The Austin Flint Murmur: Its Differentiation from the Murmur of Rheumatic Mitral Stenosis

By Jack P. Segal, M.D., W. Proctor Harvey, M.D., and Michael A. Corrado, M.D.

AFTER clinical evaluation of patients with severe aortic insufficiency, it has become evident that differentiation of the Austin Flint murmur from that of mitral stenosis is possible in the majority of cases.1-5 If carefully searched for, all of our patients with severe aortic insufficiency have an apical diastolic rumble (so-called Austin Flint murmur). By utilization of the clinical features derived from a careful history, physical examination, x-rays, and electrocardiography, it is possible in the majority of cases to determine whether the apical diastolic rumble represents an Austin Flint murmur or the murmur of organic mitral stenosis. For all practical purposes this problem in differential diagnosis only exists in those patients in whom the rheumatic etiology is the basis for their aortic insufficiency. Since surgical correction of both mitral stenosis and aortic insufficiency can now be attempted, it becomes extremely important to determine which patients with aortic insufficiency have associated mitral stenosis.

These observations are based on a careful clinical evaluation of over 400 patients with severe aortic insufficiency. The average age was about 35. Males outnumbered females in a ratio of 3 to 1. The etiology of aortic insufficiency in our series was rheumatic in over 80 per cent. The remaining cases were divided between the syphilitic, congenital and traumatic etiologies. Occasionally, aortic insufficiency was associated with a dissecting aortic aneurysm, or with the Marfan syndrome.

In every case a careful clinical evaluation consisting of a history and physical examination, electrocardiogram and chest film was performed. Additional studies such as phonocardiograms and right and left heart catheterization were sometimes performed.

To establish the diagnosis of aortic insufficiency, the following 2 criteria were essential: (1) a decrescendo, blowing aortic diastolic murmur (usually at least grade IV), and (2) a diastolic blood pressure of 40 mm. Hg or below. In almost all cases the diastolic sounds were heard down to zero. In all patients, peripheral signs of aortic insufficiency, such as the waterhammer pulse, pistol shot sound, and Duroziez's sign, were present. In all but one of the rheumatic cases the presence or absence of mitral stenosis was confirmed either at autopsy or by surgical exploration, and in some cases by both. In those cases diagnosed as syphilitic aortic insufficiency, there was no history of rheumatic fever, often the history of a primary syphilitic lesion, often a positive serology, and frequently calcification of the ascending aorta.

In table 1 are listed those clinical features important in differentiating those patients with aortic insufficiency alone from those with both aortic insufficiency and mitral stenosis combined.

The following special comments are in order:

The opening snap of the mitral valve is heard only in those patients with associated mitral stenosis. Of interest, is the fact that in patients with combined aortic insufficiency and mitral stenosis, the opening snap appears to be delayed; instead of occurring at the usual interval of .06 to .08 second after the second sound, it occurs from 0.11 to 0.12 second later.
In patients with severe aortic insufficiency 3 murmurs are almost always heard at the apex: a systolic murmur of mitral insufficiency, a very early diastolic blowing murmur transmitted from the aortic area, and the Austin Flint murmur which is usually accentuated in mid-diastole. In addition, a ventricular diastolic gallop (early diastolic gallop) is almost always heard. Very frequently a loud early systolic ejection sound of aortic origin is well heard at the apex. In fact, this sound is often mistaken for an accentuated first sound.

With aortic insufficiency alone, the apical diastolic murmur (Austin Flint murmur) is best heard in early and mid-diastole, and usually starts coincident with or immediately after the ventricular gallop. This murmur is usually of grade III intensity, but in some cases has been as loud as grade VI, and sometimes is associated with an apical diastolic thrill. Usually presystolic accentuation is not present, but with more rapid heart rates, particularly with a short P-R interval presystolic accentuation may occur. With associated mitral stenosis the diastolic rumble is "longer," extending throughout diastole and usually associated with presystolic accentuation, if sinus rhythm is present.

To summarize: the patient with an Austin Flint murmur (pure aortic insufficiency as the only lesion) is most likely male, with moderate or slight progressive dyspnea and/or angina pectoris, and normal sinus rhythm. The second sound over the pulmonic area is normal or slightly accentuated, and the first sound at the apex is often faint. A systolic ejection sound is a common finding over the base of the heart and, frequently, also over the entire precordium. At the apex a systolic murmur, ventricular diastolic gallop, early diastolic blowing murmur transmitted from the aortic area, and an apical diastolic rumble usually accentuated in mid-diastole, are heard. The electrocardiogram shows normal sinus rhythm, left ventricular hypertrophy, and frequently a prolonged P-R interval. X-ray and fluoroscopy reveal moderate enlargement of the left ventricle, no enlargement of the pulmonary artery segment, and no or very slight posterior displacement of the esophagus.

In contrast, the patient with associated organic mitral stenosis would more likely be a female with a history of moderate to marked exertional dyspnea, frequently paroxysmal, and often associated with cough and hemoptysis. The rhythm may be normal sinus, but atrial fibrillation or flutter is not an unusual occurrence. On auscultation the second sound over the pulmonic area and the apical first sound are accentuated, and an opening snap of the mitral valve is present. The apical diastolic rumble is frequently accentuated in presystole. X-ray and fluoroscopic examination reveal straightening of the left heart.

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<th>TABLE 1.—Comparison of Clinical Features in Differentiating the Austin Flint Murmur and Rheumatic Mitral Stenosis</th>
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<td>Sex</td>
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<td>More frequent in males</td>
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<td>Hemoptysis</td>
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arotic Insufficiency Alone. The following two cases represent classical instances of free 
ventricular diastolic gur.

Patient J.M. (Fig. 1). A 55-year-old white male 
with syphilitic aortic insufficiency. At approxi-
mately age 30 he had syphilis. He was asympto-
tic until 15 months prior to admission when he 
reported onset of dyspnea, orthopnea, paroxysmal 
episodes of angina pectoris, and ankle edema. On 
physical examination the skin was thin and appeared 
chronically ill. Pulse was 92 regular. Blood pressure 
was 130/80. No neck vein distention. Lungs were clear 
and heart sounds were normal. Systolic and diastolic 
thrills were present over the aortic area, where a systolic 
and diastolic murmur were palpable. The second sound 
and diastolic murmur were accentuated over the aortic area, but the 
third sound was minimally increased in intensity. The 
patient had a grade III short systolic and a grade IV diastolic 
Fmurmur at the apex of the heart. The heart sounds were 2/2 without 
coarse rales heard. The electrocardiogram showed 
right ventricular hypertrophy. Chest x-ray showed left ventricular enlargement and esophagus slightly 
shifted posteriorly. Echocardiogram showed left ventricular hypertrophy.

Fig. 1. Male, 58, with syphilitic aortic insufficiency (autopsy). Photomicrograph (upper 
left), aortic area: note ejection sound (E), diamond-shaped early systolic murmur (SM), 
and decreasing diastolic murmur (DM); at apex, note faint first sound (S1), systolic sound (S2), 
gallop (G) and Austin Flint murmur (AF). Chest x-ray shows classic left ventricular enlargement and esophagus slightly displaced posteriorly.
trocardiogram revealed left ventricular hypertrophy. Fluoroscopic examination revealed transverse enlargement of the heart, both to the right and left, with definite left ventricular enlargement. The aortic pulsations were greatly increased. There was moderate generalized posterior displacement of the esophagus on barium swallow, and moderate systolic expansion of the left atrium was seen. The clinical impression was syphilitic heart disease with aortic insufficiency, Austin Flint murmur, relative mitral insufficiency. A plastic aortic valve was inserted and the patient expired shortly after leaving the operating room. Post mortem examination confirmed the clinical diagnosis of syphilitic aortic insufficiency. The aortic valve admitted 2 fingers. A syphilitic aneurysm of the ascending aorta was also present. The mitral valve was normal.

Summary. A patient with classical syphilitic heart disease and free aortic insufficiency. At the aortic area a loud diamond-shaped aortic systolic murmur associated with a palpable thrill was present, but at autopsy no evidence of aortic stenosis was found. In contrast to the murmur of aortic stenosis, this aortic systolic murmur was short and reached its peak relatively early in systole. In all of our patients having the severe form of aortic insufficiency an associated aortic systolic murmur has been present. This murmur differs from that of aortic stenosis by being shorter in duration and occurring earlier in systole. With significant aortic stenosis the systolic murmur extends almost to aortic valve closure, and the peak of this diamond-shaped murmur occurs later in systole. On auscultation at the apex, the first heart sound was described by one observer as being accentuated. However, as shown on the phonocardiogram, an early systolic ejection sound was being mistaken for the first heart sound, which was actually faint. The phonocardiogram also confirmed the apical systolic murmur, ventricular diastolic gallop, and mid-diastolic murmur (Austin Flint). The classical x-ray and electrocardiographic findings already described were present.

![Male 32, Rheumatic A.I. (Autopsy)](image)

**Fig. 2.** A 32-year-old male with rheumatic aortic insufficiency (autopsy). Phonocardiogram, aortic area; ejection sound (E), systolic murmur (SM), and diastolic murmur (DM). Apex; loud ejection sound (E) and ventricular diastolic gallop (G). Note the systolic murmur (SM) and Austin Flint murmur (DM). Electrocardiogram shows left ventricular hypertrophy and strain.
Patient J.M. (fig. 2). A 32-year-old man with rheumatic heart disease. Increasing fatigue, dyspnea on exertion, and paroxysmal nocturnal dyspnea had been present for a year and a half, and orthopnea for 4 months. He had been treated for subacute bacterial endocarditis at the age of 23 and again at the age of 30. Physical examination: Blood pressure 160/40-0. Lungs were clear to percussion and auscultation. The heart was enlarged to the anterior axillary line. Rate 92 and regular with frequent premature ventricular contractions. At the aortic area were heard a grade III short aortic systolic murmur and a grade IV diastolic blowing murmur, loudest at the left sternal border. At the apex a grade II apical systolic, a grade III Austin Flint rumble, and a ventricular diastolic gallop were present. The liver was palpated one fingerbreadth below the right costal margin, and the spleen was not felt. One-plus pretibial edema was present. The chest x-ray revealed the heart markedly enlarged to the left with some enlargement of the left atrium, as noted on barium swallow. Electrocardiogram revealed frequent premature ventricular contractions with periods of bigeminy. The P-R interval was 0.22 second. The interpretation was left ventricular hypertrophy and strain and intraventricular conduction defect. A plastic aortic valve was inserted and approximately 3 weeks later he was discharged home. He was extremely anxious and complained of recurrent episodes of dyspnea, vertigo, and numbness of the extremities and lips. Several months later he suddenly expired. At autopsy the heart weighed 1,150 Gm. with generalized cardiac enlargement and hypertrophy, particularly of the left ventricle. Severe aortic insufficiency of the rheumatic type was pathologically confirmed. The mitral valve showed no evidence of stenosis. The chordae tendineae were long and thin and inserted in the valve in the usual way. The papillary muscle, although moderately hypertrophied, appeared not remarkable.

Summary. As demonstrated in figure 2, this patient had the classical phonocardiographic and electrocardiographic findings of severe aortic insufficiency, in this instance of the rheumatic etiology. At the apex the typical findings associated with free aortic insufficiency were present: an ejection sound followed by a systolic murmur, a blowing diastolic murmur which occurred early in diastole between the second sound and ventricular diastolic gallop, and the diastolic rumble. The phonocardiograms demonstrate the 3 mur-
murs characteristically heard at the apex in patients with free aortic insufficiency: a systolic murmur, an early diastolic murmur (the aortic insufficiency diastolic blowing murmur which was transmitted to the apex), and the Austin Flint rumble. The loud ejection sound heard at the apex is often mistaken for a loud first sound, and the diagnosis of mitral stenosis may be incorrectly entertained.

Aortic Insufficiency with Associated Mitral Stenosis. In figure 3 are represented the phonocardiogram, electrocardiogram, and x-rays of a patient with a mild aortic insufficiency and associated mitral stenosis. The patient was a 52-year-old female with a known history of rheumatic fever. For 4 years she had been aware of dyspnea on exertion which had gradually progressed in the past 6 months. She had recently observed paroxysmal nocturnal dyspnea on a few occasions. Physical examination revealed a thin female in no acute distress. Pulse 75, grossly irregular. Blood pressure 180/80-70. There was moderate neck vein distention. The heart was enlarged to the left to the anterior axillary line. At the aortic area were heard a short, soft systolic murmur and a grade IV diastolic blowing murmur transmitted to the left sternal border. At the apex the first sound was accentuated and a prominent opening snap was heard. A grade IV diastolic rumble associated with a thrill was present. The second sound at the pulmonic area was accentuated. The liver was 3 fingerbreadths below the right costal margin and the tip of the spleen was palpable. There was no edema. The electrocardiogram revealed atrial fibrillation, left axis deviation, and T-wave changes consistent with left ventricular ischemia. The x-ray of the chest showed moderate transverse enlargement of the heart, suggesting an enlarged left ventricle. There was straightening of the left heart border, indicative of an increase in the size of the pulmonary artery segment and possibly left atrial appendage. On barium swallow there was posterior displacement and compression of the esophagus. The phonocardiogram confirmed the loud snapping first sound. A loud third sound was clearly recorded 0.12 second after the second sound. In our opinion, this third sound represents a delayed opening snap. Possibly, the opening snap of the mitral valve is delayed by an increase in early ventricular diastolic pressure which may increase the pressure gradient between the left atrium and left ventricle. It is interesting to note that the opening snap has been "delayed" in all of our patients who have free aortic insufficiency and documented associated mitral stenosis. This patient's mitral valve orifice was estimated to be 1 cm² at the time of surgery.

Summary. This woman had the classic clinical picture of combined free aortic insufficiency and mitral stenosis. Her rhythm was atrial fibrillation and on auscultation she had a loud first sound, an accentuated pulmonic second sound, and a "delayed" opening snap. The diastolic rumble at the apex was long and extended to the first sound. Chest x-ray and fluoroscopy revealed straightening of the left heart border and localized esophageal displacement consistent with left atrial enlargement.

Figure 4 represents a composite of 3 patients with proven free aortic insufficiency and associated significant mitral stenosis. Patient A was a 52-year-old female with rheumatic aortic insufficiency and mitral stenosis. The phonocardiogram showed a loud first sound, a prominent third sound, and a characteristic diastolic rumble. The rhythm was atrial fibrillation. The chest film showed transverse enlargement, suggesting left ventricular enlargement and straightening of the left heart border. Patient B was a 29-year-old male with a 2-year history of recurrent episodes of hemoptysis and progressive dyspnea on exertion. Blood pressure 160/70-0. At the apex were heard a grade II apical systolic murmur, an opening snap, and a rumbling murmur occupying practically all of diastole with presystolic accentuation. These auscultatory findings were confirmed by phonocardiogram. The chest x-ray revealed enlargement of the left ventricle and straightening of the left heart border. Enlargement of the left atrium and calcification in the mitral valve area were observed on fluoroscopy. The patient expired 6 days after cardiac surgery. Autopsy was performed and confirmed the clinical impression of rheumatic heart disease with moderate aortic insufficiency and severe mitral stenosis. The mitral valve was described as slightly larger than the diameter of a lead pencil. Patient C was a 35-year-old man with a history of several years of hemoptysis, progressive dyspnea on exertion, and fatigue. Physical examination revealed a blood pressure of 120/40-0. At the apex a grade III systolic murmur, an opening snap, and a grade IV diastolic rumble with presystolic accentuation were noted. These findings are documented on phonocardiogram. Notching of the P-waves on the electrocardiogram, suggestive of left atrial enlargement, was evident. Chest x-ray revealed enlargement of the left ventricle and slight straightening of the left heart border. At surgery the mitral valve was explored and moderate stenosis was found.
These 3 cases all demonstrate the accentuated first sound present when there is associated mitral stenosis. The third sound was easily recorded in all of these patients, timing approximately 0.12 second after the second sound. The diastolic rumble usually filled all of diastole. In one patient the electrocardiogram showed atrial fibrillation, and in another, broad-notched P-waves. In all 3 cases there was definite straightening of the left heart border, suggestive of pulmonary artery segment and/or left atrial enlargement. As previously mentioned, these findings are in contrast to the patient with pure aortic insufficiency without associated mitral stenosis where the first apical sound is usually faint, the third sound occurs 0.14 to 0.16 second after the second sound (ventricular diastolic gallop), and the diastolic rumble is generally shorter and occurs in early and mid-diastole. The chest x-rays in these instances of aortic insufficiency alone do not reveal straightening of the left heart border.

**Points of Confusion.** In evaluating a number of patients with severe aortic insufficiency several points of confusion concerning the auscultatory findings have become apparent (fig. 5).

First, a loud ejection sound is frequently present over the base of the heart in patients with free aortic insufficiency. This may be heard quite well at the apex and may be misinterpreted as a loud first sound (fig. 5A). A gallop may be misinterpreted as an opening snap, and the Austin Flint murmur considered the murmur of organic mitral stenosis. The clinical points previously mentioned will help in making this differentiation. The phonocardiogram is also helpful, since the ejection sound may occur at a time later than that of the first sound.

Second, in some patients (fig. 5B) both an opening snap and ventricular diastolic gallop may be present. If this is recognized, one may suspect the presence of organic mitral stenosis. The presence of an early ventricular diastolic gallop would seem clinically to preclude a tight mitral stenosis. Figure 5C presents a woman with a tight mitral stenosis and a Graham Steell murmur. In some instances the Graham Steell murmur may be grade III or even grade IV, and confused with the murmur of aortic insufficiency. In our experience the Graham Steell murmur, except when extremely loud (grade IV), is seldom heard at the aortic area and is usually heard only in the pulmonic area and along the left sternal border. The presence of a well-maintained diastolic blood pressure would

![Fig. 4. Composite of 3 patients with aortic insufficiency and associated significant mitral stenosis. Note the loud first sound ($S_1$) "delayed" opening snap, ($S_2$), and pan-diastolic rumble in all instances. The cardiac silhouettes all show some straightening of the left heart border.](image-url)
be a point against any significant aortic insufficiency, although certainly mild aortic insufficiency may occur with a fairly normal diastolic pressure. If the patient has the classic findings of tight mitral stenosis with a loud pulmonic second sound, a normal diastolic pressure, and a decrescendo blowing diastolic murmur best heard in the pulmonic area and along the left sternal border, then one can assume that this is a Graham Steell murmur and not the murmur of aortic insufficiency.

When the heart rate is rapid (90 or above) one is more apt to misdiagnose mitral stenosis when only free aortic insufficiency is present, since the diastolic rumble frequently occupies all of diastole, often with presystolic accentuation. A patient with aortic insufficiency having normal sinus rhythm, a rapid heart rate and a short P-R interval might be particularly confusing. In this instance the short P-R interval would produce accentuation of the first sound leading to confusion with the loud first sound of mitral stenosis.

It has often been stated in the literature that in organic mitral stenosis the Q-1 interval is prolonged, and we have attempted in some instances to utilize this fact to differentiate which patients have associated mitral stenosis. In our experience, in patients with free aortic insufficiency, the first sound is often quite indistinct and difficult to clearly define on the phonocardiogram. Also, one may mistake the loud systolic ejection sound with the first sound, and thereby misinterpret a prolonged Q-1 interval. For these reasons we have not made clinical application of the Q-1 interval.

Mechanisms of Productions of the Austin Flint Murmur. Several mechanisms have been proposed for production of the Austin Flint murmur, although in general all are based on the concept of the production of relative mitral stenosis. Possibly the regurgitant aortic stream may displace the aortic leaflet of the mitral valve so that this leaflet encroaches on the mitral orifice during diastole, producing some narrowing. The passage

Fig. 5. Auscultatory points of confusion. In patient A, the loud ejection sound (E) was mistaken for a loud first sound and therefore the patient was suspected of having associated mitral stenosis. Patient B demonstrates possibility of both an opening snap (OS) and a ventricular diastolic gallop (G). Clinically, the patient had moderately free aortic insufficiency and significant mitral stenosis confirmed by surgery. Patient C had classical tight mitral stenosis with a loud Graham Steell murmur misdiagnosed as that of aortic insufficiency. She had none of the peripheral signs of free aortic insufficiency. At the apex note the loud first sound (S1), opening snap (OS), and diastolic rumble (DM).
of blood through a relatively normal mitral valve into a large dilated left ventricle may also act as a relative mitral stenosis. In addition, some investigators believe that the regurgitant aortic stream may, by increasing left ventricular diastolic pressure, displace the mitral leaflets upward toward a more closed position. The presence of mitral insufficiency would also tend to increase the flow across the mitral valve, since during systole some of the blood from the left ventricle would regurgitate into and distend the left atrium, producing both an increase in left atrial volume and pressure. In diastole, this would result in increased velocity and quantity of blood traversing the mitral orifice, again contributing to a relative mitral stenosis.

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