Remission of Symptoms in Mitral Stenosis Due to Complete Heart Block*

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A patient with severe mitral stenosis, after many years of severe disability due to heart failure, noted spontaneous improvement in symptoms. He subsequently developed Stokes-Adams attacks and was found to have complete heart block. He underwent cardiac catheterization and subsequently had a mitral valve replacement and permanent pacemaker implantation. Hemodynamic studies at the time of catheterization supported the clinical impression that his slow ventricular rate was beneficial in the presence of his severe mitral stenosis.

It comes as a surprise when a patient with serious cardiac failure reports abrupt and striking improvement in his condition, unrelated to any change in therapy. We recently investigated such a patient and his findings form the basis of this report.

CASE REPORT

This 60-year-old Caucasian man presented in 1955 to his physician with dyspnea on exertion, abdominal discomfort, flatulence, ankle edema and chest pain. He was in sinus rhythm with gross right heart failure. His venous pressure was markedly elevated; his legs grossly swollen and he had a 5 cm tender, pulsating liver. Auscultation revealed signs of mixed mitral valve disease with dominant stenosis. Tricuspid murmurs were not detected. Digitalis and diuretic therapy resulted in limited improvement only. Atrial fibrillation is known to have been present on electrocardiogram in 1981 but his ventricular rate was poorly controlled at approximately 120 per minute on crystalline digitalis glycoside (Cedilanid) 0.25 mg four times daily.

He was lost sight of until 1969, when, after 14 years of digitalis and diuretic therapy with only partial relief of his symptoms, he noted spontaneous, sudden and marked improvement in both his dyspnea and his edema. Accordingly, he stopped all therapy and returned to his work as a blockman in a butchery. He volunteered that the vigorous pulsation in his neck stopped at the time of this remission in symptoms.

In July 1970, after a year of relative improvement, he presented with syncopal attacks. Physical examination showed him to have a very slow pulse, no evidence of heart failure, and murmurs of mixed mitral valve disease. ECG showed atrial fibrillation, complete heart block, and a regular idioventricular rate of 44 per minute. X-ray films showed a large heart with a prominent left atrium and dense mitral valve calcification.

A temporary transvenous pacemaker was passed into the right ventricle at the time of cardiac catheterization. Pulmonary artery pressures and cardiac outputs were measured at several artificially paced heart rates (Fig 1). The pressure and saturation findings are listed in Table 1. Cardiac output varied between 2.5 and 3.1 liters per minute at heart rates of 45 to 75 per minute. Mitral valve orifice area was calculated to be 0.68 sq cm and no mitral incompetence was demonstrable on left ventricular angiography. At surgery the mitral valve leaflets were found to be extensively calcified, with calcium extending into the septum. The valve was excised and replaced with a No. 4 Starr-Edwards prosthesis and a permanent pacemaker was implanted at the time of thoracotomy. His postoperative course was uneventful and he has returned to work as a blockman with grade 1 disability (NYHA) at present.

Table 1—Cardiac Catheterisation Data

<table>
<thead>
<tr>
<th>Catheter Position</th>
<th>Oxygen Saturation (%)</th>
<th>Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>RA</td>
<td>—</td>
<td>cv=9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>y =6</td>
</tr>
<tr>
<td>RV</td>
<td>—</td>
<td>80/8</td>
</tr>
<tr>
<td>MPA</td>
<td>68%</td>
<td>76/29(41) to 90/46</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(42) at heart rates of 45 to 93/min</td>
</tr>
<tr>
<td>PWP</td>
<td>—</td>
<td>cv=30 − 38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>y = 24 − 30</td>
</tr>
<tr>
<td>LV</td>
<td>—</td>
<td>140/8</td>
</tr>
<tr>
<td>Aorta</td>
<td>95%</td>
<td>140/50</td>
</tr>
</tbody>
</table>

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Figures in parenthesis refer to mean pressures.

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In retrospect, this patient probably never had significant mitral incompetence. The systolic murmur was probably due to functional tricuspid incompetence resulting from right ventricular dilatation caused by mitral stenosis with secondary pulmonary hypertension. The disappearance of the pulsation in his neck which coincided with the sudden clinical improvement is thought to have been caused by a sudden reduction in tricuspid incompetence, secondary to a fall in mean pulmonary artery pressure at the time of the presumed onset of complete heart block, approximately a year prior to surgery. The effect of heart rate on mean pulmonary artery pressure and cardiac output at catheterization would seem to support this deduction, showing a rising cardiac output and a progressive decline in the abnormally raised mean pulmonary artery pressure with reduction in artificially paced heart rate (Fig 1). This contrasts sharply with the behavior of the cardiac output and the mean pulmonary artery pressure in complete heart block in persons with otherwise normal hearts or in those with abnormal myocardial function.

Tachycardia always reduces the time available in diastole for ventricular filling, and in mitral stenosis this compromises flow across the narrowed mitral valve with the result that pulmonary venous and secondary pulmonary arterial hypertension may result. This accounts, therefore, for the clinical deterioration which often results when a patient with mitral stenosis develops tachycardia, and the improvement which follows when the ventricular rate is slowed by digitalis in the presence of atrial fibrillation.

This patient illustrates the result of the unusual supervision of complete heart block with resultant bradycardia in a man with severe mitral stenosis and poorly controlled atrial fibrillation. Slowing of his ventricular rate is thought to have accounted for his dramatic symptomatic improvement and the findings at different heart rates at the time of cardiac catheterization seem to support this interpretation. It may also be argued that implantation of a pacemaker at the usual preset rate of approximately 70 per minute may worsen the condition of a patient with bradycardia due to complete heart block and unrelieved severe mitral stenosis. Such patients should not be paced at conventional heart rates without also undergoing surgery to relieve the mitral stenosis, or unless hemodynamic studies at the proposed pacemaker rate can be shown not to result in further elevation in left atrial and pulmonary artery pressures, and fall in cardiac output.

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REFERENCES

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Pulmonary Lesions in Rheumatoid Arthritis

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A solitary pulmonary nodule was discovered on roentgenographic examination in a 56-year-old man with rheumatoid arthritis of recent onset. At thoracotomy a left parahilar mass with histologic features of a necrobiotic rheumatoid nodule was found. Although rare, solitary nodules of the lung have been found but the distinction from carcinoma could not be established without removal of the lesion.

The roentgenographic discovery of pulmonary nodules accompanied by pleural effusion raises the possibility of carcinoma. Infrequently rheumatoid arthritis may be associated with such lesions. The eventual demonstration of the nature of a lung mass as a classic rheumatoid nodule prompted the following report.

CASE REPORT

A 56-year-old Caucasian man was admitted to the Naval Hospital, Philadelphia in March 1968 with a five month history of progressive painful swelling of the ankles, knees and proximal interphalangeal joints. Morning stiffness, a ten pound weight loss, inability to grasp objects and the appearance of painless nodules below the elbows prompted admission. Three years earlier the patient was admitted to the same hospital for iron deficiency anemia but no source of blood loss was found. The patient smoked over one pack of cigarettes per day for 30 years.

Physical Examination: Positive physical findings were confined to the extremities and chest. Several firm, nontender nodules were palpable along the extensor surfaces of both forearms. There was limitation of extension of both elbows and bilateral dorsal subluxation of the distal ulnae. The dorsal surfaces of the hands were concave with fusiform swelling of the third, fourth, and fifth fingers. Morning stiffness was present in the right hand and proximal interphalangeal and metacarpophalangeal joints. Thoracic motion was not limited but dullness to percussion, absent breath sounds and bronchophony were found over the posterior inferior portion of the left hemithorax. The remainder of the physical examination was unremarkable.

Laboratory Studies: The hematocrit was 39 vol percent; WBC 15,600/mm³. The peripheral smear showed slight hypochromia and targetting. The Westergren sedimentation rate was 55 mm per hour. A test with intermediate strength PPD showed 6 mm induration at 48 hours and the coccidiodin skin test 12 mm induration. The latex fixation test was negative.

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The opinions expressed herein are those of the authors and cannot be construed as reflecting the views of the Navy Department or of the Naval Service at large.

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