Premature Mitral Valve Closure
A Hemodynamic Explanation for Absence of the First Sound in Aortic Insufficiency

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Normally the mitral valve does not close until shortly after the onset of left ventricular contraction. However, certain instances of severe aortic insufficiency have been reported in which the rate of rise of left ventricular diastolic pressure was such that the ventricular diastolic pressure exceeded the left atrial pressure by mid-diastole, well before the beginning of ventricular contraction. During terminal diastole there was equilibration between left ventricular and aortic pressures. Figure 1 illustrates this phenomenon of premature mitral valve closure by comparing the simultaneous left atrial and ventricular pressures of a patient with mild mitral insufficiency and left heart failure having relatively normal hemodynamics with the similar but superimposed (redrawn) pressure tracings of the first of the three patients being reported here. McKusick has predicted that the first heart sound should be muffled in this hemodynamic situation, since the mitral valve, closure and tensing of which produces the major portion of the first sound, is already closed at the onset of ventricular contraction. Absence of the apical first sound has been observed and confirmed by phonocardiography in three patients with severe aortic insufficiency who showed premature mitral valve closure. Their clinical and physiologic data form the substance of this report.

Case Reports

Case 1

A 39-year-old white man had had recurrent chills for several years, but repeated examinations by his private physician through October 1960 had disclosed no heart murmurs. He was admitted to V. A. Hospital, Hines, Illinois, in January 1961 because of the recent onset of symptoms of left heart failure. Physical examination disclosed a blood pressure of 122/56, clubbing of the fingers, and to-and-fro murmurs all over the precordium. The first sound was absent at all areas, but the second sound was accentuated at the second left parasternal interspace (fig. 2). The pulse was Corrigan in type, and Duroziez’s sign was elicited. Chest x-rays showed cardiac enlargement, and electrocardiograms were characteristic of left ventricular hypertrophy. The P-R interval was 0.16 second with a rate of 87 per minute. On cardiac catheterization the pressures as recorded in the various heart chambers were as follows: right atrium, 6 mm. Hg; right ventricle, 50/3 to +8; pulmonary artery, 50/27; left atrium, 20 (mean), “a” wave, 34, “v” wave, 37; left ventricle, 118/53; and radial artery, 114/53 (fig. 3). The cardiac output at rest was 2.3 L/min./M.² with an effective forward stroke volume of 50 ml. and with exercise was 2.4 L/min./M.² with an effective forward stroke volume of 41 ml. Pulmonary and systemic vascular resistances were 335 and 1,300 dynes sec. cm⁻⁵ respectively.

Although 12 blood cultures were negative, he was treated with penicillin and streptomycin because of a presumptive diagnosis of bacterial endocarditis. Surgical repair of his aortic valve was planned, but sudden deterioration in his condition occurred on March 21, and he died 5 days later. At necropsy, the aortic valve was severely scarred and deformed, and only the right anterior and posterior cusps were present. Between these two cusps there was an 0.8-em. segment of aortic circumference for which there was no valve sub-
there was evidence of an old healed pancarditis manifested by perivascular onionskin type of fibrosis and fibrous adhesive pericarditis. The pathologic diagnosis was bacterial endocarditis, active, superimposed on healed deforming endocarditis with an acquired bicuspid valve, probably rheumatic.

Case 2
A 3-month-old white boy was catheterized in June 1955 and was found to have a left-to-right shunt at the pulmonary artery level and a pulmonary artery pressure equal to systemic pressure. A small ductus arteriosus, 1½ cm. in length, was ligated uneventfully. Postoperatively the clinical, electrocardiographic, and x-ray findings were compatible with severe aortic stenosis. In April 1960 a repeat catheterization revealed a pulmonary artery pressure of 36/15 mm. Hg, a left ventricular pressure of 184/16, and an aortic pressure of 90/56. Seven weeks later under cardiopulmonary bypass the aorta was opened and the aortic valve exposed. The commissure between the right coronary cusp and posterior cusp was fully developed, but the other two commissures were closed. Since there appeared to be an adequate ridge between the cusps, these commissures were opened by careful sharp dissection with a scalpel. Immediately after surgery the patient developed a loud, early diastolic murmur indicative of aortic regurgitation. In August 1961 another catheterization was performed, and the following pressures were obtained: right ventricle, 67/6 mm. Hg; pulmonary artery,
67/33; mean pulmonary capillary wedge, 18; left ventricle, 110/35; and aortic, 104/35 (fig. 4). A cineangiocardiogram with injection from the ascending aorta showed free aortic insufficiency with a markedly dilated ascending aorta. The first sound, which had been present before surgery, was no longer heard (fig. 5). The P-R interval was 0.18 second with a rate of 91 per minute. Bacterial endocarditis was considered, but several blood cultures were taken and subsequently reported negative. There was no evidence of congestive heart failure, although exercise tolerance was decreased and the mean pulmonary capillary wedge pressure was elevated to 18 mm. Hg. In May 1962 under cardiopulmonary bypass the aortic leaflets were excised and a dispersion-processed tricuspid Teflon prosthesis was sutured in place.* A few weeks later when checked by Dr. Peter Vlad of the Children's Hospital, Buffalo, the first sound was well heard.

Case 3

A 40-year-old Negro man was admitted to Hines V. A. Hospital in December 1962 for evaluation of symptoms of left heart failure that had been present for 6 months. Aortic insufficiency had been discovered in 1950. The blood pressure at that time was 130/30, and the electrocardiogram was characteristic of left ventricular hypertrophy. Because of the possibility of bacterial endocarditis he was treated with 1,200,000 units of penicillin daily for 6 weeks before receiving a medical discharge from the service. His past history recorded primary syphilis in 1945 and secondary syphilis in 1947. He had been in the military service since 1942.

The patient's tall, slender physique suggested the possibility of the Marfan syndrome, but there were no other extra-cardiac findings of this disorder. A hyperdynamic apical thrust was noted in the sixth left interspace between the midelavicular and anterior axillary lines. A first heart sound was not heard or recorded at any area over the precordium. The second heart sound was accentuated and palpable at the second left parasternal interspace. It was also heard along the left lower sternal border but was absent at the apex and the second right parasternal interspace; no splitting was detected. Grade IV/VI to-and-fro murmurs, also palpable as thrills, were present throughout the precordium. The blood pressure was 120/40, and a waterhammer pulse and Duroziez’s sign were noted. A prominent pulsus bisferiens was also present. Findings of congestive failure were limited to a pleural effusion in the lower third of the right hemithorax. Marked cardiac enlargement was present on x-ray, and the electrocardiogram again showed evidence of left ventricular hypertrophy. The P-R interval was 0.22 to 0.24 second with a rate of 68 to 83 per minute.

*This surgery was performed by Dr. William H. Muller, Jr., at the University of Virginia Medical Center, Charlottesville, Virginia.

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Figure 3B

Pressure recording in case 1 (free aortic incompetence) as the catheter is pulled back from the left ventricle to the left atrium. The "a" wave of the left atrial pressure curve averaged 34 mm. Hg and was consistently lower than the left ventricular end-diastolic pressure, which averaged 33 mm. Hg. LV, left ventricle; D, diastole; S, systole; E, end-diastole; LA, left atrium.
and the effective forward stroke volume was 35 ml. Pulmonary vascular resistance and systemic vascular resistance were 300 and 1,800 dynes sec. cm.\(^{-5}\) respectively. Left ventricular pressure and sound were recorded simultaneously by a Dallons-Teleo intracardiac micromanometer. A first sound was not present in the left ventricle (fig. 6).

Cineangiograms done from the left ventricle and the aortic root demonstrated a large end-systolic volume in the left ventricle and free regurgitation of the radiopaque dye from the aorta to the left ventricle. The ascending aorta was not dilated, a subvalvaru chamber or narrowing of the outflow tract was not demonstrated, and there was no reflux of dye into the left atrium.

The patient refused surgery and died on March 13, 1963. An autopsy was not performed.

**Discussion**

In these three patients closure and tensing of the mitral valve were presumably less forceful than normal and consequently silent because the rate of change of left ventricular pressure at the point where it exceeded left atrial pressure was less than normal. This of course implies that the rate with which the valve leaflets approach each other and hence the force with which they close is closely related to the rate of pressure change tending to drive the valves shut. In considering the auscultatory counterpart of this slow and premature valve closure, absence of the apical first sound, one additionally assumes that mitral closure is the principal component of this sound and that the intensity of sound produced by this valve closure is proportional to the force with which it closes. These are all reasonable assumptions, which are supported by physiologic and clinical data too numerous to cite here. We have no explanation for the absence of other components (sounds produced by contracting heart muscle, tricuspid closure, and pulmonic and aortic opening sounds) usually considered to participate in the production of the first heart sound complex. We are also aware of reports describing a time lag between the atrioventricular pressure crossing and the onset of the first sound and that these observations have been interpreted as evidence that the first sound occurs during ventricular isovolumetric contraction.
and is caused by mechanisms other than 'collision of the valve margins.' If a critical rate of pressure rise during isovolumetric contraction is required to set relatively indistensible cardiac structures into vibration for the production of the first sound, as suggested by these workers, this may not occur where equilibration of aortic and ventricular diastolic pressures precludes a phase of isovolumetric contraction. In any case we consider it significant that in case 2 the apical first sound was present before the surgical production of aortic incompetence, absent while aortic incompetence was present, and present again after its surgical improvement.

One would predict that in some cases showing premature mitral closure a mid-diastolic sound should be present. This might explain some of the third heart sounds present in severe aortic incompetence. It is possible in our cases that a faint sound of mitral closure was obscured by the diastolic murmur which in all three cases was clearly heard at the apex as well as the base.

Previous reports of this hemodynamic phenomenon have not included a description of the first heart sound, and the general literature on severe aortic insufficiency contains little information on this aspect. Levine and Harvey state that the first sound in severe aortic insufficiency is often faint but attribute this alteration in intensity to the frequent association of a prolonged P-R interval. They do not mention any hemodynamic correla-
**Figure 6**

Sound and pressure in the left ventricle recorded instantaneously by a Dallons-Telco intracardiac micromanometer with simultaneous left atrial and radial artery pressures as recorded by external transducers via Brockenbrough and Riley needles. The left ventricular end-diastolic pressure exceeds the left atrial pressure by 10 mm. Hg and equilibrates with the brachial artery diastolic pressure at approximately 51 mm. Hg. A first sound is not recorded in the left ventricle. External phonocardiograms also failed to record a first sound. ECG, electrocardiogram; ICP, intracardiac phonocardiogram; SM, systolic murmur; DM, diastolic murmur; LV, left ventricle; LA, left atrium; PMC, premature mitral closure; PC, premature contraction; BA, brachial artery.

Case reports of aortic valve rupture infrequently give information on the first sound and from those that do learn only that it may be present,9–14 feeble,7,15 or absent.14,16–18 While absence of the first sound is consistent with hemodynamic data presented here, reports of its unequivocal presence in instances of aortic valve rupture suggest that rather different hemodynamic situations may be present in cases having similar pathologic lesions. It is also possible that some of these “first sounds” were ejection...
sounds masquerading as true first sounds.8

The rate of rise of ventricular diastolic pressure is a function of the rate of inflow of blood into the chamber and the ventricle’s effective impedance* to filling. A rapid early rise in ventricular diastolic pressure as seen in the present cases would require then a rapid rate of aortic regurgitant inflow while a ventricle offering increased impedance to filling would accentuate this phenomenon. An increased impedance could result from either intrinsic changes in the distensibility of ventricular muscle as might be produced by ventricular hypertrophy or from a dilated ventricle with an increased residual volume operating over a stiff portion of its volumepressure curve. In this latter connection it is interesting to note the pressure-volume studies reported by Dodge et al.4,19 for an instance of this hemodynamic phenomenon. Using an angiocardiographic technic, these workers found that with an end-diastolic pressure of approximately 60 mm. Hg the simultaneous volume was 444 ml. The residual volume was 235 ml., and the stroke volume of 209 ml. was further divided into an effective forward stroke volume of 48 ml. and a regurgitant volume of 161 ml. per beat. The magnitude of these volumes is more fully appreciated when it is realized that the left ventricular end-diastolic volume in 11 patients without cause for left ventricular enlargement ranged from 60 to 131 ml.20 and that 23 to 29 per cent of this volume probably can be assumed to be the normal residual volume.21,22 These data by Dodge indicate that severe aortic regurgitation, a markedly increased residual volume, and ventricular dilatation were indeed present, but insufficient data are given to infer anything about the compliance or impedance of the left ventricle.

The similarity of the left ventricular pressure curves presented here to those found in constrictive pericarditis and other restrictive lesions involving the myocardium and endocardium is apparent. In all of these the diastolic pressure is characterized by a “dip-plateau.” In restrictive lesions without aortic insufficiency, however, the left ventricular plateau equilibrates with the left atrial pressure, whereas in severe aortic insufficiency the plateau equilibrates with the aortic diastolic pressure and exceeds left atrial pressure during mid-diastole. The former situation does not cause premature closure of the mitral valve, since there is no reason for reversal of the mitral gradient prior to ventricular systole.

Seven instances of this hemodynamic phenomenon have come to our attention in addition to the three presented here. Of these 10 cases five were adults who had a history of bacterial endocarditis,2-4,19,23 two were children who developed murmurs of aortic regurgitation following commissurotomy for congenital aortic stenosis,2 one was an adult who sustained a ruptured aortic valve as the result of trauma,24 and one was a 16-year-old girl with aortic stenosis and insufficiency but of whom there is no further information.1

The pathologic findings are known for two instances other than our own. In the one resulting from trauma, the posterior commissural attachment of the left anterior aortic cusp was torn and displaced downward toward the left ventricle.24 In another who had had an episode of bacterial endocarditis 2 1/2 years before there was a large perforation at the base of one of the aortic leaflets.3 The high incidence of bacterial endocarditis noted in this group is in agreement with the observations of Segal et al.,25 who found that 22 per cent of 83 patients with severe rheumatic aortic insufficiency had a history of bacterial endocarditis.

From available evidence we believe that the conditions conducive to the development of premature mitral closure (i.e., severe aortic regurgitation probably with an increased impedance to ventricular filling) are most often present in the acutely developing aortic incompetence as seen following traumatic rup-

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*Impedance is used rather than compliance because it embraces resistance to filling caused by elastic, frictional, and inertial properties of the ventricle and its contained blood.
ture of the valve, bacterial endocarditis, and aortic valve surgery. We further regard this phenomenon as uncommon and believe it is often of grave prognostic significance. It should be suspected if the apical first heart sound is absent in aortic incompetence.

Summary

The phenomenon of premature mitral valve closure is described in three patients with severe aortic insufficiency and related to absence of the first heart sound.

The occurrence of premature mitral valve closure is favored by severe aortic regurgitation probably with increased impedance to left ventricular filling.

Available evidence suggests that this phenomenon usually occurs in the more rapidly developing forms of aortic incompetence (bacterial endocarditis, post surgical, and traumatic rupture of the valve), that it is uncommon, and that it often implies a grave prognosis.

It should be suspected when the apical first heart sound is absent in aortic incompetence.

References

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