Mitral Valve Prolapse and Coronary Artery Disease
Clinical, Hemodynamic, and Angiographic Correlations

By Juan M. Aranda, M.D., Benjamin Beferler, M.D., Ralph Lazzara, M.D., Abraham Embl, B.S., and Humberto Machado, M.D.

SUMMARY
Among 95 patients with angina pectoris and angiographically documented coronary artery disease (CAD), prolapse of the scallops of the posterior leaflet of the mitral valve (PLMV) was noted in 30 patients. Left ventriculograms in the right anterior oblique (RAO) projection revealed isolated prolapse of the posteromedial commissural scallop (PMCS) in 12 patients and the anterolateral commissural scallop (ALCS) in two patients. Seven patients had prolapse of both PMCS and ALCS, three had prolapse of the PMCS and middle scallop (MS), and six had prolapse of all three scallops of the PLMV.

Left ventricular dilation with increased trabeculations was observed in 19 patients. Contractility determined in a quantitative fashion by segmental motion analysis was markedly impaired in 29 patients. None of the patients had angiographic evidence of mitral insufficiency. Left ventricular dysfunction was documented in 28 patients by either elevated left ventricular end-diastolic pressure (LVEDP), low cardiac index (CI) or decreased ejection fraction (EF). In two patients in whom left ventricular contractility improved after aortocoronary bypass, previously prolapsed scallops could not be identified in the postoperative ventriculogram.

Prolapsed PLMV is a frequent angiographic finding in patients with angiographically observed CAD. Impaired contractility of the ventricular myocardium and papillary muscles, left ventricular dilatation, and hypertrophy appear to play a significant role in the pathogenesis of this abnormality through distortion of the directional axis of the papillary muscles, asynergic contraction of the related free wall of the left ventricle, and changes in the normal spatial alignment necessary for mitral valve closure. The syndrome of papillary muscle dysfunction in patients with coronary artery disease represents a wider clinical spectrum than previously described.

The underlying conditions causing prolapse of the mitral valve leaflets are multiple and include the Marfan syndrome, trauma, hypertrophic obstructive cardiomyopathy, rheumatic endocarditis, congestive cardiomyopathy, atrial septal defect and periarteritis nodosa.1-5 This syndrome includes a spectrum of clinical findings which vary from a nonejection click without a systolic murmur to the presence of mitral incompetence with a holosystolic murmur and no click.6, 7 Electrocardiographic findings characterized by ST and T wave changes in the inferior leads have been observed in some of the cases reported.8 The findings are not specific, but may occur in acquired mitral valve disease due to rheumatic fever or coronary atherosclerosis.9, 10, 11 Since patients with prolapsed mitral leaflets frequently develop recurrent chest pain and ventricular arrhythmias, the possibility that the electrocardiographic changes are caused by coronary artery disease has been suggested.9

Several recent reports in the literature have emphasized angiographic-morphologic correlations12, 13 and left ventricular abnormalities14 in patients with this syndrome. However, to date the frequency of mitral valve prolapse in patients with coronary artery disease has not been systematically studied. This report describes the clinical, hemodynamic, and angiographic characteristics of patients with advanced coronary artery disease who demonstrated prolapsed scallops of the posterior leaflet of the mitral valve.

Material and Methods

Ninety-five patients, all males with angiographically documented coronary artery disease referred to the Cardiovascular Laboratory for evaluation of chest pain, arrhythmias, or congestive heart failure, provided the data for this report. An additional 12 patients studied because of atypical chest pain, who had normal resting and exercise hemodynamics, coronary arteriograms, LV contractility, and no evidence of prolapsed mitral valve, cardiomyopathy, or valvular disease served as normal controls. All patients were examined by at least two of the authors and all were examined by one of us (BB).

After informed consent was obtained, right and left heart
catheterization and selective coronary cineangiography by a
modification of the Sones technique was performed in the
postabsorptive state under mild sedation with diazepam and
meperidine. Angiographic images were recorded on 35 mm
film at 60 frames per second. Pressure was registered on an
Electronics for Medicine Multi-channel DR-16 recorder,
utilizing Statham PD-23 transducers and fluid-filled
catheters. Cardiac output was determined in duplicate by
the dye dilution technique using indocyanine green.
Isometric exercise was performed for 5 min using a hand-
grip dynamometer. The maximal voluntary contraction
was determined before the procedure was started as the average
of three consecutive maximal attempts. Measurements were
made of the intracardiac pressures and cardiac output at rest
and at four minutes of sustained handgrip at 25% of max-
imal voluntary contraction.

Left ventriculography was performed in all patients in the
right anterior oblique projection at 30°. Thirty-five to fifty
ml of methylglucamine diatrizoate (Renografin-76) was
injected into the left ventricle at a rate of 26 to 30 ml per sec-
ond, using a Cordis II power injector.

Individual systolic frames of the ventriculogram were
carefully studied. The mitral valve and the artioventricular
ring at the base of the opacified left ventricle, as well as the
posteromedial and anterolateral commissural areas, were
identified. A distinct convex bulge in the area of the
posteromedial or anterolateral commissure suggested
prolapse of the posteromedial (PMCS) or anterolateral com-
missural scallop (ALCS), respectively. A distinct central
bulge was judged to be secondary to prolapse of the middle
scallop (MS), as described by others.

All angiograms were reviewed independently by at least
two observers who assessed the contractility of the left ven-
tricle in a qualitative fashion. Regional wall segments were
classified into one of four possible categories: normal,
hypokinetic, akinetic, or dyskinetic. 1) normal — the seg-
ment had synchronous and inward movement of good
amplitude during systole; 2) hypokinetic — a portion or all
of a segment had inward movement judged qualitatively to
be diminished in amplitude; 3) akinetic — a portion or all of
the segment had complete absence of wall motion; 4)
dyskinetic — a portion or all of the segment had paradoxical
systolic outward motion.

Quantitative angiographic methods were used to deter-
mine volumes and ejection fractions (EF) using the area-
length method of Dodge modified for single plane
calculations. The 35 mm cine film was viewed on a Tagar-
no projector where a normal sinus beat was selected for
volume determination. The left ventricular end-systolic and
diastolic silhouettes were traced on paper and the areas
measured by planimetry. The correction factor for volume
determination was obtained by counting and planimetrizing
one centimeter squares in a grid that was filmed at the level
of the patient’s left ventricle using the same fluoroscopic
height that was used during the ventriculogram. The
counted area of the grid was then compared to its
planimetrized area recorded on cine film. Stroke volume
obtained by this method correlated closely with the value ob-
tained by dye dilution curves (r = 0.95).

Left ventricular hypertrophy was assessed cineangi-
ographically in the RAO by the method of Gulotta. The left
ventricular wall thickness (LVWT) in end-diastole was
measured anteriorly at a point midway between the apex
and the aortic valve. The linear correction factor was
derived by comparing the projected longitudinal diameter of
a grid to the known previously measured diameter (5 cm).

The grid was filmed as previously described. Segmental
motion analysis was performed by superimposing tracings of
end-diastolic and systolic frames using the cardiac apex and
mid-aortic valve as fixed points. The long axis (L) of the left ven-
tricle was determined from the midpoint of the aortic valve
to the ventricular apex. It was quadrisection independently
at end-systole and end-diastole by three diameters drawn
perpendicular to it, (PD, MD, and AD). Shortening of
each diameter was expressed as percent shortening of its end-
diastolic dimension.

Figure 1
Schematic representation of superimposed tracings of end-
diastolic and end-systolic frames drawn directly from the
projected left ventriculogram. The long axis (L) of the left ven-
tricle was quadrisection independently at end-systole and
end-diastole by three diameters drawn perpendicular to it, (PD, MD, and AD). Shortening of
each diameter was expressed as percent shortening of its end-
diastolic dimension.

Results
Clinical and Electrocardiographic Features
Of the 95 patients studied, 30 had angiographic evidence of prolapse of the posterior mitral leaflet (table 1). Their average age was 54 years (range 38–66). Twenty-six of the patients were significantly
disabled with Class III-IV symptoms of angina and/or
congestive heart failure (New York Heart Association
functional classification). A left ventricular lift was
palpable in 24 of 30 patients and in one it was diffuse
and paradoxical, suggesting a ventricular aneurysm.
A fourth heart sound was heard in 28 patients, a third
heart sound in seven. Early or mid-systolic apical mur-
murs (grade I–II) were detected in five patients
and recorded at the time of catheterization in three
patients. In two patients it was intermittently present.
Systolic clicks were not heard in any of the cases

Circulation, Volume 52, August 1975
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Abbreviations: Class = New York Heart Association Functional Classification; ST-T = nonspecific abnormalities; ALMI = anterolateral myocardial infarction; ASMI = anteroseptal myocardial infarction; IWM = inferior wall myocardial infarction; EAMI = extensive anterior myocardial infarction; LBBB = left bundle branch block; LAD = left anterior descending; VAP = variant angina pectoris; + = present; - = absent.

Hemodynamic Measurements

Table 2 summarizes the hemodynamic and angiographic data. Twenty-three of the 30 patients had resting left ventricular end-diastolic pressures (LVEDP) greater than 12 mm Hg, and 17 had right ventricular end-diastolic pressures (RVEDP) over 5 mm Hg. Fourteen patients had a cardiac index (CI) below 2.5 L/min/m². The ejection fraction was reduced (<57%) in 13 of 23 patients, ten had EF less than 50%. Of the 17 patients in whom the EF was normal or not measured, the CI was decreased in eight patients and the LVEDP increased in seven. Thus in 28 of the 30 cases reported, abnormal left ventricular function was demonstrated at rest.

The following values were obtained in the control group: EF = 0.73 ± .07 (range .63 – .81), LVEDV = 74.2 ± 8.6 cc³ (range 66–87), LVT = 9.5 ± 2.5 mm (range 6.3–12).

Angiographic Findings

Left ventriculography in the 30° RAO projection revealed isolated prolapse of the ALCS in 12 patients and the ALCS in two (fig. 2). Six had prolapse of both the PMCS and ALCS, three had prolapse of the studied. All patients were in normal sinus rhythm. Electrocardiographic evidence of healed transmural myocardial infarction was noted in 16 patients, 12 anterior, four inferior and nonspecific ST-T abnormalities in ten. Two patients had normal electrocardiograms, a third had extensive intraventricular conduction abnormalities, and another had variant angina pectoris with transient elevation of ST segment. Ten patients with transmural myocardial infarction had left anterior hemiblock.

Figure 2

Postoperative left ventriculogram, patient M.M., showing isolated prolapse of the anterolateral commissural scallop (ALCS) of the posterior mitral leaflet. Note the hypertrophy of the papillary muscles and their abnormal orientation within the left ventricular cavity. MS = middle scallop; PMCS = posteromedial commissural scallop; ALPM = anterolateral papillary muscle; PMPM = posteromedial papillary muscle.
### Table 2

#### Hemodynamic and Angiographic Data

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<td>106</td>
<td>14.0</td>
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<td>H</td>
<td>90</td>
<td>100</td>
<td>70</td>
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<td>+</td>
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</table>

| Average | 2.53 | 2.47 | 0.56 | 115 | 11.6 |

**Sfa:** 0.861 1.149 0.508 0.105 0.246 2.83 6.59 0.405

注：
+ = present.
- = absent.

*Lesions in left anterior descending or first diagonal branch.
†Lesions in proximal circumflex or obtuse marginal.

注释：LVEDP = 左心室末期舒张末期压力；RV EDP = 右心室末期舒张末期压力；CI = 心脏指数；EF = 射血分数；LV WT = 左心室壁厚度；LVEDV = 左心室末期舒张末期容积；AW = 前壁；IW = 后壁；H = 缓慢运动；D = 迅速运动；A = 停滞；N = 正常；RCA = 右冠状动脉；LAD = 左前降支；Circ = 环状动脉；PMCS = 前室间动脉；ALCS = 前室间动脉；MS = 中间动脉；ACB = 动脉斑块。

注：表中数据为每个变量的均值。
PMCS and MS (fig. 3), and seven had prolapse of all three scallops of the posterior leaflet (fig. 4).

Mitrral regurgitation was not observed in any of the cases reported. Left ventricular dilatation was present in 19 of 23 patients; left ventricular hypertrophy was detected in ten out of 22 patients. Hypokinetic or akinetic segments in the anterior wall were identified in 18 patients while 16 others showed these contraction abnormalities in the inferior wall. Dyskinetic areas with paradoxical movements and aneurysm formation were angiographically identified in nine patients (fig. 5).

Segmental motion analysis corroborated the qualitative observations. The end-diastolic left ventricular dimensions in the cases reported were significantly different from those observed in patients with normal LV function (table 3). The extent of shortening of the midventricular, apical and long diameter of the left ventricle was significantly reduced in patients with prolapsed scallops compared to patients with normal LV function ($P < 0.01$, $P < 0.05$, $P < 0.01$, respectively). The difference in the extent of shortening of the proximal diameter was not statistically significant ($P > 0.05$).

The ratio of the distance between the postero medial papillary muscle to mitral valve ring and apex to mitral valve ring was calculated at end-diastole and end-systole in ten patients with prolapsed scallops and in five patients with normal LV function. In the latter group the ratio varied by an average of 0.010 (table 3) while in the former it changed by an average of 0.136 ($P < 0.01$) indicating failure of the postero medial papillary muscle to shorten and sustain contraction. Thus, left ventricular dilatation, impaired segmental contractility, hypertrophy of the ventricular myocardium, or changes in the directional axis of the papillary muscles were present in all of the patients with prolapsed scallops.

Selective coronary angiograms in multiple projections demonstrated in every patient advanced
Valvular dysfunction; for example, prolapse of the posterior commissural scallop (PMCS), middle scallop (MS), and anterolateral commissural scallop (ALCS) before surgery. After ventricular resection, significant asynergy of the inferior wall and minimal prolapse of the ALCS are noted; however, the previously prolapsed PMCS and MS are not identified. The black arrows point to the left atrium opacified by contrast media injected in the left ventricular cavity.

Table 3

Analysis of Left Ventricular Segmental Motion

<table>
<thead>
<tr>
<th>Parameters (Mean)</th>
<th>Normals</th>
<th>Prolapsed scallops</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal diameter (cm)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>ED</td>
<td>11.38 ± 1.03</td>
<td>15.66 ± 2.48</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ES</td>
<td>6.96 ± 1.04</td>
<td>11.26 ± 4.08</td>
<td></td>
</tr>
<tr>
<td>% shortening</td>
<td>38.4 ± 11.71</td>
<td>32.4 ± 10.8</td>
<td>NS</td>
</tr>
<tr>
<td>Midventricular diameter (cm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED</td>
<td>12.94 ± 1.22</td>
<td>15.56 ± 1.79</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ES</td>
<td>6.52 ± 1.48</td>
<td>11.44 ± 4.09</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>% shortening</td>
<td>50 ± 6.74</td>
<td>33.1 ± 14.8</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Apical diameter (cm)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED</td>
<td>9.42 ± 0.97</td>
<td>11.61 ± 1.85</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>ES</td>
<td>3.54 ± 1.00</td>
<td>7.30 ± 3.14</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>% shortening</td>
<td>62.2 ± 11.1</td>
<td>37.6 ± 22.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Longitudinal diameter (cm)</td>
<td></td>
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<tr>
<td>ED</td>
<td>23.48 ± 1.03</td>
<td>26.92 ± 3.09</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>ES</td>
<td>18.26 ± 1.29</td>
<td>23.87 ± 4.98</td>
<td></td>
</tr>
<tr>
<td>% shortening</td>
<td>22.4 ± 5.36</td>
<td>11.8 ± 6.85</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PMPM-MVR ED</td>
<td>0.306 ± .065</td>
<td>0.233 ± 0.059</td>
<td></td>
</tr>
<tr>
<td>APEX-MVR ES</td>
<td>0.316 ± .71</td>
<td>0.369 ± 0.083</td>
<td></td>
</tr>
<tr>
<td>Δ</td>
<td>0.010</td>
<td>0.130</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Abbreviations: ED = end-diastole; ES = end-systole; PMPM = posteromedial papillary muscle; MVR = mitral valve ring; NS = not significant.

Arteriographic obstruction (>50%) in one or more major vessels. Significant obstruction of the left anterior descending artery was present in 27 patients (90%), of the circumflex artery in 17 (57%), and of the right coronary artery in 22 (73%). Eleven patients (37%) had obstruction of two major vessels and 13 (43%) had obstruction of all three major vessels. Three of the 30 patients reported (RS, MP, GH) had significant lesions of the main left coronary artery.

Eighteen patients in the group underwent aortocoronary bypass surgery while eight others had ventricular aneurysmectomy with or without aortocoronary bypass. To date nine of the 26 patients have been restudied with left ventriculography, selective coronary arteriography as well as direct visualization of the grafts. In three of the patients restudied (RB, RM, CM), previously prolapsed scallops were not evident on re-examination. One of them with an apical ventricular aneurysm and prolapsed PMCS, MS, and ALCS underwent ventricular aneurysmectomy. He sustained an inferior wall myocardial infarction during the surgical procedure. The postoperative ventriculogram revealed marked asynergy of the inferior wall, prolapsed ALCS, and minimal mitral insufficiency. The PMCS and MS were not visualized.
The auscultatory findings of mitral valve prolapse syndrome have been well documented in patients with atherosclerotic heart disease. However, there are no reports in the literature on clinical-angiographic correlations of prolapsed mitral leaflets in patients with advanced obstructive coronary artery disease. The absence of clinical-angiographic correlations may arise from the fact that there have been many discrepancies in the description of the mitral valve. There is still much controversy as to the definition of the commissural areas and arguments as to the number of leaflets of the mitral valve. Recently, Ranganathan et al. studied 50 normal mitral valves from adults. Commissures identified by commissural chordae tendineae divided the mitral valve into anterior and posterior leaflets. They found that the posterior leaflet was further subdivided into scallops by clefts or indentations. In 92% of the hearts examined, the posterior leaflet was constantly notched and was divisible into three scallops. In 84% of the cases a large middle scallop was present with two smaller scallops in either side; posteromedial and anterolateral commissural scallops. They suggested that prolapse of any part of the triscalloped structure of the posterior leaflet should be recognized in a left ventriculogram and that prolapse of commissural scallops should be observed as bulges at either extremity of the posterior leaflet area. In subsequent reports Ranganathan and Trent demonstrated the angiographic appearances of pathologic proven prolapses of the different scallops of the posterior mitral leaflet.

The selection of patients in this study was based on the angiographic observations of prolapsed scallops of the posterior leaflet of the mitral valve in patients with documented obstructive coronary artery disease. Our series differs from the series reported by Scampordonis et al. and Gulotta et al. in that their patients were selected because of the presence of nonejection clicks, late systolic murmurs, troublesome arrhythmias, or disabling chest pains. Most of their cases had normal coronary arteries and their series represented a heterogeneous clinical spectrum of patients with this syndrome in which different etiological factors accounted for the abnormalities observed. Nonetheless, our observations are in agreement with these and other reports of asymmetric disordered patterns of left ventricular contraction in patients with systolic click, late systolic murmur, and prolapse of the posterior leaflet of the mitral valve, but are different from the observations made by Ranganathan et al. In their study prolapsed middle scallop was most frequently seen while isolated prolapse of a commissural scallop occurred only once.

The explanation for these observed differences is not clear at the present time. However, the selection of patients may account for some of these differences. In a series with coronary artery disease, infarction or fibrosis of papillary muscles, left ventricular abnormalities, and normal mitral valves have been reported. In patients with prolapsed mitral valve and normal coronary arteries, necropsy studies have shown myxomatous degeneration of the valve, with elongated and thickened chordae but no intrinsic pathologic changes in the papillary muscles. This might be better understood if one considers the blood supply of the papillary muscles. The anterior papillary muscle receives its blood supply from branches of the left anterior descending and circumflex arteries, while the posterior muscle gets its blood supply from branches of the right coronary and left circumflex arteries in 70%, from the right coronary artery alone in 20%, and the left circumflex artery in 10% of the cases. The presence of severe obstructive coronary artery disease in all of our cases appears to be the most important contributing factor accounting for the differences in the number of prolapsed scallops observed, since normal mitral valve function depends on a rather complex and finely coordinated system of which the papillary muscle is one of the principal components.

The high incidence of transmural myocardial infarction, ST-T wave abnormalities, hypokinesis, and dyskinesis of various segments of the left ventricular cavity found in our series agree with the recent clinical and experimental evidence that incompetence of the mitral valve depends not only on the intrinsic dysfunction of the papillary muscles but also upon distortion of the papillary muscle foundation by asynergic contraction of the related free wall of the left ventricle. Infarction of a portion of the left ventricular wall at the base of the papillary muscle may interfere with the proper functioning of the muscle by forming an aneurysm, thus changing the directional axis of the papillary muscle. The paradoxical expansion of the wall of the aneurysm may carry the base of the muscle away from its normal position, thus changing the normal spatial three dimensional alignment necessary for proper mitral valve closure. The relatively high incidence of ventricular aneurysms in our patients agree with the above observations. In-
fraction of the base of the papillary muscle or the related free wall of the left ventricle may also interfere with its function by failure to provide adequate anchorage to the muscle and chordae tendineae during left ventricular systole. 36

Considering the above mechanisms, we were not surprised to find two patients who showed improved left ventricular contractility after aortocoronary bypass and whose previously prolapsed scallops were absent in a postoperative ventriculogram. On the other hand, prolapse of the scallops of the posterior mitral leaflet might represent an intermediate stage before clinically apparent mitral insufficiency occurs. As shown by the patient RB (fig. 5), infarction of the base of the papillary muscle and its related free wall of the left ventricle interfered with proper closure of the mitral valve by failure to provide anchorage to the papillary muscle and chordae tendineae. As a result, the previously prolapsed leaflet became insufficient.

The absence of mitral insufficiency in our group of patients contrasts sharply with the previous angiographic documentation of mild to severe mitral insufficiency in most patients with systolic click, late systolic murmur, and prolapsed posterior leaflet. 8, 14, 17, 20 In view of the findings of Ranganathan et al. 17 it is interesting to speculate that since the middle scallop is usually the largest of the posterior leaflet, prolapse of this scallop might be accompanied more frequently by mild to moderate insufficiency than prolapse of the two smaller commissural scallops. In some of our cases turbulence in the prolapsed scallop without regurgitation might have been responsible for the presence of the systolic murmur. 8

The syndrome of papillary muscle dysfunction in patients with coronary artery disease appears to represent a wider clinical spectrum than previously described. At one end of the spectrum are those patients with prolapsed scallops of the mitral valve without mitral insufficiency. In the other end of the spectrum are patients with systolic murmurs and/or systolic clicks and compensated congestive heart failure. At the other end of the spectrum are those patients with progressive cardiac decompensation and severe mitral insufficiency.

In view of the recommendations regarding antibiotic prophylaxis in patients with mitral valve prolapse syndrome, 2, 36, 38 and considering the reports of bacterial endocarditis complicating mitral regurgitation after an acute myocardial infarction, 25 the use of antibiotic prophylaxis should be considered in patients with coronary artery disease and prolapsed scallops of the posterior leaflet of the mitral valve.

Although prolapse of the posterior mitral leaflet have heterogeneous origins, impaired contractility, left ventricular hypertrophy, and dilatation appear to be the most significant contributing factors in patients with coronary artery disease by causing distortion of the papillary muscle foundation during left ventricular contraction.

Acknowledgment

We acknowledge the secretarial help of Miss Diana Smith, Miss Bonnie Agnell and the medical artistry of Mr. Marcelino Obaya.

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MV PROLAPSE & CAD

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Circulation. 1975;52:245-253
doi: 10.1161/01.CIR.52.2.245

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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