Systolic Anterior Motion in the Absence of Asymmetric Septal Hypertrophy
A Buckling Phenomenon of the Chordae Tendineae

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JUDY FIZZANO, B.S., AND MICHAEL LESCH, M.D.

SUMMARY Systolic anterior motion (SAM) of the mitral valve in the absence of asymmetric septal hypertrophy or concentric left ventricular hypertrophy has been reported in several conditions. In this report we describe the clinical and echocardiographic findings in 15 patients who demonstrated SAM without associated organic heart disease (group 1, 10 patients) or in association with mitral valve prolapse (group 2, five patients). Cross-sectional echocardiography revealed the etiology of SAM in both groups to be early systolic anterior angular motion ("buckling") of mitral chordal structures, rather than movement of the body of the anterior mitral leaflet into the left ventricular outflow tract. In contrast to normal subjects and group 1, group 2 patients had auscultatory evidence of mitral prolapse, a slightly greater mean left ventricular ejection fraction (p < 0.05) (normals, 69 ± 5.2%, group 1, 72 ± 3.8%, group 2, 75 ± 5.6%), and a greater mean diastolic mitral valve (D-E) excursion (p < 0.05) (normals, 1.8 ± 0.2 cm, group 1, 2.2 ± 0.3 cm, and group 2, 2.6 ± 0.4 cm). This spectrum of mitral excursion and left ventricular ejection fraction supports the concept that the mitral valve prolapse syndrome may have as its basis a mitral valve abnormality and/or a hyperdynamic state that predispose to both chordal buckling and mitral leaflet prolapse.

SYSTOLIC ANTERIOR MOTION (SAM) of the mitral valve was initially described in conjunction with asymmetric septal hypertrophy as one of the two echocardiographic hallmarks of idiopathic hypertrophic subaortic stenosis or hypertrophic obstructive cardiomyopathy.1-5 The presence of SAM was considered to be the M-mode echocardiographic manifestation of the left ventricular outflow obstruction in hypertrophic cardiomyopathy.1-5 Furthermore, the amount of SAM (i.e., the degree and duration of the approximation of the anterior mitral leaflet to the ventricular septum) was reported to correlate with the presence and the degree of left ventricular outflow obstruction.4 More recently, SAM has been reported without asymmetric septal hypertrophy both in the presence4-8 and in the absence6 of associated subaortic obstruction. Conditions other than hypertrophic cardiomyopathy known to be associated with SAM include membranous subaortic stenosis,10 dextraposition of the great vessels with subpulmonic obstruction,11 pulmonary hypertension,12 concentric left ventricular hypertrophy,12, 14 hyperkinetic states14, 15 (including aortic insufficiency and hypovolemia), mitral valve prolapse (MVP)16-18 and the absence of detectable heart disease.8, 15, 19

Fifteen patients who demonstrated SAM in the absence of hypertrophic cardiomyopathy on M-mode echocardiography were studied prospectively. Five of these patients had MVP; the other 10 had no evidence of organic heart disease. This report describes the clinical, phonocardiographic and echocardiographic findings in these 15 patients. Cross-sectional echocardiography defined a mechanism for the production of SAM in this group that differs from the mechanism initially reported in patients with hypertrophic obstructive cardiomyopathy.

Materials and Methods

Cardiovascular histories were obtained and cardiac examinations performed in the supine, left lateral and sitting positions in 15 patients who were referred for echocardiographic evaluation of possible MVP. Particular attention was given to symptoms of chest pain, dyspnea, dizziness, lightheadedness or palpitations and auscultatory evidence of MVP. The 15 patients in our series were divided into two groups. Group 1 was composed of 10 patients who demonstrated SAM but not MVP by M-mode echocardiography; group 2 included the five patients who demonstrated M-mode echocardiographic evidence of both SAM and MVP. Phonoangiograms were performed using an Irex Systems II recorder at a paper speed of 100 mm/sec with low-, medium- and high-frequency bandpass filters. M-mode echocardiograms were obtained in the supine and/or left lateral decubitus positions using a 2.25-MHz medium-focused transducer and a Smith Kline Ekoline 20A ultrasonoscope (interfaced to a Honeywell 1856 strip-chart recorder) or an Irex Systems II echocardiographic module and recorder. Measurements were made independently by three observers using the leading-edge method, consistent with recommendations of the American Society of
Echocardiography. Care was taken to exclude septal hypertrophy by imaging the ventricular septum from the point at which it joins with the anterior wall of the aorta to its portion below the tips of the mitral leaflets. The following parameters were measured or derived: left atrial dimension, left ventricular systolic and diastolic dimensions, ventricular septal and left ventricular posterior wall thicknesses, left ventricular ejection fraction and mitral valve D-E (early diastolic) excursion. D-E excursion was measured from the leading edge of the most anterior echo at the mitral D point to the leading edge of this echo at the E point. Left ventricular ejection fraction was calculated from left ventricular dimensions using regression equations developed by Teichholz and co-workers. The left ventricular outflow tract dimension was measured from the leading edge of the most anterior line (echo) at the mitral C point, i.e., the point of coaptation of the mitral leaflets before the onset of SAM, to the left side of the ventricular septum. M-mode echocardiographic evidence for MVP was defined as either a smooth pansystolic or a mid-to-late systolic posterior movement of the mitral echogram. Care was taken to direct the transducer in a direction perpendicular to the mitral leaflets to avoid pseudo-mitral prolapse caused by improper transducer angulation. Mitral valve echograms were performed in four of the 15 patients after inhalation of amyl nitrite to determine whether this maneuver produced an accentuation of SAM.

Cross-sectional echocardiograms were performed in all 15 patients with a Smith Kline Eko-Sector I scanner with a transducer providing a 30° sector arc. Long- and short-axis views of the heart were obtained with the transducer in the left parasternal region. The transducer was positioned in such a manner that the echo beam was perpendicular to the mitral leaflets at systolic coaptation. In addition, a four-chamber view was obtained with the transducer located at the left ventricular apex. Video recordings of each cross-sectional echo study were obtained and reviewed independently by two observers, with special attention to the ventricular septum and the mitral valvular and subvalvular structures.

Normal Controls

Fifteen healthy, nonobese control subjects without evidence of organic heart disease on the basis of history, physical examination or ECG were evaluated by M-mode and cross-sectional echocardiography.

Echocardiographic Data Adjustments

Heights and weights were recorded for each group 1 and 2 patient and each control subject, and the body surface area was estimated using the Boothby and Standiford modification of the formula of DuBois and DuBois. M-mode echocardiographic data for patients and controls were corrected for body surface area and age using regression equations described by Gardin et al. modified for measurements made using American Society of Echocardiography standards. Age-adjusted and body surface area–adjusted echocardiographic data in group 1 and 2 patients with SAM were compared with similarly adjusted data from the 15 normal subjects for all measurements and derived 95% normal confidence limits for adult normal data for all measurements except mitral valve excursion and left ventricular outflow tract dimension.

Statistical analysis of the data was performed using a two-way analysis of variance, comparing group 1 to group 2 patients and to the normal subjects.

Results

The historical and auscultatory results for groups 1 and 2 are summarized in Table 1. The mean age of patients in group 1 (43 years) and group 2 (45 years) was not significantly different. More males than females were in each group, but the study group was small. Symptoms of chest pain, dyspnea and palpitations were present in patients in both groups. Six of 10 patients in group 1 (60%) were symptomatic, whereas all five patients in group 2 had symptoms (NS).

On auscultation, none of the 10 group 1 patients (SAM only) had systolic clicks or mid-to-late systolic murmurs. In contrast, four of the five group 2 patients had the auscultatory features of MVP (p < 0.05). Thus, SAM alone, without concomitant MVP, did not produce systolic clicks or mid-to-late systolic murmurs in our patients.

Table 1. Clinical and Auscultatory Features

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Group 1 (SAM only) (n = 10)</th>
<th>Group 2 (SAM with MVP) (n = 5)</th>
<th>Normals (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Range</td>
<td>21–61</td>
<td>28–70</td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>43</td>
<td>45</td>
<td>42</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td>7</td>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>Females</td>
<td>3</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>4</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>Multiple</td>
<td>4</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Chest pain</td>
<td>1</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Palpitations</td>
<td>1</td>
<td>2</td>
<td>0</td>
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</table>

Auscultatory features

<table>
<thead>
<tr>
<th>Mid-late systolic</th>
<th>Click or murmur</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>0</td>
<td>4</td>
<td>0</td>
</tr>
<tr>
<td>Absent</td>
<td>10</td>
<td>2</td>
<td>15</td>
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</table>

Abbreviations: SAM = systolic anterior motion; MVP = mitral valve prolapse.
Echocardiography

The M-mode and cross-sectional echocardiographic features of both patient groups and the control subjects are summarized in table 2.

**M-mode Echocardiographic Findings**

When group 1 and 2 patients were compared to the 15 normal subjects, no statistically significant difference was found in any of the measured or derived echocardiographic measurements except for left ventricular ejection fraction and mitral valve D-E excursion. Group 2 had an increased left ventricular ejection fraction (p < 0.05), and both groups had increased mitral valve D-E excursion. The left ventricular ejection fractions for group 1 and 2 for the 15 control subjects were all within the normal confidence limits. Furthermore, all group 1 and 2 echocardiographic data for left ventricular systolic and diastolic dimensions, ventricular septal and left ventricular posterior wall thickness were within the 95% confidence limits. In patients 1 and 2, left atrial dimensions were subnormal, but within 10% of the lower normal limit. Figure 1 is a comparison of mitral diastolic (D-E) excursion in normal subjects and group 1 and 2 patients. The mean ± SD for normal subjects was 1.8 ± 0.2 cm, for group 1, 2.2 ± 0.3 cm, and for group 2, 2.6 ± 0.4 cm. D-E excursion in group 1 was statistically greater than in normal subjects (p < 0.001); furthermore, in group 2, D-E excursion was significantly greater than in normal subjects (p < 0.01) and group 1 (p < 0.05).

Figures 2A and 2B are examples of the M-mode.

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**Table 2. M-mode and Cross-sectional Echocardiographic Findings**

<table>
<thead>
<tr>
<th>Patient (years)</th>
<th>Sex</th>
<th>BSA (m²)</th>
<th>LA (cm)</th>
<th>LVIDd (cm)</th>
<th>LVIDs (cm)</th>
<th>IVS (cm)</th>
<th>LVPW (cm)</th>
<th>EF (%)</th>
<th>LVOT (cm)</th>
<th>MVE (cm)</th>
<th>MVP</th>
<th>Cross-sectional</th>
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</thead>
<tbody>
<tr>
<td>Group 1 (n = 10)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(SAM only)</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>54</td>
<td>F</td>
<td>1.74</td>
<td>2.8</td>
<td>4.6</td>
<td>2.9</td>
<td>1.0</td>
<td>1.0</td>
<td>70</td>
<td>2.5</td>
<td>1.7</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>61</td>
<td>M</td>
<td>1.93</td>
<td>2.9</td>
<td>4.8</td>
<td>2.8</td>
<td>1.1</td>
<td>1.1</td>
<td>73</td>
<td>3.4</td>
<td>2.4</td>
<td>-</td>
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<tr>
<td>3</td>
<td>35</td>
<td>M</td>
<td>1.97</td>
<td>4.2</td>
<td>5.4</td>
<td>3.1</td>
<td>1.1</td>
<td>1.1</td>
<td>?</td>
<td>2.4</td>
<td>-</td>
<td>+</td>
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<tr>
<td>4</td>
<td>21</td>
<td>M</td>
<td>1.82</td>
<td>3.1</td>
<td>5.3</td>
<td>2.9</td>
<td>1.1</td>
<td>1.1</td>
<td>73</td>
<td>3.4</td>
<td>2.2</td>
<td>-</td>
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<tr>
<td>5</td>
<td>45</td>
<td>M</td>
<td>2.04</td>
<td>3.7</td>
<td>5.4</td>
<td>3.3</td>
<td>1.1</td>
<td>1.0</td>
<td>69</td>
<td>3.3</td>
<td>2.4</td>
<td>-</td>
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<tr>
<td>6</td>
<td>27</td>
<td>M</td>
<td>1.88</td>
<td>3.2</td>
<td>5.2</td>
<td>3.0</td>
<td>1.0</td>
<td>1.0</td>
<td>78</td>
<td>3.7</td>
<td>2.3</td>
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<tr>
<td>7</td>
<td>61</td>
<td>M</td>
<td>2.03</td>
<td>3.6</td>
<td>5.2</td>
<td>3.3</td>
<td>1.1</td>
<td>?</td>
<td>66</td>
<td>3.0</td>
<td>1.9</td>
<td>-</td>
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<tr>
<td>8</td>
<td>26</td>
<td>F</td>
<td>1.76</td>
<td>3.6</td>
<td>4.7</td>
<td>2.8</td>
<td>1.0</td>
<td>1.0</td>
<td>71</td>
<td>3.3</td>
<td>2.0</td>
<td>-</td>
</tr>
<tr>
<td>9</td>
<td>56</td>
<td>M</td>
<td>1.97</td>
<td>?</td>
<td>4.5</td>
<td>2.6</td>
<td>1.0</td>
<td>1.0</td>
<td>73</td>
<td>3.0</td>
<td>2.0</td>
<td>-</td>
</tr>
<tr>
<td>10</td>
<td>44</td>
<td>F</td>
<td>1.70</td>
<td>3.7</td>
<td>5.2</td>
<td>2.8</td>
<td>1.0</td>
<td>1.0</td>
<td>77</td>
<td>?</td>
<td>2.8</td>
<td>-</td>
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</tbody>
</table>

Mean 43 | 1.88 | 3.4 | 5.0 | 3.0 | 1.0 | 1.0 | 72 | 3.2 | 2.2 |

* ± SD ± 15 = ± 0.1 ± 0.4 ± 0.3 ± 0.2 ± 0.05 ± 0.05 ± 3.8 ± 0.4 ± 0.3 |

Group 2 (n = 5) | (SAM and MVP) |
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>11</td>
<td>33  M</td>
</tr>
<tr>
<td>12</td>
<td>28  F</td>
</tr>
<tr>
<td>13</td>
<td>36  M</td>
</tr>
<tr>
<td>14</td>
<td>60  F</td>
</tr>
<tr>
<td>15</td>
<td>70  M</td>
</tr>
</tbody>
</table>

Mean 45 | 1.83 | 3.4 | 4.8 | 2.7 | 1.0 | 1.0 | 75 | 3.3 | 2.6 |

* ± SD ± 18 = ± 0.2 ± 0.4 ± 0.3 ± 0.4 ± 0.05 ± 0.04 ± 5.6 ± 0.3 ± 0.4 |

Normals (n = 15) |
<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Mean 42</td>
<td>1.79</td>
</tr>
</tbody>
</table>

* ± SD ± 18 = ± 0.2 ± 0.2 ± 0.4 ± 0.3 ± 0.1 ± 0.1 ± 5.2 ± 0.2 ± 0.2 |

**Abbreviations:** BSA = body surface area; LA = left atrial dimension; LVIDd = left ventricular diastolic dimension; LVIDs = left ventricular systolic dimension; IVS = ventricular septal thickness; LVPW = left ventricular posterior wall thickness; EF = left ventricular ejection fraction; LVOT = left ventricular outflow tract dimension; MVE = mitral valve early diastolic (D-E) excursion; MVP = mitral valve prolapse; MCB = mitral chordal buckle; + = present; - = absent; P = probable; ? = uncertain.
Comparison of mitral valve early diastolic (D-E) excursion in a group of normal control subjects and in group 1 (systolic anterior motion [SAM] only) and group 2 (SAM with mitral valve prolapse [MVP]) patients. The mean ± SEM for each group is depicted next to the values (in cm) for individual patients.

Figure 2. (A) M-mode echocardiogram at the level of the mitral valve from patient 6, with systolic anterior motion (SAM) only (group 1). SAM begins abruptly in early systole. RV = right ventricle; IVS = interventricular septum; LVPW = left ventricular posterior wall; AML = anterior mitral leaflet. (B) M-mode echocardiogram at the mitral valve level from patient 16, with SAM and mitral valve prolapse (MVP) (group 2). SAM in early systole is followed by late systolic MVP.
echocardiograms from group 1 and group 2 patients. Figure 2A is a characteristic M-mode echocardiogram demonstrating the mitral valve from a group 1 patient. SAM begins in early systole as an abrupt angular motion and returns to the C-D line in mid-to-late systole. The ventricular septal and posterior wall thicknesses are normal. All 10 patients in group 1 demonstrated this type of early SAM. Inhalation of amyl nitrite did not accentuate SAM in any of the patients in whom this maneuver was performed.

Figure 2B is a characteristic M-mode echocardiogram of the mitral valve from a group 2 patient. SAM begins in early systole, followed by a late systolic posterior motion (prolapse) behind the C-D line. All group 2 patients had findings of early SAM followed by late systolic MVP. Echocardiographic scans in each patient from the level of the aortic root and left atrium to the lower left ventricle demonstrated that SAM did not, in fact, represent beam-width artifact from the posterior aortic root wall or an artifact related to improper transducer angulation. None of our 15 normal subjects had SAM or MVP.

Cross-sectional Echocardiographic Findings

Of the 10 patients in group 1, nine showed definite or probable anterior buckling of mitral subvalvular (chordal) structures (MCB) by cross-sectional echocardiography (table 2). The probable MCB was composed of patients in whom systolic anterior buckling of mitral subvalvular structures was present but could not be visualized during every cardiac cycle. In patients 2, 7 and 9, the mitral chordal junction, including the tip of the anterior mitral leaflet, appeared to be involved in the MCB.

Figures 3 and 4 illustrate the phenomenon of MCB in two group 1 patients as visualized with cross-sectional echocardiography. These long-axis sequences of systolic and diastolic frames demonstrate an acutely angulated early SAM of mitral subvalvular structures giving rise to the appearance of buckling or kinking. The body of the anterior mitral leaflet moves normally (not toward the septum during systole).

Although MCB could also be visualized in short-axis and apical four-chamber views, the long-axis view was the best for imaging this phenomenon.

All five group 2 patients demonstrated MCB, followed by one of the abnormal mitral leaflet coaptation patterns described in MVP.28 Four group 2 patients demonstrated coaptation of the tips of both anterior and posterior mitral leaflets behind (cephalad to) the plane of the mitral annulus, whereas one patient demonstrated prolapse of, primarily the anterior mitral leaflet. None of the 15 normal subjects demonstrated either MCB or an abnormal mitral coaptation pattern.
Figure 5 is a diagram of the MCB phenomenon characteristic of group 1 and group 2 patients. Fourteen of the 15 patients in groups 1 and 2 studied by cross-sectional echocardiography demonstrated either definite or probable MCB.

Discussion

Our data confirm the findings of others that the presence of SAM on the M-mode echocardiogram is not diagnostic of hypertrophic (obstructive) cardiomyopathy, but is seen frequently in other forms of heart disease.\textsuperscript{10–18} Using cross-sectional echocardiography, we demonstrated MCB as the underlying mechanism for SAM in our two groups.

The findings of mitral chordal (and mitral-chordal junction) buckling in our five patients with SAM and MVP is similar to the description of one patient by Terasawa and co-workers,\textsuperscript{16} who, by cross-sectional echocardiography, noted that SAM was due to protrusion into the left ventricular outflow tract of the slackened chordae tendineae in early systole. When the slackened chordae tendineae were stretched taut in midsystole, the anterior leaflet moved to the position of maximal prolapse. Furthermore, in a recent preliminary report, Pearlman and co-workers have also described buckling of the mitral chordae (often involving the tip of the anterior leaflet as well) as the etiology for SAM in MVP.\textsuperscript{29} Our findings do not confirm the suggestion of Sahn et al. that the appearance of SAM in mitral valve prolapse is created by superimposition of the mitral annulus and mitral valve leaflet.\textsuperscript{18}

MCB differs from the SAM of the anterior mitral leaflet into the left ventricular outflow tract initially described for hypertrophic obstructive cardiomyopathy by Henry et al.\textsuperscript{30} More recently, however, other workers have emphasized other mechanisms for the production of SAM in hypertrophic cardiomyopathy. Rodger\textsuperscript{31} described SAM as a complex of echoes from the chordae tendineae, the papillary muscle and the anterior mitral leaflet, the latter structure being the furthest from and not impinging upon the septum. In addition, Tajik and co-workers\textsuperscript{32} have noted the site of obstruction in hypertrophic cardiomyopathy at various levels within the left ventricular outflow tract, involving apposition of different portions of the septum with the anterior mitral leaflet and/or chordae tendineae or papillary muscle. Therefore, it is recognized that there may be various etiologies for the SAM associated with hypertrophic obstructive cardiomyopathy.\textsuperscript{30–32}

The question of whether MCB can cause left ventricular outflow obstruction has only been partially addressed by our investigation. In four of our patients in whom amyl nitrite was administered, we could not find evidence by M-mode echocardiography of increased SAM or of left ventricular outflow tract narrowing. Crawford and associates\textsuperscript{7} described two patients with SAM in the absence of asymmetric septal hypertrophy who developed significant subaortic gradients upon provocation. One of Crawford’s patients was clearly different from our group in that his left ventricular outflow tract (measured as 2.4 cm in their figure 4) was narrower than that of any group 1 or group 2 patient in our series. Furthermore, their patient demonstrated midsystolic closure of the aortic valve leaflets, again not seen on M-mode echocardiography in any of our patients. Mintz and co-workers\textsuperscript{8} described 10 patients with SAM, without asymmetric septal hypertrophy, concentric left ventricular hypertrophy, or other structural cardiac abnormality, in whom left ventricular outflow tract obstruction could be demonstrated. Four of these patients underwent cardiac catheterization. One patient had a significant resting subaortic gradient and three patients had a provokable gradient. The patients in their series differ from our series echocardiographically in that their left ventricular outflow tracts were significantly narrower (2.2 ± 0.4 cm) than those in our patients (3.2 ± 0.3 cm, \( p < 0.01 \)). Furthermore, these workers describe a patient in whom, after provocation with amyl nitrite, the outflow tract narrows and the wall motion increases concomitantly with the occurrence of SAM.

It appears that there are two distinct subgroups of patients with SAM in the absence of asymmetric septal hypertrophy: one subgroup that does not demonstrate a left ventricular outflow gradient (nonobstruc-
tive SAM) and a second subgroup that has either a resting or provocative gradient (obstructive SAM). Our group of patients is probably similar to eight patients described in a report by Boughner and coworkers, who had SAM without asymmetric septal hypertrophy and no evidence of left ventricular outflow obstruction by Doppler or M-mode echocardiography, either at rest or after amyl nitrite inhalation. None of our patients were sufficiently symptomatic to undergo cardiac catheterization with provocative maneuvers to definitely exclude the presence of a resting or provocative left ventricular outflow gradient.

Finally, the finding of a continuum for mitral valve diastolic (D-E) excursion (and perhaps also for left ventricular ejection fraction) from normal subjects to patients with SAM only (group 1), to patients with SAM and MVP (group 2) raises the possibility that there is a spectrum of mitral valve abnormality that may become manifest as MCB alone, MVP alone, or as both conditions. One might postulate that this valvular abnormality might reflect both structural and hemodynamic factors. Mitral valve hooding, thickening, and redundant chordae tendineae (excessive chordal length relative to left ventricular long-axis dimension) have been described at necropsy in patients who had MVP. The association of a hyperadrenergic state with MVP has also been described. Perhaps the predisposition to MCB in our group 1 (SAM without MVP) and group 2 (SAM with MVP) patients reflects the relative redundancy of the chordae tendineae in these patients with or without a superimposed hyperadrenergic state. This hypothesis, however, requires further confirmation.

Acknowledgment

The authors acknowledge the expert assistance of Lillian Celic with the statistical analysis and of Tiny Kent and Melanie Merutka in preparation of the manuscript.

References

31. Rodger JC: Motion of mitral apparatus in hypertrophic car-
M-mode and Two-dimensional Echocardiographic Features in Cardiac Amyloidosis

ARISTARCO G. SIQUEIRA-FILHO, M.D., CLAUDIO L. P. CUNHA, M.D., ABDUL J. TAJIK, M.D., JAMES B. SEWARD, M.D., THOMAS T. SCHATTENBERG, M.D., AND EMILIO R. GIULIANI, M.D.

SUMMARY  Twenty-eight patients with cardiac amyloidosis were studied by echocardiography — 26 by M-mode and 13 by two-dimensional (2D) studies. All had heart failure and biopsy-proved amyloidosis. M-mode features included (1) normal left ventricular (LV) dimension in all; (2) thickened ventricular septum (88%), LV posterior wall (77%), and right ventricular (RV) anterior wall (79%); (3) decreased thickening of ventricular septum (96%) and of LV posterior wall (65%) and reduced LV global function (62%); (4) left atrial enlargement (50%); and (5) pericardial effusion (58%). Two-dimensional echocardiography provided additional features: (1) thickened papillary muscles (five of 13); (2) thickened valves (four of 13); (3) better appreciation of thickened RV wall; and (4) a characteristic “granular sparkling” appearance of thickened cardiac walls — presumably secondary to the amyloid deposit — which was noted in 12 of 13 patients. Thus, M-mode echocardiography is helpful in the recognition of cardiac amyloidosis. However, the better appreciation with 2D echocardiography of thickened cardiac walls with a “granular sparkling” appearance in patients with unexplained cardiac failure is virtually diagnostic of cardiac amyloidosis.

CLINICALLY SIGNIFICANT cardiac amyloidosis accounts for 5–10% of all forms of isolated non-coronary cardiomyopathy. Although the clinico-pathologic findings of such an unusual form of heart disease have been well described, only a few investigators have reported the echocardiographic features of amyloid cardiomyopathy, and their observations and conclusions are based on small numbers of patients. Two-dimensional echocardiography has increased our ability to diagnose several forms of cardiac disease. However, to our knowledge, there have been no reports on two-dimensional echocardiographic features of amyloid heart disease.

The purpose of this retrospective study was to analyze in detail the M-mode echocardiographic features in a relatively large group of patients with systemic amyloidosis, and to describe the two-dimensional echocardiographic features of this disease.

Methods

Patients

The study population comprised 28 patients, all of whom were examined at the Mayo Clinic. All had biopsy-proved amyloidosis. Clinical profile, ECG findings, chest roentgenogram, and pathologic findings of these patients are presented in Table 1. Thirteen patients (cases 1–13) had tissue-proved cardiac amyloidosis (eight autopsies, three biopsies during pericardectomy and two transvenous endomyocardial biopsies). Fifteen patients (cases 14–28) had evidence of heart disease in the presence of tissue-proved amyloidosis elsewhere in the body. Eleven patients had both M-mode and two-dimensional echocardiograms, 15 had only M-mode, and two had only two-dimensional examination. Six patients underwent cardiac catheterization, four of whom had coronary arteriography, which was normal in all. Detailed hemodynamics will not be discussed.

M-mode Echocardiography

M-mode echocardiograms were obtained with a commercially available ultrasonoscope (Smith Kline Instruments, Ekoline 20A) and recorded with a multichannel strip-chart recorder (Cambridge or Honeywell 1856). Studies were performed with a 2.25-MHz transducer with a repetition rate of 1000 Hz.
Systolic anterior motion in the absence of asymmetric septal hypertrophy. A buckling phenomenon of the chordae tendineae.
J M Gardin, J V Talano, L Stephanides, J Fizzano and M Lesch

Circulation. 1981;63:181-188
doi: 10.1161/01.CIR.63.1.181

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