Our case would appear to present similar anatomic findings which were recognized during life and successful therapy instituted by the transthoracic route of implantation.

While our patient was paced successfully for 23 months initially, we would agree with Kukral that the catheter position attained through the tortuous course presented by this set of anatomic relationships is tenuous at best. When a pacemaker catheter, passed from the left side, fails to pass the midline and enters the right heart via persistent left superior vena cava, an alternate method of therapy should be sought.

In an emergency situation where temporary pacing is required immediately, a femoral vein approach may be utilized to bypass the venous anomalies above the diaphragm. The anatomy of the superior vena cava may be investigated by angiography performed from the right arm or right external jugular vein. A normal superior vena cava properly communicating with the right atrium, would indicate placement of a permanent transvenous pacemaker through the right cephalic vein. The transthoracic approach would appear to offer one satisfactory alternative in the presence of an additional right superior vena cava anomaly.

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Glomerulonephritis following Streptococcal Pneumonia*

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A case of glomerulonephritis following streptococcal pneumonia in an elderly man is described. The importance of considering the Streptococcus in the differential diagnosis of bacterial pneumonias is stressed. The nonsuppurative complication of this pneumonia is documented.

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The isolated occurrence of streptococcal pneumonia is uncommon. Reported cases have occurred mainly in epidemics among military recruits.\(^1\)\(^-\)\(^3\) We describe herein an isolated case of streptococcal pneumonia and empyema complicated by glomerulonephritis.

**CASE REPORT**

The patient, a 64-year-old white man, was discharged on March 6, 1970 after six months of hospitalization for pulmonary tuberculosis of the right upper lobe without cavitation. AFB cultures from December 18, 1969 to February 2, 1970 were negative. Chest x-ray film on March 2, 1970 showed a stable right upper lobe infiltrate. The patient had a past history of atherosclerotic cardiovascular disease with angina, chronic alcoholism, and mild hypothyroidism secondary to \(^1\)\(^-\)\(^3\) therapy for hyperthyroidism. Medicines at the time of his discharge were INH, PAS and digoxin.

Nine days after discharge the patient was readmitted because of a cough producing thick purulent sputum, pleuritic chest pain, dyspnea, chills and fever beginning three days prior to admission. On physical examination the patient was in mild respiratory distress. Temperature was 102°F. Pulse was 100. Blood pressure was 140/70. The chest was dull to percussion at the right base with decreased breath sounds in that area. There were bi-basilar rales and right posterior rhonchi. The remainder of the physical examination was unremarkable. White blood cell count on admission was 26,000 with 80 percent neutrophils and 14 percent band forms. Hematocrit was 33 percent. Results of urinalysis were normal; specific gravity was 1.022 and proteinuria was absent. Serum creatinine was 1.0 gm/100 ml. Gram stain of the sputum showed gram positive cocci in chains and pairs. AFB smears were negative. Sputum cultures on admission grew group A beta hemolytic streptococci. Blood cultures were negative on admission. Chest x-ray examination showed an infiltrate and effusion involving the right mid and lower lobes (Fig 1).

The patient was initially given phenoxyethyl penicillin 2 gm per day. He remained dyspneic and febrile over the next three days. Thoracentesis on the third day of hospitalization revealed a white exudate containing many polymorphonuclear leukocytes and gram positive cocci in chains.

Culture of this fluid grew group A beta hemolytic streptococci, M-nontypable, T-type 11 and 12. No AFB were grown from pleural fluid. An ASO titer from March 21, 1970 was 1:2560 Todd units. IgA was 720 mg/100 ml, IgG 1500 mg/100 ml, and IgM 170 mg/100 ml.

On March 18, 1970 a chest tube was placed in the right pleural space. Oral penicillin was discontinued and aqueous procaine penicillin G 2.5 million units was given intravenously every six hours. Three days later the patient developed a morbilliform rash over the trunk and abdomen. Because of the persistence of the empyema it was decided to continue the penicillin. Benadryl was given and the rash resolved over two days. The patient became afebrile after two days of intravenous penicillin therapy and remained afebrile throughout the rest of his hospital course. The right lower lobe infiltrate and empyema cleared after two weeks of antibiotic therapy and closed drainage of the pleural space. Subsequent chest films have revealed no further changes (Fig 2). Measurements of vital capacity have been normal.

The patient's hematocrit gradually fell from 33 percent on March 24, 1970 to 20 percent on April 6, 1970. Stool guaiac were repeatedly negative. A direct Coombs' test was positive. Because hemolytic anemia due to penicillin was suspected the drug was stopped on April 7, 1970. Red blood cells obtained on April 6, 1970 were found to possess an antibody which could be eluted and which agglutinated penicillin sensitized red blood cells. The patient's serum was positive for penicillin hemagglutinins at 1:128 dilutions.

Urinalysis was normal on admission but on March 30, 1970 a urine specimen revealed numerous red blood cells, red blood casts, 50 mg/100 ml of protein and had a specific gravity of 1.015. BUN was 37 mg/100 ml and serum creatinine 1.7 mg/100 ml. Creatinine clearance was 62 ml per minute. Over the next two weeks the patient's blood pressure ranged from 150/95 to 180/120. Serum creatinine rose to 2.0 mg percent and creatinine clearance fell to 37 ml per minute.
Urinalysis consistently showed many red blood cells and red cell casts. An IVP was normal. Serum complement was 31 mg/100 ml (normal 123-167 mg/100 ml). A renal biopsy was performed on April 30, 1970. Light microscopy showed increased cellularity within glomeruli due to an increase in mesangial cells and polymorphonuclear leukocytes. Electron microscopy demonstrated subepithelial electron dense deposits (Fig 3). These changes were felt to be consistent with poststreptococcal glomerulonephritis.

The patient's condition gradually improved over the next two months. He became normotensive. Creatinine clearance rose to 66 ml per minute. Urinalysis still showed 10 to 20 red blood cells and occasional red blood cell casts. Complement rose to 100 mg/100 ml. ASO titers gradually fell to 1:640 (May 7, 1970). Hematocrit slowly rose to 32 percent. A repeat direct Coombs' test was negative.

He is presently asymptomatic six months after discharge. Multiple AFB cultures have been negative. Creatinine clearance on October 30, 1970 was 67 ml per minute. However, his urine on the same date still contained 10 to 12 red blood cells and three to four red blood cell casts per high power field.

**DISCUSSION**

Although group A, beta-hemolytic streptococci are an unusual cause of pneumonia, this organism should always be considered when a bacterial pneumonia is suspected. The diagnosis may easily be overlooked because streptococcal chains often fragment and appear as gram positive diplococci or cocci on gram stain. In addition a few beta streptococci in a sputum culture may be erroneously attributed to a pharyngeal carrier state. As in other bacterial pneumonias symptoms may include marked hyperthermia, cough, sputum, pleuritic chest pain and dyspnea.

This disease is often preceded by pneumococcal pneumonia or viral illnesses such as influenza or measles. Other predisposing illnesses appear to be asthma, bronchiectasis and tuberculosis. In contrast to streptococcal pneumonia, chronic illness and influenza appear to have no effect on the incidence of streptococcal pharyngitis.

Empyema is a common complication. Keefer and co-workers^ observed that 16 out of 55 patients had empyema. More recently empyema has been reported in 54 out of 95^ and 19 out of 20^ patients with streptococcal pneumonia. Bacteremia from hemolytic streptococcal pneumonia is unusual. When bacteremia is present during streptococcal pneumonia, the prognosis is poor. Fever may be prolonged and resolution of empyema slow even with adequate drainage. Intravenous penicillin in high doses is the antibiotic of choice.

The resolution of the pneumonia and empyema was rapid and uncomplicated in our patient despite the underlying pulmonary disease. His subsequent clinical course, however, was complicated by two reactions to the penicillin therapy: (1) A transient maculopapular truncal rash which appeared seven days after penicillin therapy was initiated and (2) a Coombs' positive hemolytic anemia associated with specific antipenicillin antibody which remitted when penicillin therapy was discontinued.

The onset of red blood cell casts and renal insufficiency strongly suggested poststreptococcal glomerulonephritis. A renal biopsy specimen demonstrated glomerulonephritis on both light and electron microscopy. This finding dismissed the possibility of an interstitial nephritis which has been reported following methicillin and penicillin therapy.

Glomerulonephritis following streptococcal skin infections and pharyngitis is well documented. On the other hand, glomerulonephritis following streptococcal pneumonia and empyema has not been adequately described in the past and appears to be rare. Brasiliere and associates reported only one case of glomerulonephritis among 95 patients with streptococcal pneumonia without giving details of the case. Burmeister and Overholt observed two cases of hematuria following streptococcal pneumonia treated with penicillin during an epidemic of this disease. This finding was presumed to be secondary to poststreptococcal glomerulonephritis although renal biopsies were not done.

This case report provides the first firm evidence for the occurrence of glomerulonephritis following streptococcal pneumonia. In addition the need to consider streptococcal pneumonia in the differential diagnosis of a pneumonic process in the elderly and in the nonmilitary populations is stressed.

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**Figure 3.** Electron microscopy of renal biopsy shows electron dense subepithelial deposits (a), normal basement membrane (b), and polymorphonuclear leukocyte (c).
Pulmonary Function following
Traumatic Rupture of the Bronchus*

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and L. P. Sterns, M.D., F.C.C.P.

A 33-year-old man developed severe hypoxia following a serious crush injury to the right chest. A bronchogram revealed complete obliteration of the right mainstem bronchus, but on angiography there was persistent perfusion of the right lung associated with a calculated shunt in excess of 30 percent. Other pulmonary function studies demonstrated a wide A-a O₂ gradient and a severe restrictive defect. At surgery an unusual anomalous tracheal bronchus to the right lower lobe was discovered which was also completely occluded. Following pneumonectomy the A-a O₂ gradient reverted almost to normal.

Formerly considered a rare injury, cases of partial or complete rupture of the tracheobronchial tree secondary to blunt chest trauma have been reported with increasing frequency in the past two decades.¹ The injury has sometimes been demonstrated radiographically prior to surgery²⁴ and one case report documents the results of ear oximetry and differential bronchiospirometry performed 18 months after successful anastomosis of a ruptured mainstem bronchus.³ However in general studies of pulmonary function following this type of injury have been inadequate. We recently had the opportunity to assess the physiologic derangements in a patient who had suffered rupture of the right mainstem bronchus five weeks previously.

CASE REPORT

The patient, a 33-year-old construction worker, had always been healthy, apart from a slight productive cough attributed to heavy cigarette smoking, until September 1970 when the right side of his chest was crushed between two motor vehicles. Physical examination revealed subcutaneous emphysema over the affected area, and the percussion note over the right lung was dull. Breath sounds were distant over the whole lung field. Serial chest x-ray pictures confirmed the presence of a right pneumothorax which responded slowly to tube drainage. The patient was discharged from hospital after two weeks, but further examination one month following the accident revealed there was no air entry to the right lung.

He was then referred to the University of Alberta Hospital, where at the time of his admission he appeared chronically sick, cyanosed and became dyspneic with minimal exertion. Examination of the chest confirmed the impression of complete atelectasis of the right lung.

The admission hemoglobin was 14.6 gm per 100 ml, white blood count 7,100 per mm³, sedimentation rate 21 mm/hr. Arterial blood gas studies demonstrated gross hypoxemia, the Pao being 32 mm Hg, Paco₂ 32 mm Hg, pH 7.49, oxygen saturation 67 percent. Similar results were obtained a few days later when alveolar arterial oxygen (A-a O₂) gradients were measured (Table 1). Spirometry revealed a gross restrictive defect, the loss of lung volume being confirmed by body plethysmography (Table 2).

The admission chest x-ray film revealed collapse of the right lower lobe associated with elevation of the right hemidiaphragm and displacement of the mediastinum to that side. A comminuted fracture of the body of the scapula was also noted. Three days later the right hemithorax appeared completely opaque, and the mediastinal shift had become further accentuated. A bronchogram revealed a complete cutoff of the right mainstem bronchus about 2 cm below the carina (Fig 1). However both a lung scan and a pulmonary angiogram demonstrated significant perfusion of the right lung

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**Table 1—Measurements of Alveolar Arterial Oxygen Gradients.**

<table>
<thead>
<tr>
<th></th>
<th>PO₂ mm Hg Room Air*</th>
<th>PO₂ mm Hg 100% O₂</th>
<th>A-a DO₂ mm Hg Room Air 100% O₂</th>
<th>Calculated Shunt (100% O₂)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before surgery</td>
<td>37</td>
<td>47</td>
<td>63.6</td>
<td>607</td>
</tr>
<tr>
<td>After surgery</td>
<td>68</td>
<td>500</td>
<td>27.7</td>
<td>128</td>
</tr>
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*At altitude of Edmonton 2,500 feet.