the contents of the cyst for anaerobic and aerobic bacteria, acid-fast bacteria, and fungi were sterile.

**DISCUSSION**

This case illustrates several unusual features of the natural history of bronchogenic cysts. The fact that this was a recurrent mediastinal bronchogenic cyst argues for an aggressive approach, with total resection of these lesions whenever possible. Although unreported, we suspect that recurrent cysts have occurred previously, since Adams and Thornton\(^4\) first recommended partial excision and mucosal scarification in 1943. The exposure provided by median sternotomy in this case was excellent, as has been reported by others.\(^7,8\) We have used this approach for other complicated mediastinal and pulmonary problems (including completion pneumonectomy and closure of a bronchopleural fistula), with good success. Whenever a mass is predominantly anterior to the pulmonary hilum, we believe that the exposure provided by sternotomy is superior to lateral thoracotomy. If unilateral exposure posterior to the hilum were to become necessary, a double-lumen Roberts' or Carlsen endotracheal tube can be utilized to deflate the respective lung. During surgery, if exigencies arise that mandate cardiac decompression or simultaneous bilateral pulmonary deflation (or both), total cardiopulmonary bypass can readily be instituted.

Mediastinal bronchogenic cyst is a rare cause of the superior vena cava syndrome;\(^4,8\) this patient had incipient obstruction of the superior vena cava for a second time due to a recurrent cyst. Other benign processes that can cause superior vena cava syndrome are sclerosing mediastinal fibrosis, teratoma, thymoma, aneurysm of the ascending aorta, and vena cava thrombosis.\(^6\) It is, therefore, important that the etiology of obstruction of the superior vena cava be carefully defined prior to treating a potentially benign condition with radiotherapy or chemotherapy.

Mediastinal bronchogenic cysts usually do not communicate with the tracheobronchial tree and, therefore, rarely become infected.\(^4,8\) Such was the case in this patient; however, in 1972, Schmidt and Drapanas\(^8\) reported one adult patient who died of respiratory failure within 24 hours of admission to the hospital after an unrecognized subcarinal bronchogenic cyst became infected and compressed the right main-stem bronchus. We attribute our patient's cough, orthostatic substernal pressure, and dyspnea to her bronchial compression; but she had not exhibited hyperinflation, atelectasis, or pneumonia in the right lung. Similarly, hemoptysis, as seen in this case, is only rarely associated with mediastinal bronchogenic cysts; such is not the case with bronchogenic cysts located within the pulmonary parenchyma.

Another rare complication of bronchogenic cysts is pulmonary arterial stenosis.\(^4\) No hemodynamic tracings were obtained in this case, but a gradient may have been present across the right pulmonary artery where it was compressed. The clinical significance of this is unknown, but larger cysts can cause obstruction of the main pulmonary artery or right ventricular outflow tract.\(^7\)

Exquisitely detailed definition of the anatomic extent of this lesion was revealed by the computerized axial tomographic scans.\(^10\) The displacement and compression of the superior vena cava and right pulmonary artery, as well as the right main-stem bronchial obstruction, were well demonstrated. Further experience with this new diagnostic method will clarify its applicability in the diagnosis of intrathoracic masses.

**REFERENCES**


**Legionnaires' Disease**

**Clinical and Pulmonary Histopathologic Features of a Sporadic Case**

**Michael E. Westley, M.D.; Raymond Yesner, M.D.; and David J. Pierson, M.D., F.C.C.P.**

The pulmonary histopathologic features in a sporadic case of Legionnaires' disease are shown. The changes include acute bronchitis with focal ulceration and diffuse acute interstitial pneumonitis. These changes are not those seen with typical bacterial pneumonia but are similar to changes seen when viruses, rickettsiae, chlamydiae, or *Mycoplasma pneumoniae* organisms are the infecting agents.

*From the Department of Internal Medicine, Alaska Native Medical Center, Public Health Service, Anchorage, Alaska (Dr. Westley); the Department of Pathology, Veterans Administration Hospital, West Haven, Conn (Dr. Yesner); and the Respiratory Disease Division, Department of Medicine, University of Washington, Seattle (Dr. Pierson). The opinions expressed are not necessarily those of the Public Health Service or the Veterans Administration. Reprint requests: Dr. Westley, Alaska Native Medical Center, Anchorage, Alaska 99510*
In July 1976, a total of 180 persons associated with a convention of the American Legion in Philadelphia were hospitalized with a respiratory illness that proved fatal in 29 cases.\textsuperscript{1} Reports of pulmonary pathologic abnormalities in this infection have been confined to specimens from autopsy, with nonspecific findings possibly modified by complicating bacterial infection and prolonged administration of oxygen.\textsuperscript{2,3} We report the clinical features in a sporadic case of Legionnaires' disease, in which pulmonary tissue for pathologic examination was available prior to the use of intensive therapy with supplemental oxygen or mechanical ventilation.

CASE REPORT

A 60-year-old merchant seaman developed malaise, myalgias, chills, fever, and a nonproductive cough during an eight-day voyage from California to Alaska in January 1977. Upon completion of the voyage, he was admitted to a Kodiak hospital, where physical examination revealed an acutely ill man with a temperature of 40°C (104°F) and signs of consolidation in the left anterior portion of the chest. Over the next four days the infiltrate spread to involve the right upper lobe and most of the left lung (Fig 1), despite the administration of penicillin, ampicillin, cephalothin, and gentamicin. On the fourth day of hospitalization, the patient was transferred to the Alaska Native Medical Center in Anchorage.

At the time of transfer, the patient was lethargic and disoriented and was in respiratory distress, with a pulse rate of 100 beats per minute, a respiratory rate of 35/min, blood pressure of 190/70 mm Hg, and a temperature of 39.4°C (102.9°F). Wheezes and rales were heard throughout both pulmonary fields. The level of hemoglobin was 13 gm/100 ml. The white blood cell count was 11,000/cu mm, with 71 percent mature neutrophils and 23 percent immature neutrophils. The blood urea nitrogen level was 44 mg/100 ml, and the level of creatinine was 4.4 mg/100 ml. The urine contained 100 red blood cells (RBCs) per high power field, an occasional RBC cast, and many granular casts. Levels of arterial blood gases determined with the patient breathing room air showed a pH of 7.38, an arterial oxygen pressure (PaO\textsubscript{2}) of 46 mm Hg, and an arterial carbon dioxide tension of 28 mm Hg.

Expectorated sputum contained a few neutrophils and no predominant organisms. Aerobic and anaerobic cultures of blood, sputum, and spinal fluid were obtained. Serum was obtained for determinations of viral titers during the acute phase, and swabs of the throat and rectal swabs were submitted for viral culture.

One gram of oxacillin was given intravenously every six hours. The patient continuously received therapy with supplemental oxygen via a face mask at a rate of 6 L/min, which resulted in values for PaO\textsubscript{2} of approximately 60 mm Hg.

The patient's condition failed to improve, and on the third day of hospitalization, a limited thoracotomy was performed, with biopsy of the lingula. After surgery, therapy was begun with hydrocortisone sodium succinate (500 mg/day intravenously in divided doses), and administration of oxacillin was continued. Two months following the onset of the illness, the pulmonary infiltrate had cleared, and renal function and urinary sediment were normal.

Routine bacteriologic cultures were negative. No viruses were isolated from specimens from the throat or stool or from the pulmonary tissue obtained at biopsy. Samples of serum obtained during the acute and convalescent phases of the disease (obtained on Jan 13, 1977 and Jan 27, 1977, respectively) showed no significant rises in the titers for respiratory viruses, rickettsiae, or the organism causing Q fever. The titer for Mycoplasma pneumoniae ranged from less than 1:8 to 1:16. There was a diagnostic rise from 1:16 to 1:2,048 in the

![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21011/)

**Figure 1.** Anteroposterior chest x-ray film taken four days after admission, showing extension of left-sided consolidation and involvement of right upper lobe.

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21011/)

**Figure 2.** Specimen from pulmonary biopsy, showing focal ulcerative bronchitis, with epithelial necrosis which spares basement membrane. Bronchial lumen is filled with inflammatory exudate (hematoxylin-eosin, × 500).
focal ulcerative bronchitis and extensive interstitial pneumonitis are most likely the result of infection with the bacillus of Legionnaires' disease.

The histopathologic picture in our patient’s specimen from pulmonary biopsy differs from that which is typical of acute bacterial pneumonia and is more consistent with changes characteristically found when viruses, rickettsiae, chlamydiae (bedsoniae), or Mycoplasma pneumoniae organisms are the infecting agents. Since the histologic patterns observed in such infections are non-specific, Legionnaires’ disease may perhaps be added to the list of infections causing pulmonary interstitial infiltration with mononuclear cells; further differentiation among the diseases in this group by histopathologic examination is at present not reliable.

**REFERENCES**


**Treatment of Pulmonary Melioidosis with Combination of Trimethoprim and Sulfamethoxazole**


Treatment with a combination of trimethoprim and sulfamethoxazole proved lifesaving in a patient with pulmonary melioidosis after therapeutic failure occurred with other antibiotics to which the organisms were sensitive in vitro. Antagonistic interaction of drugs occurred when the combination of trimethoprim and sulfamethoxazole was given along with other antibiotics. The combination of trimethoprim and sulfamethoxazole should be considered a major addition to the pharmacologic armamentarium for the treatment of pulmonary melioidosis.

*From the Department of Pulmonary Disease, United States Air Force Medical Center Scott, Scott Air Force Base, Illinois.

The views expressed herein are those of the authors and do not necessarily reflect the views of the United States Air Force or the Department of Defense.

**Presently at Veterans Administration Hospital, Little Rock, Ark.

Reprint requests: Dr. Fuller, USAF Hospital, Scott AFB, Illinois 62225