tracheostomy, affect our subject's response to hypoxic and hypercapnic gas mixtures; however, he was able to "reset" his respiratory center and normalize his carbon dioxide tension ($P_{CO_2}$) in a few days, with the value dropping from 75 mm Hg before respiratory arrest to 52 mm Hg after intubation to 38 mm Hg five days later. A $P_{CO_2}$ of 40 mm Hg is the upper limit of normal for our laboratory. The arterial bicarbonate level was 41 mEq/L at the time of intubation and fell into the normal range over a five-day period. Schwartz et al demonstrated that dogs chronically exposed to carbon dioxide have elevated bicarbonate levels and that upon removal from the carbon dioxide environment, the plasma bicarbonate concentration falls from a level in the high 30s to the normal range in one day, provided chloride ion is fed to the animals. Our patient was on a normal diet with no salt restrictions. We did not measure tidal volume (TV) prior to intubation, but upon intubation with the subject asleep, the TV was 200 ml and over a two-day period rose to 500 ml. The respiratory rate also increased, and the patient no longer was aware of having to work at breathing.

The maximal expiratory flow-volume curve (Fig 1) demonstrated inspiratory and expiratory airflow obstruction. Following tracheostomy, this improved, but the Kistner tube had sufficient resistance to demonstrate an inspiratory and expiratory plateau on a follow-up maximal expiratory flow-volume curve, but the limitation of flow was mild. A flap valve, which permitted speech, added sufficient resistance to affect arterial blood gas values during exercise. Should the patient return for further follow-up, another form of permanent tracheostomy and valve will be tried.

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**Progressive Extreme Bialtrial Enlargement following Mitral Valve Replacement**

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Patients with mitral valve disease and extreme enlargement of the left atrium usually exhibit significant decrease in chamber size following corrective mitral valve surgery. We describe a patient in whom extreme right and left atrial enlargement developed, and progressed following mitral valve replacement, with no evidence of prosthetic valve malfunction or tricuspid valve disease.

Extreme enlargement of the left atrium, or "giant left atrium," is relatively uncommon in patients with rheumatic mitral valve disease, with a reported incidence of 0.3 percent. Radiographic criteria for giant left atrium are based on the plain chest film, with the left atrium forming the right border of the cardiac silhouette and extending to the right rib cage. Because of an overall decrease in the incidence of rheumatic fever and the employment of early corrective surgery for symptomatic rheumatic valvular disease patients, such extreme left atrial chamber enlargement is rarely detected today. The vast majority of patients with a giant left atrium show a marked decrease in chamber size, often to near normal, following corrective mitral valve surgery. We recently encountered a patient with extreme bialtrial enlargement, which progressed following mitral valve replacement.

**CASE REPORT**

A 65-year-old woman had mitral commissurotomy for mitral stenosis in the mid 1950s. The mitral valve was replaced for recurrent mitral stenosis and mitral regurgitation with a Starr-Edwards prosthesis, model No. 6120, size 3M, in 1973. Following mitral valve replacement, she was asymptomatic for six years, and then began to experience fatigue. In December, 1981, she was hospitalized following a syncopal episode. Recurrent episodes of ventricular tachycardia were identified and subsequently found to be secondary to quinidine.

On physical examination on admission, blood pressure was 120/80 mm Hg, and the heart rate 84/minute; jugular venous distention, 8-10 cm above the sternal angle, was present. Dullness to percussion and bronchial breath sounds were detected at the right lung base. On cardiac auscultation, opening and closing sounds of the mitral prosthesis were normal; a grade 2/6 high frequency early diastolic...
The right border of the cardiac silhouette moved closer to the right rib cage between 1975 and 1981.

The electrocardiogram showed sinus rhythm with first degree AV block, no P wave abnormality, and left ventricular hypertrophy. Telemetry monitoring following admission revealed evidence of recurrent episodes of sinus arrest, unrelated to quinidine, for which a permanent transvenous ventricular pacemaker was eventually implanted. Chest x-ray films during hospitalization showed cardiomegaly with no evidence of pulmonary vascular congestion; a large opacity occupying the right lower lung field was noted, and initially was thought to represent either pleural effusion, pulmonary consolidation, or elevation of the right hemidiaphragm. Figure 1 shows chest x-ray films from 1975 (two years after valve replacement) and 1981 (present hospitalization). Between 1975 and 1981, the right border of the cardiac silhouette moved closer to the right rib cage.

Echocardiogram revealed that the left atrium was markedly dilated, at 11.5 cm; the left ventricle was dilated, with a normal fractional shortening; no prosthetic valve abnormalities were seen. There was no pericardial thickening and no pericardial effusion.

Despite the absence of a significant mitral murmur, cardiac catheterization was performed to definitively evaluate prosthetic valve function, because of the patient's fatigue and evidence of extreme left atrial enlargement. Hemodynamic data are shown in Table 1. Marked pulmonary hypertension was present, with moder-
Table 1—Hemodynamic Data

<table>
<thead>
<tr>
<th>Heart rate</th>
<th>90</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure (mm Hg)</td>
<td></td>
</tr>
<tr>
<td>Right atrium</td>
<td>14*</td>
</tr>
<tr>
<td>Right ventricle</td>
<td></td>
</tr>
<tr>
<td>systolic</td>
<td>56</td>
</tr>
<tr>
<td>filling</td>
<td>11*</td>
</tr>
<tr>
<td>end-diastolic</td>
<td>16</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td></td>
</tr>
<tr>
<td>systolic</td>
<td>53</td>
</tr>
<tr>
<td>diastolic</td>
<td>25</td>
</tr>
<tr>
<td>Pulmonary capillary wedge</td>
<td>19</td>
</tr>
<tr>
<td>Left ventricle</td>
<td></td>
</tr>
<tr>
<td>systolic</td>
<td>190</td>
</tr>
<tr>
<td>filling</td>
<td>11</td>
</tr>
<tr>
<td>end-diastolic</td>
<td>19</td>
</tr>
<tr>
<td>Resistance (dynes<em>sec</em>cm⁻²)</td>
<td></td>
</tr>
<tr>
<td>Total systemic</td>
<td>1856</td>
</tr>
<tr>
<td>Total pulmonary</td>
<td>649</td>
</tr>
<tr>
<td>Pulmonary vascular</td>
<td>296</td>
</tr>
</tbody>
</table>

Prosthetic mitral area gradient: 8 mm Hg
Cardiac index: 2.8 liters/min/m²
Calculated prosthetic mitral valve area: 2.36 cm²

*Right atrial and right ventricular filling pressures were identical on pull-through.

ate elevation of the pulmonary capillary wedge pressure; total pulmonary resistance and systemic vascular resistance values were significantly elevated. Significant V waves were not present in the right atrial or pulmonary capillary wedge pressure tracings. The prosthetic mitral diastolic area gradient was 8 mm Hg, with a calculated prosthetic valve area of 2.36 cm².

On left ventricular angiography, the left ventricle was dilated, with minimal mitral regurgitation; overall left ventricular ejection fraction was slightly diminished at 50 percent. The prosthetic valve poppet movement was normal. Right ventricular angiogram showed a dilated right ventricle, with no evidence of tricuspid regurgitation. Right atrial angiogram (Fig 2) showed a markedly dilated right atrium, with delayed films showing an even larger left atrium extending to the right rib cage beyond the outline of the right atrium; both atria occupied a large area of the lower right hemithorax corresponding to the opacity seen on plain chest films. The aortic root angiogram showed grade 1-2 aortic regurgitation. In January, 1983, the patient expired following cardiac arrest. Post-mortem examination confirmed marked enlargement of the left and right atria (Fig 3).

**Discussion**

Giant left atrium has been noted to occur with rheumatic mitral valve disease; mitral regurgitation is usually the predominant valve lesion.1,2 Extreme right atrial enlargement is almost always due to tricuspid regurgitation,1 and occasionally to tricuspid stenosis.4 We describe a patient in whom extreme enlargement of both atria developed, and progressed after mitral valve replacement, with no evidence of significant prosthetic valve malfunction, and no evidence of tricuspid stenosis or regurgitation.

The pulmonary capillary wedge (and by inference left atrial) pressure elevation in our patient was related mostly to the presence of a prosthetic mitral valve; the prosthetic valve gradient of 8 mm Hg was slightly higher than the expected gradient for the model 6120, size 3M Starr-Edwards mitral prosthesis.3 The severe pulmonary hypertension was due to both an elevated pulmonary capillary wedge pressure and an elevated pulmonary vascular resistance. The elevated pulmonary vascular resistance had apparently persisted following mitral valve replacement. Right ventricular failure and right atrial pressure elevation resulted from the pulmonary hypertension. Thus, the presence of a prosthetic mitral valve with intrinsic obstruction to flow, with secondary left and right atrial pressure elevations, could have played an important role in the progressive biatrial enlargement.

Although extreme atrial enlargement is relatively uncommon, awareness of this entity is of considerable clinical importance, particularly in view of the potential for diagnostic confusion. Patients with extreme atrial enlargement have undergone attempted thoracocentesis for suspected pleural effusion with disastrous consequences,1 exploratory thoracotomy for suspected mediastinal tumor,7 and attempted pericardiocentesis for suspected pericardial effusion.7 Although right-sided pleural effusion was initially suspected in our patient, thoracocentesis was avoided after decubitus chest films (showing no "layering out" of fluid) and an echocardiogram (showing marked left atrial enlargement) provided clues that the right lower lung field opacity did not represent pleural effusion. With the widespread use of echocardiography, serious mistakes in differential diagnosis are unlikely today. If, however, noninvasive tests are inconclusive, cardiac catheterization and angiography should be performed for diagnostic clarification.

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