Lesson 250: PreAnesthetic Assessment of the Patient With Esophageal Rupture

WRITTEN BY:
Peregrina L. Arciga, MD; Adeaye Windokin, MBBS
1 Assistant Professor, Department of Anesthesiology, Charles R. Drew University of Medicine and Science, Los Angeles; Assistant Professor, Department of Anesthesiology, David Geffen School of Medicine, University of California, Los Angeles.
2 Third-year resident, Department of Anesthesiology, King Drew Medical Center, Los Angeles

REVIEWED BY:
Elizabeth A.M. Frost, MD, Department of Anesthesia, Mount Sinai School of Medicine, New York, NY. Date reviewed: January 2006

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NEEDS STATEMENT
Esophageal rupture is a complication that is increasing in frequency, in part because of the expanded use of endoscopic techniques. Any delay in treating these critical patients may be catastrophic. Definitive guidance is scarce for the clinical anesthesiologist who may be called to provide emergency care for these patients. The topic has been identified by committee as important information for anesthesiologists.

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Anesthesiologists

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The term esophageal rupture or perforation was initially used in 1724 by Hermann Boerhaave, who described the classic case of Baron John van Wassenaer, Grand Admiral of Holland. The Baron, who suffered from in digestion, sought to “cleanse the stomach gently” by vomiting, and in the attempt sustained a ruptured esophagus. He died 18.5 hours later, and at autopsy Boerhaave found a tear in the distal esophagus with food material in both pleural cavities. Since that description, more than 200 instances of spontaneous perforation have been reported.

Esophageal rupture is a potentially devastating injury. Rapid diagnosis and therapy provide the best chance for survival. Delays in diagnosis are common, however, which increases morbidity and mortality.

Classification
Dehisence of the esophagus has been variously described. For example, Mallory-Weiss syndrome describes a spectrum of esophageal injury due to laceration or tearing. If it presents as hematemesis or melena that typically occurs after many hours or days of severe vomiting and retching, and is traceable to one or several siltlike lacerations of the gastric mucosa, longitudinally situated at or slightly above the esophagogastric junction. Boerhaave’s syndrome is defined as a spontaneous rupture or perforation of the esophagus. Both esophageal perforation and laceration can be iatrogenic—in particular the result of esophageal instrumentation—but can also occur spontaneously.

An increase in diagnostic and therapeutic endoscopic procedures has made instrumentation the most common cause of iatrogenic esophageal perforation. Instrumentation associated with esophageal intubation, the placement of nasogastric tubes, the placement of an esophageal-tracheal supraglottic ventilatory device, or transesophageal echocardiography can be complicated by esophageal perforation—especially in emergency situations and in patients with anatomic distortions such as hiatal hernia or achalasia.

The term spontaneous rupture of the esophagus had been given to all perforations involving the entire thickness of the esophagus, whenever such perforations were not associated with external trauma, instrumentation, or ingestion of a foreign body. Most lesions result from “overstrain”—most commonly from vomiting, neurogenic (achalasia), or idiopathic causes. The descriptor spontaneous no longer seems tenable, and the term atraumatic panmural rupture of the esophagus can be substituted. The use of panmural or complete distinguishes Boerhaave’s syndrome from Mallory-Weiss syndrome.

Epidemiology
In the United States, Boerhaave’s syndrome occurs rarely, with an estimated incidence of 1 in 6,000+ patients. It accounts for 15% of cases of esophageal rupture or perforation. Iatrogenic

PREANESTHETIC ASSESSMENT

Dr. Elizabeth A.M. Frost, who is the editor of this continuing medical education series, is Clinical Professor of Anesthesiology at Mount Sinai School of Medicine in New York, NY. She is the author of Clinical Anesthesia in Neurosurgery/Butterworth Heinemann, Boston) and numerous articles. Dr. Frost is also past president of the Anesthesia History Association and former editor of the journal of the New York State Society of Anesthesiologists, Sphere. She is also editor of the book series based on this CME program: Preanesthetic Assessment, Volumes 1 through 3 (Birkhauser, Boston) and 4 through 6 (McMahon Publishing, New York City).

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The incidence of perforation was 1.7%.

Iatrogenic perforation is generally caused by foreign bodies or traumatic perforations. Esophageal rupture secondary to blunt external trauma is very rare, with few cases reported worldwide.

### Table 1. Etiology of Esophageal Rupture

<table>
<thead>
<tr>
<th>Causes</th>
<th>Factors</th>
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<tbody>
<tr>
<td>Therapeutic endoscopy</td>
<td>Dilation, hemostasis, stent placement, foreign body removal</td>
</tr>
<tr>
<td>Diagnostic endoscopy</td>
<td>Underlying esophageal lesions, experience level of endoscopist, difficulty of instrumentation</td>
</tr>
<tr>
<td>Spontaneous/barogenic</td>
<td>Vomiting, straining, weight lifting, coughing, childbirth, seizures</td>
</tr>
<tr>
<td>Esophageal pathology</td>
<td>Esophageal ulcer, esophagitis, stricture, hernia, achalasia, carcinoma, infection</td>
</tr>
<tr>
<td>Other</td>
<td>Direct trauma, foreign body</td>
</tr>
</tbody>
</table>

Even with prompt therapy, the mortality rate is very high, varying from 30% to 50%. With a delay in diagnosis, the mortality rate exceeds 90%. Mortality rates from perforation caused by instrumentation (ie, iatrogenic perforation) are lower than from other causes (15%-20%), although they are clearly still significant.

Boerhaave’s disease is generally associated with vomiting—customarily after drinking and eating binges. It is more commonly observed in males than females. Spontaneous esophageal perforation is usually observed in patients aged 40 to 60 years. Boerhaave’s syndrome is very rare in younger patients. Isolated case reports have documented its occurrence in children; 4 cases have been reported in neonates. Some investigations have found that the esophagus is 13 times stronger in neonates and 4 times stronger in children than in adults. Interestingly, perforations usually occur on the right side in neonates, whereas 90% occur on the left side in adults. Iatrogenic perforation is generally associated with a preexisting pathology; thus, it is typically found in patients who are 50 years and older.

### Pathophysiology

The hypopharynx and esophagus are the most common sites of perforation during upper endoscopy primarily because the esophageal wall is thin and, unlike the rest of the alimentary tract, lacks a reinforcing serosal layer, which usually contains collagen and elastic fibers. Thus, the wall is weaker and may rupture at a lower intraluminal pressure. Vomiting usually precedes the development of spontaneous perforation. In addition, the blood supply to the esophageal wall is tenuous, making the esophagus even more prone to perforation. Because the esophagus is surrounded by loose intramural connective tissue, an infectious and inflammatory response can disseminate easily to nearby vital organs, thereby making the esophageal perforation a medical emergency.

More than 90% of cases, perforation occurs in the lower third of the esophagus; most frequently, the tear is in the left posterolateral region (90%). The prediction for left-side perforations may be caused by the lack of adjacent supporting structures, thinning of the musculature of the lower esophagus, and anterior angulation of the esophagus at the left diaphragmatic crus. Tears usually extend superiorly. Perforation of the esophagus resulting from instrumentation or trauma does not appear to have specific site predilection.

### Etiology

The cause of esophageal perforation in the case presented could have been either from spontaneous perforation secondary to vomiting and retching during attempts to evacuate the esophagus, or from the endoscopic instrumentation performed to remove the foreign body (food bolus) from the esophagus. An initial tear secondary to retching also may have been worsened by instrumentation (Table 1).

### Instrumental Perforation

Diagnostic endoscopy, performed almost exclusively with flexible fiber-optic endoscopes, carries a low risk for perforation. Therapeutic interventions such as dilatation, hemostasis, stent placement, or foreign body removal can dramatically increase the risk for perforation. Most endoscopic perforations occur in patients undergoing therapeutic endoscopy with the presence of underlying esophageal lesions. Difficult instrumentation, the experience level of the endoscopist, and the presence of traumatic bleeding are additional risk factors for perforation.

### Noninstrumental Perforation

Spontaneous or high-pressure rupture of the esophagus can be associated with esophageal pathologies such as esophageal ulcers, peptic esophagitis, strictures, hiatal hernias, achalasia, carcinoma, and foreign bodies. Candidiasis, herpetic and immunodeficiency infections, and severe reflux esophagitis are also causes of pathologic perforations. Regarding the present case, it was conjectured that the esophagus may have been weakened by chronic inflammation, peptic esophagitis, alcoholism, or poor nutrition and thus had become more susceptible to rupture. In acute cases, rupture caused by pressure exerted from retching or vomiting against a closed upper sphincter seems to be plausible.

Vomiting is the most frequent cause of barogenic rupture of the esophagus (eg, Boerhaave’s syndrome), but it is certainly not the only cause; this has led to some debate about the name of the syndrome. Diverse etiologies have been reported for Boerhaave’s syndrome, including straining, weight lifting, severe coughing, childbirth, blunt trauma, seizures, and ulcer’s ulcers from central nervous system disease, seaseass, postoperative vomiting, esophagitis with perforated ulcer, asthma, smooth muscle hypertrophy of the esophagus, and use of the drug disulfiram. When taken by alcoholics who are attempting to stop drinking, disulfiram causes violent vomiting if alcohol is consumed. Rupture of the esophagus has occurred after ingestion of a mushroom that contains a chemical similar to disulfiram.

Spontaneous rupture of the esophagus is never truly spontaneous in that there is a precipitating factor. The phrase does connote, however, that the rupture is not caused by direct trauma, foreign body, or instrumentation. The term atraumatic panural rupture of the esophagus distinguishes Boerhaave’s syndrome from atraumatic tear of the esophagus, which is the Mallory-Weiss tear. Most frequently, the causative mechanism is a rapid rise in intraluminal pressure with sudden distention of the distal esophagus. Protracted vomiting leads to fatigue of the vomiting center in the sensory nucleus of the vagus nerve in the floor of the fourth ventricle. The result is discoordination of the vomiting reflex—a complex act requiring the synchronous relaxation and contraction of many voluntary and involuntary muscles. Physiologic obstruction occurs because of a failure of relaxation of the upper and lower esophageal sphincters at the moment of greatest propulsive force. Cadaver studies have shown that it is not the total pressure exerted but the rapid rise that bursts the esophagus. In fact, pressures up to 9.63 psi have been achieved with hydrostatic dilators in the distal esophagus without damage.

The distal part of the esophageal wall is essentially the same with Mallory-Weiss syndrome, although it is a mucosal tear manifested by profuse bleeding because of the rich vascularplexus that is also torn. The remainder of the wall remains intact.

### Clinical Manifestations

Vomiting is the most striking feature—characteristically exercit- ing and poorly relieved by narcotics (Table 2). In approximately...
50% of cases, the classic sequence of forcible vomiting, mild hematemesis, and subternal chest pain present. Left chest pain is usually pleuritic and may radiate to the epigastrium, subternal area, or back. In 4 out of 12 cases in one series, there was left shoulder pain caused by phrenic nerve irritation of the left hemidiaphragm. The pain may increase during swallowing, which may cause coughing if there is a pleural tear. Occasionally, the patient collapses with the onset of severe pain. Vomiting usually ceases with the onset of pain, or there may be no actual vomitus but the patient experiences retching. Dyspnea is usually present and respi- rations are typically rapid and shallow because of severe pain and splintering. Hydropneumothorax or tension pneumo- thorax may contribute significantly to respiratory impair- ment. Patients often complain of extreme thirst as seen in hypo- volemic states. Some degree of peripheral collapse occurs in two thirds of cases. Patients are typically anxious, cool, clam- my, slightly cyanotic, and tachycardic, from a combination of hypovolemia and possible bacteremia. Blood pressure may fall and patients may become mildly febrile in the early stages, or vital signs may be within normal limits within the crucial first 15 minutes of the occurrence of the mediastinal pleura. A very common clinical picture is that of an acute abdo- men with marked reflex rigidity of the upper abdominal mus- cles, epigastric tenderness and rebound, and absent peristalsis. Indeed, the most common misdiagnosis is that of a perforated duodenal ulcer. A key point is that the dis- ease progresses, the abdominal signs diminish in proportion to the increasing chest signs—but hopefully the clinician does not lose valuable time waiting for this progression. Another clinical presentation is that of spontaneous pneu- mothorax with all the characteristic signs, such as tracheal deviation, hyperresonance, loss of retrosternal dullness, and decreased breath sounds. Also, the clinician must be aware that physical examination of the chest may be completely normal in the early stages of Boerhaave’s syndrome. Palpable crepitus in the suprasternal notch or neck is frequently overlooked or late in appearance, although it is present in roughly two thirds of cases. This important clini- cal finding is emphasized in the tetrad of Goff, which includes chest pain, respiratory distress, and prostration. Hamman’s sign, an auscultatory mediastinal crunch, is heard in 20% of cases and can be mistaken for the friction rub of pericarditis. Mackler’s triad (vomiting, chest pain, and subcutaneous emphysema) is uncommon and thus unreliable for diagnosis.

**Diagnosis**

**Laboratory Studies**

Laboratory tests offer little help in the diagnosis of esophageal perforation because initially the results are nor- mal after a perforation. Probably the most common find- ing—although it is nonspecific—is leukocytosis. While leukocytosis is present in the majority of cases, 14 of 47 patients described by Abbott et al had white cell counts less than 10,000, although 7 of those 14 had a leftward shift (increased number of immature neutrophils or band forms, which suggested acute inflammation) in the differential count. One half of the patients in this group had a hematocrit level greater than 50% as a result of volume contraction. Acidose secondary to incipient sepsis may develop.

The amylase level of the pleural effusion would be very high if measured because the effusion is mainly swallowed saliva. This can mimic acute pancreatitis, which also includes a left pleural effusion as part of the disease process. Saliva is which is rapidly obtainable, yields the most information. The most common error in diagnostic evaluations is the failure to obtain a chest X-ray when an esophageal perforation pre-

sents as an acute abdomen. Chest radiographs may be high- ly suggestive of perforation; however, radiographs are normal in 10% to 15% of cases. Almost 80% of cases have chest X-ray findings of a left pneumothorax plus an effusion. In at least 90% of cases, there is an effusion with or without pneumothorax (which is bilateral in 7% of the time). The pres- ence of mediastinal air must be determined; it is found in 66% of cases. Air may be retrocardiac and easily missed; widening of the mediastinum is occasionally seen. The so-called V sign of Naclerio, another subtle X-ray finding that is easily over- looked, represents air in the fascial planes of the medi- astinum and diaphragmatic pleura behind the heart. It is both an early and reliable finding when it occurs. Subcutaneous emphysema in the soft tissues of the neck or chest wall may be seen. The mediastinum may be shifted, especially if there is a tension pneumothorax. On rare occasions, atelectasis alone secondary to splitting of the chest wall is the only radi- ographic finding. Although the results of chest radiographs are abnormal in as many as 97% of cases, the diagnosis of perforation can only be made in approximately 25%.

Esophagography with barium or water-soluble contrast material (eg, diatrizoate meglumine–diatrizoate sodium solu- tion)—readily available in most emergency departments—results in positive findings in less than 75% of cases. Therefore, a lack of extravasation does not necessarily exclude a finding of perforation. In this false negative group, a key point is that if there is still a high index of suspicion of esophageal perforation after negative contrast study results. However, endoscopy plays a very limited role in the diagnosis of esophageal perforation because it is time-consuming, somewhat risky, and general- ly unnecessary for the diagnosis, and for the most part, patients are too ill and unstable to undergo the procedure. False-negative esophagograms are thought to occur because either either the material (eg, barium) is too viscous to leak out, passage of the material is too rapid (eg, diatrizoate megu- mine–diatrizoate sodium solution), or the perforation is blocked by edema, clots, or food particles. If anteroposteri- or and lateral projections are negative, oblique views should be obtained to ensure that a leak is not hidden by contrast material within the lumen.

There is considerable controversy regarding which medi- um is preferable—barium or water-soluble contrast. Some, including Abbott and van Heerden, believe that barium is the material of choice, while others contend that it is irritating to the mediastinum and pleura and is difficult to remove at surgery. In general, use of a water-soluble contrast agent (eg, diatrizoate meglumine–diatrizoate sodium solution) is preferred initially because barium can cause an inflammatory response in the mediastinum. As the contrast agent extrav- asates into the mediastinum, the barium crystals become coated with a fibrin membrane, followed by fibrosis and gran- uloma formation. Swallows of diatrizoate meglumine–diatrizoate sodium are useful for confirming a diagnosis of esophageal perforation, but the swallowing must be done in left and right decubitus positions. Even when the procedure is properly performed, the rate of false-negative results is 10% to 25%. Because diatrizoate meglumine–diatrizoate sodium solution can cause severe pneumonitis, barium should be used if this diagnosis is thought to be likely.

Barium is also preferred if there is suspicion of a tracheo- esophageal fistula. If an examination with diatrizoate meglu- mine–diatrizoate sodium is negative, dilute barium should be considered because it has better coating properties and is more sensitive in the detection of smaller leaks. The size of the rent in the X-ray can be very misleading, and the clinician should not defer surgery on grounds that the perforation is small. One small perforation seen on X-ray measured 8 cm in length at (foracotomy/)

Computed tomography (CT) of the chest can be useful in selected patients in whom contrast radiography fails to con- firm the diagnosis. The diagnosis of perforation is confirmed by CT if the scan shows air in the mediastinum, abscess cavi- ties adjacent to the esophagus, or an actual communication of an air-filled esophagus with an adjacent mediastinal air- fluid collection. CT studies can be particularly helpful in showing mediastinal changes when the perforation has already sealed.

### Table 2. Clinical Signs and Symptoms

<table>
<thead>
<tr>
<th>Symptom</th>
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<tbody>
<tr>
<td>Pain</td>
</tr>
<tr>
<td>Retching/vomiting</td>
</tr>
<tr>
<td>Dyspnea</td>
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<tr>
<td>Pneumothorax</td>
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<tr>
<td>Thirst</td>
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<tr>
<td>Anxiety</td>
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<tr>
<td>Mild cyanosis</td>
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<tr>
<td>Hypotension</td>
</tr>
<tr>
<td>Hypotension</td>
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<tr>
<td>Rigidity of abdominal muscles</td>
</tr>
<tr>
<td>Palpable crepitus</td>
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Other Studies
Esophagoscopy can often be used to visualize perforations if they are iatrogenic.

Differential Diagnosis
The incidence of diagnostic error can be as high as 50%.
Perforated peptic ulcer is the most frequent misdiagnosis, but can be distinguished from esophageal perforation by a history of ulcer, pneumoperitoneum, and a gradual increase in the severity of chest symptoms in most cases compared to abdominal findings. Both perforated organs require surgery, but ulcer disease requires laparotomy while esophageal perforation is best approached transthoracically.

The second most common diagnostic error is the confusion of esophageal rupture with acute myocardial infarction. The hazards of an unnecessary thoracotomy in the face of an acute myocardial infarction are obvious and underscore the need for an electrocardiogram in the initial evaluation of suspected esophageal ruptures. It is also helpful in recognizing pericarditis and pulmonary embolus, which can mimic esophageal rupture to some degree. Hammond's mediastinal crunch can sound like a pericardial friction rub. In addition, patients with esophageal rupture may have pain when sitting forward as with pericarditis. The dyspnea, tachycardia, cyanosis, chest pain, and circulatory collapse of pulmonary embolus bear striking similarity to the manifestations of esophageal rupture and represent a dangerous pitfall. Other types of thoracic disease such as dissecting aortic aneurysm (usually in hypertensive patients and associated with loss of peripheral pulses) and spontaneous pneumothorax (rarely associated with severe pain, vomiting, or subcutaneous emphysema) should be ruled out, since thoracotomy plays little role in their management.

Acute pancreatitis is often associated with alcohol abuse, vomiting, left pleural effusion, and chest and abdominal pain. A normal serum amylase level makes pancreatitis unlikely; however, high serum amylase has been reported in only 1 case of esophageal perforation.

Other abdominal processes to be considered are biliary colic, mesenteric vascular accident, rupture of the subphrenic abscess, acute pylonephritis, and incarcerated diaphragmatic hernia. One can readily appreciate the value of a limited investigation of the upper gastrointestinal tract with contrast material in the emergency room to quickly separate esophageal perforation from this array of more common disease entities.

Patient Management
Patients with esophageal perforation are frequently hemodynamically unstable. Because the airway may be compromised, full resuscitative facilities should be readily available at bedside. As soon as the diagnosis is considered, an investigation should be initiated to ascertain the site and extent of the rupture. A tube thoracostomy may be urgently used to decompress the chest. The fluid removed is usually stomach contents, and occasionally pus—especially if a delay in diagnosis has occurred. An emergency cardiothoracic surgical consultation must take place.

The management of esophageal perforation is either surgical or medical. Surgery has been the most common approach for the treatment of esophageal perforation, but in selected cases medical management of the patient may give better results. Regardless of the type of therapy chosen, it has been shown repeatedly that delays in diagnosis and treatment are associated with increased morbidity and mortality.

Nonsurgical management of esophageal perforations has been associated with a 20% to 38% mortality; however, in carefully selected patients, the mortality rate can approach zero. The difficulty with nonsurgical management is the prospective identification of perforations that will remain contained versus those that will cause ongoing contamination with subsequent uncontrolled infection.

The selection criteria for medical treatment as reported by Cameron et al were disruption contained in the mediastinum, minimal symptoms, and minimal signs of sepsis. A poor clinical state may also be a reason for medical treatment. Medical management consists of the following: intravenous fluids, broad-spectrum antibiotics (eg, imipenem-cilastatin), nasogastric suction, fasting status, and adequate drainage with tube thoracostomy. The early use of nutritional supplements is helpful.

Barrett described the first successful surgical repair of the esophagus in 1947. Surgery, including primary repair, exclusion and diversion, or resection (esophagectomy), remains the mainstay of treatment. Surgical repair within 12 to 24 hours is associated with a mortality rate of 34%, while a delay of 24 hours is associated with 64% mortality. Direct repair of the rupture and adequate drainage of the mediastinum and pleural cavity provide the best survival rates. Surgery in these patients usually consists of initial rapid resuscitation followed by primary repair of the esophageal tear with interrupted resorbable sutures with reinforcement by intercostal flap. Thoracic esophageal perforation repairs can be reinforced by an autologous pleural flap or by pedicle muscle flaps from the intercostal muscles, chest wall musculature, diaphragm, or a mobilized pedicle of omentum. Reinforcement with vascularized tissue may decrease the rates of fistula formation and mortality (13% and 6%, respectively) compared to those of unreinforced primary repair (39% and 25%, respectively).

Depending on the site of the lesion, either a left or right thoracotomy is usually performed, followed by pleural and mediastinal lavage with debridement of necrotic tissue, and insertion of pleural and mediastinal drains. A mini-laparotomy is also performed for a decompressive gastrostomy and a feeding jejunostomy. In patients operated on within 24 hours of rupture, primary closure and wide drainage of the mediastinum are effective, and are recommended as the gold standard for treatment. Alternative surgical techniques include metallic stent, esophagectomy, controlled fistula with a T-tube, well-vascularized pedicle tissue flap using omentum, fundoplasty, and esophageal exclusion.

Anesthetic Care
An electronic search of the literature did not reveal specific directions for anesthetic management of this condition. The administration of anesthesia to the patient with esophageal rupture must take into account the criticality of the situation. Morbidity and mortality increase exponentially with treatment delay. Rapid resuscitation through large-bore cannulas with intravenous warm fluids must be started before anesthesia induction. The administration of colloids will quickly restore intravascular volume. Blood products should be administered as soon as possible.

During the induction of anesthesia, the anesthesiologist should consider the high risk for aspiration in the patient. Any increase in intra-abdominal pressure should...
be avoided, to prevent further contamination through the esophageal rupture. The process of induction should be smooth. Coughing and straining should be avoided, as they may increase the chances of further tearing in tissue that is already inflamed and friable. The insertion of a nasogastric tube is not recommended, as any instrumentation can aggravate the injury to the esophagus or pass through the rent in the esophagus. A rapid-sequence induction is mandatory. Securing the airway during the awake state is a possible alternative but should be attempted cautiously because retching, vigorous coughing, or straining during the procedure may aggravate the esophageal tear. These patients are also often volume depleted and in the early stages of septic shock, and may become hemodynamically unstable with any reduction in afterload.26 Etomidate and ketamine are suitable induction agents. Even so, hypotension may still occur and hydration prior to induction of anesthesia should always be administered. Intraoperative muscle relaxants should be readily available.31

The choice of muscle relaxants for intubation is between succinylcholine and a rapidly acting nondepolarizer. A theoretical disadvantage of succinylcholine is that it may increase intra-abdominal pressure caused by fasciculations, and thus increase the risk for reflux through the esophageal perforation. A defasciculating dose of nondepolarizer does not reliably attenuate such a rise in intra-abdominal pressure. Rocuronium provides excellent intubating conditions within 25 to 35 seconds and is a reasonable alternative. The onset of maximum single twitch depression after the administration of 3 to 4 times ED₉₅ of rocuronium resembles the onset of action of intravenous succinylcholine at 1 mg/kg. However, if there is any doubt about the patient’s airway, or if the airway has to be secured as fast as possible, succinylcholine is still the gold standard.

Cricoid pressure (Sellick’s maneuver) has been associated with esophageal rupture, and its use during rapid sequence induction and intubation in patients with esophageal rupture is controversial.32,33 The risk for worsened contamination through the esophageal perforation if reflux occurs should be weighed against the risk for contamination of the lungs in a patient who already has one lung damaged.

Most esophageal repairs involve a thoracotomy. Lung separation is usually required to facilitate surgical access and can be performed by the use of a double lumen tube, or a single lumen tube with a bronchial blocker.34 The choice of endotracheal tube depends on the patient’s airway anatomy and the anesthesiologist’s preference, and does not matter as much as the skill of the operator. Proper confirmation of placement should be performed using a fiber-optic bronchoscope.33

Monitoring should involve standard American Society of Anesthesiologists’ monitors, the cannulation of an artery both for continuous blood pressure monitoring and frequent blood gas analyses, and insertion of a central venous catheter to aid assessment of fluid status, and for infusion of inotropes. A urinary catheter is essential to monitor urinary output. A warming blanket, fluid warmer, and a heat moisture exchange device should be used to maintain body temperature. Special attention is required during retrosternal manipulation because direct pressure on the heart may produce arrhythmias or hypotension.

Major blood loss is anticipated in these patients. Serial hemoglobin and hematocrit measurements can provide an accurate assessment of blood loss. Appropriate measures should be taken to correct blood volume by the infusion of colloids, crystalloids, and blood components. Various factors—including prolonged surgery, lung congestion, large fluid shifts, hypothermia, a long surgical incision, and pain—may cause postoperative hypoventilation, hypoxia, and atelectasis. Elective postoperative mechanical ventilation is suggested in these patients.

### Postanesthetic Care

Postoperatively, these patients are usually monitored in the intensive care unit because they are highly prone to cardiorespiratory complications. If there is any doubt about the adequacy of respiratory exchange, mechanical ventilation should be maintained until an improvement in respiratory status prior to extubation. Postoperative care requires meticulous attention to intravenous fluid replacement, continuation of antibiotics, and continuous drainage of the pleural and mediastinal spaces as necessary. Postoperative care also includes nasogastric tube decompression of the stomach until postoperative ileus resolves, after which enteric feeding is slowly advanced through a jejunostomy or Duboff tube. Early feeding can also be given parenterally. An esophagogram is obtained 5 to 10 days postoperatively to document the absence of leaking and allow oral intake. Long-term surveillance for evidence of sticture formation, reflux, or carcinoma is also recommended.

### Management of the Case

On preanesthetic assessment, the patient was noted to be obese (109 kg) and edentulous, with a Mallampati classification of III. He was classified as American Society of Anesthesiologists physical status IV-E, secondary to longstanding hypertension, uncontrolled diabetes, esophageal perforation with pneumothorax and tracheal deviation, and esophageal moniliasis with mediastinitis.

After routine monitors were attached, an epidural catheter for postoperative pain management was inserted with the patient in a sitting position. Despite a Mallampati classification of III, the patient had good mouth opening, compliant mandibular space, and adequate neck mobility. After appropriate oxygenation, rapid-sequence induction of anesthesia was performed with etomidate and succinylcholine. In the presence of 2 anesthesiologists, endotracheal intubation was performed with a 41-French double-lumen tube. Correct positioning was confirmed by the differential inflation/deflation/auscultation method, in addition to the use of fiber optics. The use of fiber optics for confirmation, although perhaps not a “standard of care” with the use of double-lumen tubes, has revealed that the incidence of malpositioning using differential clamping and auscultation is between 37% and 36%. Difficulties with the use of double-lumen tubes (eg, deflation of the nondependent lung, ventilation of the dependent lung, or separation of the 2 lungs) occur in up to 25% of cases.41

An arterial cannula was inserted for continuous arterial blood pressure monitoring and blood gas sampling. The left subclavian line was connected to a transducer for central venous pressure monitoring. Hourly blood glucose monitoring was performed. Intravenous insulin was administered to maintain the blood glucose level below 150 mg/dL. Anesthesia was maintained using oxygen in isoflurane, with intermittent doses of fentanyl for analgesia and rocuronium for muscle relaxation. Intermittent doses of bupivacaine 0.25% were given via the epidural catheter to supplement the general anesthetics.

Surgery, which consisted of a left thoracotomy with repair of the esophageal perforation, feeding jejunostomy, decompression gastrostomy, and replacement of a chest tube, was performed uneventfully. Thereafter, the patient was placed in a supine position. A blood gas analysis indicated a PaO₂/FiO₂<200 (PaO₂ of 134 mm Hg on FiO₂ of 100%). It was decided to continue endotracheal intubation and ventilation of the patient. The double-lumen tube was exchanged for a single-lumen tube. The patient was transferred to the intensive care unit.

Postoperatively, the patient’s course was turbulent because respiratory failure developed, and there was a need for inotropic support. He made a gradual recovery and was discharged home on the 27th postoperative day.

### Conclusion

In the absence of clear treatment guidelines for these patients and based on our experience in this case, it is suggested that anesthetic considerations include measures to avoid aspiration and further aggravation of the esophageal tear. In addition, the early, rapid, and continued resuscitation from a morbid inflammatory condition is recommended.
Lesson 250: PreAnesthetic Assessment of the Patient With Esophageal Rupture

Post-test

1. Which of the following is a true statement regarding esophageal perforation?
a. The incidence of Boerhaave’s syndrome in the United States is about 1:1,000 patients.
b. At least 80% of cases show a right pneumothorax plus effusion.
c. A hemoglobin less than 8 g/dL

d. all of the above

2. Factors that increase the risk for esophageal perforation:
a. Chest radiographs are abnormal in only 15% to 20% of cases
b. Chest radiographs are abnormal in only 15% to 20% of cases
c. Increased serum amylase

d. leukocytosis

d. all of the above

3. Important clinical findings with esophageal rupture include:
a. Thrombocytosis
b. Thrombocytosis
c. Increased serum amylase

d. leukocytosis

d. all of the above

Lesson 250 continued from page 57

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