The Austin Flint Murmur and the a Wave of the Apexcardiogram in Aortic Regurgitation

By Elizabeth Parker, M.D., Ernest Craige, M.D., and W. P. Hood, Jr., M.D.

SUMMARY

Hemodynamic data from 45 patients with either aortic regurgitation (with and without the Austin Flint murmur) or aortic regurgitation and mitral stenosis were correlated with the a-wave percentage amplitude of the apexcardiogram. Changes in the a wave correlated well with corresponding changes in left heart pressures but not with variations in volume. Of 15 patients in whom the ratio of the a wave to the total excursion during systole (a/H ratio) was ≥13%, 11 had left ventricular end-diastolic pressures (LVEDP) >20 mm Hg. All patients with a/H ratios >15% had abnormal LVEDP. The converse was not true; 13 patients had elevated LVEDP with normal a/H ratios. Patients with aortic regurgitation and mitral stenosis had a/H ratios similar to those of normal subjects.

The Flint murmur showed a significant correlation with left ventricular volume changes. Its presence was valuable in prediction of a large regurgitant volume and a high left ventricular stroke volume. It was also present in association with elevated left atrial mean pressure and elevated LVEDP.

Thus, two ancillary findings in aortic regurgitation, a high a wave in the apexcardiogram and the Austin Flint murmur, are of value in anticipating alterations in left ventricular hemodynamics as determined by left-sided catheterization and volume studies.

Additional Indexing Words:
Phonocardiogram Left ventricular end-diastolic pressure Left ventricular volume

ASSESSMENT of the severity of aortic regurgitation by external means has usually depended on the intensity of the typical blowing diastolic murmur plus left ventricular enlargement and characteristic peripheral signs. The electrocardiogram and cardiac silhouette by X-ray have provided valuable supplementary information. Two other accompanying features of aortic regurgitation—the Austin Flint murmur and the a wave in the apexcardiogram—may also provide information in assessment of the degree of physiologic derangement.

The Austin Flint murmur may be detected in association with aortic regurgitation of moderate to severe degree. It has been variously described as an apical mid-diastolic rumble and as a presystolic murmur closely resembling that of mitral stenosis. The murmur can be documented by phonocardiography and has recently been evaluated by O'Brien and Cohen1 with regard to associated hemodynamic events in the left atrium and left ventricle in patients with severe aortic regurgitation.

The apexcardiogram may also be used in the assessment of left ventricular disease,
although application of this method to problems of aortic regurgitation has been infrequent. In studies of the apex tracing in aortic valvular disease and cardiomyopathy, Epstein...
et al. noted the frequent occurrence of a large $a$ wave in aortic valvular disease and observed that a large atrial systolic wave could be of value in differentiating the Austin Flint murmur from that of mitral stenosis.

The purpose of this paper is to examine the relationship of these two ancillary manifestations in aortic regurgitation—the Austin Flint murmur and the $a$ wave in the apexcardiogram—to hemodynamic alterations in the left side of the heart. Pressure and flow measurements combined with ventricular volume studies afford an unusual opportunity for correlative studies. The object of such investigation is to provide a more accurate assessment of the pathophysiologic mechanisms associated with certain aspects of the physical examination and their graphic counterparts recorded in the apex- and phonocardiogram.

**Methods**

Forty-five patients were chosen for study. These were divided into two groups.

**Group A. Predominant aortic regurgitation**

This group comprised 32 patients in whom the diagnosis was established by left-sided cardiac catheterization and cineangiography. In nine of the 32, supplementary information was provided at surgery. In 25 patients there was pure aortic regurgitation and in seven, there was an associated mild mitral regurgitation. The Austin Flint murmur was present in 20 of the patients in group A and was absent in 12. There were 27 males and five females, ranging in age from 17 to 65 years. The valvular disorders were ascribed to rheumatic heart disease in 12 patients, syphilis in eight, cystic medial necrosis in two, traumatic aneurysm in two, and aneurysm or aortic root dilatation of unknown origin in eight. All the patients in this group were in normal sinus rhythm. The average blood pressure was 148/51.

**Group B. Mixed aortic regurgitation and mitral stenosis**

This group was made up of 13 patients in whom both aortic regurgitation and mitral stenosis were present, with the latter being the predominant lesion. All were studied by left heart catheterization, and the diagnosis was confirmed by surgery in nine cases. In two of these there was also a postmortem examination. This group included two males and 11 females, ranging in age from 18 to 49 years. The etiology of the valvular lesions in all the patients in this group was rheumatic. All were in normal sinus rhythm, although in one case there was a prolonged P-R interval. The average blood pressure in this group was 118/62. Estimated mitral valve area at catheterization ranged from 0.7 to 2.5 cm$^2$, with an average of 1.25 cm$^2$.

Phonocardiograms were recorded on a Cambridge MC IV Multichannel Data System Recorder with Cambridge microphones. Phonocardiograms were taken in two locations simultaneously in conjunction with an electrocardiogram and a pulsatile record, either an indirect carotid tracing or an apical tracing. Variable filters provided a cut 8 db per octave above and below center frequency, which was set at 150–300 Hz, depending on the type of murmur being recorded. The areas to be studied phonocardiographically were determined first by auscultation, since the early diastolic murmur might be maximal at either the left or right sternal edge and the apical rumble was often audible over only a very limited area. The Austin Flint murmur is defined as an apical diastolic rumble—either mid-diastolic, presystolic, or both in timing—occurring in a setting of predominant aortic regurgitation where the presence of mitral stenosis can be eliminated. The Flint murmur was determined to be present when it could be heard on auscultation and demonstrated in the phonocardiogram (fig. 1).

**Figure 1**

Severe aortic regurgitation of unknown etiology. Preoperative phono- and apexcardiograms (above). An intense, early diastolic murmur (EDM) is shown in the phonocardiogram at left sternal edge (PCG-LSE), while at the mitral area (MA) a separate later murmur (MDM) of somewhat lower frequency is seen. This is an Austin Flint murmur. The apexcardiogram (APEX) shows a very large a wave (a), which results in an a/H ratio of 39.9% (see fig. 2). Postoperative tracing (below) was made after successful repair of the aortic regurgitation by a plastic procedure which did not require a prosthetic valve. The a wave and diastolic murmurs have been eliminated.

Additional abbreviations: $Q =$ a point on the phonocardiogram corresponding to the beginning of the QRS complex in the ECG; $2 =$ second heart sound.

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Apex tracings were made with the patient in the left lateral decubitus, with a Hellige Pulse Microphone Transducer.\textsuperscript{*} The transducer was held by hand against the chest wall at the point of maximal impulse. Patients were permitted to breathe quietly during recording. An oscilloscope on the multichannel recorder was used to verify proper positioning of the transducer. Paper speed was set at 100 mm/sec. The manner in which amplitude of the waves of the apexcardiogram was measured is illustrated in figure 2. The method used is that of Benchimol and Dimond,\textsuperscript{4} and it was also employed by Epstein et al.\textsuperscript{2} The height of the a wave was calculated as a fraction of the total outward movement in systole (H), and expressed as a percentage or \( a/H \) ratio.

\textsuperscript{*}Fritz Hellige & Co., Freiburg, West Germany. This apparatus has a flat response at frequencies from 50 Hz down to 0.3 Hz, and a time constant of 1.2 sec.
all determinations, the average of at least three complexes was used. Similar measurements were made on 30 normal controls (table 1), who were medical students, house staff members, and technicians, ranging in age from 21 to 49 years.

Catheterization data were available for all 45 patients but for none of the normal subjects. The catheterization studies were performed close in time to but not simultaneously with the noninvasive observations.

Left ventricular volume calculations were made for 25 of the 45 catheterized patients by the following methods:

(1) Biplane angiocardiography was performed at 6 films/sec for 5 sec. The site of injection of contrast material varied with the physiologic state. For injection into the left ventricle, aortic root, or left atrium, 50 to 60 ml of 75% sodium meglumine diatrizoate was used, whereas in the pulmonary artery 60 to 80 ml of a 90% solution was employed. Lead III of the electrocardiogram and a photocell output for timing film exposures were recorded during the biplane study. Left ventricular pressure, measured through a retrograde no. 8F catheter connected to a Statham P23Db transducer, was recorded during or immediately before the period of filming. In those patients whose ventricular pressure was not measured simultaneously with filming, brachial arterial pressure was monitored before, during, and after the biplane study to confirm constant systolic pressure. All recordings were made on a Sanborn 568-1100M photographic recorder at a paper speed of 100 mm/sec. The zero point of end-diastolic pressure was considered to be midway between anterior and posterior chest walls, and was taken as post-a-wave pressure.5

(2) Left ventricular volume was calculated from each film pair by the area-length method of Dodge et al.6 Each value so obtained was corrected for the volume of trabeculae carneae and papillary muscles, methods previously described by Rackley et al.7 Corrected values from several cardiac cycles were rearranged according to time after onset of the QRS complex into a single composite volume curve. We averaged several maximal and minimal values to arrive at end-diastolic (EDV) and end-systolic volumes (ESV), respectively, and left ventricular stroke volume (LVSV) was calculated as the difference between these two values. Effective stroke volume (Eff SV) was measured independently by the Fick oxygen method in duplicate. Regurgitant volume (RV) was determined as the difference between LVSV and Eff SV.

(3) In 36 patients left ventricular and brachial arterial pressures were recorded simultaneously, while in one patient simultaneous left ventricular and aortic pressures were obtained. Pullback tracings across the aortic valve were recorded in 32 patients. In no patient was a systolic pressure gradient across the aortic valve demonstrated. In eight patients direct left atrial pressure was obtained, and in 36 patients pulmonary capillary (PC) wedge pressures were recorded. Left ventricular (LV) pressure

### Table 1

Mean of Each Parameter from Hemodynamic and Apexcardiographic Data for Groups A, B, and C*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Group A Without Flint murmur</th>
<th>Group A With Flint murmur</th>
<th>Group B</th>
<th>Group C</th>
<th>Pooled S</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats/min)</td>
<td>71.3</td>
<td>79.4</td>
<td>79.8</td>
<td>65.2</td>
<td>13.4</td>
</tr>
<tr>
<td>a/H ratio (%)</td>
<td>10.9</td>
<td>18.0</td>
<td>10.9</td>
<td>10.1</td>
<td>6.3</td>
</tr>
<tr>
<td>Mean PCW or LA pressure (mm Hg)</td>
<td>9.7</td>
<td>16.3</td>
<td>20.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LA peak a-wave pressure (mm Hg)</td>
<td>15.3</td>
<td>22.7</td>
<td>26.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>17.9</td>
<td>24.3</td>
<td>10.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDV (ml)</td>
<td>260.4</td>
<td>343.3</td>
<td>151.2</td>
<td>105.4</td>
<td></td>
</tr>
<tr>
<td>ESV (ml)</td>
<td>168.4</td>
<td>185.8</td>
<td>72.6</td>
<td>94.9</td>
<td></td>
</tr>
<tr>
<td>LVSV (ml)</td>
<td>99.8</td>
<td>157.5</td>
<td>88.6</td>
<td>26.7</td>
<td></td>
</tr>
<tr>
<td>Eff SV (ml)</td>
<td>61.5</td>
<td>65.3</td>
<td>68.2</td>
<td>18.7</td>
<td></td>
</tr>
<tr>
<td>RV (ml)</td>
<td>36.3</td>
<td>92.2</td>
<td>22.6</td>
<td>24.4</td>
<td></td>
</tr>
</tbody>
</table>

*Group A = patients with aortic regurgitation; Group B = patients with mixed aortic regurgitation and mitral stenosis; Group C = normal subjects.

Abbreviations: HR = heart rate; a/H ratio = height of the a wave, calculated as a fraction of the total outward movement in systole; PCW = pulmonary capillary wedge; LA = left atrial; LVEDP = left ventricular end-diastolic pressure; EDV = left ventricular end-diastolic volume; ESV = left ventricular end-systolic volume; LVSV = left ventricular stroke volume; Eff SV = effective stroke volume; RV = regurgitant volume; S = pooled within-group standard error.
Mean LVEDP for those patients in group A without the Austin Flint murmur was 17.9 mm Hg, while for those with the murmur, it was 24.3 mm Hg. Mean LVEDP for group A as a whole was 21.6 mm Hg. There was a tendency for the elevated LVEDP in group A to be associated with a higher than normal a/H ratio in the apexcardiogram. The correlation between the a/H ratio and LVEDP was significant for all patients with aortic regurgitation, both with those with the Austin Flint murmur (correlation coefficient, 0.63) and those without the murmur (correlation coefficient, 0.59). Of 15 patients with a/H ratios of 13% or more, all had LVEDP above 10 mm Hg. Of these 15, 11 showed LVEDP exceeding 20 mm Hg (range 23–40 mm Hg). The converse, however, was not true: 13 patients with elevated LVEDP had normal to low a/H ratios. It is noteworthy that of this group only two patients had pressures as high as 36–40 mm Hg, while the remainder demonstrated relatively low LVEDP, in the range of 14–18 mm Hg.

In group B (patients with aortic regurgitation and mitral stenosis) a significantly lower LVEDP (mean, 10.8 mm Hg) was recorded than in group A. The a/H ratio was likewise lower, 10.9 (±5.3), a value almost identical to that observed in the normal group.

LA Peak a-Wave Pressure (Fig. 4, Table 1)

The peak a-wave pressure in LA or PC wedge pressure pulses was 22.7 mm Hg in those patients in group A with the Austin Flint murmur, but only 15.3 mm Hg in those without the murmur.

In group A, a high a/H ratio in the apexcardiogram reflected an elevated LA or PC peak a-wave pressure (fig. 4). As was previously noted in the correlation with LVEDP, however, a high pressure in the LA or PC a wave was often associated with a normal a/H ratio. Thus, a high a/H ratio in the apexcardiogram was of predictive value with respect to left heart pressure, but the converse was not always true, there being numerous examples of elevated pressures with normal a/H ratios in the apexcardiogram.
AUSTIN FLINT MURMUR AND THE A WAVE IN AR

Comparison of a/H ratio of the apexcardiogram with left atrial (or PCW) peak a-wave pressure. A high a/H ratio is associated with an elevated left atrial or PC wedge peak a-wave pressure. The a-wave pressure in LA or PCW is higher in those patients with the Flint murmur (triangles) than in those without it (circles). In group B (mitral stenosis plus aortic regurgitation) a high left atrial pressure is not reflected in a high a/H ratio.

In group B, the presence of mitral stenosis in addition to aortic regurgitation was associated with an average peak a-wave pressure in LA or PC wedge of 26.8 mm Hg. As might be expected, this was not reflected in an elevation of a/H ratio in the apexcardiogram. As noted previously, the mean a/H ratio in this group was the same as that of the normal subjects.

Relation of Apexcardiogram and Phonocardiogram to Left Heart Volumes

Table 1 presents a summary of the results of left ventricular volume studies from the 25 patients for whom such data were obtained.

Left Ventricular Volume Measurements and the Apexcardiogram

In group A (patients with predominant aortic regurgitation) there was a poor correlation between the EDV and the a/H ratio (r = 0.38 for patients without the Flint murmur and 0.36 for patients with the murmur). Seven of 11 patients with EDV greater than 300 ml had a/H ratios within the normal range. Similarly, in group B (mixed aortic regurgitation and mitral stenosis) a lack of correlation was found between the volume parameters and the a/H ratio. All patients in group B were found to have EDV greater than 100 ml. Only two of this group, however, had a/H ratios above normal.

Although there was a slightly better correlation between the a/H ratio and RV for both patient groups than had been apparent for the EDV, the relationship of the apexcardiographic ratio to RV was inconsistent. A lack of relationship was also demonstrated between the LVSV and the a/H ratio as determined from the apexcardiogram.

A significant relationship was found in the patients of group A between the presence of the Austin Flint murmur and the RV. All nine patients with the Austin Flint murmur for whom volume determinations had been made had an RV exceeding 50 ml, and the mean RV for this group was 92.2 ml, as compared to a mean of 36.3 ml for patients with aortic regurgitation but no Austin Flint murmur.

The only other measurement derived from ventricular volume studies with a significant correlation to the presence of the Flint murmur was the LVSV. In the presence of the murmur, the mean LVSV for patients with predominant aortic regurgitation was 157.5 ml, as compared with 99.8 ml for those without the murmur.

Discussion

Estimation of the severity of aortic regurgitation is increasingly important at present owing to the possibility of surgical intervention with insertion of prosthetic valves. It is possible to establish the degree of physiologic derangement very accurately by means of left heart catheterization and ventricular volume measurements. However, these procedures cannot conveniently be done repeatedly. To the extent that external noninvasive techniques can anticipate findings at catheterization, they can be used to follow patients serially and detect deterioration so as to
permit optimal timing of catheterization and surgery.

Of the two manifestations of the physical examination investigated in this study—the Austin Flint murmur and the presystolic $a$ wave forming part of the apical impulse—the murmur is the better known.

As originally described by Flint in 1862, the murmur was presystolic in timing and indistinguishable from that of mitral stenosis.\(^8\,^9\)

Subsequent observations have reflected a variety of opinions, not only as to its genesis but as to its timing as well. Flint's own explanation related the murmur to vibrations set up in the mitral leaflet by atrial contractions, presupposing partial closure of the mitral valve secondary to ventricular dilatation. His theory is remarkably similar to more recent concepts of "relative" or functional mitral stenosis. Herrmann in 1926 observed that lesions of the posterior aortic cusps most consistently produced the Austin Flint murmur, and explained the rumble on the basis of functional mitral obstruction resulting from partial closure of the anterior mitral leaflet.\(^10\)

The most generally accepted explanation of the Austin Flint phenomenon is that it appears to involve vibration of the anterior mitral valve leaflet in response to filling from both inflow and outflow tracts of the left ventricle, much as the reed of an oboe vibrates in the double air stream to which it is exposed.

Although the timing of the murmur is usually given as mid-diastolic, a presystolic accentuation may also be found. This latter situation implies augmentation of the slow-filling wave by atrial contraction, and reemphasizes the marked similarity of the Flint rumble and the murmur of mitral stenosis. The latter is likewise frequently described with two components, both a mid-diastolic accentuation and the presystolic crescendo attributed to atrial contraction. It is interesting to note that Flint, himself, placed considerable emphasis on atrial contraction in his original explanation of the murmur.\(^8\)

O'Brien and Cohen\(^1\) measured simultaneous LV and LA or PC wedge pressures in patients with an apical mid-diastolic rumble in the presence of aortic regurgitation. An early diastolic gradient between the LA (or PC wedge) and LV was consistently found, with a reversed gradient in end-diastole from LV to the LA. All of their patients with the Austin Flint rumble showed elevated LVEDP and elevated LA or PC wedge pressures, while patients without the Flint murmur demonstrated neither the reversed end-diastolic gradient nor significantly elevated LVEDP. These findings were thought to be consistent with the generally accepted explanation that the Flint rumble is due to the aortic

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**Figure 5**

Relationship of left-sided pressures at catheterization to phonocardiographic events. (Nonsimultaneous records taken at identical heart rates.) The left ventricular tracing (LV) is largely off the paper in order to bring out the gradients existing with left atrial (LA) pressures in diastole. The LV pressure falls rapidly in early diastole to levels well below the LA pressure, leading to an LA-LV gradient of 18 mm Hg 0.06 sec after onset of diastole. In end-diastole, however, an LV-LA gradient of 6.4 mm Hg is seen at a time when the Austin Flint murmur (AF murmur) is showing a presystolic crescendo.
regurgitant stream impinging upon the anterior leaflet of the mitral valve, hindering its complete opening. Although the presystolic component was not conspicuous in their cases with the Flint murmur, O'Brien and Cohen felt that presystolic mitral regurgitation might be present as indicated by the pressure relationship.

In only one patient in our group with aortic regurgitation were simultaneous LA and LV pressures recorded (fig. 5). According to the method of O'Brien and Cohen, the LA-LV gradient measured at 0.06 sec after onset of diastole was approximately 18 mm Hg, while the LV-LA gradient at end-diastole was 6.4 mm Hg. In contrast to the observation of O'Brien and Cohen that the Austin Flint murmur in patients with reversed gradients was limited to mid-diastole, we recorded an Austin Flint murmur with marked presystolic accentuation in this particular patient.

Similar findings had been reported by other investigators. Lochaya et al. described three cases of severe aortic regurgitation in which a reversal of the pressure gradient across the mitral valve was associated with presystolic mitral regurgitation and a late diastolic murmur. Rees et al. and Kelly et al. observed instances of elevated mean LA pressure and substantially increased LVEDP in cases of severe aortic regurgitation such that not only an end-diastolic reversal of the gradient but also equalization of LV and aortic pressures occurred. Premature mitral valve closure in mid-diastole was presumed to have resulted from aortic regurgitation into a left ventricle with severely diminished compliance. Premature mitral closure was thought secondarily to enhance myocardial contraction, serve as a barrier to continued aortic reflux, and protect the pulmonary circuit from the elevations in LVEDP.

All 20 patients with the Austin Flint murmur displayed LVEDP exceeding our normal reading of 10 mm Hg (fig. 2). Four of these, however, showed only moderate elevations (11–14 mm Hg). LA or PC wedge mean pressure similarly exceeded 10 mm Hg in 15 of the 20 patients with the Flint murmur.

This inconsistent relationship between the presence of the Flint murmur and left-sided pressures led to a consideration that elevated end-diastolic volume might play a significant role in the genesis of the Flint murmur, as indicated in the studies of Dodge et al. In support of this thesis we found all but one patient with the Flint murmur, for whom volume determinations had been made, to have LV end-diastolic volume exceeding 200 ml. Regurgitant volume in all patients with the murmur was greater than 50 ml, and the marked difference observed between mean RV for patients with and without the Austin Flint murmur would imply that the murmur, when present, indicates very high levels of RV. The increase in LVEDP uniformly observed for the subgroup with the murmur would presumably result from a combination of increased volume in late diastole and decreased left ventricular distensibility in association with aortic regurgitation of significant degree. Atrial systole results in an exaggerated pressure rise in the ventricle by operating over a stiffer portion of the volume-pressure curve.

The apexcardiogram a wave (fig. 1) may be seen as the late diastolic response of the left ventricle to atrial systole. It occurs simultaneously with the left atrial a wave and with a fourth heart sound and disappears with atrial fibrillation. It is altered in timing in partial A-V block, moving earlier with the P wave, and is inconspicuous or absent in mitral stenosis. Measurement of amplitude of the a wave in terms of some standard unit would be highly desirable and might be accomplished by reference to a reproducible impulse. Such methods have been found to be of value in separation of the relatively gross differences in amplitude of the apical movement in systole of "hyperdynamic" impulses from qualitatively similar normal subjects. However, the comparatively small height of a waves and simplicity have led to general acceptance of the method of expressing a-wave height as a ratio of the total excursion of apical movement (a/H ratio).

Epstein et al. have reviewed the history of
graphic recordings of precordial movement and present excellent examples of apex tracings illustrative of certain cardiovascular disorders. In cases of aortic regurgitation with a diastolic rumble of uncertain etiology, a large a wave in the apexcardiogram may be interpreted as evidence for an isolated aortic lesion.

The large a wave inscribed in aortic regurgitation is not, however, unique for that lesion. It may be seen as well in other left ventricular disorders, including coronary disease, hypertension, aortic stenosis, and coarctation. In their study of 59 patients with ischemic heart disease, Benchimol and Dímond observed an abnormal apex a wave in nearly every subject with ischemic heart disease, attributing it to an abnormal ventricular filling response secondary to increased ventricular resistance to atrial systole. They found a close association between increased LVEDP, LA pressure, and LA residual volume and tall apex a waves, with a significant increase in a-wave amplitudes of arteriosclerotic subjects after exercise. With reference to aortic regurgitation, this association correlates well with the observation of West et al., who noted high levels of myocardial oxygen extraction in severe aortic insufficiency and felt that relative ischemia occurred even in the presence of increased coronary flow.

Epstein et al. similarly demonstrated a significant association of large apex a waves, as expressed by the a/H ratio, and rising LVEDP and LA pressures, and attributed these findings both to increased LA activity and to the effect of atrial systole on the non-compliant left ventricle of aortic valve disease, both stenosis and regurgitation, and of cardiomyopathy.

In our group of 32 patients with aortic regurgitation, we found a significantly positive correlation between rising LA and LV pressures and increasing a/H ratios (figs. 2 and 3). The relationship was best with LVEDP. The correlation between an abnormally large a/H ratio in the apexcardiogram and the presence of the Flint murmur, however, was not good.

It is interesting to note that, while LVEDP correlated well with the a/H ratio for all patients with aortic regurgitation, LA a-wave peak pressures showed much better correlation with the a/H ratio of those patients who also had the Flint murmur (e.g., r for a/H and LA a-wave peak pressure = 0.18 for patients without the murmur and 0.60 for those with the murmur). This may imply that, whatever actual physiodynamics obtain in the generation of the Flint murmur, a significant barrier to forward flow from LA to LV does indeed exist. From our data it is not possible for us to delineate with any accuracy the mechanism involved, but the concept of "functional mitral stenosis" would appear to be a valid one. Mean PC wedge or LA pressures in the group of patients with isolated aortic regurgitation and the Flint murmur were strikingly similar to those seen in our group with mixed aortic regurgitation and mitral stenosis.

The influence of a large regurgitant volume in holding the anterior leaflet of the mitral valve in a half-closed position is supported by preliminary echocardiographic studies in the authors' laboratory (Fortuin NJ, Craige E: Unpublished data). The movements of the valve can be displayed as one aspect of a multi-channel recording along with phonocardiograms, electrocardiograms, and carotid pulse. These observations support the classical concept of Herrmann with regard to the genesis of the murmur. We believe that, in most instances, both the mid-diastolic and presystolic elements of the murmur are caused by antegrade flow over a partially opened valve. The observations of Ueda et al. that the presystolic component of the Flint murmur dropped out when cardiac rhythm changed from normal sinus to atrial fibrillation, supports the importance of atrial systole (antegrade flow) rather than diastolic mitral regurgitation, which presumably would occur without regard for atrial rhythm.

More than half of the patients with aortic regurgitation, for whom volume studies were available and who demonstrated left ventricu-
lar EDV in excess of 300 ml, had a/H ratios within the normal range. This is not surprising in view of the fact that LVEDP and EDV are not necessarily closely related, as previously shown by Rackley et al.\textsuperscript{18} from our laboratory. Thus, the a/H ratio in the apexcardiogram, related more closely to pressure abnormalities, and the Flint murmur, related to volume abnormalities, are not necessarily closely correlated with each other.

The absence of a significant a wave in the apexcardiogram of certain patients with severe aortic regurgitation has been attributed by Di-Matteo et al.\textsuperscript{19} to the presence of such a high LVEDP that the force of atrial contraction is inadequate to register a pressure wave in the ventricle. These authors cite the earlier work of Colvez et al.\textsuperscript{20} who, in 1959, demonstrated LVEDP of 38–40 mm Hg in severe aortic regurgitation, with reversal of LV-LA pressure gradients, leading to precocious closure of the mitral valve before atrial systole could take place.

These studies demonstrate that two ancillary findings in aortic regurgitation, a high a wave in the apexcardiogram and the Austin Flint murmur, are of value in anticipating alterations in left ventricular hemodynamics as determined by left-sided catheterization and volume studies.

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References


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ELIZABETH PARKER, ERNEST CRAIGE and W. P. HOOD, JR.

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