Table 1—Pulmonary Function and Arterial Blood Determinations

<table>
<thead>
<tr>
<th>Date</th>
<th>Thoracotomy-Decortication</th>
<th>Arterial Blood Gases</th>
<th>FVC %</th>
<th>PEFR %</th>
<th>FEV%</th>
<th>MMEFR L/min</th>
<th>PEFR L/min</th>
<th>MBC %</th>
<th>RV %</th>
<th>TLC %</th>
<th>RV/TLC %</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/10/73</td>
<td>2/2/73</td>
<td>pH</td>
<td>7.44</td>
<td>7.44</td>
<td>57</td>
<td>57</td>
<td>80</td>
<td>80</td>
<td>104</td>
<td>104</td>
<td>78</td>
</tr>
<tr>
<td>2/22/73</td>
<td>3/6/73</td>
<td>PO2 mm Hg</td>
<td>56</td>
<td>57</td>
<td>35</td>
<td>35</td>
<td>56</td>
<td>56</td>
<td>90</td>
<td>90</td>
<td>78</td>
</tr>
<tr>
<td>5/10/73</td>
<td>4/4/73</td>
<td>PCO2 mm Hg</td>
<td>47</td>
<td>47</td>
<td>24</td>
<td>24</td>
<td>68</td>
<td>68</td>
<td>80</td>
<td>80</td>
<td>68</td>
</tr>
</tbody>
</table>

- **FVC** = forced vital capacity
- **FEV** = forced expiratory volume
- **MMEFR** = maximum mid-expiratory flow
- **PEFR** = peak expiratory flow rate

Immediately drained, which was putrid and contained a white blood cell count of 58,000/ml, with 95 percent lymphocytes and 5 percent polymorphonuclear cells. Drainage of pus from the chest tube continued for three weeks thereafter. Gram stains of the empyema fluid revealed gram + bacilli and cocclobacillary forms without the detection of sulfur granules. Numerous spuas cultures grew normal flora. Aerobic and anaerobic cultures of the empyema fluid grew only *A. naeclundii*, the identity of which was confirmed by the Mycology Laboratory of the New York City Department of Health.

The patient ran high remittent temperatures of up to 38.9°C over the first 17 days of her hospitalization, but became afebrile by the end of the third week of penicillin therapy. She was also treated with a lincomycin analogue (Clindamycin) in addition to penicillin for four weeks. After nine weeks of penicillin therapy she underwent thoracotomy for a decortication procedure. The pathologic examination of resected fibrous tissue showed only chronic nonspecific inflammation. The penicillin therapy was continued for another week, and the patient was discharged without any medication. She has been asymptomatic over the three months since discharge, with recovery of her pulmonary function (Table 1).

**DISCUSSION**

The patient showed several of the clinical findings characteristic of previous reports of actinomycosis. While she had no definite history of loss of consciousness or aspiration, and was not an alcoholic, she did have carious teeth and pyorrhea. She also clearly demonstrated the tenacity of the fungus to maintain an established suppurative infection despite massive antibiotic and drainage therapy, which eventually necessitated decortication. The finding of the *A. naeclundii* organism is unique because even *A. israeli* is rarely found to be the only organism present in a suspected actinomycotic lesion. This frequently makes it difficult to exclude a pathogenic role for accompanying anaerobic bacteria. The cellular and colonial morphologic characteristics of *A. naeclundii*, while very similar to those of *A. israeli*, can be differentiated by cultural characteristics, primarily their ability for aerobic growth. It is most important to identify this organism rapidly, for it appears to be similar to *A. israeli* in both its response to antibiotic therapy and its ability to destroy lung tissue.

**ACKNOWLEDGMENT**: We would like to thank Mr. Neville Trowers, Head of the Bacteriology Laboratory, for his assistance in the diagnostic studies.

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**Successful Pregnancy in a Patient with Severe Superior Vena Cava Syndrome**

*M. Dennis Barton, M.D.,* **and Watson A. Bowes, Jr., M.D.†**

A 37-year-old parturient woman with presumed mediastinal fibrosis survived an antepartum pulmonary embolism, antepartum circulatory collapse with loss of consciousness and postpartum pneumonia. Her infant is developing normally 18 months after delivery.

**The gravid uterus of late pregnancy normally causes aortocaval compression. Venous compression is more marked, but aortic compression also occurs, especially in the supine position. The symptoms may be alleviated by changing the parturient patient to a left lateral position, or by shifting the weight of the uterus to the left. The occurrence of this supine hypotensive syndrome (SHS) in patients with fixed cardiac output, or**

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exhibited pulselessness and loss of consciousness. The pupils
were not demonstrated on right. Probable conclusion is that vena cava occlusion extended below
azygous vein junction with superior vena cava.

previously impaired venous return can have predictably
severe results. This case demonstrates the consequences
of the SHS, pulmonary embolism and postpartum
pneumonia in a pregnant patient with presumed mediastinal fibrosis.

**CASE REPORT**

A 37-year-old woman, gravida 3, para 2, entered a Colorado
community hospital in her 29th week of pregnancy on

At the age of 21 the patient had had a febrile illness. She
had since exhibited a superior vena caval syndrome, with
intermittent episodes of swelling in her chest, neck and arms.
Radiographic studies in 1966 (Fig 1) were interpreted as
showing a bilateral thrombosis of the subclavian veins, the
innominate veins and the superior vena cava.

Two days prior to her admission, the patient began to
complain of dyspnea, chest pain and syncope.

On admission, the patient was cyanotic, with systolic blood
pressures of 70 to 90 mm Hg. A radioactive technetium lung
scan showed no perfusion of the right lung, and decreased
perfusion of the anterior superior aspect of the upper lobe of
the left lung. A diagnosis of pulmonary embolism was made.
She was treated with heparin.

The patient was transferred to the University of Colorado Medical Center in her 37th week of pregnancy. Admission
examination showed facial cyanosis and mild proptosis. Breath sounds were decreased over the right lung. Dilated
veins were present over the neck, chest wall and abdomen.

An admission radiograph of the chest showed the medias-
tinum shifted to the right, crowding of the ribs, and elevation
of the diaphragm on the right side.

Four days after admission, a physician was drawing blood
while the patient was supine. The patient lost consciousness.
No peripheral pulse was felt, nor was an apical heart beat
heard. External cardiac massage was applied and the patient
regained consciousness. After two to three minutes, she again
exhibited pulselessness and loss of consciousness. The pupils
dilated. External cardiac massage was repeated and con-
tinued for one minute until the patient regained conscious-
ness.

The cause of this episode was ascribed to SHS. The
syndrome was exacerbated by the peculiar venous drainage
exhibited by the patient. She was told to avoid the supine
position and there was no recurrence of her symptoms.

When her pregnancy was at term, heparin was withheld
for 18 hours and labor was induced by amniotomy. She was
kept in the left lateral position throughout labor and delivery,
and delivered a 2,970 gm boy. The Apgar score was nine at
one minute and nine at five minutes. Anticoagulant therapy
was re instituted 12 hours after delivery.

On the first postpartum day, the patient again complained
of chest pain. There was no change in the chest x-ray film
findings. On the third postpartum day her infant developed a
Streptococcus group B infection. On the same day the patient
developed a right upper lobe infiltrate and a right pleural
effusion. Uterine culture grew B streptococci and sputum
cultures both B streptococci and Klebsiella species.

The patient responded to penicillin therapy, and intercostal
tube blocks were done to relieve chest pain. By the 11th
postpartum day a radioactive technetium lung scan still
showed absence of activity over the right lung.

She underwent a radioactive xenon ventilation study. This
revealed rapid bilateral lung activity, but delayed xenon
washout on the right side. The patient was discharged on
May 13, 1971 after 90 days of hospitalization.

**DISCUSSION**

The clinical picture presented here is that of superior
vena cava syndrome caused by mediastinal fibrosis. The cause of mediastinal fibrosis is frequently unknown.

Goodwin et al recently reviewed 38 cases of mediasti-
nal fibrosis with a proved etiology. They found 26 cases
caused by *Histoplasma capsulatum* (histoplasmosis) and
12 cases caused by *Mycobacterium tuberculosis*. At
present no other causative agents have been identified.
Mediastinal fibrosis can present as either an expanding mass of granulomatous lymph nodes or an exuberant proliferation of fibrous tissue beyond the confines of the lymph nodes. The latter form is responsible for the reported cases of pulmonary artery occlusion associated with this disorder. The absence of the right pulmonary artery, discovered early in the patient's course, was at first attributed to pulmonary embolism. It is quite possible that part or all of the right pulmonary artery occlusion in this patient is due to mediastinal fibrosis. The acute onset of chest pain and dyspnea, coupled with the first abnormal antepartum left lung scan, then a normal postpartum left scan, suggests that the left lung embolization had occurred.

Pulselessness and loss of consciousness were presumably due to the supine hypotensive syndrome. Lees et al report a pregnant patient with supine hypotension showing a mean blood pressure reduced to 40 mm Hg, and a cardiac index reduced to less than 2.0 liter/sq M/min. Bradycardia was noted, but not loss of consciousness. Fainting has been recorded in patients with the supine hypotensive syndrome. An acute rise in cerebral venous pressure, further decreasing cerebral blood flow, may have played a part in this patient's symptoms.

Antepartum pulmonary embolism is an infrequent but serious complication of pregnancy. Venous stasis and hypercoagulability both occur with pregnancy. The hypercoagulability has been ascribed to increased fibrinogen, increased factors 7, 8, 9, and 10, and a reduction of fibrinolytic activity during pregnancy. Indeed, pulmonary embolism is the second leading cause of maternal mortality in England. Anticoagulation with heparin is the recommended therapy. Heparin does not cross the placenta, at least in amounts that affect the fetus. Anticoagulant therapy was discontinued in sufficient time to allow delivery.

Cardiac arrest and successful resuscitation with cardiac defibrillation have occurred in pregnancy, with fetal survival. It is of note that the Apgar score was nine at one minute and nine at five minutes with this patient's son despite a brief period of circulatory collapse six days prior to delivery. At 18 months of age, the child's growth and development are normal.

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