On the Mechanism of the Austin Flint Murmur

By Nicholas J. Fortuin, M.D., and Ernest Craige, M.D.

SUMMARY

We studied mitral valve motion in 15 patients with aortic regurgitation and an Austin Flint murmur by recording simultaneously the phonocardiogram, apexcardiogram, and mitral valve echocardiogram. In 14 patients the murmur was presystolic, and in 13 of these a mid-diastolic component was present as well. The mid-diastolic component occurred after the period of rapid ventricular filling as the mitral valve was closing. The presystolic component occurred either as the mitral valve was rapidly closing following normal atrial opening movement or during the entire period of atrial systole. In this latter situation the valve did not open completely with atrial systole. All of these patients demonstrated a prominent A wave on the apexcardiogram. In patients with a two-component Flint murmur, isometric handgrip exercise greatly accentuated the murmur, altered the pattern of valve motion so that atrial systole was no longer effective in opening the valve, and reduced the size of the apex A wave.

One patient had only a mid-diastolic Flint murmur. The mitral valve was open for only a brief period in mid-diastole and did not reopen with atrial systole. Cardiac catheterization documented elevation of ventricular diastolic pressures above left atrial pressures during most of diastole. Amyl nitrite reduced diastolic ventricular pressure and permitted the mitral valve to open with atrial systole. The apex A wave, which was small at rest, increased in size after amyl nitrite.

Our findings suggest that the Flint murmur is due to antegrade flow across the mitral valve. The rumble occurs during rapid closure of the valve as flow velocity is increasing although actual volume of flow may be decreasing.

Additional Indexing Words:

Echocardiography Ultrasound Aortic regurgitation Mitral valve
Apexcardiography Phonocardiography Amyl nitrite

Several explanations have been proposed for the apical diastolic rumbling murmur which often accompanies pure aortic regurgitation since the initial description of the murmur by Austin Flint in 1862.¹⁻⁵ Most of these have postulated that an abnormal position of the mitral valve leaflets is of importance in producing the murmur. Flint and later Herrmann suggested that the murmur was due to the presence of a functional mitral stenosis resulting from incomplete opening of the anterior mitral leaflet because of abnormal ventricular filling or impingement on the leaflet by the aortic regurgitant jet during diastole.¹⁻⁵ Others have ascribed the murmur to vibrations set up in the anterior mitral leaflet as it oscillates between the antegrade stream of blood from

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the left atrium and the retrograde stream from the aorta. More recently, several groups have observed late diastolic mitral regurgitation in patients with severe aortic regurgitation and have suggested that this may be responsible for the murmur. Simultaneous pressure measurements in the left ventricle and left atrium have demonstrated that, with severe aortic regurgitation, diastolic left ventricular pressure may exceed left atrial pressure and thus predispose to late diastolic mitral regurgitation.

We undertook the present investigations to elucidate further the mechanism of production of this murmur by correlating the position and motion of the anterior leaflet of the mitral valve with the Flint murmur. To do this we recorded simultaneously the phonocardiogram, mitral valve echocardiogram, and apexcardiogram in 15 patients with aortic regurgitation and a Flint murmur. We obtained further studies of the relationship of the murmur to valve position by alteration of the

### Table 1

**Description of Patients, Flint Murmur, Mitral Valve Motion, and ACG**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Etiology</th>
<th>Austin Flint murmur</th>
<th>Mitral valve motion</th>
<th>ACG (A/H%)</th>
<th>DFP (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57</td>
<td>BE</td>
<td>Crescendo presystolic</td>
<td>Double-domed, normal atrial opening and rapid presystolic closure</td>
<td>14</td>
<td>−160</td>
</tr>
<tr>
<td>2</td>
<td>23</td>
<td>RHD</td>
<td>2-component, crescendo presystolic</td>
<td>Double-domed, normal atrial opening and rapid presystolic closure</td>
<td>13</td>
<td>−70</td>
</tr>
<tr>
<td>3</td>
<td>16</td>
<td>RHD</td>
<td>2-component, crescendo presystolic</td>
<td>Double-domed, normal atrial opening and rapid presystolic closure</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>31</td>
<td>RHD</td>
<td>2-component, primarily presystolic</td>
<td>Double-domed, incomplete opening with atrial systole</td>
<td>11</td>
<td>−60</td>
</tr>
<tr>
<td>5</td>
<td>56</td>
<td>BE</td>
<td>2-component, primarily presystolic</td>
<td>Double-domed, incomplete opening and rapid closure with atrial systole</td>
<td>17</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>23</td>
<td>RHD</td>
<td>2-component, primarily presystolic with crescendo into S1</td>
<td>Double-domed, normal atrial opening and rapid presystolic closure</td>
<td>13</td>
<td>+20</td>
</tr>
<tr>
<td>7</td>
<td>32</td>
<td>RHD</td>
<td>2-component, primarily presystolic with crescendo into S1</td>
<td>Double-domed, normal atrial opening and rapid presystolic closure</td>
<td>13</td>
<td>−40</td>
</tr>
<tr>
<td>8</td>
<td>60</td>
<td>Unknown</td>
<td>2-component, primarily presystolic with crescendo into S1</td>
<td>Double-domed, normal atrial opening and rapid presystolic closure</td>
<td>25</td>
<td>−110</td>
</tr>
<tr>
<td>9</td>
<td>27</td>
<td>RHD</td>
<td>2-component primarily presystolic</td>
<td>Double-domed, incomplete opening with atrial systole</td>
<td>12</td>
<td>−70</td>
</tr>
<tr>
<td>10</td>
<td>25</td>
<td>BE</td>
<td>2-component</td>
<td>Double-domed, incomplete opening with atrial systole</td>
<td>17</td>
<td>−150</td>
</tr>
<tr>
<td>11</td>
<td>58</td>
<td>Unknown</td>
<td>2-component</td>
<td>Double-domed, incomplete opening with atrial systole</td>
<td>18</td>
<td>+20</td>
</tr>
<tr>
<td>12</td>
<td>77</td>
<td>Unknown</td>
<td>2-component, primarily presystolic</td>
<td>Double-domed, normal atrial opening with rapid presystolic closure</td>
<td>23</td>
<td>−70</td>
</tr>
<tr>
<td>13</td>
<td>26</td>
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<tr>
<td>14</td>
<td>64</td>
<td>Rheumatoid arthritis</td>
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<td>Double-domed, incomplete opening with atrial systole</td>
<td>−</td>
<td>+10</td>
</tr>
<tr>
<td>15</td>
<td>23</td>
<td>BE</td>
<td>Middiastolic</td>
<td>Single-domed with premature closure in middiastole</td>
<td>5</td>
<td>−280</td>
</tr>
</tbody>
</table>

Abbreviations: BE = bacterial endocarditis; RHD = rheumatic heart disease; ACG = apexcardiogram; DFP = diastolic filling period.
murmur with isometric handgrip exercise and the inhalation of amyl nitrite.

Methods

The 15 patients studied had severe aortic regurgitation as determined by the clinical criteria of increased cardiac size, a diastolic blood pressure of less than 60 mm Hg, and the presence of peripheral signs of excessive diastolic runoff. No other valvular lesions were felt to be present by clinical examination. Mitral stenosis was excluded by the mitral valve echocardiogram which in all cases demonstrated a diastolic slope in excess of 100 mm/sec. Aortic valve replacement was subsequently necessary in four of these patients and in two additional patients was recommended but refused. The ages and clinical etiologic diagnoses of the aortic valve disease are described in table 1. All patients had a low-pitched mid- or late diastolic rumbling murmur at the apex which differed in timing from the aortic regurgitant murmur and could be recorded phonocardiographically.

Phonocardiograms (PCG) were recorded on a Cambridge multichannel recorder using Cambridge (Leatham) microphones. Phonocardiograms were obtained from the area at the cardiac base to the right or left of the sternum where the aortic diastolic blowing murmur was best heard and simultaneously at the cardiac apex with the patient in a slight left lateral decubitus position. Variable filters provided in the amplifier unit of the recorder were set at medium frequency for recordings in the mitral area and high frequency for the recordings at the base. Apex tracings (ACG) were made with a Hellige pulse transducer, as described previously. The height of the A wave of the ACG was considered in relation to the entire apical excursion and recorded as an A/H ratio. The mitral valve echocardiogram was obtained by means of a Smith-Kline Ekoline 20 ultrasound unit employing an 0.75-inch transducer by technics described previously. An electronic gating circuit allowed display of the mitral valve signal on the phonocardiographic record together with the PCG, ACG, and a single electrocardiographic lead. Recordings were made at a paper speed of 100 mm/sec.

In patients 3, 10, and 11, PCG, ACG, and mitral valve echo were recorded after 1.5 minutes of isometric exercise with a handgrip dynamometer at 60 to 70% of maximal capacity. Patient 15 inhaled amyl nitrite, and records were made 30 seconds later during the peak effect of the drug. In this patient the effects of amyl nitrite on left atrial and left ventricular pressure were assessed within 24 hours of the phono-echo study at the time of diagnostic left-heart catheterization. Left

![Figure 1](http://circ.ahajournals.org/)

**Figure 1**

Combined phono-echo record from patient with moderate aortic regurgitation without an Austin Flint murmur. See text for further discussion. PCG = phonocardiogram; PA = pulmonary area; HF = high frequency; EDM = early diastolic murmur; 1 = first heart sound; 2 = second heart sound. Heavy time lines = 0.2 sec.
ventricular volumes were also measured in this patient from biplane angiocardiograms obtained after injection of radiographic contrast agent into the aortic root. The method of Dodge et al.\textsuperscript{15} was used to compute the volumes.

The duration of the diastolic filling period (DFP) was determined in each case from phono-echo record as the time from the initiation of valve opening on the echogram to the time of completion of mitral valve closure. The mean of five measurements was recorded as the final value in each patient. Since this period is inversely related to heart rate, each subject's value was compared with a regression equation relating DFP to heart rate obtained from 12 normal subjects (N.J. Fortuin: Unpublished data). DFP was expressed as the difference above (+) or below (−) the normal predicted value for the subject's heart rate.

Results

In figure 1 a normal mitral valve echogram recorded simultaneously with PCG and ECG from a patient with mild aortic regurgitation without an Austin Flint murmur is presented. Shortly after the second heart sound, the mitral valve begins to open, rapidly achieves its maximum opening excursion, immediately begins a gradual closing movement, reaches a brief period of stable near-closure in late diastole, and then abruptly reopens with atrial systole. This is followed by a more rapid closing movement which results in almost complete valve closure prior to the onset of ventricular systole.

In 14 patients the Austin Flint murmur was recorded as presystolic, and in all but one of these a middiastolic component was present as well (table 1). In these patients the A wave of the ACG was prominent (A/H ratio 11 to 25%), and the mitral valve echogram demonstrated two diastolic opening movements as in the normal situation (table 1). The presystolic component of the Austin Flint murmur was often crescendo in character, peaking into a soft first heart sound (fig. 2). In these instances the mitral valve opened normally with atrial systole, but the closing movement was more rapid than normal. As illustrated in figure 2, the crescendo murmur is coincident with the rapid closing movement of the mitral valve. In other patients the presystolic component to the murmur was longer, beginning with the opening movement of the mitral valve with atrial systole and proceeding as a

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure2.png}
\caption{Phono-echo record from patient 1 to illustrate presystolic Flint murmur (PSM). Note that PSM has its onset at peak of valve opening and becomes louder as valve is closing. Distortion of mitral valve echo in systole and early diastole is due to artifact in the tracing. MA = mitral area; MF = midfrequency; AA = aortic area; SEM = systolic ejection murmur.}
\end{figure}
crescendo-decrescendo murmur (fig. 3). In these subjects the valve did not open fully with atrial systole and did not have an excessively rapid closing movement (see figs. 3 and 5).

The midsystolic component of the Austin Flint murmur could be readily differentiated from the aortic diastolic murmur by its lower frequency and because it became louder as the early diastolic murmur became softer. This murmur occurred just after the valve had reached its peak opening in early diastole and became louder as the early systolic murmur became softer. This murmur occurred just after the valve had reached its peak opening in early diastole and became louder as the early systolic murmur became softer. This murmur occurred just after the valve had reached its peak opening in early diastole and became louder as the early systolic murmur became softer.

Isometric handgrip exercise resulted in increased systolic blood pressure and heart rate and augmented the systolic and diastolic murmurs of aortic regurgitation in three patients with a two-component Flint murmur and double-domed valve motion pattern. Figure 4 illustrates the effects on heart sounds and the mitral valve echograms. The Austin Flint murmur, which is recorded at the mitral area, has its onset well after the early diastolic murmur. It begins at the time of mitral valve opening and ends with mitral valve closure. The mitral valve echogram is remarkably altered. In comparison with the resting pattern (fig. 3), there has been almost total obliteration of the opening movement due to atrial systole. In figure 5 the effects of isometric exercise on the mitral valve echogram and simultaneous ACG are demonstrated in another patient. Again, the opening movement of the valve due to atrial systole is lost. In addition, the A wave of the ACG, which was prominent before handgrip exercise, is much diminished in size. In a third patient (no. 3), who had clinically milder

Figure 3

Phono-echo record in patient 11 with two-component Austin Flint murmur (AFM). The AFM has its onset in middiastole as the early diastolic aortic murmur (EDM) is diminishing and occurs while the mitral valve is closing. The second component of AFM occurs coincident with atrial systole. At the time of this murmur, the mitral valve opens incompletely. Abbreviations as in previous figures.

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Aortic regurgitation than the previous two, isometric handgrip exercise intensified the murmurs but did not alter the opening movement of the mitral valve produced by atrial systole. In this patient the A wave of the ACG increased in size.

In patient 15 (fig. 6), the Austin Flint murmur was confined to middiastole. Unfortunately, this murmur, which was loud and easily recognizable by auscultation, could not be well differentiated from a loud early diastolic regurgitant murmur by phonocardiography. However, the PCG did document the absence of presystolic murmur. The ACG had only a small A wave, and the mitral valve echogram displayed only an early diastolic opening movement followed by rapid closure. The valve was open for only a brief period in diastole and did not reopen with atrial systole. During cardiac catheterization simultaneous recording of left ventricular pressure with left atrial and central aortic pressures (fig. 7) demonstrated that left atrial pressure exceeded left ventricular pressure only in early diastole, and thereafter left ventricular pressure exceeded left atrial pressure. In addition, aortic pressure equilibrated with left ventricular pressure during left diastole. The severity of the aortic regurgitation in this patient was further documented by quantitation of an enormous increase in end-diastolic volume and a large regurgitant volume (table 2). Forward stroke volume measured by the Fick technic was normal.

The administration of amyl nitrite to this patient significantly altered the pattern of valve motion and permitted the valve to reopen with atrial systole (fig. 8). During catheterization amyl nitrite caused a marked reduction in left ventricular pressure during all of diastole (fig. 9). This patient underwent aortic valve replacement, and after surgery mitral valve motion was normal (fig. 10).

The difference of the DFP from that predicted for heart rate for each patient is...
(Top) Resting record of patient 10, who also had a two-component Flint murmur. Note similarity of valve motion pattern to that in figure 3. Simultaneous apex-cardiogram (Apex) demonstrates prominent rapid filling wave (F) and A wave. (Bottom) Record obtained after 1.5 min of isometric handgrip exercise. Note loss of mitral valve opening movement due to atrial systole and diminution of size of apex A wave.
Austin Flint originally described a presystolic, "blubbing" murmur associated with severe aortic regurgitation. The presystolic timing of the murmur was agreed on by several subsequent observers, although others have emphasized the middiastolic component of the murmur. We have recorded the murmur at both times in our patients but have found that most often it is a

**Table 2**
Cardiac Catheterization Data from Patient 15

<table>
<thead>
<tr>
<th>Pressures</th>
<th></th>
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<tbody>
<tr>
<td>Aortic</td>
<td>145/60 mm Hg</td>
</tr>
<tr>
<td>Left ventricular</td>
<td>150/0/60 mm Hg</td>
</tr>
<tr>
<td>Left atrial</td>
<td>a = 46 mm Hg</td>
</tr>
<tr>
<td></td>
<td>v = 50 mm Hg</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>Mean = 30 mm Hg</td>
</tr>
<tr>
<td></td>
<td>55/20 mm Hg</td>
</tr>
<tr>
<td>Volumes</td>
<td></td>
</tr>
<tr>
<td>End-diastolic</td>
<td>436 ml</td>
</tr>
<tr>
<td>End-systolic</td>
<td>242 ml</td>
</tr>
<tr>
<td>Total stroke</td>
<td>194 ml</td>
</tr>
<tr>
<td>Forward stroke (Fick)</td>
<td>63 ml</td>
</tr>
<tr>
<td>Regurgitant</td>
<td>131 ml</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>45%</td>
</tr>
<tr>
<td>Ventricular mass</td>
<td>442 g</td>
</tr>
</tbody>
</table>

described in table 1. Ten of the 15 patients had shortened DFP.

**Discussion**

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two-component murmur with both middiastolic and presystolic timing. In only one case, a patient with severe hemodynamic effects, was the murmur confined to middiastole. The preponderance of patients with presystolic murmurs in this study may be a reflection of patient selection since most of our subjects had a relatively stable hemodynamic situation and mild symptoms. Patients studied at the time of cardiac catheterization in preparation for cardiac surgery generally have a more severe hemodynamic burden from aortic regurgitation and would be expected more often to manifest only a middiastolic murmur.

In our patient (no. 15) with a middiastolic Flint murmur, the mitral valve was open for only a brief period of time during diastole and failed to reopen with atrial systole. This kind of valve motion has also been noted by Pridie et al. in patients with severe, acute aortic regurgitation.21 The early diastolic closure of the valve occurred because of a rapid rise in left ventricular diastolic pressure to levels which exceeded left atrial pressure during most of the diastole (fig. 7). This reversal of the normal left atrial-ventricular pressure difference produced premature valve closure in middiastole and prevented reopening of the valve by atrial contraction. The rapid rise in ventricular diastolic pressure was the result of massive and rapid distention of the ventricle by free aortic reflux and antegrade flow across the mitral valve. The magnitude of the reflux was documented in this case by angiographic volume measurements (table 2).

**Figure 8**

Simultaneous mitral valve (MV) echo and apex tracing in patient 15 during peak effect of inhaled amyl nitrite. In comparison with figure 6, the valve now reopens with atrial systole (A), and the A wave of apex tracing has become larger. E = peak opening movement of the valve; O = nadir of the apex tracing.

**Figure 9**

Effects of amyl nitrite on left atrial and ventricular pressures. At peak effect of drug, there has been a fall in left ventricular diastolic pressure so that left atrial pressure coincides with left ventricular pressure during all of diastole.
Antegrade ventricular filling, which was of normal volume (table 2), occurred only during the brief period in diastole during which left atrial pressure exceeded left ventricular pressure and the mitral valve was open. Since the Austin Flint murmur was heard only in middiastole in this patient, it is likely that antegrade flow across the valve was responsible for production of the murmur. We propose that the murmur occurred because the shortened diastolic filling period with normal antegrade flow resulted in increased velocity of antegrade flow across the valve. Velocity of flow was further augmented because the mitral valve was closing and thus reducing its orifice size during much of the filling period. Thus, a turbulent flow jet of great velocity was produced, which is precisely the situation that Rushmer suggests is responsible for most heart murmurs.22

Extension of this line of reasoning can be used to explain the mechanism of the Flint murmur in patients in whom the hemodynamic consequences of aortic reflux are not so severe. These patients generally manifest a two-component or solely presystolic murmur. In most of them, the diastolic filling period is abbreviated because of prolonged ejection time. We have found that the middiastolic component of the murmur occurs as the mitral valve is closing rapidly. This closure movement is excessively rapid because of the rise in ventricular pressure which results from combined filling of the ventricle from aorta and left atrium.21 We postulate that as a result of the early aortic reflux, left atrial emptying is incomplete during the period of rapid ventricular filling and blood continues to flow across the mitral valve beyond this period as the mitral valve is closing. Continued flow across a narrowing orifice may be responsible for the early diastolic left atrial-left ventricular pressure gradient which has been described in patients with the Flint murmur by others9 and was seen in patient 15 in this study (fig. 7). The narrowing mitral valve orifice and continued antegrade flow result in increased velocity of flow, even though the actual amount of flow may be diminished. Turbulence set up in the antegrade stream is then responsible for production of a murmur. This mechanism of increasing flow velocity while the valve orifice size is diminishing has been proposed by Criley23 to explain the crescendo quality of the presystolic murmur of mitral stenosis and may also be applicable to the middiastolic rumble which accompanies mitral regurgitation.24, 25
large left-to-right shunts, or high output states. The middiastolic component of the Austin Flint murmur and the murmurs accompanying these latter conditions are similar low-frequency rumbling noises which occur in middiastole after the period of rapid ventricular filling. A unifying mechanism of production, as proposed here, would help to explain why they have similar auscultatory characteristics.

This explanation may also be applied to the presystolic component of the Flint murmur. Excessively rapid closure of the mitral valve following atrial systole, as noted in some of our patients, probably occurs because of a rapid rise in late ventricular diastolic pressure. Continued antegrade flow across a rapidly narrowing orifice therefore produces increasing flow velocity and a crescendo Flint murmur (fig. 2). In other patients the presystolic component of the murmur begins earlier with the onset of atrial systole (fig. 3). In this situation atrial contraction may be more forceful because of increased distention which occurs as a result of incomplete atrial emptying in early diastole. In these patients the valve does not open fully with atrial systole, presumably because of increased ventricular pressure in late diastole. The combination of increased force of atrial systole and incomplete valve opening (figs. 2 and 4) results in turbulent flow and a rumbling murmur.

Isometric handgrip exercise, which elevates peripheral resistance, would be expected to increase the relative volume of aortic reflux in patients with aortic regurgitation. We found that this maneuver resulted in a marked increase in the intensity of the murmurs associated with aortic regurgitation, particularly the Austin Flint murmur. The pattern of mitral valve motion was also altered, with almost complete loss of the opening movement as a result of atrial systole in two patients. Thus, the two-component Flint murmur was changed to a single loud murmur in middiastole. The valve motion pattern during isometric exercise resembled that seen in patient 15 at rest. This probably occurred because the added aortic reflux caused elevation of left ventricular diastolic pressure above left atrial pressure in the latter portion of diastole, as in patient 15, and therefore prevented the normal valve opening motion with atrial systole. In one patient isometric handgrip did not alter the atrial opening movement in the mitral valve echogram. In this patient the added reflux was probably not sufficient to elevate left ventricular diastolic pressure above left atrial pressure. This patient may have been operating on a lower level of the diastolic ventricular pressure volume curve than the other two; similar increments in volume therefore would not result in a similar increase in left ventricular pressure.

As in the resting situation, with isometric exercise the Flint murmur occurred only when the mitral valve was open. Because of the altered left ventricular dynamics the valve was open for a shorter period of time, and thus velocity of flow across it could be expected to increase. Elevated left atrial pressures with resultant increased force to the antegrade stream may also be in part responsible for the increased intensity of the murmur in this situation.

We used amyl nitrite to alter the Flint murmur and valve motion pattern in patient 15. This drug reduces peripheral resistance and would be expected to reduce the volume of aortic reflux. In patients with aortic regurgitation, the Flint murmur has been reported to decrease and left ventricular diastolic pressure to fall following inhalation of amyl nitrite. In our patient amyl nitrite reduced diastolic ventricular pressure, so that the mitral valve could reopen with atrial systole. The duration of diastolic filling was thereby prolonged, and atrial systole could then make its contribution. This observation may explain why the Flint murmur is softer in this type of situation.

A previous study from our laboratory described a correlation between the presence of an Austin Flint murmur and the severity of aortic regurgitation as determined by left ventricular volume measurements. The observations reported here suggest a relationship
between the timing of the Flint murmur and the hemodynamic severity of the aortic leak. Thus, with mild or moderate reflux, no or only a slight alteration in ventricular diastolic pressures occurs, and no Flint murmur is heard. With moderately severe reflux, left ventricular pressure rises in late diastole following atrial systole, resulting in rapid valve closure after a normal opening movement and a crescendo presystolic Flint murmur (fig. 2). With increasingly severe aortic regurgitation, ventricular pressures are elevated earlier in diastole, and the mitral valve cannot open fully with atrial systole; a longer presystolic murmur is heard in this situation as well as a middiastolic rumble (fig. 3). With very severe chronic aortic regurgitation or acute aortic regurgitation, left ventricular pressures are elevated in middiastole and exceed left atrial pressures. The mitral valve is open only during middiastole; therefore, the Flint murmur occurs only at this time (fig. 6). In any patient the timing and intensity of the Flint murmur and the pattern of mitral valve motion can be moved forward or backward along this scale by the use of isometric exercise or amyl nitrite, respectively. These simple maneuvers may therefore aid in the bedside evaluation of the hemodynamic severity of the aortic reflux.

The studies of valve motion reported here do not support the theory which postulates that the Flint murmur is due to the left ventricular inflow obstruction by mitral leaflets held in a semiclosed position during diastole by the regurgitant stream.\(^5\) Similarly, the suggestion that the murmur is due to "relative mitral stenosis" occasioned by a normal-sized valve and large ventricular cavity is not supported by our studies.\(^{18, 20}\) The fact that the Flint murmur occurs only when the mitral valve is open makes it unlikely that diastolic mitral regurgitation could be responsible for the presystolic component of the murmur, as suggested recently by others. Actually, Flint himself came the closest to predicting the pattern of valve motion described here.\(^1, 32\) He postulated that the mitral valve leaflets were forced into a closed position during mid-

diastole as a result of distention of the ventricle by antegrade and regurgitant flow and proposed that the murmur occurred in presystole as the atrium contracted against a nearly closed mitral valve.

In our previous study of aortic regurgitation, we found that a large A wave in the apexcardiogram was associated with elevated left ventricular end-diastolic pressures (LVEDP) but that in some patients with high LVEDP the A wave was not increased.\(^{14}\) Correlation of mitral valve motion with the ACG in the present study provides an explanation for this discrepancy. In all patients in whom the mitral valve opened with atrial systole, the A wave was prominent. However, when LVEDP became so high that it exceeded left atrial pressure and the mitral valve did not open with atrial systole, as in patient 15, the ACG A wave was small.\(^{33}\) This situation was induced in two patients (no. 10 and 11) with handgrip. In patient 3, on the other hand, the A wave increased during isometric handgrip, and the valve echogram showed that the valve continued to open normally with atrial systole. When the LVEDP of patient 15 was lowered with amyl nitrite and the mitral valve could open with atrial systole, a large A wave appeared on the ACG. Thus, the mitral valve echogram in conjunction with the ACG allowed evaluation of the relationship between left atrial and ventricular diastolic pressures.

These studies provide evidence that the Austin Flint murmur, which is commonly heard in patients with severe aortic regurgitation, is in essence a flow rumble produced by antegrade flow across the mitral valve. Further hemodynamic studies done in conjunction with the noninvasive methodology employed here will very likely provide additional insight into some of the observations reported.

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