

Esophageal Perforations: New Perspectives and Treatment Paradigms

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Despite significant advances in modern surgery and intensive care medicine, esophageal perforation continues to present a diagnostic and therapeutic challenge. Controversies over the diagnosis and management of esophageal perforation remain, and debate still exists over the optimal therapeutic approach. Surgical therapy has been the traditional and preferred treatment; how-

ever, less invasive approaches to esophageal perforation continue to evolve. As the incidence of esophageal perforation increases with the advancement of invasive endoscopic procedures, early recognition of clinical features and implementation of effective treatment are essential for a favorable clinical outcome with minimal morbidity and mortality. This review will

attempt to summarize the pathogenesis and diagnostic evaluation of esophageal injuries, and highlight the evolving therapeutic options for the management of esophageal perforation.

Key Words: Esophageal trauma, Esophageal perforation, Minimally invasive thoracoscopic surgery, Esophageal stenting, Esophageal endoclippling.

J Trauma. 2007;63:1173–1184.

PATHOPHYSIOLOGY

External Trauma

Penetrating esophageal trauma occurs mainly in the cervical esophagus, and morbidity is usually associated with vascular, tracheal, and spinal cord injuries.^{1,2} Hirshberg et al. evaluated 34 patients with transcervical gunshot wounds, and found 2 patients (6%) with esophageal injuries.¹ Similarly, Demetriades et al. examined 97 patients with transcervical gunshot wounds, and reported injury to the aerodigestive tract in 6% of patients.² Sheely et al. documented a 22-year experience in over 700 patients with penetrating neck trauma, and found 39 patients (5.5%) with cervical esophageal injury.³ Of these patients, there were 55 associated injuries, with trachea being the most often injured structure (Table 1). In contrast, the thoracic esophagus is less vulnerable to external trauma because of its central anatomic location and small compact size. Cornwell et al. reported that intrathoracic esophageal injuries occurred in 0.7% of 1,961 patients with gunshot wounds to the chest.⁴ A retrospective multi-institutional study during a 10.5-year period involving 405 patients with known penetrating esophageal injuries showed that the predominant mechanism of injury was gunshot or shotgun wounds in 78.8% of patients and stab wounds in 18.5% of patients.⁵ Cervical esophageal injuries were most common (57%), and thoracic and abdominal esophageal injuries occurred in 30% and 17%, respectively. Combined

injuries to the esophagus occurred in 4% of patients, and the overall mortality rate was 19%.⁵

Esophageal perforation from external blunt trauma is an exceedingly rare event. The most common cause is related to high-speed motor vehicle crashes.^{6–9} Beal et al. examined 96 reported cases of blunt esophageal trauma, and found that the site of perforation occurred in the cervical and upper thoracic esophagus in 82% of patients.¹⁰ The clinical outcome of these patients is influenced by the delay in diagnosis, complications from the esophageal perforation, and associated injuries. Because the signs and symptoms of early esophageal perforation are subtle, accurate and prompt diagnosis can be challenging in patients presenting with multiple blunt injuries. Therefore, a high index of suspicion is required when treating patients sustaining high-energy trauma to the neck or torso.

Spontaneous Esophageal Rupture (Boerhaave's Syndrome)

In 1724, Dr. Hermann Boerhaave described a case of spontaneous esophageal rupture in the autopsy of Baron von Wassenauer, Grand Admiral of the Dutch Fleet. Boerhaave's patient was a previously robust admiral, who developed a sudden excruciating chest pain while straining to vomit after indulging himself with food and wine. The details of Boerhaave's findings have been recorded in the report of Derbes published in 1955.¹¹ The incidence of Boerhaave's syndrome is relatively rare, although likely an underreported phenomenon, with 300 cases reported in the literature worldwide.¹² Boerhaave's syndrome is thought to arise from a rapid increase in intraluminal esophageal pressure through a patent lower esophageal sphincter during vomiting. This sudden increase of intraluminal pressure is the result of failure of relaxation of the cricopharyngeal muscle. As such, transmural rupture of the esophageal wall occurs, commonly in the left posterolateral wall of the lower third esophagus located 2 cm

Submitted for publication December 27, 2006.

Accepted for publication March 13, 2007.

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DOI: 10.1097/TA.0b013e31805c0dd4

Table 1 Associated Injuries in Penetrating Cervical Esophageal Trauma

Associated Injuries	Number
Trachea	20
Spinal cord	6
Thyroid	7
Thyroid cartilage	5
Jugular vein	4
Recurrent laryngeal nerve	4
Common carotid artery	3
Cricoid cartilage	3
Lung	2
Larynx	1
Total	55

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to 3 cm proximal to the gastroesophageal junction. This area is structurally and inherently weakened as the longitudinal muscle fibers taper out before passing onto the stomach wall.

Foreign Body

Barber et al. described a variety of objects and materials that were inappropriately ingested, especially by children and individuals with psychiatric disorders.¹³ The common offenders are chicken or fish bones, partial dentures, plastic eating utensils, and metal safety pins. Objects less than 2 cm in size may traverse the normal adult esophagus without problems.¹⁴ Once in the stomach, most objects can pass through the rest of the gastrointestinal tract. About 80% to 90% of ingested foreign bodies pass the gastrointestinal tract spontaneously.¹⁵ Of those that mandate intervention, less than 1% need open surgery, whereas the remainder can be removed by endoscopic manipulation.¹⁵ Lam et al. retrospectively reviewed 5,848 patients with foreign body ingestion, and found 8 patients (0.001%) with esophageal perforation secondary to bone impaction in the cervical esophagus; 3 patients presented with clinical evidence of esophageal injury at the time of presentation, and 5 patients were diagnosed with iatrogenic perforation during esophagoscopy.¹⁶

In some instances, sharp or jagged foreign bodies lacerate the wall partially or completely. Most commonly, such laceration or perforation occurs in the normal anatomic narrowings of the esophagus. These narrowing points typically occur at the cricopharyngeal muscle of the upper esophageal sphincter, level of aortic arch, left mainstem bronchus, and lower esophageal sphincter. Up to 5% of patients may present with acute airway obstruction when the foreign body is impacted near the upper esophageal sphincter, causing compression of the trachea.¹⁷

Iatrogenic Esophageal Perforation

The diagnostic and therapeutic contributions of endoscopic procedures during the last two decades have made

instrumentation as the most common cause of esophageal perforation, accounting for 59% of patients in recent series.¹⁸ Esophageal perforation during upper endoscopy is estimated to occur at a frequency of 0.03%, compared with 0.11% during rigid endoscopy.^{19,20} The more common sites of iatrogenic esophageal perforation are at the normal anatomic narrowings of the esophagus. Perforation most often occurs in the hypopharynx or cervical esophagus secondary to exertion of force in attempting to pass the endoscope through the cricopharynx.²¹ The risk of esophageal injury also increases when therapeutic manipulations are undertaken during endoscopic intervention. Perforation of the distal esophagus is most frequently related to esophageal dilation performed for esophageal strictures or achalasia. The incidence of distal esophageal rupture after pneumatic dilation for achalasia ranges from 2% to 6%.²² Such a complication is often associated with previous pneumatic dilation or the use of high inflation pressures.²³

Endoscopic sclerotherapy for esophageal varices leads to esophageal perforation in 1% to 3% of patients.²⁴ Salo et al. reported esophageal perforation and mediastinal gas gangrene after sclerotherapy of bleeding from a Mallory-Weiss laceration.²⁵ The local necroinflammatory reaction after sclerosant injection contributes to the transmural necrosis of esophageal wall.^{26,27} Avoidance of perforation may be achieved by attempting to control the depth, volume, and concentration of injection to prevent extensive and prolonged ischemia to the surrounding tissue.²⁸

Perforation of the esophagus can result from a variety of other forms of endoscopic instrumentation. Iatrogenic esophageal perforation during transesophageal echocardiography has been well documented with an incidence of 0.18%.²⁹⁻³¹ Rarely, rupture of distal esophagus may occur with improper placement and inflation of the gastric balloon of a Sengstaken-Blakmore tube to control bleeding esophageal varices.^{27,32} Other forms of esophageal intubation associated with perforation include nasogastric tube placement,³³ endotracheal tubes,^{34,35} endoscopic retrograde cholangiopancreatography,³⁶ and endoscopic ultrasound-guided interventions.^{37,38}

Esophageal injuries during surgical procedures in close proximity to or directly involving the esophagus occur infrequently. Operative procedures associated with esophageal injuries include fundoplication, vagotomy, hiatal hernia repair, lung transplantation, pneumonectomy, thyroid resection, tracheostomy, thoracic aneurysm repair, esophageal leiomyoma enucleation, mediastinoscopy, and cervical spine surgery.³⁹⁻⁴⁷ When injury to the esophagus is recognized intraoperatively, direct primary repair is almost always successful with minimal morbidity.

Chemical-Induced Esophageal Perforations

Most of the chemical-induced esophageal injuries occur from accidental ingestion of caustic agents by young children, usually under 5 years of age. Occasionally, adults may ingest these agents in an attempted suicide. The severity and site of caustic esophageal injury depend not only on the

nature, amount, and concentration of substance ingested, but also on the duration of mucosal contact.^{48,49} Because of a relative delay in transit time, the anatomic narrowings of the esophagus are most susceptible to caustic burns. Spitz and Lakhoo reported that caustic injury results in a hypotensive lower esophageal sphincter with constant reflux, leading to a prolonged exposure of the distal esophagus to ingested substances.⁵⁰ The greater period of contact in the lower esophagus reflects the more extensive injury occurring in this area.

Although alkaline and acid ingestion are the most common chemicals implicated in caustic burns, there are several important fundamental differences between alkali and acid injury.^{51,52} Alkali ingestion causes more esophageal than gastric injury, but the converse is true for acid ingestion. Alkali causes liquefactive necrosis, causing a deep burn, whereas acid produces coagulative necrosis, forming an eschar that limits tissue penetration.⁵³ Alkali ingestion induces in pylorospasm with regurgitation of the caustic agent into the esophagus, followed by cricopharyngeal muscle spasm and propulsion back into the stomach, aggravating both the esophageal and gastric burns.⁵⁴ On the other hand, acid passes through the esophagus more quickly than alkalis. In the stomach, acid also triggers immediate pylorospasm, pooling the acid in the distal antrum and producing severe gastritis that may progress within 24 hours to 48 hours to full-thickness necrosis and perforation.

Caustic injury to the esophagus has been divided into three phases.^{50,51} The initial phase is characterized by inflammation, edema, and necrosis during the initial few days after injury. This is followed by sloughing of esophageal debris with mucosal ulceration, accompanied by the development of granulation tissue and collagen deposition, and subsequent re-epithelization lasting about 3 weeks to 4 weeks. During this second phase, the esophageal wall is weakest and prone to perforation. In the third phase, cicatrization and stricture formation may progress for many weeks as the destroyed esophageal submucosa and muscularis are replaced with scar tissue.

CLINICAL PRESENTATION

The signs and symptoms of early esophageal injury can be vague and nonspecific. Therefore, a high index of suspicion is critical to avoid delays in establishing an accurate diagnosis. The clinical presentation depends on the cause, location of the injury, size of the perforation, degree of contamination, length of time elapsed after injury, and presence of associated injury.⁵⁵ Nesbitt and Sawyers reviewed esophageal injuries from all causes during a 50-year period, and found pain to be the most common symptom (71%), followed by fever (51%), dyspnea (24%), and crepitus (22%).⁵⁶ Other investigators have also reported similar trends.^{57,58} Dissection of air along the subcutaneous planes or into the mediastinum is a hallmark of esophageal perforation. Occasionally, a systolic crunching sound, the "Hammon's sign", can be heard over the cardiac apex and left sternal

border. Subcutaneous emphysema after thoracic esophageal perforation is detected by palpation in 30% of patients, whereas emphysematous crepitus in the neck after cervical esophageal perforation is detected by palpation in 60% of patients.⁵⁹ Interestingly, subcutaneous emphysema, chest pain, and vomiting constitute the Mackler's triad, a pathognomonic sign for spontaneous esophageal rupture observed in less than half of the cases.

With cervical esophageal perforation, neck ache and stiffness are common clinical findings, but pain is typically less severe. Soilage of oropharyngeal flora through the retroesophageal space is limited because of esophageal attachment to the prevertebral fascia.⁶⁰ In thoracic esophageal perforation, retrosternal or chest pain lateralizing to the side of perforation is common. The initial contamination of the visceral mediastinum is followed by subsequent perforation of the mediastinal pleura. The left pleural space is usually involved with distal esophageal perforation, whereas the right pleural space is commonly violated with proximal esophageal perforation. The influx of gastric contents into the mediastinum initiates an intense inflammatory response and cytokine activation, resulting in mediastinitis accompanied with fluid sequestration, hypotension, and sepsis.⁶¹ With intra-abdominal esophageal perforation, dull epigastric pain radiating to the back may occur if the perforation is posterior and communicates with the lesser sac. More commonly, sharp, unrelenting, epigastric pain is often associated with anterior perforation with subsequent widespread peritoneal contamination. The early onset of systemic signs such as tachycardia, tachypnea, and fever are the usual clinical features. Rapid deterioration with signs of systemic inflammatory response such as leukocytosis, sepsis, and shock develop within hours of presentation.

DIAGNOSIS OF ESOPHAGEAL PERFORATION

Radiographic studies are invaluable in establishing the diagnosis of esophageal perforation. If cervical esophageal perforation is suspected, a lateral neck X-ray may demonstrate air in the prevertebral facial planes before it is detectable by chest radiograph.⁴⁰ In thoracic or intra-abdominal esophageal perforation, posterior and lateral chest radiographs, and upright abdominal series should be obtained. Chest radiograph is suggestive in 90% of patients with esophageal perforation, but may be normal immediately after the injury.⁶² Han et al. reported that radiographic evidence of mediastinal emphysema requires at least 1 hour after the initial injury to become discernable, whereas pleural effusion and mediastinal widening may take several hours to evolve.⁶² Panzini et al. documented that 75% of patients manifested abnormal radiographic findings on chest radiograph within 12 hours of instrumental esophageal perforation, and pneumomediastinum was the most common radiographic finding.⁶³ An interesting roentgen finding, the "V sign", associated with spontaneous esophageal rupture has been described.⁶⁴ It reflects localized mediastinal emphysema in the left lower mediastinum along the aorta and above the left diaphragm,

forming the characteristic “V sign”. Aside from mediastinal emphysema, other suspicious radiographic findings include mediastinal air-fluid level, hydropneumothorax, mediastinal widening, and pleural effusions.⁶³ Pleural effusions are located on the right if the perforation is in the midesophagus, whereas left-sided pleural effusions are commonly associated with distal esophageal perforation.

Esophageal perforation may also be identified by pleural fluid analysis. Diagnostic pleural fluid findings are food particles, a pH of less than 6.0, or the presence of an elevated amylase level.⁴⁰

Contrast esophagography is the study of choice for suspected esophageal perforation, but has an overall false-negative rate of 10%.⁶⁵ Traditionally, water-soluble contrast agents, such as gastrografin (meglumine sodium), have been recommended over barium sulfate as the contrast of choice. There is a concern that extravasation of barium sulfate into the mediastinum can lead to an intense inflammatory response, resulting in fibrosing mediastinitis.^{66–68} In addition, the long-term presence of barium in the mediastinum makes interpretation of future mediastinal imaging difficult, whereas gastrografin is rapidly absorbed.⁶⁹ However, gastrografin, if aspirated, can induce a severe necrotizing pneumonitis and pulmonary edema because of its inherent hypertonicity.⁷⁰ Also gastrografin extravasates in only 50% of cervical and 80% of thoracic esophageal perforations.⁷⁰ As the higher density and better mucosal adherence of barium allow the detection of smaller esophageal perforation,⁷¹ it will detect 60% of cervical and 90% of surgically confirmed intrathoracic esophageal perforations.^{59,72} Our preference is to obtain a barium study as the initial test as any extravasation will likely lead to surgical intervention. Local inflammation and edema of the injured esophagus may preclude a positive barium study. Therefore, if the clinical suspicion remains high, serial repetition of barium studies beginning several hours after the first attempt is warranted.

Chest computed tomography (CT) is an indispensable adjunct in diagnosing esophageal perforation. In the settings of a negative esophagram with a high clinical suspicion, critically ill patients unable to undergo esophagography, or atypical symptoms of esophageal injury at presentation, thoracic CT imaging is useful to confirm the diagnosis. Typical CT findings include mediastinal or extraluminal air, esophageal thickening, pneumomediastinum, esophagopleural fistula, pleural effusions, abscess cavities adjacent to the esophagus, and communication of an air-filled esophagus with an adjacent mediastinal air-fluid collection.^{73–75} Of these, extraluminal air is the most common CT finding associated with esophageal perforation, occurring in 92% of cases.⁷⁴

Although esophagoscopy is not recommended as the primary diagnostic study of choice, it is useful in providing a direct visualization of the perforation. Horwitz et al. showed that flexible esophagoscopy had a sensitivity of 100% and a specificity of 83% in the evaluation of penetrating esophageal injury.⁷⁶ However, a missed perforation hidden in a mucosal

Table 2 Management of Esophageal Perforation

Drainage only
Esophagectomy
Immediate reconstruction
Delayed reconstruction
Exclusion and diversion
Minimally invasive repairs
Esophageal stenting
Endoclip application
Video-assisted thoracoscopic surgery
Nonoperative treatment
Primary closure
Primary closure with buttressing of repair
Pleural flap
Pericardial fat pad
Diaphragmatic pedicle graft
Omentum onlay graft
Rhomboid muscle
Latissimus dorsi muscle
Intercostal muscle
T-tube drainage

fold and the potential to convert a small mucosal or submucosal tear into a large perforation during air insufflation argue against the use of esophagoscopy in the diagnosis of esophageal injuries.⁷⁷

MANAGEMENT OF ESOPHAGEAL PERFORATION

The fundamental principles of management in esophageal perforation include elimination of septic focus, provision of adequate drainage, augmentation of host defenses by antibiotics, and maintenance of adequate nutrition.⁷⁸ Therapeutic interventions aimed to achieve these goals vary with the cause, location, and severity of the perforation, as well as the time interval between perforation and intervention (Table 2). The overall health status and physiologic reserve of the patient, extent of associated injuries, and underlying esophageal pathologic findings are also the critical determinants of successful therapy.

Surgical Therapy

Surgical intervention includes primary closure with or without autogenous tissue reinforcement, esophageal resection, exclusion and diversion, esophageal T-tube, and drainage alone (Fig. 1). Cervical esophageal perforation can be treated by drainage alone.⁷⁹ Drainage alone is less successful with thoracic or abdominal perforation because containment of contamination is difficult. Intrathoracic esophageal disruption requires aggressive mediastinal and pleural drainage. The parietal pleura is opened along the entire length of the esophagus, and both the mediastinum and pleural space are debrided, irrigated, and drained by thoracostomy.

The surgical technique for perforation of the cervical esophagus involves a cervical incision along the anterior border of left sternocleidomastoid from the level of the cricoid cartilage to the sternal notch. The sternocleidomastoid

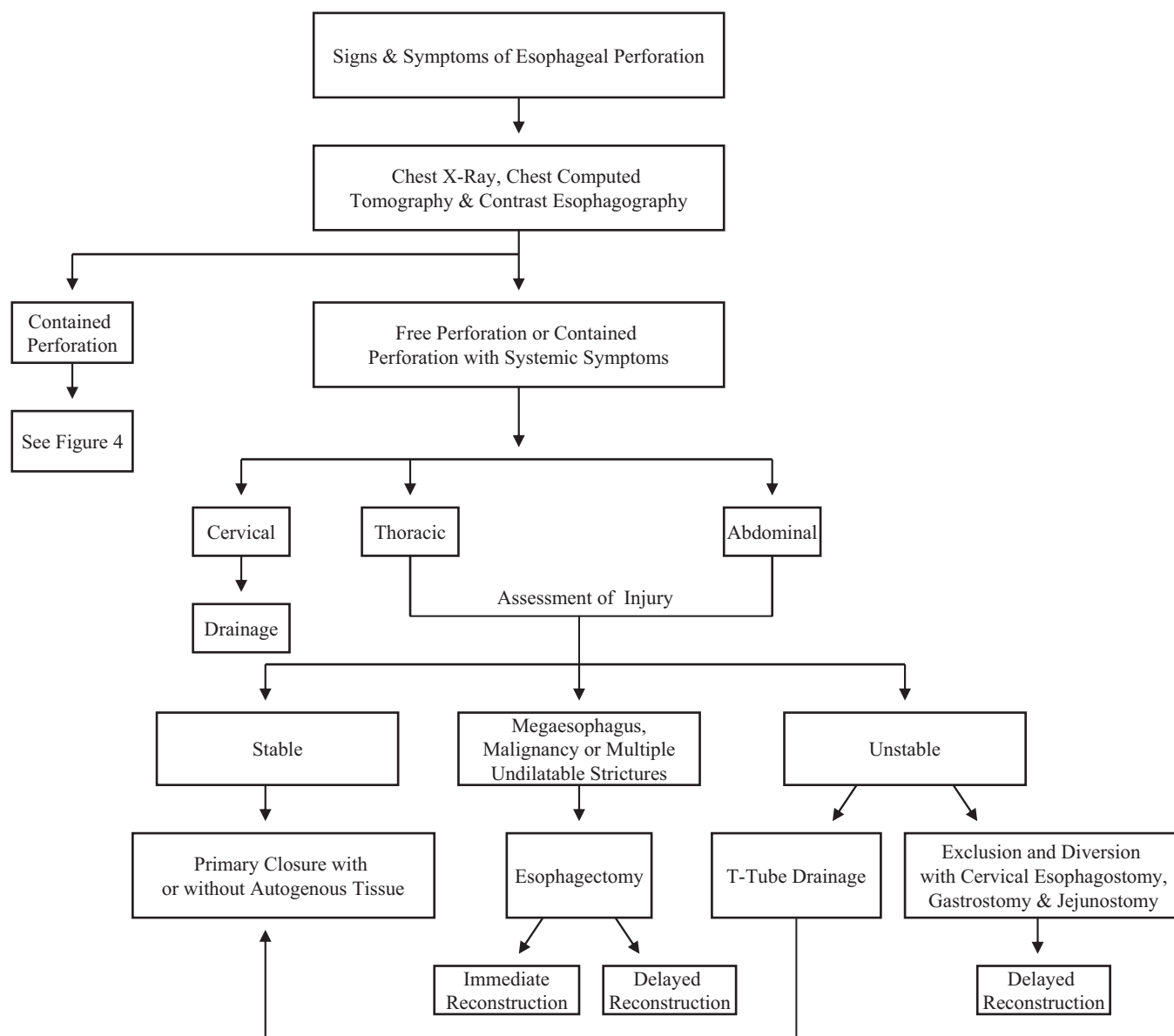


Fig. 1. Algorithm for surgical therapy of esophageal perforation.

and carotid sheath are retracted laterally, and the trachea and thyroid are displaced medially to expose the esophagus (Fig. 2). Blunt, finger-dissection technique is used to gain access to the prevertebral space, taking care to avoid injury to the recurrent laryngeal nerve in the tracheoesophageal groove. Alternatively, the prevertebral space can be approached behind the carotid sheath (Fig. 3). Access to perforation in the middle third of the esophagus is through a right thoracotomy in the fifth or sixth intercostal space, and perforation in the lower third is best approached through a left thoracotomy in the sixth or seventh intercostal space. Perforation at the esophagogastric junction can be accessed by either a left thoracotomy or upper midline laparotomy.

Primary repair is the preferred surgical treatment of choice in thoracic or abdominal esophageal perforation. Successful outcome requires the debridement of necrotic tissue,

full exposure of the mucosal defect after longitudinal esophagomyotomy, and approximation of mucosal and submucosal edges over a 40F to 46F bougie in a tension-free closure. Muscular layer is then reapproximated using a running or interrupted absorbable suture technique.⁸⁰ A variety of vascularized autogenous tissues, including pleural flap, diaphragmatic pedicle graft, omentum onlay graft, rhomboid and latissimus dorsi muscles, intercostals muscles, and pericardial fat pad have been used to buttress the primary repair.^{81–85} Our preference is to buttress the esophageal repair with a pedicled intercostal muscle flap, developed during the initial chest incision. Although reinforcement with vascularized tissue may decrease fistula formation and mortality,⁸⁵ postoperative esophageal leaks after reinforced primary repair can be as high as 83% in patients presenting after 24 hours of perforation.⁸⁶

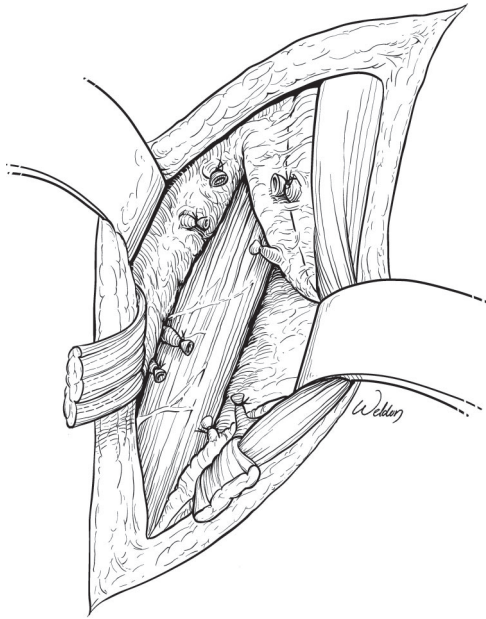


Fig. 2. Exposure of the cervical esophagus.



Fig. 3. Surgical approaches to the cervical esophagus.

Of paramount significance is the elimination of distal obstruction distal to the site of primary repair commonly seen in strictures and achalasia. Moghissi and Pender reported that primary repair without treatment of distal obstruction resulted in a mortality of 100%, whereas treatment of both perforation and distal obstruction reduced the mortality to 29%.⁸⁷ Therefore, intraoperative dilation should be attempted for distal strictures, and esophagomyotomy opposite the site of perforation should be accomplished for achalasia after primary repair of perforation.⁸⁸ When perforation occurs in the presence of severe gastroesophageal reflux, an antireflux procedure can be considered and used to bolster the esophageal repair. Multiple sites of distal obstruction not amendable to

correction at the time of primary repair constitute an indication for esophagectomy.

If primary repair is not possible at the time of surgery because of severe mediastinitis or underlying esophageal pathologic findings, surgical options include esophageal resection with immediate or delayed reconstruction, or exclusion and diversion. Esophagectomy should be considered as the procedure of choice for perforations associated with megaesophagus, carcinoma, caustic ingestion, or severe undilatable reflux strictures.⁵⁴ Segmental resection as a therapy for esophageal perforation is undertaken as a prelude to either immediate or delayed reconstruction using transposed stomach or colon.⁸⁹ The decision to restore gastrointestinal continuity in a single stage must be made on an individual basis. If the underlying pathologic process is a localized resectable cancer, or an undilatable or malignant stricture, resection with immediate reconstruction is indicated.⁹⁰ However, perforation caused by caustic ingestion requires segmental resection, cervical esophagostomy, and placement of jejunostomy. Several investigators have also recommended cervical esophago-gastric anastomosis during the primary operation in selected patients with intrathoracic perforation to restore gastrointestinal integrity.^{89,91} With this approach, the esophagogastric anastomosis is performed outside the soiled mediastinal field, and postoperative anastomotic leak can be managed by cervical drainage.

Exclusion and diversion techniques have been employed in patients with extensive mediastinal contamination, grossly devitalized esophagus, or hemodynamic instability unable to tolerate definitive repair or resection.^{92,93} Traditional techniques include cervical esophagostomy, gastrostomy, jejunostomy, mediastinal or pleural drainage, and exclusion of the perforated esophageal segment to prevent further contamination. This approach has evolved to one that preserves esophageal continuity by the placement of either a staple line or removable ligature distally in conjunction with cervical esophagostomy.⁹⁴ Nonetheless, the ongoing septic focus, need for a second operation to restore gastrointestinal continuity, and difficulties with subsequent esophageal reconstruction have all limited the technical application of this approach.^{79,85}

In patients with esophageal injuries that cannot be repaired at the time of surgery or hemodynamic instability unable to tolerate definitive repair, management with an esophageal T-tube has been advocated.⁷⁹ The T-tube creates a controlled esophagocutaneous fistula, allowing drainage of the esophagus and time for surrounding tissues to heal. Although continued leakage can progress to sepsis and chronic fistula formation,^{59,79,85,95} several investigators have reported successful clinical outcome with the use of esophageal T-tube.^{79,95} In our experience, the placement of a large T-tube has become one of the most versatile techniques for the complicated esophageal perforation, avoiding the suboptimal results associated with ligation or exclusion.

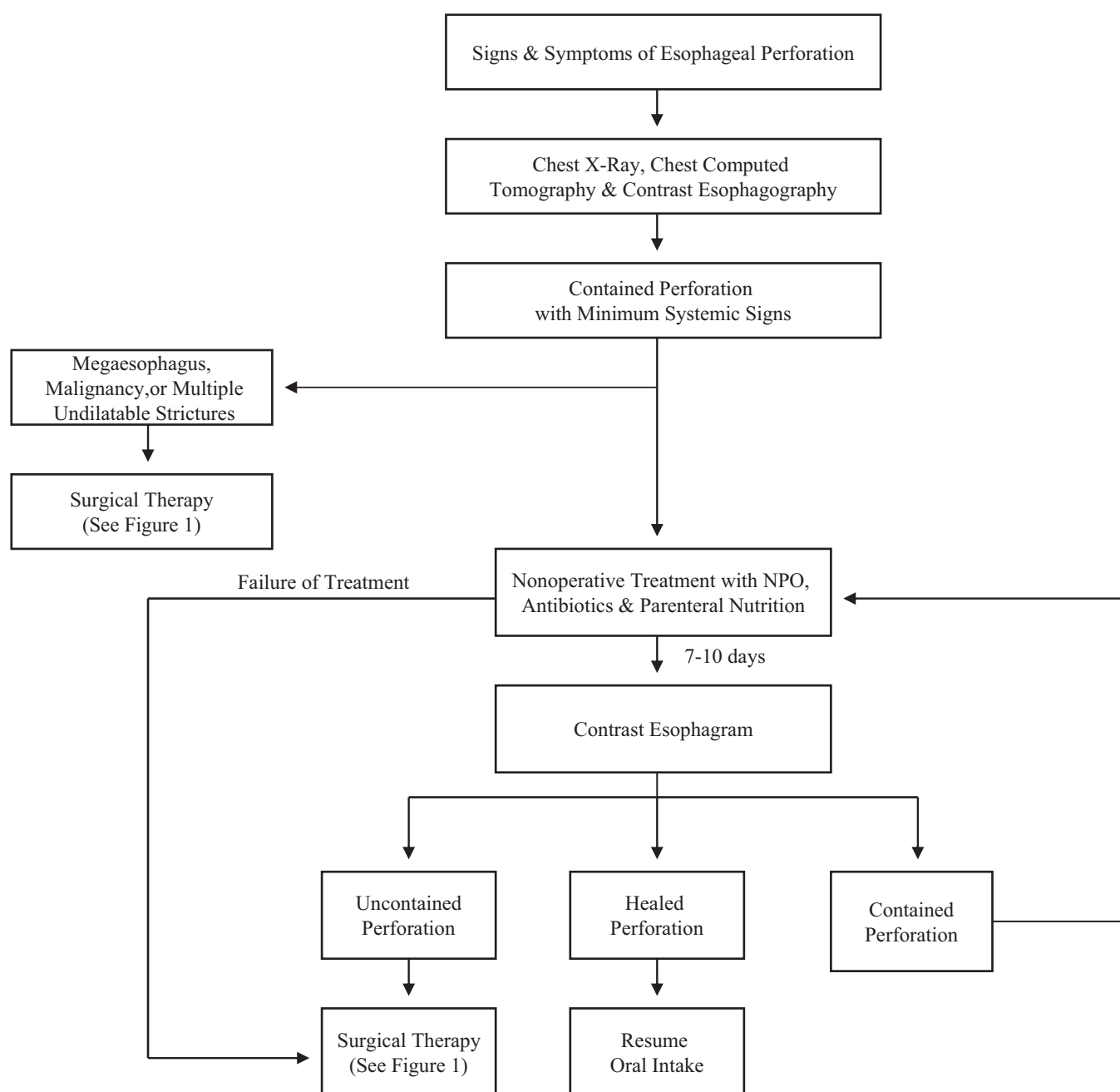


Fig. 4. Algorithm for nonoperative treatment of esophageal perforation. NPO, nil per os.

Brinster et al. reviewed published case series from 1990 to 2003, and found that the overall mortality associated with esophageal perforation in 726 patients was 18%.¹⁸ Surgical primary repair, with or without reinforcement, is the most successful therapeutic modality with an averaged mortality of 12%. This is followed by esophagectomy with a mortality of 17%. In contrast, other surgical therapies are associated with a higher mortality rate. A mortality of 24% was observed with the various exclusion and diversion procedures, and drainage alone was associated with a mortality of 37%.¹⁸

Nonoperative Treatment

Nonoperative approach for esophageal perforation is acceptable in selected patients with well-contained perforation and minimal mediastinal soilage, and includes maintenance of oral hygiene, cessation of oral intake, broad-spectrum antibiotics, and parenteral nutritional support (Fig. 4). Mediastinal or pleural fluid collections are drained with chest tubes. This therapeutic modality has been successful in treating cervical tears after instrumentation, well-circumscribed intramural dissections after pneumatic dilatation, small post-operative anastomotic leak, chronic perforation with minimal

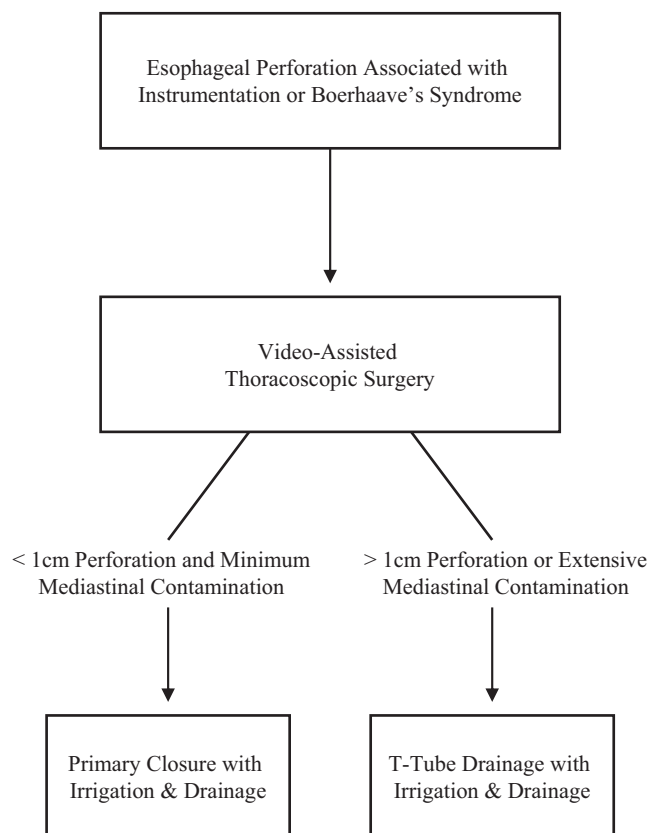
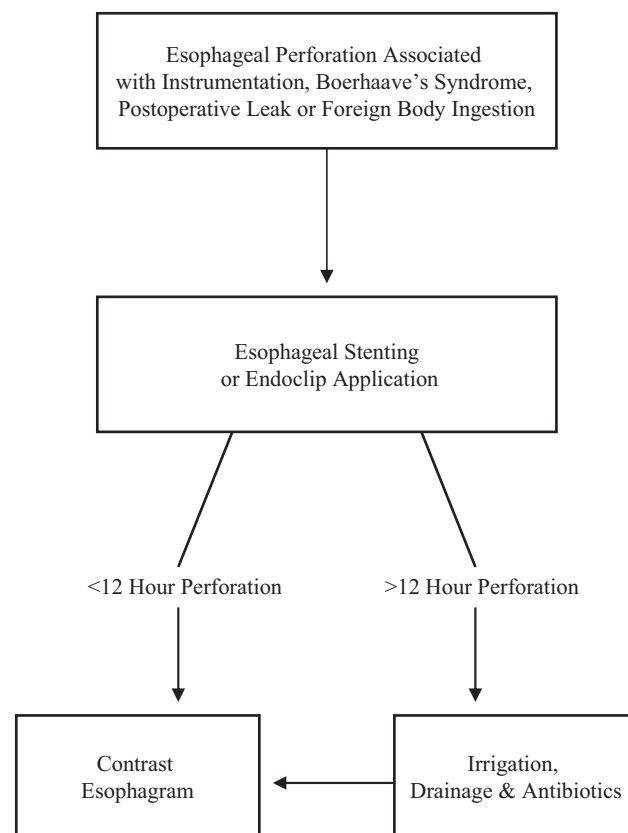
A**B**

Fig. 5. Algorithm for management of esophageal perforation with video-assisted thoracoscopic surgery (A) and endoscopic stenting and clipping (B).

clinical symptoms, and intramural tears secondary to varix sclerosis when the periesophageal fibrosis prevents the development of mediastinitis.^{96,97}

Cameron et al. proposed criteria for nonoperative management: disruption contained in the mediastinum or between the mediastinum and visceral lung pleura, drainage of the cavity back into the esophagus, and minimal signs of clinical sepsis.⁹⁸ Altorjay et al. extended these criteria, including the detection of early perforation, or a well-circumscribed late perforation, findings of esophageal tissue defect not neoplastic, not in the abdominal cavity, and not accompanied by simultaneous obstructive esophageal disease, and availability of imaging modalities and thoracic surgical expertise.⁹⁷ Even with strict adherence to these criteria for nonoperative treatment, up to 20% of patients managed nonoperatively develop multiple complications within 24 hours and required surgical intervention.⁹⁷

Brinster et al. retrospectively reviewed the clinical outcome from case series in 154 patients managed with nonoperative treatment, and reported an averaged mortality of 18%; the average mortality in 322 patients treated with primary repair was 12%.¹⁸ Nonoperative treatment of selected patients with contained esophageal perforation and minimal

contamination can be a safe and effective option. These patients, however, require diligent clinical assessment with a high index of suspicion for failures of nonoperative therapy. Failure to improve or deterioration in the clinical status during surveillance requires prompt surgical intervention.

Minimally Invasive Techniques

Video-Assisted Thoracoscopic Surgery

Minimally invasive thoracoscopic surgery offers a magnified view of the entire thoracic cavity and excellent access to all mediastinal compartments. The use of this technique in the setting of esophageal injury has been largely limited to instrumental or spontaneous esophageal perforation^{99–101} (Fig. 5). In addition, Chung and Ritchie emphasized that minimally invasive surgery is preferred in patients who are too ill to tolerate radical surgical debridement and drainage; it is less invasive and provides an expeditious life-saving alternative with good clinical outcome.¹⁰² Nguyen et al. proposed the fundamental goals in thoracoscopic surgery: identification of esophageal perforation, debridement of necrotic debris, control of leak, and wide drainage of mediastinum.⁹⁹ The usual thoracoscopic approach employs three or four trocars positioned conventionally through the right chest. A

left video-assisted thoracoscopic surgery or transabdominal approach can be used in distal esophageal perforation, or when the leak is demonstrated to extend into the left chest.^{100,101} Intraoperative endoscopy is an invaluable adjunct in identifying the site of perforation. The suspected region can be submerged under irrigation during endoscopic insufflation to precisely localize the site of perforation. Once identified, the devitalized margins of the perforation are debrided. If the defect is <1 cm surrounded by viable tissue, a primary closure can be performed with interrupted sutures. In the case of a larger perforation surrounded by inflamed tissue, wide drainage is advocated with the placement of a T-tube to control the soilage. Although minimally invasive thoracoscopic surgery for esophageal injury has become the preferred approach in many institutions, its role in the diagnosis and management of esophageal perforation requires further clinical investigation. As more experience is acquired in video-assisted esophageal surgery, minimally invasive approach to esophageal perforation will continue to emerge as an evolving technique for managing a challenging and potentially life-threatening problem.

Endoscopic Stenting and Clipping

In 1959, Celestin described the palliation of esophageal malignancy with a plastic endoprosthesis introduced at laparotomy.¹⁰³ In the 1970s, Atkinson and Ferguson suggested that endoscopic placement of plastic prosthesis (Celestin tube) for inoperable esophagogastric neoplasms provided a simple and relative safe alternative of relieving dysphagia and improving nutrition.¹⁰⁴ The last three decades have witnessed a tremendous growth in the indications for endoluminal prosthesis, including anastomotic tumor recurrence after surgery, primary or secondary tumors within the mediastinum causing extrinsic esophageal compression, tracheoesophageal fistula, and benign esophageal strictures.^{105–107} Successful management of early esophageal perforation with endoluminal stenting has also been described in the settings of endoscopic instrumentation, postoperative repair, foreign body ingestion, and Boerhaave's syndrome.^{108–122} The insertion of an endoscopic prosthesis may provide as an effective therapeutic maneuver, or temporary relief of symptoms to allow a more definitive treatment at a later date. The management of these patients should be individualized and through a multidisciplinary team approach. Endoscopist, thoracic surgeon, and radiologist should interact in a coordinated effort in the planning of therapeutic decisions and participating in the clinical evaluation of treatment outcome.

Endoluminal stents are made from Nitinol (titanium-nickel alloy) or stainless steel compressed into a small caliber introducer system.¹⁰⁵ Most stents are available in an uncovered form or with a plastic coating on the inside and/or the outside of the stent. Uncovered stents are less liable to migration, but commonly associated with tumor ingrowth. Newer generations of metallic self-expanding stents are equipped with antireflux valves and retrievable threads, and

capable of expansion at body temperature to conform to the esophageal wall. The central covering of the stent seals the perforation, and the uncovered metal ends allow integration into the esophageal wall.

Fisher et al. examined 15 consecutive patients with spontaneous or iatrogenic esophageal perforation treated with self-expandable metal stents.¹²³ All patients were eventually discharged to home except for one patient died of aspiration pneumonia after 6 days of stent insertion. The stents were extracted after an average of 4 weeks, and the perforations were well healed and remained sealed. The authors emphasized that stent insertion should be performed expeditiously once the diagnosis is established to reduce the extent of mediastinal contamination. Thoracoscopic irrigation and drainage, and antibiotic administration should be instituted if the stent is not inserted within the first 12 hours of perforation (Fig. 5).

Endoscopic clipping has been historically used for the control of gastrointestinal bleeding.¹²⁴ In 1995, Wewalka et al. described the treatment of esophageal perforation with endoscopic clipping after pneumatic dilation for achalasia.¹²⁵ Since then, successful endoscopic closure of esophageal perforation with metallic clips has been reported for perforations associated with instrumentation, foreign body ingestion, and Boerhaave's syndrome.^{126–129} (Fig. 5). This mode of treatment is suitable only for selected patients with small (<1.5 cm) clean perforation, and minimal symptoms of infection. Although the length of time between the occurrence and the diagnosis of perforation is an important prognostic factor, recent reports advocated clipping of mature perforation in special circumstances.^{130,131} Raymer et al. applied endoscopic metallic clips to close mature perforations associated with fistulae after controlling mediastinal drainage.¹³⁰ Similarly, Abe et al. described a case of delayed esophageal perforation with mediastinitis from foreign body ingestion; endoscopic nasomediastinal drainage followed by metallic endoclip closure of perforation was performed in a patient whom refused surgical intervention.¹³¹ Six days after endoclip application, esophagography demonstrated no further leakage, and the patient was eventually discharge to home.

CONCLUSION

Esophageal perforation is a critical and potentially life-threatening event with considerable morbidity and mortality. The management of esophageal perforation, although controversial, requires a thoughtful and individualized approach. When the diagnosis is made early, an unconfined esophageal leak is a surgical emergency, and surgery therapy is still considered the "gold standard". The main principles of surgical intervention are rapid closure of the esophageal leak, drainage of mediastinal or pleural collections, and administration of parenteral nutrition and broad-spectrum antibiotics. However, there is no consensus in regard to the optimal therapy when the perforation is confined in the absence of systemic infection. Several treatment methodologies have

evolved over the years, including nonoperative treatment, minimally invasive thoracoscopic surgery, endoscopic stenting, and metallic endoclip application. In contrast to nonoperative management of esophageal perforation, minimally invasive techniques have the advantages to prevent further contamination of mediastinal and pleural spaces, and resume oral intake in a timely fashion. The introduction of these therapeutic modalities is potentially a major step forward in the management of esophageal perforation. The clinical efficacy of minimally invasive techniques is promising enough to warrant further clinical investigation directed at defining these evolving modalities as invaluable ancillary adjuncts in the management of early esophageal perforation.

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