tricuspid area restraint seems to be an effective and sufficient cause to account for the observed diastolic murmur.*

With regard to the systolic murmur, it is to be noted that the tumoral mass remained in the right ventricle for no less than the initial two thirds of the systole, thus allowing the tricuspid valve closure to be incomplete and to cause tricuspid incompetence which in turn would be the main origin of the observed systolic murmur. At the end of systole, as the tumor had been completely lodged in the right atrium, the systolic murmur consequently ceased (Fig 1).

We want to emphasize the conspicuous functional disturbance capable of causing such a high level sound energy dissipation as observed in this asymptomatic patient, having its origin in a loosely attached and highly mobile, pediculated, right atrial metastatic tumoral mass. Thus, the unusual feature in this case is the presence of such intense, harsh, systolic and diastolic murmurs all over the precordium, originating on a tumoral mass loosely attached to a low pressure cardiac chamber, a finding not previously reported in the medical literature available to the authors.

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Spontaneous Pulmonary Torsion*

Robert M. Shorr, M.D.; and Aurelio Rodrigues, M.D.

A patient with systemic manifestations of sepsis after above-knee amputation developed massive left pulmonary atelectasis and pneumonia. These persisted despite repeated bronchosopies. At thoracotomy, 180° torsion of the left lung was found. The patient improved temporarily but died later of sepsis, probably related to severe hip osteomyelitis.

Torsion of the lung is a rare, potentially lethal event that has been recognized and sporadically reported in the literature. Predisposing factors include trauma,¹ accessory pulmonary lobe,² diaphragmatic hernia,³ thoracic surgical procedures,⁴ pneumothorax, and obstructing bronchial neoplasms. We present the first case of spontaneous com-

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Figure 1. Admission chest x-ray film showing mild left lower lobe atelectasis.

Figure 2. Preoperative chest x-ray film showing massive left atelectasis of upper and lower lobes with shift of mediastinum toward the left.
Table 1—Clinical Findings—Arterial Blood Gas Measurements

<table>
<thead>
<tr>
<th></th>
<th>Preoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIO₂</td>
<td>.60</td>
<td>.50</td>
</tr>
<tr>
<td>PO₂</td>
<td>88 mm Hg</td>
<td>115 mm Hg</td>
</tr>
<tr>
<td>PCO₂</td>
<td>36 mm Hg</td>
<td>40 mm Hg</td>
</tr>
<tr>
<td>pH</td>
<td>7.43</td>
<td>7.37</td>
</tr>
<tr>
<td>Shunt</td>
<td>35%</td>
<td>20%</td>
</tr>
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Over the next several weeks, the patient developed persistent left pulmonary atelectasis and pneumonia that were not responsive to repeated bronchoscopy, high-frequency ventilation, and independent lung ventilation. Bronchoscopy at this time revealed persistent narrowing of the left mainstem bronchi. The patient was taken to the operating room for an exploratory thoracotomy. A complete 180° torsion of the entire left lung was found, as well as massive necrotizing pneumonia of the upper lobe and part of the lower lobe. Because of these severe findings and technical difficulties, left pneumonectomy was performed. The patient initially recovered from this procedure, and oxygenation as well as pulmonary shunt improved (Table 1). However, sepsis from a presumed source of osteomyelitis of the hip continued, and the patient died on the eighth postoperative day.

**DISCUSSION**

Torsion of the lung is a rare phenomenon with lethal complications if unrecognized. Sporadic reports of such events following trauma³ and thoracic surgical procedures⁴-⁷ have been published over the past 50 years. The etiology of pulmonary torsion in certain clinical settings can be easily explained. In torsion associated with trauma, Daughtry⁴ postulated that severe deceleration injuries tear the thin inferior pulmonary ligament, with subsequent rotation of injured lobes. Iatrogenic torsions have also been reported, with an etiology of unrecognized intraoperative lobar rotation by retraction. When irreversible ischemia is recognized early, it can be prevented and the twisted segment can be surgically rotated to its normal anatomic position.

Most cases of pulmonary torsion include scenarios of unremitting pneumonia and/or atelectasis with nondiagnostic bronchoscopic findings, and seem to involve the left side more commonly; the clinical sequence of the case reported in this paper did follow this pattern. The etiology of a spontaneously twisted lung is difficult to explain. The inferior pulmonary ligament must be congenitally absent, as was the case in our present report. Another contributing factor may be a pneumatic process that creates pleural adhesions, causing rotation about the pulmonary hilum. Preoperative diagnosis in the present case failed, despite successive bronchoscopic examinations that revealed a narrowed mainstem bronchi that, in retrospect, was the torced segment of the bronchial tree. Intraoperative diagnosis was complicated by the massive necrotizing pneumonia associated with this unusual anatomic finding. In retrospect, an index of suspicion, accompanied by the bronchoscopic findings, may have led to a diagnosis of torsion or at least prompted further tests. One could postulate that a bronchogram with or without pulmonary angiograms or a perfusion scan would be diagnostic of this rare entity.

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**Percutaneous Drainage of an Infected Lung Bulla in a Patient Receiving Positive Pressure Ventilation**

Nathan C. Dean, M.D.; Mark C. Stein, M.B.B.Ch., B.Sc.; and Michael S. Stulbarg, M.D.

Percutaneous drainage of an infected bulla was performed under fluoroscopy in a patient who was receiving positive pressure ventilation. The procedure was without complications, and the patient was later weaned from mechanical ventilation.

Several reports of percutaneous catheter drainage of infected lung abscesses have appeared in recent years, but none has involved patients receiving mechanical ventilation, and none involved infected bullae.⁴ We report the catheter drainage of a bulla infected with Pseudomonas aeruginosa in a patient receiving positive pressure ventilation.

**CASE REPORT**

A 55-year-old man was admitted to the hospital because of dyspnea following a three-day history of fever, productive cough, and nonpleuritic chest pain. He had been documented to have chronic airways obstruction with recent pulmonary function tests showing a FEV₁ of 1 L and a FVC of 2.2 L. He had never received mechanical ventilation, but did have chronic dyspnea on exertion (one fourth flight of stairs), an 80 pack year smoking history, and had taken oral corticosteroids continuously for several years. Medications prior to admission were prednisone, 20 mg twice a day, albuterol, theophylline, furosemide, and tobutamide. He had undergone right thoracotomy for a benign nodule in 1977.

Physical examination revealed an obese man with a respiratory rate of 40 per minute and a temperature of 39.5°C. Mild digital

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