anatomic relationship between the esophagus and pericardium, this is at first surprising. Undoubtedly, the explanation lies in the infrequency of esophageal ulceration in achalasias.

All authors emphasize the importance of early diagnosis and surgical intervention for treatment of esophagopericardial fistula. Clinical findings highly suggestive of esophagopericardial fistula include precordial pain, fever, dyspnea and the presence of a water-wheel (bruit de moulin) murmur. However, such clinical manifestations vary and, as in our patient, may be overshadowed by major, life-threatening complications of pericardial infection. This emphasizes the central role of radiographic studies in establishing diagnosis.

Diagnosis of pneumopericardium on plain erect chest radiographic examination is not difficult provided a high index of suspicion is maintained. Two previous reports have stressed the importance of obtaining an erect view of the chest in identifying the abnormal air collection within the pericardium. In our patient also, pneumopericardium was not present on the bedside supine film of the chest prior to barium swallow study.

Once pneumopericardium is recognized, both esophagographic and esophagogoscopic studies should be performed to demonstrate possible fistulae. No barium was seen to enter the pericardium during the barium swallow study in our patient. This is not surprising as the patient was examined in a supine position, where gravity would not encourage passage into the pericardium. However, it emphasizes the need for endoscopic examination, which not only demonstrates such anterior fistulae, but may also reveal their nature. During esophagogoscopic study, it is important that the amount of air used should be limited as complications from massive air leakage via the fistula could result. These include pneumomediastinum, pneumothorax or, most significantly, increasing pneumopericardium causing cardiac tamponade.

Other etiologies of pneumopericardium include injury to the chest, cardiopulmonary surgery, lung abscess, gas-producing infection of the pericardium and fistulae with either the bronchial tree or exterior. All such entities are easily excluded by clinical history and chest roentgenographic examination.

Our case illustrates that pneumopericardium may occur in achalasia as a result of esophagopericardial fistula due to an ulcer. It should be considered in the differential diagnosis of pneumopericardium in these patients and, if a fistulous tract is not identified on swallow examination, endoscopic study should be performed.

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Endocarditis of a Tricuspid Prosthesis Causing Valvular Stenosis and Shunting Through a Patent Foramen Ovale*

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A young intravenous drug user presented with Staphylococcus aureus endocarditis involving the tricuspid valve, which was replaced with a Hancock bioprosthesis. She presented again with fever and dyspnea five months later and was found to be cyanotic. Recurrent endocarditis involving the prosthesis with right-to-left shunting through a patent foramen ovale was documented by echo and confirmed at autopsy.

Staphylococcus aureus endocarditis involving the tricuspid valve is a relatively common problem in intravenous

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drug abusers.\textsuperscript{13} Although the illness usually has an acute course, the occurrence of mortality appears to be low.\textsuperscript{13} We report a case of tricuspid endocarditis with an unusual, fatal complication.

**Case Report**

A 22-year-old woman first presented in August 1984 with fever. She had no prior medical history but had been using heroin and cocaine intravenously on a daily basis for one year. Multiple blood cultures all grew \textit{S. aureus}. An echocardiogram demonstrated a large vegetation on the tricuspid valve. Despite adequate doses of nafcillin, her course was complicated by persistent fevers, multiple pulmonary emboli, hemolytic anemia, and glomerulonephritis. A tricuspid valve replacement was performed with a Hancock porcine heterograft. Her postoperative course was uneventful, and she was discharged on Oct 16, 1984. The patient denied subsequent drug abuse. On Feb 9, 1985, she was hospitalized with fever, shortness of breath, and pleuritic chest pain. She was febrile to 38.3°C (101°F), cyanotic, and tachypneic on admission. No murmurs were heard. Arterial blood gas values revealed pH, 7.53; Pco\textsubscript{2}, 22; Po\textsubscript{2}, 37 (79 percent saturation). A repeat with 100 percent inspired oxygen was pH, 7.53; Pco\textsubscript{2}, 24; Po\textsubscript{2}, 38 (80 percent saturation). A chest x-ray film showed clear lung fields and no evidence of congestive heart failure. The patient was treated with ampicillin, nafcillin, and gentamicin and anticoagulated for possible pulmonary emboli. Subsequently, an echocardiogram showed a massive vegetation on her tricuspid valve prosthesis and a markedly enlarged right atrium. All blood cultures grew \textit{S. aureus}. She remained hypoxic despite an Fio\textsubscript{2} of 100 percent and positive end-expiratory pressure. A perfusion lung scan showed multiple segmental perfusion defects. Contrast echocardiography (Fig 1) showed a significant right-to-left shunt at the atrial level. In spite of high doses of pressor agents, she continued to deteriorate and died on Feb 13, 1985.

At autopsy, the heart weighed 380 g, with marked right atrial dilation. An intact porcine xenograft tricuspid valve was identified. Examination of the atrial aspect of the prosthetic valve revealed one small thrombus (0.3 cm) attached to a strut, but the sutures were intact, and there was no evidence of dehiscence or annular abscess. The ventricular aspect of the tricuspid valve, however, was remarkable for a large obstructive polypoid vegetation involving the entire circumference of the annulus and projecting 3.5 cm into the right ventricular cavity (Fig 2). In addition, the foramen ovale was broadly patent with a luminal circumference of 2.0 cm, and the endocardium overlying the channel was irregular and focally thickened. The other valves were unremarkable. The lungs showed extensive evidence of pulmonary congestion.

**Discussion**

This young intravenous drug abuser died from profound shock and hypoxia which resulted from endocarditis of her prosthetic tricuspid valve. The hypoxia was due to a combination of multiple pulmonary emboli (both septic and bland) and a right-to-left shunt through a patent foramen ovale.

Patent foramen ovale are commonly found at autopsy (probe patent 29 percent, pencil patent 6 percent). Significant right-to-left shunting is often due to right ventricular failure and right atrial hypertension, usually secondary to pulmonary emboli or chronic obstructive lung disease.\textsuperscript{4} In this case, the right atrial hypertension was most likely due to the pulmonary emboli and the tricuspid stenosis caused by a massive vegetation. Although hemodynamically significant valvular stenosis appears to be a rare complication in patients with endocarditis,\textsuperscript{5} it is reportedly a more frequent problem with infected porcine valve heterografts.\textsuperscript{6} However, we believe this is the first case where a large vegetation caused tricuspid stenosis, which in turn caused a significant shunt through a patent foramen ovale, as demonstrated by contrast echocardiography.

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**References**


![Figure 1. Apical four chamber view of the heart demonstrating an enlarged right atrium (RA), a right-to-left shunt into the small left atrium (LA) with contrast (arrow) appearing in the atrium and left ventricle (LV).](image1)

![Figure 2. View of the tricuspid prosthetic valve seen from the right ventricular cavity. The prosthesis is covered along the struts and valve leaflets by an obstructing polypoid infective vegetation which projects into the ventricle.](image2)
Broncholith Removal using the YAG Laser*

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An 85-year-old woman presented with a broncholith in the intermediate bronchus that could not be extracted with either flexible or rigid bronchoscopes. A YAG laser was used to fragment this broncholith so that it could be removed in pieces through a bronchoscope. Chemical composition and morphology of the broncholith were determined. Fragmentation of the large, impacted broncholith with the laser eliminated the necessity for a thoracotomy in this elderly woman.

The presence of calcium in the hilar nodes is frequently seen and often due to old tuberculosis or histoplasmosis. However, broncholithiasis and expectoration of calcified particles is not commonly encountered in clinical practice.

When broncholithiasis requires treatment, bronchoplasty or resection of the lung distal to the area of erosion is the traditional surgical approach. Surgery may be needed to control the complications of hemoptysis, cough, bronchorhea, recurrent pneumonia or abscess, and bronchiecstasy. Occasionally, freely movable particles within the bronchus can be removed through a bronchoscope.

While lasers are used for the treatment of carcinoma or stenosis which obstructs the trachea or bronchi and for control of bleeding superficial vessels, laser bronchoscopic treatment is a relatively new technique. We were aware that lasers could fragment urologic stones* and decided, therefore, to use a YAG laser to fragment a large impacted broncholith within the intermediate bronchus of an elderly patient. We subsequently studied the chemical composition and morphology of the stone before and after impact by the laser.

CASE REPORT

An 85-year-old woman presented to the emergency room with complaints of an uncontrollable cough and hemoptysis. On initial examination, she had inspiratory wheezing and was in moderate respiratory distress. Her breath sounds were diminished at the right lung base, where there were also intermittent inspiratory wheezes. Chest roentgenographic examination revealed numerous calcified lymph nodes in the right and left hilar regions (Fig 1A). A lung scan showed decreased perfusion and delayed ventilation with air trapping in the right lower lung zones. At fiberoptic bronchoscopic examination, a large, irregular, freely movable broncholith rested in the lumen of the intermediate bronchus. With retrieval instruments, we could pull it as far as the cricoid cartilage, but no further because of the normal narrowing of the airway at this point. The broncholith was therefore pushed back into the lumen of the intermediate bronchus to allow the patient to ventilate with her left lung. A rigid bronchoscope was then tried because of its more powerful biopsy forceps. Only a few small pieces of the broncholith could be chipped away. We decided to fragment the broncholith with a YAG laser, which easily broke the broncholith into pieces (Fig 2) and facilitated their removal. When the patient was reexamined one month later,

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FIGURE 1. Postero-anterior chest roentgenogram A) before, and B) after removal of broncholith.