A massive fibrinous clot would be visible through the filter's plastic surface, but smaller thrombi would probably pass unrecognized. Clotting within the filter represents one reason for diminished hemofiltrate output. Clotting can cause pulmonary embolism, or the acute loss of VVB.

Renal failure occurs in up to 75% of patients dying of cirrhosis. CAVH and CVVH have been shown to improve pulmonary and hemodynamic function during and after liver transplantation.

Intraoperative hemofiltration was used to help treat hypervolemia. Placement of the hemoconcentration filter across the VVB circuit in parallel to the patient provided transmembranal pressure gradients and hemofiltrate flow rates associated with CAVH without cannulation of the arterial circulation. There is also no need for venous cannulation beyond that required for VVB. The technique can effect a rapid decrease in intravascular volume and may be performed in selected patients without systemic anticoagulants.

References

Anesthesiology 1999; 90(5):111–5
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Respiratory Compromise and Dramatic Chest X-ray Changes during General Anesthesia in a Patient with a Bronchogenic Cyst

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BRONCHOGENIC cysts are rare congenital anomalies that may cause obstructive emphysema. We present the case of an infant in whom acute respiratory distress was caused by a bronchogenic cyst. Dramatic changes in the chest radiograms, from hyperinflation of the left lung and atelectasis of the right lung to hyperinflation of the right and atelectasis of the left, were observed within a short period of time during general anesthesia. Surgical excision provided complete relief.

Case Report

An 8-month-old, 8-kg boy was admitted to a nearby hospital with a 4-week history of a croupy cough, wheezing, and fever. He was the full-term product of an uncomplicated pregnancy and delivery. He had similar symptoms when he was 5 months old and was treated for asthma and pneumonia for 1 month. At admission, he was tachypneic (60–70 breaths/min) with marked inspiratory stridor unaffected by posture. There were faint breath sounds on the left side of the chest and slight wheezing was heard on the right side. Oxygen saturation measured by pulse oximetry ($Sp_O_2$) was 74% and did not respond to

Anesthesiology, V 90, No 3, Mar 1999
intravenous steroids, aminophylline, and 100% oxygen by mask. Chest radiography showed hyperinflation of the left lung, atelectasis of the right middle lobe, and a shift of the mediastinum to the right without evidence of a pneumothorax. His trachea was intubated and he was transferred to our hospital for further treatment. At arrival, respiration was manually assisted at a rate of 40–50 breaths/min with 100% oxygen. The SpO₂ varied between 92 and 100%, and the changes in SpO₂ were not related to changes in position. Computed tomography (fig. 1) revealed pneumomediastinum, hyperinflation of the left lung, and a 3 cm mass compressing the left main stem bronchus. Bronchoscopy and excision of the mass during general anesthesia were planned.

In the operating room, the heart rate, blood pressure, arterial oxygen saturation (SaO₂) and end-tidal carbon dioxide (EtCO₂) were 190 beats/min, 105/65 mmHg, 90%, and 77 mmHg, respectively. Anesthesia was induced in the supine position with 2 or 3% sevoflurane in 100% oxygen. No muscle relaxant was used. Respiration was assisted manually. An arterial blood gas analysis showed a pH of 7.15, a pressure of carbon dioxide (PaCO₂) of 99 mmHg, and a pressure of oxygen (PaO₂) of 258 mmHg. When the respiratory rate was increased from approximately 30–50 breaths/min at a peak airway pressure of 20–30 mmHg, the EtCO₂ decreased to 42 mmHg. At this point, the initial chest radiograph taken in the operating room was similar to the one taken earlier at the referring hospital, but subcutaneous emphysema now extended from the right lateral abdominal wall to the right side of the neck (fig. 2).

Examination with a flexible fiberoptic bronchoscope failed to detect any foreign body or an obstruction of the trachea. SpO₂ gradually decreased to approximately 90% during the bronchoscopy and returned to 98% after resumption of ventilation. However, the SaO₂ dropped precipitously to approximately 70% during the third bronchoscopy and did not respond to vigorous ventilation (respiratory rate 60–70 breath/min, minute volume 7–8 l/min). A high airway pressure was required (peak airway pressure 50–60 mmHg) to maintain the EtCO₂ below 50 mmHg. The blood pressure decreased from 90/50 to 60/45 mmHg. At this time, the arterial blood gases on 100% oxygen were pH of 7.48, PaCO₂ of 57.8 mmHg, and PaO₂ of 45.4 mmHg. Within approximately 10 min, the SpO₂ returned to almost 90%. The arterial blood pressure remained at 70–90/40–50 mmHg and the heart rate remained at 110–170 beats/min. Subsequent chest radiography (fig. 3), taken approximately 90 min after the first, showed dramatic changes: the right lung was now hyperinflated with a leftward shift of the mediastinum and atelectasis of the left lung. An emergency thoracotomy was planned because of the patient's respiratory distress and the radiogram changes.

A right posterolateral thoracotomy was performed with the patient in the left lateral decubitus position. A bronchogenic cyst, approximately 3 × 2 cm and containing jelly-like mucus, originating from the right lateral wall of the trachea 1 cm above the carina, was observed impinging on the esophagus. Immediately after excision of the cyst, there was improvement in lung compliance, SpO₂, and EtCO₂. A low inspiratory pressure of 15 mmHg was sufficient to ventilate the lungs. The arterial blood gas analysis revealed a pH of 7.59, a PaCO₂ of 26.7 mmHg, and a PaO₂ of 312 mmHg. The patient's postoperative course was uneventful. A histologic examination confirmed the diagnosis of a bronchogenic cyst.

**Discussion**

Hyperinflation of the lung occurs in many pathologic processes, including atelectasis with compensatory em-
Fig. 3. Radiograph showing hyperinflation of the right lung, atelectasis of the left lung, subcutaneous emphysema, and a leftward shift of the mediastinum. An endotracheal tube is in the trachea and the gastric tube is displaced to the right.

physema, congenital pulmonary cyst, pneumatocele, pneumothorax, diaphragmatic hernia, congenital lobar emphysema, and obstructive lobar emphysema. A bronchogenic cyst causes obstructive emphysema by compressing the tracheobronchial tree. Rarely, a bronchogenic cyst with a tracheobronchial communication expands tremendously, mimicking a pneumothorax.

The unique aspect of this patient was the dramatic changes in the chest radiography that occurred within a short period of time. The cause of the changes is a matter of speculation. One possibility is that manipulation with the bronchoscopecopy may have moved the trachea, the cyst, or both, changing the location of where the cyst obstructed the airway. Other factors might also have changed the location, such as high intrathoracic pressure, the change in the patient's position, a cranial shift of the diaphragm, or a reduction in the transverse cross-sectional area of the thorax caused by anesthesia. Compression of the left bronchus might have acted similar to a check-valve, causing hyperinflation of the left lung seen in figure 2. Hyperinflation of the right lung would similarly have resulted from a change in the location of the compression to the right bronchus. If there was communication between the bronchogenic cyst and the trachea, the high pressure in the trachea during the inspiration phase of manual ventilation might have raised the intracystic pressure, causing the cyst to expand. The larger the cyst, the more it would compress the airway. Murray described a neonate who had a large air-filled bronchogenic cyst with a bronchial communication and in whom acute respiratory distress developed.

This case has implications for the management of anestheisa in patients with a bronchogenic cyst. Bronchogenic cyst should be considered in the differential diagnosis of hyperinflation of the lung especially with unusual changes in radiographic findings.

References