Anomalous Insertion of Papillary Muscle Directly Into Anterior Mitral Leaflet in Hypertrophic Cardiomyopathy
Significance in Producing Left Ventricular Outflow Obstruction

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Background. Obstruction to left ventricular outflow in hypertrophic cardiomyopathy (HCM) is usually due to systolic anterior motion of the mitral valve. Occurrence of structural mitral valve abnormalities in HCM and their significance in producing outflow obstruction (even in the absence of typical systolic anterior motion) has not been fully appreciated.

Methods and Results. Analysis of 78 mitral valves excised from patients with obstructive HCM showed that 10 (13%) had anomalous insertion of one or both left ventricular papillary muscles directly into the anterior mitral leaflet. This malformation was identified by echocardiography, which demonstrated direct continuity between the hypertrophied papillary muscle and mitral leaflet, resulting in a long rigid area of midcavity narrowing that appeared to be solely or largely responsible for outflow obstruction. Basal subaortic pressure gradients were large (70–150 mm Hg). Mitral valve replacement reduced the outflow gradient substantially to 0–15 mm Hg in four patients with postoperative cardiac catheterization. However, two other patients who underwent septal myotomy/myectomy had persistent symptoms and incomplete relief of obstruction (gradients 60 and 70 mm Hg) because of continued midcavity apposition of papillary muscle and ventricular septum.

Conclusions. Anomalous papillary muscle insertion into anterior mitral leaflet represents a mechanism of obstruction to left ventricular outflow in patients with HCM and differs considerably from typical dynamic obstruction caused by mitral valve systolic anterior motion that occurs in many other patients with HCM. Recognition of this malformation emphasizes the diverse morphological expression of HCM and also has important clinical implications for patients requiring operation because the gradient is likely to persist even after adequate myotomy/myectomy; consequently, mitral valve replacement would appear to be the operation of choice in most such patients. (Circulation 1991;84:1188–1197)

Obstruction to left ventricular outflow in patients with hypertrophic cardiomyopathy (HCM) usually results from systolic anterior motion of the mitral valve and midsystolic contact of mitral valve with ventricular septum.1–9 The valve itself has often been described as free of intrinsic disease.10–14 In the course of recent morphologic investigations of mitral valves in HCM, we observed a variety of structural mitral valve alterations including increased leaflet area and asymmetric or segmental leaflet elongation.15 Also, a subset of patients was identified as having a congenital deformity of the mitral valve apparatus not fully appreciated as a cause of subaortic obstruction16,17 in which one or both papillary muscles inserted directly into the ventricular surface of the anterior mitral leaflet. This study describes clinical recognition of this anomaly and focuses on its echocardiographic features and their diagnostic, hemodynamic, therapeutic, and prognostic implications.

Methods

Selection of Study Patients

The cardiovascular registry of the Pathology Branch, 1982–1989, was reviewed and, of the 78 mitral valve specimens removed at operation or necropsy from patients with obstructive HCM in suitable morphological condition, 10 (13%) were identified as having a
Results

Morphology of Mitral Valve Apparatus

Each of the 10 valves had direct insertion of the cephalad portion of one or both left ventricular papillary muscles into the ventricular surface of the anterior mitral leaflet (Figures 1–4). Anomalous papillary muscle insertion involved the commissural region in each valve but also extended to contiguous portions of the anterior and posterior mitral leaflets (Figures 1–4). Chordae tendineae were absent in the area of direct anomalous muscle insertion but otherwise were present in usual numbers with normal anatomic relation. In eight of the 10 patients, a single papillary muscle inserted into the mitral leaflet near either the anterolateral (six patients) (Figures 2, 3B, and 4C) or posteromedial commissure (two patients) (Figure 2). In the other two valves (patients 1 and 4 in Table 1), both papillary muscles inserted into the anterior mitral leaflet, each near its respective commissure. Mitral leaflet size was normal or only slightly increased in each patient (6.8–13.0 cm²; mean, 10.2 cm²).

Only one (1%) of the 100 valves from control patients had direct insertion of a papillary muscle into the ventricular surface of the anterior mitral leaflet (a 36-year-old man with a normal heart).

Echocardiographic—Morphological Correlations

In nine of the 10 study patients, the morphology of the mitral valve apparatus (initially defined by visual inspection of the excised valve specimen) was subsequently identified by retrospective analysis of the echocardiographic studies. In the other patient (who was the last evaluated), anomalous papillary muscle insertion was identified prospectively before surgery by both transthoracic and intraoperative echocardiography (Figure 4).

In each of the 10 patients (as viewed in the parasternal and apical views), the anterior mitral leaflet appeared to be in continuity with one or both papillary muscles, creating the image of a large mass inserting directly into the ventricular surface of the anterior mitral leaflet (Figures 3–7); delicate thin echos characteristic of chordae tendineae were not evident. In addition, the aberrant papillary muscle showed an exaggerated anterior displacement within the left ventricular cavity (Figures 3–7). Consequently, the mitral apparatus was situated in particularly close proximity to (or in direct apposition with) the ventricular septum, producing extreme reduction in end-diastolic left ventricular outflow tract dimension. During systole, a long area of midseptal contact was produced by the anomalous papillary muscle with the most caudal extent located about 3–6 cm from the aortic valve (Figures 4–6).

In six of the 10 patients, the anomalous papillary muscle was very large and occupied substantial portions of the anterior mitral leaflet body beyond the commissural region (Figures 3B and 4C); there was complete absence of associated mitral systolic anterior motion on echocardiogram (Figures 4–7). The
FIGURE 2. Photograph of mitral valve specimens (with ventricular aspect exposed) excised at operation from four patients with obstructive hypertrophic cardiomyopathy showing anomalous papillary muscle insertion directly into anterior mitral leaflet (arrows). Papillary muscles are of various sizes and insert into the region of either the posteromedial (valve at upper right) or anterolateral commissure (the other three valves).
other four patients had relatively small anomalous papillary muscles that were situated almost entirely in the commissural area, involving the anterior leaflet less extensively and sparing the distal margins (Figure 2); also, the distal portion of anterior leaflet appeared to make contact with the septum, usually in a passive fashion (with a relatively flat motion pattern) as the papillary muscle moved forward in systole.

The anatomic relation between papillary muscle and mitral valve was often not evident in the standard parasternal long-axis plane oriented through the center of the left ventricular cavity (Figure 5). Only when the cross-sectional plane was angulated slightly medially or laterally with respect to the center of the cavity (toward the posteromedial or anterolateral commissures) was the true anatomic continuity between papillary muscle and mitral leaflet visualized. Also, when the left ventricle was scanned slowly from base to apex in the short-axis plane, the anomalous papillary muscle could be visualized as directly contiguous with anterior mitral leaflet, without imaging interposed structures, which appeared to be chordae tendineae (Figure 3A).

**Hemodynamic Findings**

All 10 study patients had marked obstruction to left ventricular outflow under basal conditions measured at cardiac catheterization; subaortic pressure gradients were 70–150 mm Hg (average, 93). Left ventricular end-diastolic pressures ranged from 8 to 26 mm Hg (average, 17) and were greater than 15 mm Hg in five patients.

**Clinical Findings**

Each of the 10 patients had severe symptoms of cardiac dysfunction: exertional dyspnea in eight, angina in eight, and syncope in four. All 10 underwent operation to relieve symptoms and left ventricular outflow tract obstruction. Eight patients had mitral valve replacement as a primary operative procedure; four of these eight underwent postoperative cardiac catheterization studies and each showed abolition or marked reduction in basal outflow gradient to 0–15 mm Hg.

The other two patients had septal myotomy/myectomy (Patients 1 and 4 in Table 1); postoperative echocardiograms showed an apparently adequate resection of septal muscle (1.8–2.5 cm wide; 3.0–3.5 cm long), which extended caudal to the mitral leaflet tips in the long-axis plane. Nevertheless, large basal outflow gradients (60 and 70 mm Hg) remained after operation, a result of persistent muscular apposition between the anomalous papillary muscle and septum in the midcavity region beyond the caudal extent of the myotomy/myectomy. One of these two patients died 4 days after operation as a consequence of persistent congestive heart failure and outflow obstruction. The other patient had a second more extensive myotomy/myectomy performed 10 years later (because of persistent symptoms and obstruction), which also did not achieve a reduction in the outflow gradient.

Seven of the 10 patients showed evidence of genetic transmission of HCM in at least one first-degree relative based on echocardiographic and clinical
In about 15% of mitral valves removed from patients with obstructive HCM, we identified anomalous insertion of one or both left ventricular papillary muscles directly into the anterior mitral leaflet. The findings of the present integrated morphological and echocardiographic investigation describe the clinical significance of this congenital mitral valve malformation responsible for obstruction to left ventricular outflow in some patients with HCM.

These patients had a distinctive left ventricular geometry with a strikingly narrowed outflow tract and greatly exaggerated anterior displacement of the papillary muscles beyond that usually present in HCM. Consequently, subaortic obstruction appeared to occur largely by virtue of systolic apposition between the hypertrophied papillary muscle and septum in the midportion of the left ventricular cavity; typically the papillary muscle (as well as the contiguous distal mitral leaflets in some patients) came forward together as an intact single, rigid structure to make contact with the septum, creating a long area of midcavity apposition. It appeared that the absence of chordae tendineae between papillary muscle and mitral leaflet restricted leaflet motion and usually prohibited the distal leaflet from achieving the flexibility necessary to bend and produce the pattern of systolic anterior motion typical of patients with obstructive HCM. This finding was particularly true of those valves in which the anomalous papillary muscle was large and extensively involved the anterior leaflet.

In nine of our 10 study patients, the initial echocardiographic assessment failed to identify the morphological abnormality of the mitral apparatus. This was partly because anomalous papillary muscle insertion directly into mitral valve had not been previously recognized as an established cause of subaortic obstruction in HCM. However, on retrospective analysis of the echocardiograms, we did identify this malformation. Critical to echocardiographic diagnosis was recognition that the aberrant papillary muscle inserts into the commissural regions of the mitral valve (and not into the central portion of the anterior leaflet). Therefore, standard parasternal long-axis...
TABLE 1. Clinical, Hemodynamic, and Echocardiographic Data in 10 Patients With Hypertrophic Cardiomyopathy and Direct Insertion of Papillary Muscle Into Anterior Mitral Leaflet

| Patient No. | Age (yr) | Sex | Cardiac operation | Preop Functional class (NYHA) | Preop LV-SA PSG (mm Hg) | Preop LV (S/D) (mm Hg) | Preop Postop Thickness (mm) | Preop Postop Cavity (mm) | Postop follow-up (months) | No. anomalous papillary muscles | Mitral leaflet area (cm²) | SAM (preop) | Family history of HCM |
|------------|---------|-----|------------------|-----------------------------|------------------------|------------------------|-----------------------------|-----------------------------|----------------------------|-----------------------------|-----------------------------|-------------|---------------------|
| 1          | 7       | M+M| 3 2              | 150 120 240/8 200/14 . . .  . . .  168 | ... 9.9 - - |                     |                               |                             |                             |                             |                     |   |       |
| 22         | 7       | M+M| 3 3              | 50* 70 190/10 210/10 22 16 30 44 34 | ... 9.9 - - |                     |                               |                             |                             |                             |                     |   |       |
| 24         |         | 1, 1, 1 | 3 3            | 75 0 215/21 120/26 14 16 33 48 6† | 1 ... - |                     |                               |                             |                             |                             |                     |   |       |
| 2          | 31      | MVR| 3 1              | 118 ... 120/14 18 16 28 40 1 | 1 11.8 - + |                     |                               |                             |                             |                             |                     |   |       |
| 3          | 31      | MVR| 3 2              | 90 15 185/24 125/19 19 18 16 36 67 | 1 13.0 + + |                     |                               |                             |                             |                             |                     |   |       |
| 4          | 33      | F  | 3 1              | 100 60† 218/26 28 20 21 37 0.06‡ | 2 7.9 - + |                     |                               |                             |                             |                             |                     |   |       |
| 5          | 39      | F  | 3 2              | 70 0 200/23 115/14 18 14 17 32 43 | 1 12.2 + + |                     |                               |                             |                             |                             |                     |   |       |
| 6          | 39      | F  | 3 2              | 70 0 160/16 140/7 21 13 23 40 68 | 2 6.8 - + |                     |                               |                             |                             |                             |                     |   |       |
| 7          | 45      | M  | 3 1              | 100 ... 190/10 19 18 21 38 41 | 1 12.0 + + |                     |                               |                             |                             |                             |                     |   |       |
| 8          | 49      | M  | 3 2              | 85 ... 170/21 16 ... 14 32 18 | 1 11.0 - - |                     |                               |                             |                             |                             |                     |   |       |
| 9          | 54      | MVR| 3 2              | 70 ... 170/10 18 20 29 42 6‡ | 1 11.5 ... |                     |                               |                             |                             |                             |                     |   |       |
| 10         | 62      | F  | 3 3              | 90 5 170/24 140/24 18 15 28 43 13‡ | 1 7.6 + + |                     |                               |                             |                             |                             |                     |   |       |

NYHA, New York Heart Association; LV, left ventricle; SA, systemic artery; S, systolic; D, diastolic; VS, ventricular septum; LVFV, left ventricular free wall; SAM, systolic anterior motion; HCM, hypertrophic cardiomyopathy; M+M, ventricular septal myotomy/myectomy; MVR, mitral valve replacement; CABG, coronary artery bypass grafting; M, male; F, female; -, present; --, absent.

*Gradient measured at time patient was taking propranolol.
†Gradient estimated by continuous wave Doppler echocardiography; recorded 2 days after operation and probably represents an underestimation because patient was clinically in a state of low cardiac output and congestive heart failure.
‡Death (patients No. 1 and 4 due to congestive heart failure and No. 9 and 10 due to sudden cardiac death; patient No. 10 also had marked pulmonary hypertension present before as well as after operation: 85/30 mm Hg; mean, 59).

Each of our 10 patients had severe symptoms of congestive heart failure refractory to medical treatment as well as marked basal obstruction to left ventricular outflow. Recognition in such patients that outflow obstruction is due to a malformation of the mitral apparatus may have important implications with regard to planning operative strategy. Our data suggest that conventional septal myotomy/myectomy (Morrow procedure) is not the optimal procedure for relieving obstruction in this subgroup of patients. Indeed, the two patients in this study who underwent myotomy/myectomy with an apparently adequate muscular resection nevertheless had only partial reduction in subaortic gradient and persistent marked outflow tract obstruction caused by continued systolic contact between anomalous papillary muscle and ventricular septum. In addition, these two patients continued to have substantial cardiac symptoms and ultimately died from heart failure. We have also evaluated two other patients with obstructive HCM (who are not formally part of this study) and the characteristic echocardiographic images of anomalous papillary muscle insertion; the malformation was described by the surgeon at operation but the mitral valve specimen was not available for examination because neither valve replacement nor necropsy was performed. After septic myotomy/myectomy, these patients had persistence of severe symptoms (or

cross-sectional planes directed through the midportion of left ventricular cavity usually fail to image the abnormal anatomic relation between mitral leaflet and papillary muscle and the true geometric proximity of these structures to the ventricular septum. Accurate identification depends on sweeping the transducer medially or laterally from the center of the cavity to obtain nonstandard cross-sectional planes. Consequently, in a patient with HCM, the combination of left ventricular outflow tract obstruction and absence of mitral systolic anterior motion would strongly suggest the possibility of midcavity obstruction caused by anomalous papillary muscle insertion. Indeed, in the last (or 10th) patient in our series, anomalous papillary muscle insertion was diagnosed prospectively by both transthoracic and intraoperative echocardiography applying these anatomic and spatial principles derived from retrospective analysis of the other nine patients.

Direct insertion of papillary muscle into the anterior mitral leaflet represents a congenital malformation with selective failure of chordal development at about 12 weeks' gestation. At this stage of normal cardiac development, the papillary muscles are contiguous with the mitral leaflets. Under normal circumstances, cephalad portions of the papillary muscles are first transformed into thick muscular chordae tendineae, which ultimately become delicate fibrous chordae positioned between the mitral leaflets and the papillary muscles. The arrest in normal embryological development demonstrated by our patients is not pathognomonic of HCM but may occur occasion-
postoperative death) and subaortic obstruction caused by papillary muscle–septal contact.

The standard septal myotomy/myectomy operation\textsuperscript{11,25} probably fails to reduce the magnitude of the subaortic gradient adequately in patients with anomalous papillary muscle because it is fundamentally designed to relieve obstruction by enlarging outflow tract cross-sectional area and reducing dynamic systolic anterior motion of the mitral valve\textsuperscript{26–29} (by virtue of minimizing the Venturi phenomenon).\textsuperscript{16} Conversely, obstruction in our patients is not due to dynamic mitral leaflet motion,\textsuperscript{1,9,16,26,28} but rather is a form of midcavity obstruction\textsuperscript{30–32} that occurs by virtue of a different pathophysiological mechanism. This muscular obstruction occurs deep within the ventricle and is largely inaccessible to the standard myotomy/myectomy resection, which is commonly performed through an aortotomy.

Awareness that congenital papillary muscle insertion may occur in patients with obstructive HCM is critical to its identification either by echocardiography or at the time of operation. Inspection of mitral valve anatomy by the surgeon may confirm the presence of this anomaly and also aid in the assessment of whether a particularly extensive myotomy/myectomy or mitral valve replacement\textsuperscript{33–36} would most likely afford relief of obstruction. Indeed, in the study patient most recently evaluated, the decision to perform mitral valve replacement was predicated on the prospective identification (before operation) of direct papillary muscle insertion.

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