Abnormal connections between distal portions of the respiratory airways and other anatomic structures usually produce significant pulmonary compromise. These interconnections may have an acute onset and lead to catastrophic consequences. Subacute or chronic presentations may also occur and will be emphasized in this review. It is usually not possible to localize the precise anatomic location of such tracks. Therefore, the generic term, "bronchial fistula," will be used even though the actual interconnection may be distal to true bronchi or may be unknown. Tracheal fistulas will be omitted from this discussion.

The frequency of bronchial fistulas in most disease states is unknown but their occurrence is unusual. The poor prognosis uniformly associated with longstanding fistulas complicated by infection, however, mandates their inclusion in the differential diagnosis of a large number of patient presentations. The consequences of regional or systemic infection, hypoxemia, and progressive lung injury due to persistent contamination of the pulmonary parenchyma by foreign material may produce significant morbidity. For the purposes of this review, bronchial fistulas will be grouped as those joining the lung with abdominal organs, the pleural space, vascular structures including the heart, and the central nervous system.

**ABDOMINOBRONCHIAL FISTULAS**

A variety of abnormal connections between the lung and abdominal organs occur usually as a result of infection, trauma, or malignancy. These fistulas most often produce pulmonary rather than visceral symptoms even through the primary disease initiating the fistula originates in the abdominal organ. It is postulated that inflammation, infection, or tumor invasion within the abdomen causes diaphragmatic and lung penetration. The pulmonary consequences are nonspecific and include consolidation, atelectasis and hypoxemia. Although some diagnostic clues may exist, confirmation of a fistula is usually made by chance or after persistent searching with different diagnostic techniques.

**Bronchobiliary Fistulas**

First reported in 1850, this fistula is rare with an incidence prior to antibiotic use of 4 percent and 10 percent in bacterial liver and subphrenic abscesses, respectively. Antibiotic usage has reduced this incidence considerably. Causes of bronchobiliary fistulas are shown in Table 1.

Presenting symptoms include nonspecific systemic manifestations of chronic infection and nonspecific abdominal or respiratory complaints of cough, sputum production, dyspnea, and abdominal pain. Bilioptysis (biliptysis, bile ptyalism), bitter-tasting and bile-stained sputum, is the important diagnostic sign of a bronchobiliary fistula. Although reported rarely in patients with sickle cell disease during a hemolytic crisis, the occurrence of bilioptysis always warrants further evaluation for a fistula. The amount of bile and hence quantity and discoloration of expectorated fluid or sputum may be variable, and direct measurement of sputum bilirubin is therefore useful. Other important diagnostic aids include recovery of hydatid cysts or debris and amebae from sputum. A characteristic

---

*From the Department of Anesthesiology and Critical Care Medicine, University of Pittsburgh School of Medicine, Pittsburgh.
†Professor, Department of Anesthesiology and Critical Care Medicine.
‡Assistant Professor, Department of Anesthesiology and Critical Care Medicine.
Table 2—Causes of Bronchoesophageal Fistulas

<table>
<thead>
<tr>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malignant esophageal, pulmonary, and mediastinal tumors</td>
</tr>
<tr>
<td>Congenital</td>
</tr>
<tr>
<td>Infections—tuberculosis, atypical mycobacteria, syphilis, actinomycoses, histoplasma</td>
</tr>
<tr>
<td>Trauma</td>
</tr>
<tr>
<td>Surgery (esophagogastrectomy)</td>
</tr>
<tr>
<td>Prolonged tracheal intubation</td>
</tr>
<tr>
<td>Endoscopy with sclerotherapy</td>
</tr>
<tr>
<td>Achalasia</td>
</tr>
<tr>
<td>Lye ingestion</td>
</tr>
<tr>
<td>Chemotherapy or radiation therapy with tumor lysis</td>
</tr>
<tr>
<td>Emesis (Boerhaave’s syndrome)</td>
</tr>
</tbody>
</table>

"chocolate sauce" sputum is described with amebic liver abscesses that have penetrated into the lung.

Bronchobiliary fistulas are usually located in the right upper quadrant of the abdomen, penetrate the posteromedial aspect of the right diaphragmatic dome, involve lower lobe structures, and are often associated with empyemas and subdiaphragmatic abscesses. Unusual left-sided fistulas have occurred. Identification of the fistula may be possible with transhepatic or T-tube cholangiography, radionuclide (HIDA) scanning, bronchography, endoscopic retrograde cholangiopancreatography (ERCP), or by a contrast fistulogram.

Surgical correction of the defect and resection or drainage of infected material from the thorax and abdomen usually are required. Percutaneous aspiration and catheter drainage in conjunction with antibiotics and endoscopic stone removal and sphincterotomy have been successful in controlling infection and promoting fistula closure without surgery.

Esophagobronchial Fistulas

Fistulas between the esophagus and lower airway most commonly are associated with malignant tumors of the esophagus, but other causes (Table 2) also occur. Symptoms often combine the local and systemic effects of the primary disease complicated by acute and chronic aspiration. Symptoms may be present for many years as with congenital defects and usually reflect aspiration, especially of liquids. Ono’s sign (decreased incidence of aspiration when food is taken in the supine position) may be diagnostically helpful.

Barium studies of the esophagus are the preferred diagnostic method but are associated with frequent respiratory spillage. Because bronchoesophageal fistulas usually occur on the anterior esophageal surface, esophagography in the prone position may be helpful. Computed tomography, esophagography, and bronchoscopy may be useful in some patients but appear less sensitive. Bronchoscopy performed as the patient swallows methylene blue may diagnose and localize the fistulous tract. Conversely, aerosolized methylene blue may be visualized during esophagoscopy.

Bronchoesophageal defects commonly are not diagnosed until adolescence or adulthood because of delayed opening of the fistula due to incomplete or inconstant anatomic defects between the two structures. Sequestered pulmonary tissue, bronchogenic cysts, or esophageal diverticulae may also cause these fistulas that usually occur between the right lung and the esophagus.

Tuberculosis and syphilis have historically been important causes. Erosion from mediastinal or peribronchial lymph nodes to the esophagus has been the presumed mechanism for mycobacterial spread and fistula formation. Similarly, gumma in the mediastinum may produce a syphilitic fistula and rarely esophageal syphilis or erosion from a syphilitic aortic aneurysm may occur. An infectious cause may be presumed when histologic findings of chronic inflammation occur without culture or tissue evidence of invading organisms.

Treatment of nonmalignant fistulas usually is surgical and must be supported by appropriate antimicrobial therapy, parenteral nutrition, etc. Sclerosis using sodium hydroxide or silver nitrate, occlusion with tissue glue or collagen, and transbronchial cautery have had variable success. Surgical excision of fistulas due to malignant neoplasms may also be considered. Better results were reported when the tumor and fistula were surgically bypassed than when resection and preservation of the esophageal lumen was attempted. When palliation for reduction of aspiration is the goal, a variety of prosthetic stents are available that can be inserted at surgery or via a transoral route so as to occlude the fistula tract. Prevention of reflux and closure of the fistula with such stents may fail and may be associated with additional morbidity such as erosion of the stent into the aorta.

Gastrobronchial Fistulas

In 1985, a review of gastrobronchial fistulas uncovered only 14 cases, thereby identifying this as a rare event. The causes again reinforce the general sequence of injury from surgery or trauma, inflammation as from peptic ulcer disease or heterotopic pancreatic tissue, infection, or neoplasm leading to tissue disruption. Communication with the respiratory tract leads to chemical injury and infection of the lung.

Clinical symptomatology is related to the cause of gastric inflammation and the secondary manifestations of pulmonary injury. There are no pathognomonic signs either of the gastric or lung involvement, although hemoptysis may be more common in this type of abdominobronchial fistula. An interesting diagnostic observation is a bronchial secretion pH of 2 to 3 (normal pH is 6 to 8) in one case report. Contrast studies from either the lung or stomach usually
establish the diagnosis. Surgical repair has been uniformly successful.

Other Abdominobronchial Fistulas

Fistulas between other abdominal organs and the lung occur with a frequency worthy of case presentation. Among these are small bowel, large bowel, kidney, pancreatic, and splenic fistulas.

An enterobronchial fistula has been reported in a patient after small-bowel resection complicated by a left subphrenic infection. He presented with nonspecific signs of infection and weight loss but also reported the "sensation of gas rising upwards from his left upper abdomen into his left hemithorax followed by a taste of food in his mouth." Next, another patient developed a similar fistula after splenectomy for malignant histiocytosis. In both cases, *Escherichia coli* was the predominant sputum pathogen. The diagnoses were made by contrast studies and surgical repair was successful in both.

Colonobronchial fistulas have been reported following colitis, pyelonephritis, neoplasm, or tuberculosis complicated by abscess formation. These fistulas are left sided and lead to invasion of the lung by gram-negative bacteria.

Nephrobronchial fistulas usually are caused by bacterial or echinococcal perirenal abscess, renal tuberculosis, trauma, or nephrolithiasis. A urineiferous odor to the patient's breath or sputum may be a helpful clue. Rarely nephrobronchial and colonobronchial fistulas coexist.

A recent case report documents the occurrence of a fistula between a splenic abscess and a left lower lobe bronchus. The abscess and pleural space were drained percutaneously with subsequent closure of the fistula. No surgery was required.

An elevated sputum lipase above serum levels was indicative of a pancreaticobronchial fistula in a patient with a pancreatic pseudocyst. Although pleural fluid is commonly contaminated by pancreatic enzymes in such patients, the formation of a fistula is considered rare and usually follows a surgical procedure or biopsy.

Bronchopleural Fistulas

Bronchopleural fistulas (BPF) can be categorized as follows: (1) postoperative complications following pneumonectomy or lung resection; (2) secondary to chronic inflammation/infection; and (3) following internal or external chest trauma. Although each type may require different diagnostic and therapeutic approaches, all are associated with both acute or chronic manifestations. The spectrum of clinical presentation, diagnosis, and treatment presents a challenge often extending from the intensive care unit to the chronic care facility.

A persistent BPF occurs postoperatively in two to 13 percent of patients and may appear within hours to years after surgery. Conditions that appear to predispose to lung friability and hence fistula formation include preceding, concomitant, or subsequent infection; chemotherapy; radiation treatment; poor nutrition; or general debility caused by the primary disease. A common denominator of these mechanisms is ischemia of lung parenchyma leading to disruption. Controversy exists about surgical techniques for lung resection in such patients, particularly regarding the use of staples and the type of stapler used. The CO₂ and Nd:YAG laser techniques have been advocated to decrease air leaks postoperatively. Underlying friability of the parenchyma, however, is usually more at fault for the occurrence of a BPF than is surgical technique.

The diagnosis of a BPF after pneumonectomy may depend on signs of respiratory distress, extra-alveolar air, or sequential roentgenographic evidence of mediastinal movement or air-fluid levels in the operated-on hemithorax. After lesser lung resections, persistent gas flow through the thoracostomy tube is indicative of a BPF.

Several operative strategies may be used to close a BPF after pneumonectomy or lung resection. In addition to primary closure of the fistula, reinforcement with pleura, thymus, pericardium, muscle pedicle, or abdominal omentum is often done. If the lung remaining in the hemithorax is unable to expand due to pleural thickening, a decortication procedure may also be performed. Thoracoplasties have also been used to close large persistent fistulas, although this option has been more commonly exercised for late-occurring fistulas. Because further surgery to close the BPF may be neither successful nor well tolerated, a variety of nonsurgical options exist that will be discussed below.

Fistulas occurring late after surgery are often associated with an empyema and closely resemble those fistulas characteristic of chronic infection. The patient usually presents with signs of pulmonary infection and respiratory failure. Late fistulas after pneumonectomy may be diagnosed roentgenographically by the reappearance of air in the vacant hemithorax, injection of an indicator such as methylene blue into the pleural space with its recovery from the sputum, accumulation of radionuclide in the pleural space after inhalation of xenon, or when bronchography shows spillage of contrast into the vacant hemithorax. Later fistulas after lobectomy or smaller resections may also present with acute or subacute symptoms of infection and pneumothorax.

Surgery must be preceded by drainage of the empyema, antibiotics, nutrition, and aggressive respiratory care. Reinforced closure of the original anatomic stump and expansion of any residual lung may
then be possible. Often, however, because of the patient's debility, limitations imposed by the earlier surgery, continued infection, or recurrent tumor, closure of these fistulas is not possible. Obliteration of the pleural space or provision of long-term or permanent bronchopleurocutaneous drainage then becomes the surgical objective. A staged thoracoplasty with or without a myoplasty is such an option. Alternatively, an open thoracostomy window may be used as a long-term drainage route. This procedure usually incorporates a skin-to-pleura suture line to promote epithelialization of the track.\(^\text{26}\) Residual lung tissue may or may not collapse within the hemithorax depending on its adherence to the parietal pleura. This thoracostomy window may undergo eventual spontaneous secondary closure or, after resolution of the infection, surgical removal of the window or covering the window with a myocutaneous flap is also possible.\(^\text{27}\)

Tubercular, fungal, or pyogenic infections, radiation therapy, or chemotherapy may also produce fistulas due to increased tissue inflammation and necrosis leading to greater tissue friability.\(^\text{28}\) These patients are treated as described for late occurring postoperative fistulas complicated by empyema. Closure of a permanent cutaneous fistula may be of secondary concern as treatment of these infections may be difficult and leave the lung permanently damaged.

Fistulas due to external blunt or penetrating trauma or internal expansion rupture (barotrauma) are most often managed by closed thoracostomy drainage. They usually resolve spontaneously except if surgery is mandated because of injury to the trachea, pulmonary vessels, or large bronchi. Surgery for closure of fistulas in more peripheral airways is especially unrewarding as localization of the fistula may be difficult and closure may require a lung resection. Coincident infection or decreased pulmonary function due to contusion or other lung disease may also limit surgery as an option.

Several issues especially apply to this etiologic group but may also pertain to any fistulas. The amount of gas lost from the lung via the thoracostomy tube during mechanical ventilation is ordinarily estimated as the difference between inspiratory and expiratory tidal or minute volumes measured at the airway. Sophisticated methods to quantitate gas flow directly through the fistula are available\(^\text{29,30}\) and may allow better titration of therapy. Gas recovered from the fistula may have participated in physiologic gas exchange as evidenced by an increased amount of carbon dioxide in gas leaving the chest.\(^\text{29,30}\) Measurement of the carbon dioxide content of this gas may be important when evaluating carbon dioxide production during nutritional assessment or dead space measurements. The magnitude of such gas exchange was dramatically demonstrated by a patient who had a long-term thoracostomy window and, with his mouth and nose occluded, could meet his respiratory needs by ventilating only through the bronchocutaneous fistula.\(^\text{30}\)

Finally, the efficiency of various chest drainage systems has been evaluated.\(^\text{31,32}\) Those with a high intrinsic internal resistance may fail to evacuate the pleural space when very large fistulas are present. Other extraordinary techniques to maximize air removal from the pleural space include mercury placed in the suction control chamber of commercial drainage units or use of high-capacity electronically driven suction units. High suction applied to the pleural space, however, may actually increase gas flow through the fistula.\(^\text{35}\)

Nonsurgical care of the patient with a BPF can be categorized as follows: (1) compensatory support until spontaneous closure occurs; (2) therapy directed toward reducing gas flow through the fistula so that spontaneous closure will be accelerated; and (3) direct interventions to close the track. The most common clinical approach is to provide compensatory support. The patient is permitted to exert the additional work of breathing required to sustain respiratory function or the mechanical ventilator is adjusted to supply sufficient ventilation and oxygen while spontaneous closure of the BPF is awaited. Most fistulas do close spontaneously.\(^\text{33}\)

When the fistula is large enough to compromise oxygenation or ventilation, attempts to reduce gas flow or close the BPF will be necessary. Direct closure techniques can be applied from the airway using a bronchoscope or from the pleural end of the fistula via a thoracoscope. Both methods are often preceded by bronchoscopy to localize the anatomic subsegment that is continuous with the fistula(s). Localization can be accomplished by inserting the bronchoscope or a balloon catheter (fogarty, pulmonary artery, or septostomy) into each segmental orifice sequentially and observing when bubbling has ceased in the water-seal chamber of the collection unit. More elegant appearing methods include bronchography or indicator methods wherein xenon is injected into segmented bronchi and measured over the thoracostomy tube\(^\text{39}\) or methylene blue is instilled via a bronchoscope and its appearance is observed on the pleural surface using a thoracoscope.\(^\text{34}\)

Closure methods are shown in Table 3. Most are based on a temporary physical occlusion of the airway until an inflammatory response to the foreign material effects a permanent seal. All carry a risk that the obstructed segment may become infected or the obstructing object may act as a one-way valve leading to distal expansion rupture of other previously normal lung units. Simultaneous use of a bronchoscope and thoracoscope combines the two methods.

Therapy to decrease gas flow via the fistula in anticipation of spontaneous closure may be used when
massive embolus or smaller more insidious emboli is difficult as the primary manifestation of this fistula may be nonspecific signs of shock. The diagnosis may be made only at emergent thoracotomy. Unexpected cardiac arrest after initiation of positive pressure ventilation or during ventilation at high airway pressures may be the first sign of air emboli. Hemothorax and other evidence of lung injury should remind the clinician that migration of gas into a vein may also occur. The amount of gas entering the veins depends on the pressure gradient between the air space and the vein after the track is opened. Therefore, high airway pressure and low venous pressure may lead to more embolization. When emboli are suspected, treatment should include posturing the patient in the left lateral decubitus position and ventilation through an endotracheal tube placed in the contralateral mainstem bronchus if only one side of the chest is injured. Successful surgical therapy to rapidly identify and control the bronchovenous fistula is advocated and incorporates proximal cross-clamping at the lung hilum or, when possible, isolating the injured segment more distally with clamps. Thereafter, a pulmonary resection is usually necessary.

Rupture of the pulmonary artery by a balloon flotation catheter occurs in less than 0.2 percent of patients but produces a mortality of 45 to 65 percent. Caused by high lateral pressures on the vascular wall from the inflated balloon or from erosion of the catheter tip through the artery, a bronchopulmonary artery fistula may produce minor or catastrophic hemothysis. Factors that appear to potentiate rupture are patient age older than 60 years, lung or vascular fragility, anticoagulation, and perhaps pulmonary hypertension. Urgent responses to extensive bleeding include positioning the patient with the lung in which the catheter is located in the dependent position; bronchial intubation to maintain respiration; occlusion of the involved airway with balloon catheters so as to provide tamponade or limit blood spillage to other lung units; pulmonary artery occlusion or embolization; and surgery. Hemorrhage may recur over a several-day period due to formation of a false aneurysm in the pulmonary artery.

Many other vascular fistulas, as shown in Table 5, occur due to the sequence of inflammation/infection producing tissue destruction and penetration. Symptoms of vascular fistulas may be surprisingly chronic with intermittent and often copious hemothysis extending over days to a few years. Pain occurs infrequently and often systemic symptoms of the primary disease or secondary infection predominate. Most fistulas join the lung and aorta and may be suggested by an aneurysm seen on a chest roentgenogram. Aortography is the preferred diagnostic method. Premortem diagnosis has also been made by the following:

### Table 3—Closure Methods for Fistulas

<table>
<thead>
<tr>
<th>Methods</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachoscopy</td>
</tr>
<tr>
<td>Lead fishing weights or shot</td>
</tr>
<tr>
<td>Balloon occlusion (septostomy catheter)</td>
</tr>
<tr>
<td>Tissue adhesive (bucrylate, Histoacryl)</td>
</tr>
<tr>
<td>Silver nitrate to directly visualized stump</td>
</tr>
<tr>
<td>Fistulas</td>
</tr>
<tr>
<td>Fibrin glue</td>
</tr>
<tr>
<td>Autologous blood instillation to form an</td>
</tr>
<tr>
<td>obstructive clot and doxycycline as an</td>
</tr>
<tr>
<td>irritant</td>
</tr>
<tr>
<td>Absorbable gelatin sponge (Gelfanm)</td>
</tr>
<tr>
<td>Thoraoscopy</td>
</tr>
<tr>
<td>Instillation of talc into fistula</td>
</tr>
<tr>
<td>Fibrin glue</td>
</tr>
<tr>
<td>Tissue glue—Histoacryl</td>
</tr>
</tbody>
</table>

urgent intervention is needed, multiple fistulas are present, or full closure techniques fail. Such treatment is directed toward reducing the pressure gradient across the fistula by changing gas pressure at one or both ends of the BPF. This is accomplished either by reducing the force created by positive airway pressure or by altering the subambient pressure in the pleural space. These techniques have been extensively reviewed and are summarized in Table 4. Various combinations of these techniques are also employed but none has been evaluated critically to prove efficacy or a beneficial effect on mortality or morbidity.

### Bronchovascular Fistulas

Fistulas between the lung and blood vessels or the heart usually present clinically with life-threatening hemothysis or air embolization. Therefore, these connections differ histologically from the other fistulas discussed as established tissue tracks do not exist. Two examples of such acute and catastrophic connections include blunt and penetrating wounds to the lung and rupture of the pulmonary artery into the airway as caused by a balloon flotation catheter.

Disruption of airway and vascular structures during blunt or penetrating trauma may introduce air into the pulmonary venous circulation leading to lethal systemic air emboli. The diagnosis of an acute

### Table 4—Treatment to Decrease Gas Flow through a BPF

<table>
<thead>
<tr>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduce positive airway pressure</td>
</tr>
<tr>
<td>Minimize tidal volume, positive end-expiratory</td>
</tr>
<tr>
<td>pressure (PEEP), expiratory retard, inspiratory</td>
</tr>
<tr>
<td>flow, and inspiratory time during mechanical</td>
</tr>
<tr>
<td>ventilation</td>
</tr>
<tr>
<td>Emphasize spontaneous breathing modes of</td>
</tr>
<tr>
<td>ventilation</td>
</tr>
<tr>
<td>Independent lung ventilation via a double-humen</td>
</tr>
<tr>
<td>bronchial tube</td>
</tr>
<tr>
<td>High-frequency jet ventilation</td>
</tr>
<tr>
<td>Change pleural pressure</td>
</tr>
<tr>
<td>Increase or decrease chest tube suction</td>
</tr>
<tr>
<td>Application of PEEP or inspiratory closure</td>
</tr>
<tr>
<td>valves on chest tubes</td>
</tr>
<tr>
<td>Decubitus body position with the fistula</td>
</tr>
<tr>
<td>dependent</td>
</tr>
</tbody>
</table>

484

Thoracic and Extrathoracic Bronchial Fistulas (Powner, Bierman)
Table 5—Bronchovascular Fistulas

<table>
<thead>
<tr>
<th>Fistulas</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atriobronchial fistula without hemoptysis associated with pyogenic mediastinal abscess</td>
</tr>
<tr>
<td>Tubercular lung abscess eroding into the aorta from the left main bronchus producing lethal hemoptysis</td>
</tr>
<tr>
<td>Intermittent hemoptysis from an aortobronchial fistula after placement of a graft for treatment of an aortic aneurysm</td>
</tr>
<tr>
<td>Aortobronchial fistula due to atherosclerotic, syphilitic, or tubercular aneurysms; aortic; coarctation; or prior thoracic aortic surgery</td>
</tr>
<tr>
<td>Fistula between the left upper lobe and left ventricle after resection of an LV aneurysm producing nonfatal hemoptysis</td>
</tr>
<tr>
<td>Superior vena cava–right upper lobe fistula from a hyperalimentation catheter</td>
</tr>
<tr>
<td>False vascular aneurysms after correction of congenital heart anomalies producing lung fistulas</td>
</tr>
<tr>
<td>Azygos vein–right upper lobe fistula from a ventriculocatheter drain to hydrocephalus</td>
</tr>
<tr>
<td>Pulmonary artery–right middle lobe fistula at the site of a necrotic lymph node in chronic lymphocytic leukemia</td>
</tr>
</tbody>
</table>

(1) methylene blue infusion into a venous catheter followed by sputum staining; (2) tracheal instillation of radiopaque contrast with visualization in the heart under fluoroscopy; (3) thoracotomy; and (4) arteriography or venography demonstrating spillage into the lung. Surgical repair of bronchovascular fistulas is usually successful if the patient survives the episode(s) of hemoptysis and the diagnosis can be made.

Bronchial-Subarachnoid Fistulas

Bronchial-subarachnoid fistulas have been rarely reported following radiation therapy, postoperatively, and in patients with lung carcinoma. These patients may present with the nonspecific neurologic changes of confusion and headache presumably due to the observed pneumocephalus. Neurosurgical closure of the fistula has been accomplished in some cases but overall mortality was high due to the underlying tumor and secondary infection.

Conclusion

Fistulas may join the airway with the pleural space or other thoracic and abdominal organs or structures. The manifestations of such abnormal connections may be acute and produce dramatic symptoms. Alternatively, bronchial fistulas may also appear with a chronicity and subtlety that will challenge the best investigative efforts. Few signs are specific and very few diagnostic tests provide the high degree of accuracy needed to forestall the considerable morbidity and mortality fistula cause. Therefore, those physicians responsible for both emergent and continuing patient care must be alert to the possibility of this diagnosis and informed as to their many manifestations.

References

26. Galvin IF, Gibbons JRP, Maghout MH. Bronchopleural fistula:...
a novel type of window thoracostomy. J Thorac Cardiovasc Surg
1988; 96:833-35
27 Eerola S, Virkkula L, Varselaa E. Treatment of postpneumonec-
tomy empyema and associated bronchopleural fistulas. Scand J
28 Powner DJ, Cline CD, Rodman GB. Effect of chest-tube suction
on gas flow through a bronchopleural fistula. Crit Care Med
1985; 13:99-101
29 Bishop MJ, Benson MS, Pierson DJ. Carbon dioxide excretion
via bronchopleural fistulas in adult respiratory distress syn-
drome. Chest 1987; 91:400-02
30 Pregant DJ, Aldrich TK, Fell SC, Heller S, Kamholz SL. The
maintenance of total ventilatory requirements through a chronic
bronchopleural cutaneous fistula. Am Rev Respir Dis 1987;
136:1001-02
31 Rusch WV, Capps JS, Tyler ML, Pierson DL. The performance
of four pleural drainage systems in an animal model of broncho-
32 Baumann MH, Sahn SA. Medical management and therapy of
bronchopleural fistulas in the mechanically ventilated patient.
Chest 1990; 97:721-29
33 Pierson DJ, Horton CA, Bates PW. Persistent bronchopleural
air leak during mechanical ventilation. Chest 1986; 90:321-3
34 Aasebo U. Thoracoscopic closure of distal bronchopleural fistu-
35 Powner DJ. Pulmonary barotrauma in the intensive care unit. J
36 Yee ES, Verrier ED, Thomas AN. Management of air embolism
in blunt and penetrating thoracic trauma. J Thorac Cardiovasc
Surg 1983; 85:661-68
37 Carlson TA, Goldenberg IF, Murray PD, Tadavarthy SM,
Walker M, Gobel FL. Catheter induced delayed recurrent
pulmonary artery hemorrhage. JAMA 1989; 261:1943-45
38 Jondeau G, Lacombe F, Rocha F, Rigaud M, Hardy A, Bourdarias
J. Swan-Ganz catheter-induced rupture of the pulmonary artery.
39 Grazzitti PJ. Atriobronchial fistula: an unusual complication of
intravenous feeding in the presence of a mediastinal abscess.
Anaesthesia 1987; 42:669-70
40 Masjedi M, Davoodian F, Forouzesh M, Abtahi SJ. Bronchomaro-
tic fistula secondary to pulmonary tuberculosis. Chest 1988;
94:199-200
41 Wheeler AP, Loyd JE. Fatal hemoptysis: aortobronchial fistula
as a preventable cause of death. Crit Care Med 1989; 17:1228-
30
42 O'Donnell A, Tsou E, Katz N. Ventriculobronchial fistula: a
rare cause of intermittent massive hemoptysis. J Cardiovasc
Surg 1989; 30:378-80
43 Demey HE, Colemont L, Hartoko TJ, Roodhoft MI, Ysebaert
DK, Bossaert LL. Venopulmonary fistula: a rare complication
of central venous catheterization. JPN 1987; 11:580-82
44 Garniek A, Morag B, Schmahmann S, Rubinstein Z. Aortobron-
45 Isamat F. Bronchovenous fistula as a late complication of a
ventriculooatriostomy. J Neurosurg 1969; 31:574-75
46 Stoller JK, Picker LJ, Weiss ST, Thurer BL, Kasdon EJ.
Pulmonary artery-bronchial fistula complicating chronic lymph-
47 Demeter SL, Cordasco EM. Aortobronchial fistula: keys to
successful management. Angiology 1980; 31:431-35
48 Lerner EJ, Bilaniuk LT. Spontaneous bronchial-subarachnoid
fistula. AJNR 1989; 10:5103
49 Swaid SN, Windham TL, Morawetz RB. Pneumocephalus
secondary to spontaneous bronchial-subarachnoid fistula. Neu-
rosurgery 1983; 13:72-3
A more detailed reference list is available from the authors.

Thoracic and Extrathoracic Bronchial Fistulas (Powner, Bierman)