ing in the middle third of the trachea with direct extension into the esophagus, the mediastinum, and the hilar nodes (Fig 2).

**DISCUSSION**

Tracheal carcinomas are notoriously silent tumors and so are not diagnosed until late in the course, often after a 10-month to 15-month delay from their initial symptoms. It has been estimated that for every tracheal tumor, there are 75 laryngeal tumors and 180 lung tumors, but the reason for this differential incidence is not known.

Tracheal cancer is described as occurring in one of three cell types. The squamous cell variety is the most common, followed by the adenoid cystic and the mucous-secreting cell types. Fifty percent of tracheal tumors are found in the lower third of the trachea, 33 percent in the upper third, and only 17 percent in the middle third of the trachea. Cough, hemoptysis, and wheezing are the most common initial symptoms, and many patients have a history of being treated for asthma for some time with little success. The prognosis of tracheal cancers depend upon the cell type and the size of the lesion. Those patients with either squamous cell or mucus-secreting cell carcinomas that are large in size have a poor prognosis.

In 1973, Miller and Hyatt described the technique of evaluating obstruction of the upper airway by using flow-volume loops. It has recently been shown that by using maximal expiratory flow-volume loops obtained with the patient breathing a mixture of 80 percent helium and 20 percent oxygen, one can diagnose obstruction of the upper airway in the presence of diffuse airway obstruction. Helium, being a less dense gas than air, will not alter flow in the peripheral airway where the flow regime is laminar but will increase turbulent flow. At higher lung volumes in the presence of upper airway obstruction, the flow regime is turbulent, and so breathing a helium-oxygen mixture would be expected to improve the flow rates over this part of the curve. This improvement in flow rates at larger lung volumes caused by breathing a helium-oxygen mixture might be used, therefore, to diagnose upper airway obstruction even when the flow-volume loop obtained with the subject breathing air is typical of more diffuse airway obstruction.

**REFERENCES**


**Echocardiography in Lutembacher’s Syndrome**

LTC Joseph A. Quash, MC, USA; LTC James E. Davia, MC, USA; COL Carlos M. de Castro, MC, USA; LTC Thomas E. Bowen, MC, USA; and COL David C. Green, MC, USA

The findings in a patient with surgically proven Lutembacher's syndrome (coexisting mitral stenosis and atrial septal defect) are presented. In addition to a typical pattern of mitral stenosis, the preoperative echocardiogram revealed paradoxical septal motion, thereby providing, prior to cardiac catheterization, a basis for the suspicion of an associated lesion due to diastolic overload of the right ventricle.

*From the Cardiology and Thoracic Surgery Services, Walter Reed Army Medical Center, Washington, DC.
The coexistence of an atrial septal defect and mitral stenosis is known as Lutembacher's syndrome. While appearing in the average clinical practice with noteworthy infrequency, it is essential that the correct diagnosis be established in order that appropriate surgical correction of both components of the syndrome may be accomplished. Herein is presented a case of Lutembacher's syndrome in which the echocardiographic examination was a useful procedure in establishing the diagnosis.

**Case Report**

The patient, a 26-year-old white woman, was referred to Walter Reed Army Medical Center in April 1975 for a closed mitral commissurotomy. There was no previous history of rheumatic fever. She was first told of a heart murmur at the age of 16 years. The patient had no cardiac symptoms until approximately two years prior to this admission, when she noted progressive fatigue, shortness of breath, and dyspnea on exertion. There was no previous history of bacterial endocarditis or systemic illness. There was no history of pain in the chest, orthopnea, or nocturnal dyspnea.

The patient weighed 58 kg (128 lb), and her height was 168 cm (5 ft 6 in). Her blood pressure was 110/70 mm Hg, and her pulse rate was 84 beats per minute and regular. A grade 2/6 systolic ejection murmur was heard in the third left intercostal space. A grade 2/6 diastolic rumble with presystolic accentuation was heard at the apex but became louder at the lower left sternal border. No other murmurs were present.

The electrocardiogram was suggestive of bialtrial enlargement but was otherwise within normal limits. The chest x-ray film revealed straightening of the left cardiac border but no other evidence for left atrial enlargement. The pulmonary fields were plethoric, and the main pulmonary artery was slightly prominent. The overall cardiac size was within normal limits, except for retrosternal fullness in the lateral projection. Cardiac catheterization revealed a 2.7:1 left-to-right shunt at the atrial level and an elevated pulmonary arterial wedge pressure (Table 1). An echocardiogram demonstrated mitral stenosis and paradoxical septal motion (Fig 1). The right ventricular dimension was 1.84 cm/sq m, which is above the normal range of 0.5 to 1.2 cm/sq m.

**Table 1—Data from Cardiac Catheterisation**

<table>
<thead>
<tr>
<th>Location</th>
<th>Percent</th>
<th>Oxygen Satura-</th>
<th>Pressure, mm Hg*</th>
<th>Exercise Pressure, mm Hg*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior vena cava</td>
<td>68</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Right atrium</td>
<td>85</td>
<td>a = 4; v = 2 (3)</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>87</td>
<td>36/4</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>85</td>
<td>24/8 (16)</td>
<td>55/24</td>
<td>...</td>
</tr>
<tr>
<td>Pulmonary arterial wedge</td>
<td>...</td>
<td>(8)</td>
<td>(26)</td>
<td>...</td>
</tr>
<tr>
<td>Left atrium</td>
<td>95</td>
<td>a = 14; v = 8 (7)</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>95</td>
<td>110/5</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

*Numbers within parentheses are means.

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**Discussion**

In a patient with the physical findings of mitral stenosis, a coexisting atrial septal defect should be suspected if a systolic ejection murmur is present at the upper left sternal border together with radiologic evidence of increased pulmonary vascularity. The combination of these two cardiac abnormalities, known as Lutembacher's syndrome, may be difficult to diagnose because
the hemodynamic predominance of one lesion may mask the clinical findings of the other lesion.\(^2\)

The physical findings in Lutembacher’s syndrome are characterized by variability from case to case. The most constant features are a systolic ejection murmur at the upper left sternal border, a loud first heart sound, and fixed splitting of the second heart sound.\(^1\)\(^-\)\(^5\) Virtually all cases will have a diastolic rumble at the apex, but presystolic accentuation of this murmur and an opening snap are much less common.\(^1\)\(^-\)\(^4\) Failure to find all of the classic physical findings of mitral stenosis may be due to the decompressing effect of the atrial septal defect on the left atrium.\(^5\)

The radiologic findings are more characteristic of an atrial septal defect than of mitral stenosis in that enlargement of the pulmonary artery, right atrium, and right ventricle are common, whereas left atrial enlargement and evidence of pulmonary venous hypertension are unusual.\(^1\)\(^-\)\(^4\) The vascular pattern is rather that of pulmonary plethora. The most characteristic electrocardiographic finding is biatrial enlargement.\(^1\) In addition, a QR or rSR' pattern in lead V\(_1\) is frequently found.

Although the physical findings, chest x-ray film, and ECCs suggested the presence of Lutembacher’s syndrome in our patient, the echocardiogram was the most useful noninvasive diagnostic procedure in establishing the diagnosis. Mitral stenosis was diagnosed on the basis of a significantly reduced E-F slope in the presence of diastolic anterior movement of the posterior leaflet of the mitral valve\(^7\) (Fig 1). Paradoxic septal motion, which is not found in isolated mitral stenosis, provided a clue for the presence of a coexisting atrial septal defect;\(^6\) however, it must be emphasized that the paradoxic septal motion is not diagnostic of an atrial septal defect, since it may be found in other lesions due to diastolic overload, as well as in a variety of other situations.\(^6\) Mitral stenosis with pulmonary hypertension and secondary tricuspid regurgitation also might produce paradoxic septal motion, but the normal pulmonary arterial pressure and the absence of large V waves in the tracing of the right atrial pressure render this possibility highly unlikely in our patient. We are unaware of other reports of Lutembacher’s syndrome in which the echocardiographic findings have been described.

The echocardiogram in our case illustrates that paradoxic septal motion in the presence of a pattern for mitral stenosis should provide a clue to the presence of Lutembacher’s syndrome and should direct attention toward the detection of an atrial septal defect at cardiac catheterization. Since the echocardiographic examination is simple, easy to perform, and noninvasive, we recommend that it should be a routine part of the evaluation in patients with the physical findings of mitral stenosis or atrial septal defect.

**References**


**Tuberculous Pneumonia with the Syndrome of Inappropriate Secretion of Antidiuretic Hormone***

**Cause of the Adult Respiratory Distress Syndrome**

Steven A. Sahn, M.D., F.C.C.P.,** and Kelley M. Skeff, M.D.

Bilateral tuberculous pneumonia with the syndrome of inappropriate secretion of antidiuretic hormone was the cause of the adult respiratory distress syndrome in an elderly patient. Early recognition and prompt therapy enabled the patient to make a complete recovery without the necessity for mechanical ventilation. With the shift of care of tuberculous patients out of the sanatorium, the practicing physician should be aware of the varied manifestations of tuberculosis.

The adult respiratory distress syndrome is a distinct clinical entity resulting from an episode of widespread pulmonary injury. It is characterized by progressive respiratory distress (dyspnea and hyperventilation), progressive reduction in pulmonary oxygen transport, progressive pulmonary infiltrates on the chest x-ray film, and a reduction in compliance and lung volumes.\(^7\) The syndrome has been associated with trauma, sepsis, shock, toxic inhalation, drug ingestion, aspiration, pancreatitis,