly relative to the coaptation line of the mitral leaflets and medially or laterally relative to the midportion of the leaflet body. Posterior displacement will increase the distance from the tip of the papillary muscle to the body of the anterior leaflet resulting in increased tension in this region. The corresponding distance from the papillary muscle tip to the body of the posterior leaflet should remain relatively unchanged until the dyskinesis becomes profound. Thus, the anterior leaflet should selectively be pulled toward the cardiac apex and its complete systolic closure prevented. In contrast, little additional tension will be exerted on the posterior leaflet and its closure pattern should remain relatively normal. In the long-axis view, incomplete coaptation of the mitral valve leaflets due to displacement of the point of coaptation toward the apex of the left ventricle in the presence of a discrete left ventricular aneurysm or left ventricular dilatation has been recently reported. These patients were not studied in the four-chamber view. In our patients, this incomplete coaptation and displacement of the anterior mitral valve leaflet was not shown in the long-axis view, but was clearly revealed in the four-chamber view.

Applying the same reasoning to the medial or lateral component of the dyskinetic process suggests that maximal tension should occur in the central portion of the mitral leaflets. When both components are combined, the maximal tension should occur in the midportion of the anterior leaflet. The degree of abnormal tension in the midportion of the anterior leaflet should depend on the presence and degree of dyskinesis and be relatively independent of the papillary muscle involved except for the direction of pull on the diseased papillary muscle. Because of the uneven tension exerted on the anterior and posterior leaflets, the anterior leaflet should be involved uniformly, whereas posterior leaflet involvement would be expected only with more severe degrees of dyskinesis. Although no method quantitates severity of dyskinesis and relates this to a degree or pattern of abnormal closure, an association should be expected.

![Figure 5. Diagram of the left ventricle in a long-axis projection illustrating the effects of dyskinesis at the base of a papillary muscle on the chordal attachments to the anterior and posterior mitral leaflets. When the ventricle is displaced posteriorly during systole (interrupted lines), the distance from the tip of the papillary muscle to the body of the anterior mitral leaflet increases. This should increase the tension on a chordae tendineae connecting these two points.](http://circ.ahajournals.org/)

*A2* = aorta; *LA* = left atrium.
This theoretic model explains the cause and pattern of abnormal leaflet closure and helps to explain why this abnormal leaflet closure is observed predominately in the four-chamber view. Any structure is optimally visualized echocardiographically when it lies perpendicular to the central axis of the exploring sound beam. When viewed from the normal left parasternal transducer position, the central ray of the scan plane will be oriented perpendicular to the coaptation line of the mitral leaflets and the chordae tendineae. The bodies of the mitral leaflets, however, are oblique to the scan plane passing through it at an angle that increases relative to true perpendicular as the leaflets approach their insertion into the atrioventricular ring. In contrast, from the apical window, the scan plane is aligned parallel to the coaptation line of the mitral leaflets and more perpendicular to the leaflet bodies. The leaflet bodies are, therefore, recorded using their axial rather than the lateral resolution of the system. Because axial resolution is far better than lateral resolution, small changes in leaflet position should be better appreciated in this orientation. In addition, in the apical four-chamber view the scan plane transects the body of the anterior mitral leaflet in an oblique medial-to-lateral orientation. Thus, a great deal more of the leaflet body is visualized from this transducer location and it is oriented in a more optimal plane to evaluate the relative position of the leaflet bodies in systole. The closure line is not shown clearly in this projection and, as suggested by the theoretical model, this is not where the most obvious pathology should lie.

Finally, although abnormal leaflet closure occurred in a high percentage of patients with newly arising mitral regurgitation and was universally associated with dyskinesis in the region of a papillary muscle, there were two cases in which the clinical syndrome of papillary muscle dysfunction was present but abnormal leaflet closure could not be visualized. The reason for these false negatives is unclear, but there are several possibilities. First, although most of the cross-sectional studies were performed prospectively after the acute infarct, analysis of the patient's history and physical examination before infarction was retrospective. Therefore, mitral regurgitation might have been present before infarction but was either overlooked clinically or not indicated in the medical record. Second, abnormal leaflet closure could have actually been present in these cases, but the degree of abnormality was beyond the discriminatory abilities of either the observer or the cross-sectional technique. The observation that dyskinesis was present at the base of the papillary muscle in one of these cases and the uniform association of left ventricular dyskinesis and incomplete closure in each of the other 23 cases suggests that the latter explanation may have been appropriate in at least one instance.

In three other cases, incomplete leaflet closure and dyskinesis at the base of the papillary muscle were observed in the absence of clinical evidence of mitral regurgitation. As hemodynamic or angiographic studies were not performed in these cases, clinically silent mitral regurgitation might have been present. In addition, as the cross-sectional and clinical examinations were not performed simultaneously, the mitral regurgitation may have been present only transiently or developed after the clinical evaluation. The strong association, however, between left ventricular dyskinesis and abnormal leaflet closure provides anatomic support for the actual occurrence of the clinical syndrome of papillary muscle dysfunction.

In conclusion, therefore, this study suggests that de novo mitral regurgitation in patients with prior myocardial infarction is almost always due to dyskinesis involving the left ventricular myocardium beneath one of the papillary muscle groups, producing increased tension on the anterior mitral leaflet and preventing its normal closure. Mitral valve prolapse occurs rarely, if at all, in the setting of myocardial infarction in the absence of chordal or papillary muscle rupture. Studies demonstrating prolapse in infarct patients may merely reflect the random association of these two common clinical entities.
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Circulation. 1981;63:565-571
doi: 10.1161/01.CIR.63.3.565
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 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:
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