Sudden, Severe Aortic Insufficiency

By E. Douglas Wigle, M.D., and Claude J. Labrosse, M.D.

The experimental production of sudden, severe aortic insufficiency causes left ventricular diastolic pressure to become markedly elevated so that it exceeds left atrial pressure and results in diastolic closure of the mitral valve. Ten cases of severe aortic insufficiency have been reported in which left ventricular pressure exceeded left atrial pressure during diastole, causing premature mitral valve closure. In all of these 10 cases the severely elevated left ventricular diastolic pressure equaled the aortic pressure by end-diastole. This latter phenomenon was not observed under experimental conditions.

The etiology of the aortic insufficiency in these 10 cases strongly suggested to Meadows et al. that the sudden onset of the lesion and its severity were important factors in the development of these unusual hemodynamic features. In five cases there was a history of bacterial endocarditis, in two the aortic insufficiency developed after surgery for aortic stenosis. Traumatic rupture of the aortic valve was found in one case, the aortic insufficiency developed relatively suddenly in a previously hypertensive patient in another. No mention of the valve pathology was available in the tenth case.

The present report details the clinical and hemodynamic findings in 14 cases of severe aortic insufficiency in whom left ventricular pressure exceeded left atrial pressure during diastole. The severely elevated left ventricular diastolic pressure equaled aortic pressure by end-diastole in nine cases (cases 1 to 9) and was less than aortic diastolic pressure in the remaining five (cases 10 to 14). The etiology of the lesion was bacterial endocarditis in 10 instances, prolapse of all three aortic valve leaflets associated with lesions of the ascending aorta in two instances, and rupture of the valve following muscular strain in one instance. The valve pathology in the remaining case is unknown. This experience suggests that the unusual hemodynamic aberrations in these cases was related to the suddenness of onset as well as the severity of the aortic reflux. The poor prognosis attending such aortic valve lesions renders the prompt recognition of sudden, severe aortic insufficiency of considerable importance.

The presence of additional mitral insufficiency in nine of these 14 cases permitted comparison of the effects of this lesion in patients with the previously observed effects of superimposing mitral insufficiency on experimental acute aortic insufficiency. Welsh et al. observed that when mitral insufficiency was superimposed on acute aortic insufficiency the left ventricular end-diastolic pressure decreased, whereas left atrial pressure increased.

Material and Methods

Complete details of history and physical examination were available in all 14 patients. One or both of the authors examined 12 of these patients, careful note being made of (1) the auscultatory intensity of the first heart sound at the apex, (2) the presence or absence of diastolic sounds, and (3) the character of the systolic and diastolic murmurs. Phonocardiograms were obtained in seven cases and apexcardiograms in four cases. In two cases, both phonocardiograms and apexcardiograms were recorded at the time of left heart catheterization during the simultaneous recording of left atrial and ventricular pressures. Twelve-lead electrocardiograms were available in all cases as were chest x-rays and fluoroscopic examinations.

Left heart catheterization was carried out by combining percutaneous retrograde aortic with transseptal left heart catheterization in nine cases. Right heart catheterization was simultaneously performed in two of these cases. Right atrial...
SUDDEN, SEVERE AORTIC INSUFFICIENCY

and right ventricular pressures were recorded via the transseptal left heart catheter in the remaining seven cases in which this catheterization technique was used.\textsuperscript{17} Two cases underwent transthoracic left heart catheterization by the Bjork technic\textsuperscript{20} and, in these, retrograde aortic catheterization was subsequently performed for angiographic purposes. Three cases (cases 5, 6, and 14) had retrograde aortic and left ventricular catheterization alone. In these three cases, left atrial pressure was not recorded, but left ventricular end-diastolic pressure was assumed to exceed left atrial pressure because of the extreme elevation (48 to 55 mm. Hg) of the former. All pressures were recorded on an Electronics for Medicine photographic recorder with matched Statham P 23 db strain-gauges. Aortic regurgitation was demonstrated to be severe in all cases by aortic root cineangiography. In six of the nine cases undergoing combined retrograde aortic and transseptal left heart catheterization the severity of the aortic insufficiency was confirmed by dye-dilution technic, indocyanine green dye being injected 2 cm. above the aortic valve via the retrograde catheter, retrograde flowing dye being sampled from the left ventricle via the transseptal catheter, forward flowing dye being recorded by an ear oximeter placed on the right ear.\textsuperscript{17} The presence and severity of mitral insufficiency was estimated in nine cases (cases 2 to 4 and 7 to 12) by injection of indocyanine green dye into the left ventricle. Regurgitant dye was sampled by withdrawal of blood from the left atrium, forward flowing dye being sampled by an oximeter placed on the right ear.\textsuperscript{17, 21} The degree of aortic or mitral insufficiency, with these dye-dilution technics, was estimated by comparing the area under the forward and regurgitant curves and was graded mild, moderate, moderately severe, or severe (one plus to four plus—table 2), on this basis.\textsuperscript{17, 21} During the phase of left ventricular opacification following aortic root cineangiography, opacification of the left atrium was taken to indicate the presence of mitral insufficiency. The severity of the mitral leak could not be assessed by this method, nor could the absence of left atrial opacification be taken as absolute evidence of the absence of mitral insufficiency. Cardiac outputs by the Fick principle were determined in cases 4, 7, and 12.

Results

The clinical findings in the 14 cases are tabulated in table 1 and the results of hemodynamic investigation in table 2. There were 13 males and one female. The age range was from 23 to 50 years. In patient 1, who first developed aortic insufficiency following severe muscular strain, the left coronary cusp was torn and deficient at the time of surgery. In case 12 a chronic aortitis of undetermined cause\textsuperscript{22} was found at surgery. All three aortic valve cusps were prolapsed into the left ventricle. In case 13 the aortic cusps were prolapsed following dissecting aneurysm of the ascending aorta. The etiology and pathology of the aortic valve lesion was unknown in case 7. The remaining 10 cases developed aortic insufficiency following bacterial endocarditis. In seven of these 10 cases a large perforation in one or more aortic valve leaflets was observed at the time of cardiac surgery or at postmortem examination. The aortic valve was not visualized in the remaining three cases that developed aortic insufficiency following bacterial endocarditis.

Hemodynamic Investigation

The clinical characteristics of this group of patients are, to a considerable extent, dependent upon the hemodynamic abnormalities. The left ventricular end-diastolic pressure was extremely elevated in all cases (average 45 mm. Hg, range 30 to 58 mm. Hg). In every case, left ventricular pressure exceeded left atrial pressure during diastole (figs. 1A, 2, and 3), and usually did so well before end-diastole. In cases 1 to 9, the left ventricular end-diastolic pressure averaged 48 mm. Hg (range 39 to 58 mm. Hg) and equaled aortic pressure by end-diastole (fig. 1B). In cases 10 to 14, the left ventricular end-diastolic pressure averaged 40 mm. Hg (range 30 to 50 mm. Hg) and was less than aortic pressure at end-diastole. There did not appear to be any significant difference in the severity of the aortic insufficiency either by cineangiography or dye-dilution estimation, between cases 1 to 9 and cases 10 to 14. Mitral insufficiency, however, was demonstrated to be present in cases 10 to 14 either by dye-dilution technic or cineangiography, or both. In cases 1 to 9, mitral insufficiency was present in four, and absent in five cases. In the five cases without superimposed mitral insufficiency (cases 1, 2, and 5 to 7), the left ventricular end-diastolic...
### Table 1

**Clinical Findings in Sudden, Severe Aortic Insufficiency**

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| E.C.G.: LAH | 0 | 0 | 0 | + | 0 | + | 0 | + | 0 | + | 0 | + | 0 | + | + | + |
|-------------|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| LVH | + | + | + | + | + | + | + | + | + | + |
| PR interval | .16 | .20 | .17 | .19 | .20 | .20 | .21 | .24 | .17 | .20 | .22 | .24 | .22 | .19 |
| Mean QRS axis | −10 | +20 | +50 | +70 | +60 | +50 | −30 | +60 | +45 | +60 | −40 | 0 | −25 | −40 |
| X-Ray: Aorta | N | N | N | 2/4 | 2/4 | 1/4 | 2/4 | 1/4 | N | N | 1/4 | 1/4 | 2/4 | 2/4 | 2/4 |
| Left atrium | 0/4 | 1/4 | 2/4 | 1/4 | 1/4 | 2/4 | 2/4 | 2/4 | 2/4 | 2/4 | 2/4 | 2/4 | 2/4 | 2/4 | 1/4 |
| Right atrium | 0/4 | 1/4 | 2/4 | 1/4 | 2/4 | 1/4 | 2/4 | 2/4 | 1/4 | 1/4 | 2/4 | 2/4 | 1/4 | 1/4 |
| Right ventricle | 0/4 | 0/4 | 2/4 | 1/4 | 2/4 | 2/4 | 2/4 | 1/4 | 2/4 | 1/4 | 2/4 | 2/4 | 1/4 |
| CT ratio, % | 52 | 58 | 53 | 62 | 72 | 62 | 75 | 61 | 54 | 65 | 64 | 56 | 52 | 69 |

*Time from recognition of A.I. to onset of symptoms.
†See text.
‡Intensity of murmurs and x-ray size of cardiac chambers graded 1 to 4.

Abbreviations: 0, absent; −, no observation; +, present; SR, spontaneous rupture of aortic valve; CA, chronic aortitis of undetermined cause; DA, dissecting aneurysm; CT ratio, cardiothoracic ratio; LAH, left atrial hypertrophy; BE, bacterial endocarditis; Noc., nocturnal; LVH, left ventricular hypertrophy; RV, right ventricle; P, perforation of one or more aortic cusps visualized at surgery or post mortem; N, normal; LV, left ventricle.

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**Clinical Investigation**

Thirteen of the 14 patients noted dyspnea on exertion and 12 had suffered dyspnea on exertion in one or more occasions. Anginal chest pain was noted in only three cases and in two of these it was secondary to dissecting aneurysm. The interval between the onset of symptoms and the onset of aortic insufficiency was characterized by a progressive deterioration, especially following exertion. Their clinical course was usually one of progressive deterioration, especially following exertion. Clinical pitting and fatigue on exertion were prominent.

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**Sudden, Severe Aortic Insufficiency**

In the nine cases with this superimposed lesion, the left ventricular systolic pressure averaged 48.4 mm. Hg, whereas in the nine cases without this superimposed lesion (cases 3, 4, and 8 to 14) this pressure averaged 27.9 mm. Hg. The mean left ventricular pressures were greater than 45 mm. Hg when mitral insufficiency was present and 41 mm. Hg or less when there was no mitral insufficiency and less than 8 mm. Hg without mitral insufficiency. End-diastolic right ventricular pressure, a higher left ventricular end-diastolic pressure, was present in seven of the eight cases with additional mitral insufficiency. Pulmonary arterial and right ventricular pressures were greater than 45 mm. Hg.
### Hemodynamics in Sudden, Severe Aortic Insufficiency

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<td>122/44</td>
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*All pressures measured in mm. Hg.
†Mitril insufficiency demonstrated by left atrial opacification during left ventricular opacification following aortic root angiogram (see text).
S/D, systolic/end diastolic; AI, aortic insufficiency; MI, mitral insufficiency; LV, left ventricle; LA, left atrium; RA, right atrium; RV, right ventricle; PA, pulmonary artery; Angio., angiographic estimation of aortic insufficiency—graded 1+ to 4+ in severity (see text); Dye, Dye-dilution estimation of aortic or mitral insufficiency—graded 1+ to 4+ in severity (see text).
inent symptoms in the majority of cases. The severity of dyspnea and fatigue tended to be greater in those cases with additional mitral insufficiency than in those without this lesion.

All cases had peripheral signs of severe aortic insufficiency as evidenced by prominent carotid pulsations, a waterhammer pulse, capillary pulsation, pistol-shot sounds over the femoral arteries, and Duroziez's sign. These peripheral signs were less severe when the patient was in the stage of congestive heart failure. Palpable evidence of left ventricular hypertrophy was present in all cases. A palpable right ventricular heave was noted only in those cases with mitral insufficiency and secondary pulmonary hypertension. A double outward movement of the left ventricle in diastole ("double diastolic apex beat") was both visible and palpable in cases 4, 8, 9, 11, and 12.

The auscultatory features of this series of cases were most helpful in making a clinical diagnosis of sudden severe aortic insufficiency. As described by Meadows et al.\(^8\) a distinct first heart sound at the cardiac apex (first or mitral component of the first sound) was not heard in any case. Occasionally a faint first heart sound could be heard at the lower left sternal border and was believed to represent the second (tricuspid) component of the first heart sound. Phonocardiograms at the cardiac apex usually failed to reveal any first heart sound (fig. 3A), although on one occasion a faint sound was recorded (fig.

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**Figure 1**

A, left. Simultaneous left ventricular and left atrial pressures demonstrating the left ventricular pressure exceeding left atrial pressure during diastole. B, right. Simultaneous left ventricular and aortic pressures demonstrating the equalization of these pressures by end-diastole. These recordings were made 3 months after the sudden onset of severe aortic insufficiency due to rupture of the aortic valve that occurred following muscular strain. The left coronary cusp of the aortic valve was torn and deficient at the time of surgery.

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**Figure 2**

Case 2. Simultaneous left ventricular and left atrial pressures demonstrating the former exceeding the latter well before end-diastole. The systolic portion of the left ventricular pressure is not shown due to the high recording sensitivity. The effect of a forceful left atrial contraction against a closed mitral valve, on the left atrial pressure, is evident. E.D.P., end-diastolic left ventricular pressure.
Case 11. A, upper. Simultaneous left ventricular (L.V.) and left atrial (L.A.) pressure recordings and apex phonocardiogram demonstrating the occurrence of a diastolic sound (Sx) at the point where left ventricular diastolic pressure exceeded left atrial pressure. This sound is believed to be due to diastolic closure of the mitral valve. B, lower. Simultaneous left ventricular (L.V.) and left atrial (L.A.) pressure recordings and apexcardiogram (A.C.G.) demonstrating the occurrence of an inward movement of the cardiac apex (arrow) synchronous with the time that left ventricular exceeded left atrial pressure. This inward movement of the cardiac apex was believed related to the diastolic closure of the mitral valve and was at times visible and palpable clinically. The inward apical motion occurred between two outward diastolic apical movements (See fig. 6 and text).

4A). In this instance, the faintly recorded sound had one component that was believed to be tricuspid valve closure (fig. 4A). Following surgical repair of the aortic valve in this case, with a return to normal of the hemodynamics (fig. 5), a loud apical first heart sound occurred.

Figure 3

Figure 4

Case 1. A, upper. Phonocardiogram from a case of severe aortic insufficiency due to spontaneous rupture of the aortic valve following muscular strain. The

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sound was present, and both mitral and tricuspid components could be recognized at the lower left sternal border (fig. 4B).

In 11 cases an early diastolic sound was audible (fig. 4A). In the two cases in which apex phonocardiograms were done at the time of simultaneous recording of left atrial and ventricular pressures, this sound occurred at the time the left ventricular pressure exceeded left atrial pressure during diastole (fig. 3A) and was believed due to diastolic (premature) closure of the mitral valve. In one instance this diastolic sound appeared to have two components, the first component being a third heart sound, the second component, the premature mitral valve closure sound. When an apexcardiogram was recorded simultaneously with left atrial and ventricular pressures, an inward movement of the cardiac apex occurred at the time that left ventricular pressure exceeded left atrial pressure during diastole, i.e., at the time of diastolic closure of the mitral valve (fig. 3B). This diastolic inward movement of the cardiac apex is better demonstrated in the apexcardiogram shown in figure 6. This inward movement was preceded and followed by sharp diastolic outward movements of the cardiac apex (fig. 6). The initial outward movement was believed due to rapid ventricular filling from the left atrium and from the aorta. The second outward movement was believed related to the continuing aortic

recordings at the cardiac apex have been darkened for clarity of illustration. The most notable features are the loud diastolic sound (S₃) believed due to diastolic closure of the mitral valve and the absence at the cardiac apex of the first (mitral) component of the first heart sound. A small second (tricuspid) component to the first heart sound (S₁) was recorded maximally at the left sternal border in the fourth intercostal space (LSB4). D.M., diastolic murmur; S₂ second heart sound. B. lower, Postoperative phonocardiogram, following surgical repair of the torn and deficient left coronary cusp of the aortic valve, showing a loud first heart sound at the cardiac apex and two components to the first sound (mitral and tricuspid valve closure sounds) recorded at the left sternal border in the fourth intercostal space (LSB4). The diastolic sound (seen in fig. 4A) disappeared following corrective surgery, which returned the hemodynamics virtually to normal (fig. 5).

Figure 5

Case 1. Simultaneous left ventricular (L.V.) and aortic pressure recordings following spontaneous aortic valve rupture (left) and surgical repair of the aortic valve (right) demonstrating the return to normal of the left ventricular end-diastolic pressure (E.D.P.).

Figure 6

Case 8. Apexcardiogram (A.C.G.) demonstrating the diastolic inward movement of the cardiac apex (arrow) between two outward movements (I and II). The inward movement of the apex occurred at the time left ventricular pressure exceeded left atrial pressure and was believed associated with diastolic closure of the mitral valve (fig. 3B). The initial outward movement (I) was believed due to the rapid phase of ventricular filling from the left atrium, plus aortic insufficiency. The second outward movement (II) was believed due to continuing aortic insufficiency. This double outward motion of the cardiac apex in diastole could be visualized and palpated clinically in this case, hence the term “double diastolic apex beat.” Because of the premature mitral valve closure there is no atrial filling or “a” wave.
insufficiency following the premature mitral valve closure. This phenomenon of two diastolic outward movements of the left ventricular apex, interrupted by an inward movement occurring simultaneously with diastolic closure of the mitral valve, has been termed a “double diastolic apex beat” (table 1). This feature was visible and palpable clinically in five cases and was evident during left ventricular opacification following aortic root cineangiography in cases 5 and 7.

There was an aortic systolic ejection murmur in all cases, in the absence of any hemodynamic evidence of aortic stenosis (table 1). In 12 cases this murmur was grade II/IV or greater in intensity and frequently was very harsh. The aortic diastolic murmur was almost invariably harsh in quality, being grade II/IV or greater in intensity in all cases. An Austin Flint murmur was audible at the cardiac apex in nine of the 14 cases. The presence of this latter murmur and the harshness of the aortic diastolic murmur made detection of the diastolic sound at the cardiac apex difficult at times. Because the systolic and diastolic murmurs were frequently of similar harshness, and because of the absent apical first heart sound and the presence of a diastolic sound, the auscultatory differentiation of systole and diastole was frequently very difficult. It was usually necessary to palpate the carotid pulse at the time of auscultation in order to be certain of the phases of the cardiac cycle.

Case 1 was investigated following aortic valve rupture (due to muscular strain) and following surgical correction. The latter resulted in a return of the hemodynamics to normal (fig. 5), even though a mild degree of aortic insufficiency remained. The apical first heart sound reappeared and the diastolic sound disappeared following the corrective surgery (fig. 4).

Case 4 was investigated prior to the occurrence of bacterial endocarditis and following perforation of the aortic valve. Prior to endocarditis there was hemodynamic evidence of mild aortic stenosis (fig. 7, left) and angiographic evidence of moderately severe aortic insufficiency. Following perforation of the aortic valve (proved at cardiac surgery), this patient developed the hemodynamic characteristics of sudden severe aortic insufficiency (fig. 7, right). In addition, the apical first heart sound disappeared as did the ejection click, a diastolic sound appeared, a “double diastolic apex beat” was visible, palpable, and demonstrable by apexcardiography, and the aortic diastolic murmur acquired harsh overtones.

A distinct apical pansystolic murmur was audible in seven of nine cases in whom mitral insufficiency was subsequently detected during hemodynamic assessment (tables 1 and 2). The loudness of this murmur usually reflected the severity of the mitral lesion but not invariably (cf. case 9, tables 1 and 2). Patient 4 when first seen, following perforation of the aortic valve during bacterial endocarditis, had no apical pansystolic murmur. Two months later a grade II/IV pansystolic apical murmur was present and the

Figure 7
Case 4. Simultaneous left ventricular and aortic pressure recordings prior to aortic valve perforation (left) and following valve perforation from bacterial endocarditis (right). At cardiac surgery a large perforation was present in the noncoronary cusp and a small perforation present in the right coronary cusp of the aortic valve. Although there was moderately severe aortic insufficiency (and mild aortic stenosis) prior to endocarditis (left), the left ventricular end-diastolic pressure was only minimally elevated. Following endocarditis, the left ventricular end-diastolic pressure was extremely elevated and equaled aortic diastolic pressure (right). These tracings were recorded 1 year apart and the tracing at the right was obtained 5 months after the development of endocarditis.
Sudden, Severe Aortic Insufficiency

Patient shortly thereafter developed left and right heart failure. The presence of mitral insufficiency was confirmed during hemodynamic assessment. Postmortem examination in this case revealed normal mitral valve leaflets and dilatation of the mitral annulus. Postmortem examination of the mitral valve in patients 9, 10, and 13, each of whom had evidence of mitral insufficiency, revealed a dilated mitral annulus, a normal anterior mitral cusp, and a somewhat shortened posterior cusp. In none of the patients in whom the mitral valve was examined postmortem was there evidence of involvement of the mitral valve by rheumatic fever. These observations suggest that the occurrence of mitral insufficiency in these cases of sudden, severe aortic insufficiency may be secondary to left ventricular dilatation.

The electrocardiogram revealed evidence of left ventricular hypertrophy and strain in all cases. Left atrial hypertrophy was evident in six cases; five of which had evidence of mitral insufficiency. Every case was in sinus rhythm and although the P-R interval was greater than 0.20 second in five cases (table 1), the absence of the first heart sound at the cardiac apex was not believed related to this delay in atrioventricular conduction.

Radiologic assessment revealed evidence of cardiac enlargement in every case. The cardiothoracic ratio varied from 52 to 75 per cent (table 1). Left ventricular enlargement was particularly notable although the degree of enlargement of this chamber was extremely variable (fig. 8). Left atrial enlargement was most evident in those cases with superimposed mitral insufficiency. In five cases the aorta was considered to be normal in size, and in no case was it dramatically enlarged as is often the case in long-standing aortic insufficiency (fig. 8). Calcification of the aortic valve was present only in patient 4, in whom it had been present prior to perforation of the aortic valve.

Discussion

The extremely elevated end-diastolic left ventricular pressure in these cases of sudden.

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Figure 8

Posteroanterior chest x-rays in case 1 (left) and case 2 (right) demonstrating the marked variation in heart size that was observed in this series of cases. The x-ray in case 1 was taken 1 year after spontaneous rupture of the aortic valve following muscular strain. The x-ray in case 2 was taken 5 years after a bout of bacterial endocarditis. At surgery large perforations were present in two of the three aortic valve cusps. Neither case had evidence of mitral insufficiency.
severe aortic insufficiency was believed due to the imposition of a severe diastolic overload on the relatively unprepared left ventricle. It has been demonstrated that in ventricles unaccustomed to a diastolic overload, the end-diastolic pressure rises sharply at a smaller diastolic volume than in ventricles that have been chronically dilated (i.e., in aortic or mitral insufficiency). The chronically dilated ventricle is at times able to accommodate large diastolic volumes without a significant rise in end-diastolic pressure. This change in the pressure-volume characteristics of the chronically dilated heart is ill understood, but may be related to the phenomenon of stress relaxation and be due to "slippage" of myocardial fibers and possibly myocardial filament "disengagement."

The reason that sudden severe aortic insufficiency results in extreme end-diastolic pressure elevation may be initially related to a lack, or inadequate degree, of stress relaxation for the degree of aortic reflex present. The pericardium could also limit the degree of ventricular dilatation. By either or both of these mechanisms, resistance to diastolic filling would be increased and end-diastolic pressure would rise sharply. Subsequently, a variable degree of ventricular dilatation would occur (fig. 8, table 1) which would be accompanied by an increase in the degree of aortic insufficiency. This sequence would account for the very large end-diastolic volumes that have been observed in some longstanding cases of severe aortic insufficiency of sudden onset.

Aside from the cases herein reported, two other cases of aortic insufficiency of sudden onset have been encountered that did not exhibit the described hemodynamic abnormalities. In one of these there was only a small perforation of one aortic valve cusp at the time of surgery. In addition, the hemodynamics of 357 cases of chronic aortic insufficiency from this laboratory were reviewed and none exhibited the unusual hemodynamic changes herein reported. Thus it would appear that the aortic insufficiency must be both relatively sudden in onset and severe in degree before the described syndrome develops.

The hemodynamic similarity between experimental acute aortic insufficiency and sudden severe aortic insufficiency in human beings is striking. In both instances, left ventricular diastolic pressure rises sharply to exceed left atrial pressure. In the experimental preparation the ventricular diastolic pressure did not, however, equal aortic diastolic pressure. Whether or not the failure of the latter to occur in the experimental situation was related to the fact that the pericardium was open, is not known.

The similarity between experimental and clinically occurring acute aortic insufficiency may extend to the effects of superimposed mitral insufficiency. In the experimental preparation the addition of this latter lesion resulted in a lowering of left ventricular end-diastolic pressure and a rise in left atrial pressure. In this series when mitral insufficiency was present, the left ventricular end-diastolic pressure was lower and the mean left atrial pressure was higher, than when this added lesion was not present. Whether or not the lower left ventricular end-diastolic pressure and the failure to equal aortic diastolic pressure in cases 10 to 14 was related to the presence of mitral insufficiency, was not known; but this was considered possible. If this were so, the fact that in cases 3, 4, 8, and 9 with additional mitral insufficiency the left ventricular end-diastolic pressure still equaled aortic diastolic pressure would indicate that the added mitral lesion did not always result in significant lowering of the left ventricular diastolic pressure.

Mitrval insufficiency in these cases could occur in systole or in diastole (once left ventricular diastolic pressure exceeded left atrial pressure). Diastolic mitral insufficiency would be particularly effective in lowering end-diastolic left ventricular pressure in that the left atrium would serve as an additional reservoir for the aortic regurgitant volume of blood. In the experimental study of superimposing mitral insufficiency on acute aortic insufficiency, the preparation was such
that diastolic mitral insufficiency could have occurred.\textsuperscript{1}

Previous authors have commented on the protective role played by the abnormal hemodynamic situation in these cases. By equaling the aortic pressure the diastolic left ventricular pressure would limit the degree of aortic insufficiency present.\textsuperscript{9} By exceeding left atrial pressure and closing the mitral valve in diastole the elevated ventricular diastolic pressure prevented drastic rises in left atrial and pulmonary venous pressures.\textsuperscript{1}

As well as serving a protective function, the abnormal hemodynamics explain the major features of the clinical syndrome. The diastolic closure of the mitral valve caused a sound and a simultaneous inward movement of the left ventricle in diastole. Having closed in diastole, the mitral valve does not close again at the onset of systole, hence the absence of the mitral component of the first heart sound at the cardiac apex.\textsuperscript{8} These features, together with the harsh aortic systolic and diastolic murmurs, account for the difficulty in differentiating systole from diastole by auscultation alone.

**Summary**

Fourteen cases of severe aortic insufficiency are presented in which an extremely elevated left ventricular pressure exceeded left atrial pressure during diastole. In 13 of the 14 cases there was proof, or strong suggestive evidence, that the aortic insufficiency was sudden in onset. In nine cases the left ventricular pressure equaled aortic pressure by end-diastole. In five cases left ventricular pressure failed to equal aortic pressure by end-diastole. The similarities between the hemodynamics of experimental and clinically occurring sudden severe aortic insufficiency, including possibly the effect of superimposed mitral insufficiency, was at times striking. The clinical picture in these cases was sufficiently distinctive that the abnormal hemodynamics could be predicted and the presence of sudden, severe aortic insufficiency suggested. The absence of the first heart sound at the cardiac apex, the presence of an early diastolic sound and "double diastolic apex beat," the harshness of the aortic systolic and diastolic murmurs, all served to make auscultatory differentiation of systole and diastole at times extremely difficult. This difficulty in differentiating the phases of the cardiac cycle by auscultation alone was in itself rather characteristic of sudden severe aortic insufficiency. The poor prognosis in these cases makes early recognition mandatory and early surgery advisable.

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**References**


Notes for the Lumleian Lecture of April 17, 1616, by William Harvey

"It is plain from the structure of the heart that the blood is passed continuously through the lungs to the aorta as by two clacks of a water bellows to raise water. It is shown by application of a ligature that the passage of blood is from the arteries into the veins. Whence it follows that the movement of the blood is constantly in a circle, and is brought about by the beat of the heart. It is a question, therefore, whether this is for the sake of nourishment or of heat, the blood cooled by warming the limbs, being in turn warmed by the heart."—William F. Hamilton, M.D., and Dickinson W. Richards, M.D. Circulation of the Blood. Edited by Alfred P. Fishman, M.D., and Dickinson W. Richards, M.D. New York, Oxford University Press, 1964, p. 74.
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