Rupture of the Aortic Valve

By William L. Proudfit, M.D., and Lawrence J. McCormack, M.D.

RUPTURE OF THE AORTIC VALVE has been recognized in the past, but interest in the diagnosis has been purely academic. Clinical interest in this rare lesion has been stimulated by the recent development of a surgical method of treatment. The following case is presented and discussed to emphasize the clinical signs of rupture of the aortic valve.

CASE PRESENTATION

A 56 year old city fireman had been well until he was in an automobile accident on August 5, 1950. At that time his blood pressure was found to be elevated. On Sept. 7, 1950, physical examination revealed no significant abnormalities except for a blood pressure of 180/100 mm. Hg. He remained in fairly satisfactory condition, except for elevation of blood pressure to about 200/125 mm. Hg, until May 1951, when a cerebral thrombosis occurred; recovery was fairly satisfactory.

In July 1951, a presystolic gallop rhythm was noted but no cardiac murmurs. On January 31, 1952, he was feeling quite well, but his blood pressure was 176/104 and the presystolic gallop rhythm still was present. On Sept. 19, 1952, he was next seen because of dyspnea and orthopnea of several weeks' duration. On examination, his blood pressure was 190/94, a gallop rhythm was present and, for the first time, there was a loud, musical, cooing, diastolic murmur that was best heard along the left border of the sternum. A systolic blowing murmur of moderate intensity also was present, and there was a rumbling mid and late diastolic murmur at the cardiac apex. An electrocardiogram showed only slight left axis deviation and a flat T wave in lead AVL. The left axis deviation was less marked than that recorded on Sept. 9, 1950. On roentgen examination of the chest, the transverse diameter of the heart measured 19.6 cm. (2.6 cm. greater than two years previously), and the cardiac configuration was that of left ventricular hypertrophy.

Treatment consisting of a low-salt diet, digitalization, and mercurial diuretics resulted in prompt improvement. He remained fairly well during the following 14 months. His blood pressure varied from 126 to 190 mm. Hg systolic, and from 50 to 70 mm. Hg diastolic, and the gallop rhythm disappeared. He was last seen on Nov. 11, 1953, at which time he had no complaints. Physical examination showed no change from the previous findings and the blood pressure was 150/50. On the morning of Nov. 18, 1953, the patient was found dead in bed. He had had no complaints on retiring the previous evening.

DISCUSSION

Dr. William L. Proudfit (member of the Staff of the Department of Cardiovascular Disease): The diagnosis of rupture of the aortic valve is not a difficult one to make in most cases. The cardinal sign of rupture of the aortic valve is the occurrence of a musical aortic diastolic murmur. Usually the murmur is very loud, prolonged and widely transmitted, and is accompanied by a thrill. A systolic murmur also may be present. The musical character of the diastolic murmur is a result of its being almost a pure tone—rather than a mixture of tones or a noise. The tone may be higher pitched in early diastole than in the latter part of diastole and the pitch may vary from case to case. Vibrations of 130 to 340 per second were found in the series reported by Gelfand and Bellet.1 In our case the diastolic murmur had a frequency of about 250 cycles per second. Various descriptive terms have been applied to the murmur, most commonly, "cooing dove or sea gull." The murmur bears a striking resemblance to the last 3 notes of the 5 note call of the mourning dove. However, the pitch of the birdcall is higher, the frequency being...
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about 470 cycles per second in the call we have studied, and the frequency is constant for the duration of the note. Frings of the Pennsylvania State University, an authority on the calls of sea gulls, points out in a personal communication that there is a variety of calls, but none of them resembles the tape-recorded sound created by the musical diastolic murmur of a ruptured aortic valve.

The musical aortic diastolic murmur is well transmitted to the cardiac apex, but Austin Flint murmurs also are common in cases of rupture of the aortic valve. The musical murmur beginning early in diastole may be heard at the apex and, in addition, there may be a low rumbling mid and late diastolic murmur, as was noted in the case just presented.

The diastolic pressure usually is low in aortic valvular rupture, and Korotkoff's sounds may be heard to zero diastolic pressure. Peripheral signs of aortic insufficiency are present when the diastolic pressure is low. It is interesting that in this case both the systolic and diastolic pressures decreased after rupture of the valve.

The prognosis of the disease is poor, and death usually occurs within a few weeks or months; occasional patients may live for many months or even several years. Congestive heart failure, which is resistant to treatment, is the principal complication and results in death.

There is no clue in the history as to an etiologic basis for rupture of the aortic valve. Syphilitic valvular disease is a predisposing factor in many cases of rupture of the aortic valve. Eversion of the aortic valve, which is indistinguishable clinically from rupture, is a complication also of syphilitic disease. Rheumatic cardiac disease rarely may predispose to rupture of the valve. Bacterial endocarditis may cause a perforation of a leaflet with ensuing traumatic rupture of the aortic valve. Spontaneous rupture of otherwise normal-appearing valves may be seen. Hypertension is a possible predisposing factor.

Rupture or eversion of the aortic valve may be accompanied by sudden onset of pain, dyspnea or shock coincident with the performance of some strenuous physical activity. Later, dyspnea due to left ventricular failure is the predominant symptom, though anginal pain may occur.

The case presented is unusual in that the initial symptoms were well controlled by treatment during the terminal 14 months of life. The cause of sudden death was not apparent.

Dr. McCormack, will you describe the findings of the postmortem examination?

DR. LAWRENCE J. McCORMACK (member of the Staff of the Department of Pathology): The pertinent necropsy findings were limited to the heart, lungs and kidneys.

The heart weighed 930 Gm. There was a massive left ventricular preponderance with the apex being made up entirely of the left ventricle. Dissection of the coronary circulation demonstrated a normal distribution with some atherosclerosis producing luminal narrowing up to 25 per cent. The opened right atrium was not remarkable; the tricuspid valve measured 14 cm. in circumference and possessed normally formed, thin, pliable valvular leaflets. The right ventricle measured 10.0 cm. in depth and its wall was 4.0 mm. in thickness. The pulmonary valve measured 7 cm. in diameter and was not remarkable. The opened left atrium was smooth and thin walled. The mitral valve measured some 11.0 cm. in circumference with thin, pliable leaflets. The chordae tendineae were not remarkable. The left ventricular myocardium was pale and thickened to 2.5 cm. (fig. 1). The trabeculae carnea were flattened, and there was some

Fig. 1. Gross specimen of heart with left ventricle opened. Myocardial hypertrophy and cardiac dilatation are the prominent features.
dilatation of the left ventricle. Just inferior to the left cusp of the aortic valve could be seen 3 transverse fibrous thickenings, measuring up to 1.0 cm. in length and 0.2 cm. in thickness. The thickenings in actuality were small cusped structures with their free margins pointing toward the aortic valve. The aortic valve measured 7.0 cm. in diameter. Its 3 cusps were equal in size. The cardiac surface of the left cusp was reddened and appeared slightly thickened. The anterior cusp showed a small fenestration near its point of union with the aorta on the left side. The fenestration measured 4.0 by 1.0 mm.; the free margin of the cusp was intact. The left cusp showed a similar fenestration but, in addition, the left point of its attachment had pulled free from the aortic annulus and possessed a bulbous enlargement at the free tip (fig. 2). Histologic preparation demonstrated an increased cellularity of the right and left cusp margins; they also were thickened by a deposition of fibrin. No disease was demonstrable in the aortic wall at the point of attachment. Sections through the thickened areas of the endocardium showed them to be composed of fibroblasts. The myocardium possessed enlarged muscular fibers, but no necrosis or fibrosis was noted.

The lungs were heavy, weighing 1505 Gm. The external surface was mottled gray, red and black. Palpation demonstrated some increased consistency. The cut surface was grayish red in color, and copious amounts of frothy fluid could be expressed from the surfaces. Microscopically, the lung showed a granular coagulum within many of the alveoli.

The kidneys were of normal size, with a combined weight of 400 Gm. Their external surfaces were finely granular and the cut section demonstrated the same fine granularity with good demarcation of cortex and medulla. The cortex measured 0.7 cm. in thickness. Histologically, the changes were limited to the medium-sized and small arterioles. The medium-sized ones showed a marked degree of intimal thickening, but the small arterioles were only moderately thickened and uniformly hyalinized.

**Anatomic Diagnoses**

Rupture of left semilunar cusp of aortic valve with resultant aortic insufficiency.
Cardiomegaly (930 Gm.), predominantly left ventricle.
Cardiac dilatation, acute.
Fenestration, small, anterior cusp of aortic valve.
Jet lesion, endocardium.
Trauma of aortic valvular cusps.
Acute pulmonary edema (1505 Gm.).
Arteriolar nephrosclerosis.

**Dr. Proudfit:** What evidence is there that the aortic insufficiency was functionally significant?

**Dr. McCormack:** Evidences that the valvular lesion was of functional significance are several. First of all, the flailing valvar margin striking the other semilunar cusps produced a moderate degree of nonspecific endocardial reaction. The 3 cusplike structures on the endocardial wall inferior to the valve are classic for a so-called "jet" lesion and are produced by an abnormal stream of blood striking the endocardium. The massive myocardial hypertrophy seems out of proportion to the amount of hypertension the patient had and is much more consistent with the cardiac size of aortic insufficiency.

**Dr. Proudfit:** It is important to recognize rupture or eversion of the aortic valve because
of the possibility of treating it effectively. Radical measures are justified because the prognosis is grave. Recently, Leonard, Harvey and Hufnagel\textsuperscript{2} reported treatment of traumatic rupture by insertion of a Hufnagel plastic valve in the aorta, distal to the origin of the left subclavian artery. Postoperatively, the diastolic blood pressure returned to normal and the patient was able to resume his usual employment. Because a direct approach to the aortic valve for repair of the defect is not possible at the present time, the Hufnagel operation should be employed until a more satisfactory surgical treatment has been devised.

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