ered a recurrence rather than a relapse, as relapses tend to occur within two to four weeks after discontinuation of therapy. Additionally, this repeat episode coincided precisely with the reuse of parenteral heroin after clinical cure had been affected. Though phage typing of the organism was not done, the serum minimal inhibitory concentration level was distinctly greater during the sixth episode, 2 mEq methicillin/ml vs 16 mEq/ml.

In summary, this case emphasizes two distinct and important points. First, recurrent infective endocarditis is most frequently encountered in parenteral drug abusers and may lead to progressive destruction of valve structures with ultimate death. This patient, indeed, required prosthetic aortic valve replacement during his third episode of endocarditis and died with multiple aortic ring abscesses and a fenestrated mitral valve. The second important point is that appropriately timed surgical intervention in patients with active infective endocarditis may not only result in a bacteriologic cure when resistant or persistent infections are present, but will prevent relentless hemodynamic compromise from continued valve destruction. This patient epitomizes the usually fatal clinical course of individuals developing recurrent infective endocarditis.

ACKNOWLEDGMENTS: The authors wish to express their appreciation to Dr. Han-Seob Kim for his help with the pathology specimen, and to Mrs. Jeannie Ford for her skillful secretarial assistance.

REFERENCES


Ruptured Aneurysm of Aortic Sinus of Valsalva into Right Ventricle*

Nobuyuki Anzai, M.D.; Manabu Yamada, M.D.; Naofumi Fujii, M.D.; Yoichiro Kasama, M.D.; and Sosuke Miyazawa, M.D.

We report a patient with ruptured aneurysm of the aortic sinus of Valsalva into the right ventricle, whose heart murmur represented only a diastolic element without aortic regurgitation. Surgery revealed a small opening through myocardium of the ventricular septum without ventricular septal defect. During systole, the opening was constricted and presumably closed with myocardial contraction, and left-to-right shunt might have occurred only in diastole. This might lead to only a diastolic murmur.

The murmur of ruptured aneurysm of the aortic sinus of Valsalva into the right ventricle is frequently continuous or to and fro. We have encountered a patient with ruptured aneurysm of the aortic sinus of Valsalva into the right ventricle, whose heart murmur was heard only during diastole without aortic regurgitation. We describe the mechanism of this unusual manifestation of heart murmur.

CASE REPORT

The patient was 21-year-old man who had been healthy until the end of March, 1975, when he noticed a heart murmur. He was referred to the Higashi Nagano National Hospital with the chief complaint of palpitation and precordial distress on May 16, 1975.

On admission, his height was 170 cm and weight 62 kg. His pulse rate was 94 beats/min and regular. His blood pressure was 128/50 mm Hg. On auscultation, a grade 2/6 diastolic murmur, loudest at the 2nd, 3rd and 4th intercostal spaces along the left sternal border, was heard (Fig 1). A thrill was also palpable at the same area. No abnormality was noted on examination of blood, urine, or tests of hepatic and renal function. The chest x-ray film showed moderately increased pulmonary blood flow, with a cardiothoracic ratio of 49 percent. The electrocardiographic findings revealed the following: regular sinus rhythm; P-R interval 0.14 sec; QRS axis +60°; no evidence of left or right ventricular hypertrophy. Right cardiac catheterization disclosed that oxygen saturation was increased in the right ventricle. The left-to-right shunt was 55 percent. During left cardiac catheterization, ventricular tachycardia occurred. Left ventricular pressure was difficult to obtain. (Table 1). Retrograde aortography revealed regurgitation of the contrast medium from the right coronary sinus into the outflow tract of the right ventricle without aortic regurgitation (Fig 2). The patient underwent surgery on May 5, 1975. An aneurysmal mass, 1 cm in diameter and 1 cm in length, was seen at the right ventricular outflow tract about 1 cm beneath the pulmonic valve. The mass was incised at the base. This resulted in a defect of the sinus wall approximately 3 mm in diameter. The opening was

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The murmur of ruptured aneurysm of aortic sinus of Valsalva into the right heart frequently shows continuous or to-and-fro motion. The character of the murmur, however, depends upon the size of the aneurysm and fistulous tract, magnitude of left-to-right shunt, site of opening, associated congenital lesions and severity of cardiac failure. Therefore, the murmur is not always continuous or to and fro. In cases of a small opening, only a systolic murmur is manifested, simulating an unruptured aneurysm in which only a systolic murmur is heard.

In the ruptured aortic aneurysm of the sinus of Valsalva into the right heart, the continuous murmur is frequently accentuated in the diastolic element. This diastolic accentuation is similar to that of a coronary A-V fistula, in which coronary blood flow is augmented during diastole and heart murmur is accentuated in diastole.

Anatomic and hemodynamic similarity exists between a coronary artery, a coronary A-V fistula and a ruptured aortic aneurysm of sinus of Valsalva into the right ventricle. Anatomically, they originate in the aortic sinuses and terminate in the right heart. Hemodynamically, the pressure in the aortic sinuses is maximal during diastole, and during systole it is disproportionately decreased by Venturi effect.

Figure 1. Phonocardiogram demonstrating only diastolic element without systolic element.

Figure 2. Lateral view of retrograde aortogram demonstrating regurgitation of contrast media from the right coronary sinus into the outflow tract of the right ventricle without aortic regurgitation.

**DISCUSSION**

**Table 1—Data from Intracardiac Catheterisation**

<table>
<thead>
<tr>
<th>Position</th>
<th>Maximum/Minimum pressure, mm Hg</th>
<th>Oxygen Saturation, percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>24/16 (16)</td>
<td>69.7</td>
</tr>
<tr>
<td>Inferior vena cava</td>
<td>20/11 (15)</td>
<td>68.4</td>
</tr>
<tr>
<td>Right atrium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Upper</td>
<td>20/8 (16)</td>
<td>70.0</td>
</tr>
<tr>
<td>Middle</td>
<td>22/9 (15)</td>
<td>66.4</td>
</tr>
<tr>
<td>Lower</td>
<td>22/8 (15)</td>
<td>68.4</td>
</tr>
<tr>
<td>Right ventricle</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Outflow</td>
<td>50/5 (30)</td>
<td>85.8</td>
</tr>
<tr>
<td>Apex</td>
<td>60/0 (30)</td>
<td>96.5</td>
</tr>
<tr>
<td>Inflow</td>
<td>60/5 (30)</td>
<td>85.8</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wedge</td>
<td>14/7 (9)</td>
<td>89.7</td>
</tr>
<tr>
<td>Branch</td>
<td>40/20 (32)</td>
<td>84.5</td>
</tr>
<tr>
<td>Main</td>
<td>60/20 (36)</td>
<td>83.0</td>
</tr>
<tr>
<td>Aorta</td>
<td>96/50 (60)</td>
<td>96.5</td>
</tr>
</tbody>
</table>

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However, both a coronary artery and a coronary A-V fistula must traverse the myocardium and the factor of myocardial relaxation, which plays an important role in augmenting coronary blood flow during diastole, is present. The myocardial relaxation factor, however, is not present in the ruptured aneurysm of the aortic sinus of Valsalva into the right heart. However, if the aneurysmal opening is within the right ventricle, a mechanism similar to the myocardial relaxation factor might be a result of the decrease in intraventricular pressure during diastole. In hemodynamics, the analogy to a coronary artery and a coronary A-V fistula is applied to a ruptured aneurysm of the aortic sinus of Valsalva into the right ventricle.\textsuperscript{1,5}

Therefore, in the ruptured aneurysm of the aortic sinus of Valsalva into the right ventricle, the pressure gradient between the opening of the right ventricle and aortic sinuses is maximal during diastole and minimal during systole. Under these circumstances, maximal flow of left-to-right shunt through the opening would be during diastole and contributed to diastolic accentuation of continuous murmur. This accentuation correlated with the cineangiogram which demonstrated that most of the flow was in diastole.\textsuperscript{6} This case showed only a diastolic murmur without aortic regurgitation. In this case, since the opening was small without a ventricular septal defect and was through the myocardium of the ventricular septum, during systole the opening might be constricted and presumably closed with myocardial contraction. The pressure gradient between the aortic sinuses and right ventricle might disappear in systole and the left-to-right shunt did not occur during systole through opening. Therefore, the blood flow through the opening was only during diastole and contributed only to a diastolic murmur. Furthermore, in this case, ventricular septal defect and aneurysmal obstruction of right ventricular outflow tract, which lead to systolic murmur, were not seen.\textsuperscript{6,6} Thus, this patient demonstrated only a diastolic murmur without aortic regurgitation.

REFERENCES

3 Lin TK, Crockett JE, Dimond EG: Ruptured congenital aneurysm of the sinus of Valsalva. Am Heart J 51:446, 1956
8 Wiggers CJ: Physiology in Health and Disease (ed 5).

Acute Respiratory Failure following Severe Arsenic Poisoning*

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A 47-year-old man had an episode of severe respiratory failure after acute intoxication with arsenic. Features of the initial clinical presentation included nausea, vomiting, and diarrhea, acute psychosis, diffuse skin rash, and marked pancytopenia. A peripheral neuropathy then developed which resulted in severe weakness of all muscles of the limbs, the shoulder and pelvic girdles, and the trunk. The neuropathy continued to progress despite treatment with dimercaprol (BAL in oil). Five weeks after the initial exposure, the patient was no longer able to maintain adequate ventilation and required mechanical ventilatory support. Improvement in the patient's neuro muscular status permitted successful weaning from the ventilator after one month of mechanical ventilation. Long-term follow-up revealed no further respiratory difficulty and slow improvement in the strength of the peripheral muscles.

The clinical features of arsenic poisoning have been well described and include a peripheral neuropathy which may be severe.\textsuperscript{4} Although respiratory failure may complicate a wide range of neuropathic processes, including the Guillain-Barre syndrome, polymyelitis, and acute intermittent porphyria, there are no detailed reports of respiratory failure as a sequel of acute arsenic intoxication.

We report a case of respiratory failure due to extreme weakness of the respiratory muscles in a patient who survived an acute intoxication with arsenic. The report details the time course of this complication of arsenic poisoning and documents the eventual reversibility of the respiratory failure.

CASE REPORTS

A 47-year-old farmer was admitted to a local hospital with nausea, vomiting, and diarrhea. During the fourth hospital day, mental changes consisting of delusions and hallucinations developed, and the patient was referred to the Minneapolis Veterans Administration Medical Center.

The patient, at the time of transfer, was unable to give any coherent history. An interview with the patient's wife established that he was a farmer and had exposure to several kinds of insecticides. He used no drugs and was not a heavy drinker of alcohol.

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