

Evolving Options in the Management of Esophageal Perforation

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Esophageal perforation remains a devastating event that is difficult to diagnose and manage. The majority of injuries are iatrogenic and the increasing use of endoscopic procedures can be expected to lead to an even higher incidence of esophageal perforation in coming years. Accurate diagnosis and effective treatment depend on early recognition of clinical features and accurate interpretation of diagnostic imaging. Outcome is determined by the cause and location of the injury, the presence of concomitant esophageal disease, and the interval between perforation and initiation of therapy.

Esophageal perforation continues to present a diagnostic and therapeutic challenge despite decades of clinical experience and innovation in surgical technique. Accurate diagnosis and early treatment are essential to the successful management of patients with this increasingly frequent condition, but presentation is often ambiguous and resembles that of other disorders. The diagnostic errors and delayed treatment that result significantly increase morbidity and mortality. Optimal therapy, especially after delayed diagnosis, continues to evolve. Since the original description of esophageal perforation more than 250 years ago, diagnosis remains challenging, management remains controversial, and mortality remains high.

Material and Methods

A review of the literature by search of the MEDLINE database limited to human studies published in English was performed with the keywords "esophageal perforation," and "Boerhaave's syndrome," which were then matched to "surgery," "nonoperative," and "primary repair." Articles cited in the references retrieved by MEDLINE search were reviewed. The etiology, pathogenesis, and clinical, diagnostic, surgical, and nonoperative features of esophageal perforation, as well as the current mortality rate associated with each respective treatment modality in case series between 1990 and 2003, were examined.

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The overall mortality associated with esophageal perforation can approach 20%, and delay in treatment of more than 24 hours after perforation can result in a doubling of mortality. Surgical primary repair, with or without reinforcement, is the most successful treatment option in the management of esophageal perforation and reduces mortality by 50% to 70% compared with other interventional therapies.

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Historical Background

The signs and symptoms of esophageal perforation have been described in the literature for centuries. In 1946, Barrett [1] reviewed the first report of spontaneous esophageal perforation: Hermann Boerhaave's 1723 detailed account of the barogenic esophageal rupture suffered by the High Admiral of the Dutch Navy, Baron van Wassenaer, was due to intense and prolonged vomiting following excessive ingestion of food and alcohol. The first successful surgical repairs following esophageal perforation were accomplished by Barrett [2] and by Olson and Clagett [3] in 1947. Satinsky and Kron [4] performed the first successful esophagectomy following perforation in 1952. As early recognition of signs and symptoms improved and antibiotics became widely available, the mortality associated with esophageal perforation declined through the 1960s and 1970s. The incidence of esophageal perforation has increased with the advancement of invasive diagnostic technology and the etiology has changed from mostly spontaneous or traumatic to mostly iatrogenic.

Etiology

In 559 patients from recent series, iatrogenic injury to the esophagus was the most frequent cause of esophageal perforation, with instrumentation accounting for 59% of all patients [5–17]. Spontaneous perforations accounted for 15% of all patients. Other injuries included foreign body ingestion (12%), trauma (9%), operative injury (2%), tumor (1%), and other causes (2%). The relationship of cause to location is illustrated in Figure 1. Types of instrumentation that commonly cause esophageal perforation include esophagoscopy, sclerotherapy, variceal

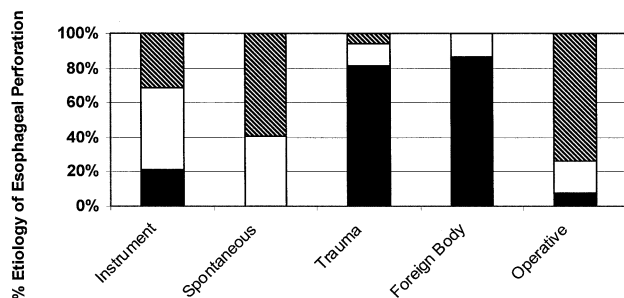


Fig 1. Association of cause to anatomic location of esophageal perforation in 250 patients from recent series [5, 6, 8, 14, 90]. ▨ = abdominal; □ = thoracic; ■ = cervical.

ligation, pneumatic dilation, bougienage, and laser therapy. Over the past 30 years, the actual risk of esophageal perforation during flexible esophagoscopy has remained low and is estimated to occur at a frequency of 0.03% during flexible upper endoscopy compared with 0.11% during rigid endoscopy [18, 19]. Simultaneous dilation during endoscopy increases the risk of perforation to between 0.09% for Maloney-Hurst-type dilators and 2.2% for the Celestin-type dilator [18, 19]. Endoscopic sclerotherapy for esophageal varices leads to perforation in 1% to 6% of patients in which the sclerosing agent induces a transmural necroinflammatory injury in the esophagus [20]. Decreasing the volume and concentration of sclerosant may reduce this risk [20]. Esophageal perforation can also occur during endoscopic variceal ligation, when the esophageal mucosa is caught and torn between the overtube and the endoscope [20]. The risk of perforation during pneumatic dilation for achalasia is 1% to 5%, with higher inflation pressure and previous pneumatic dilation increasing this risk [21]. The placement of nasogastric tubes [22], endotracheal tubes [23], Sengstaken-Blakemore or Minnesota tubes for tamponading variceal bleeding [20], or endoesophageal prostheses [24] can also cause esophageal perforation.

In a normal esophagus, the location at greatest risk of instrumental injury is Killian's triangle, which is formed by the inferior constrictor pharyngeus and the cricopharyngeus muscles. In this region, the posterior esophageal mucosa is unprotected by muscularis, and is separated from the retroesophageal space by buccopharyngeal fascia only. Cervical osteophytic spurs, kyphosis of the spine, or hyperextension of the neck can further increase the risk of perforation in this area [25]. Instrumental perforation also occurs at anatomic areas of narrowing such as the distal esophagus just proximal to the gastroesophageal junction and the impingement of the aortic arch and left main stem bronchus. Biopsy sites and areas just proximal to benign or malignant strictures are also at risk for perforation.

Intraoperative injury to the esophagus can occur during surgical procedures in close proximity to or directly involving the esophagus. Pneumonectomy [26], vagotomy [27], hiatal hernia repair [28], cervical spine operations or fractures [25], transesophageal echocardiography

[29], and atrial surgery [30] have been associated with esophageal perforation.

Penetrating trauma causes perforations mainly in the cervical esophagus, and morbidity and mortality are usually due to associated injuries [31]. Perforation secondary to blunt trauma is exceedingly rare, but may present following motor vehicle accident [32] or attempted Heimlich maneuver [33]. Ingestion of foreign bodies or caustic materials can produce perforation in areas of anatomic narrowing such as the cricopharyngeus, the impingement of the aortic arch and left main stem bronchus, and in the distal esophagus just proximal to the lower esophageal sphincter. Spontaneous, or barogenic, esophageal perforation results from a sudden increase in intraesophageal pressure like that associated with hyperemesis, or, much less frequently, childbirth, seizure, prolonged coughing or laughing, or weightlifting [34]. The rupture usually involves the left wall of the supradiaphragmatic esophagus, dissects all esophageal layers in a longitudinal manner, and frequently drains into the left pleural or peritoneal cavity [35]. Erosion by primary or metastatic esophageal carcinoma [36], Barrett's ulcers [37], surrounding infection [38], or immunodeficiency [39] can also cause esophageal perforation.

Clinical Presentation

The cause and location of the injury, as well as the interval between perforation and diagnosis, determine the clinical features of esophageal perforation. Diagnosis is often difficult because presentation is inconclusive and often mimics that of other disorders such as myocardial infarction, peptic ulcer perforation, pancreatitis, aortic aneurysm dissection, spontaneous pneumothorax, or pneumonia. Common clinical manifestations of esophageal perforation include chest pain, dysphagia, dyspnea, subcutaneous emphysema, epigastric pain, fever, tachycardia, and tachypnea. Any combination of these signs and symptoms following instrumentation of the esophagus or respiratory tract implies perforation until proven otherwise.

Cervical perforation of the esophagus is generally less severe and more easily treated than intrathoracic or intraabdominal perforation. Spread of contamination to the mediastinum through the retroesophageal space is slow after cervical perforation, and attachments of the esophagus to the prevertebral fascia in this region limit lateral dissemination of esophageal flora. Patients with cervical perforations can present with neck pain, cervical dysphagia, dysphonia, or bloody regurgitation. Subcutaneous emphysema is commonly found on physical examination and appears radiographically in 95% of patients of cervical esophageal perforation [40].

Intrathoracic perforations rapidly contaminate the mediastinum. The rupture may immediately extend into the pleural cavity, most frequently on the left, or the pleura may withstand the injury. If the integrity of the pleura is maintained, gastric contents infiltrate the mediastinum and produce characteristic mediastinal emphysema and inflammation, and eventually cervical subcutaneous emphysema. This initial, chemical mediastinitis is followed

by bacterial invasion and severe mediastinal necrosis. Rupture of the overlying pleura by mediastinal inflammation or by the initial perforation directly contaminates the pleural cavity, and pleural effusion results. As a result of negative intrathoracic pressure, gastric fluids and bacteria are drawn farther into the pleural space. Contamination disseminates, and sequestration of fluid and hypovolemia result [41]. Chest pain, tachycardia, tachypnea, fever, and leukocytosis occur during the ensuing inflammatory response, and systemic sepsis and shock develop within hours.

Perforations of the **intraabdominal esophagus** are uncontained and result in **contamination** of the **peritoneal cavity**. Patients report back pain and an inability to lie in the supine position. Epigastric pain is frequently present and may be referred to the shoulders due to irritation of the diaphragm secondary to perforation. Patients may present with acute abdomen. As in intrathoracic perforation, the early onset of systemic signs such as tachycardia, tachypnea, and fever with rapid deterioration to systemic sepsis and shock is characteristic.

Diagnosis

Early diagnosis of esophageal perforation reduces the rate of complication and **mortality** significantly [5, 42, 43]. Up to **50%** of patients are **atypical**, however, and consequent **diagnostic errors** lead to **delayed** treatment [10]. The presence of esophageal perforation should always be suspected with development of cervical, thoracic, or abdominal pain following endoscopy.

Diagnosis of cervical esophageal perforation is aided by lateral roentgenogram of the neck, which may demonstrate air in the prevertebral fascial planes before it is detectable by radiograph of the chest or physical examination [44]. When thoracic or abdominal esophageal perforation is suspected, upright abdominal as well as posteroanterior and lateral chest radiographs should be obtained immediately.

Plain chest roentgenogram suggests esophageal perforation in 90% of patients, but can be **normal** if taken **early** [44]. The presence of pleural effusions, pneumomediastinum, subcutaneous emphysema, hydrothorax, hydro-pneumothorax, or subdiaphragmatic air heightens suspicion of esophageal perforation. **Soft tissue and mediastinal emphysema require at least 1 hour to develop after perforation**, whereas pleural effusions and mediastinal widening take several hours to evolve [44]. Panzini and colleagues [45] reported that **75% of patients had abnormal chest roentgenograms within 12 hours** of instrumental perforation. Further, pneumomediastinum was present in 60%, and 33% had a density adjacent to the descending aorta in the left cardiophrenic angle with a loss of descending aorta contour [45].

Contrast esophagography remains the standard in diagnosis of esophageal perforation. Water-soluble contrast agents, such as gastrografin, are advocated for first line screening of suspected perforation due to their rapid absorption, although gastrografin may effectively extravasate in just 50% of cervical and 75% to 80% of thoracic perforations [46]. Additionally, if aspirated, gastrografin

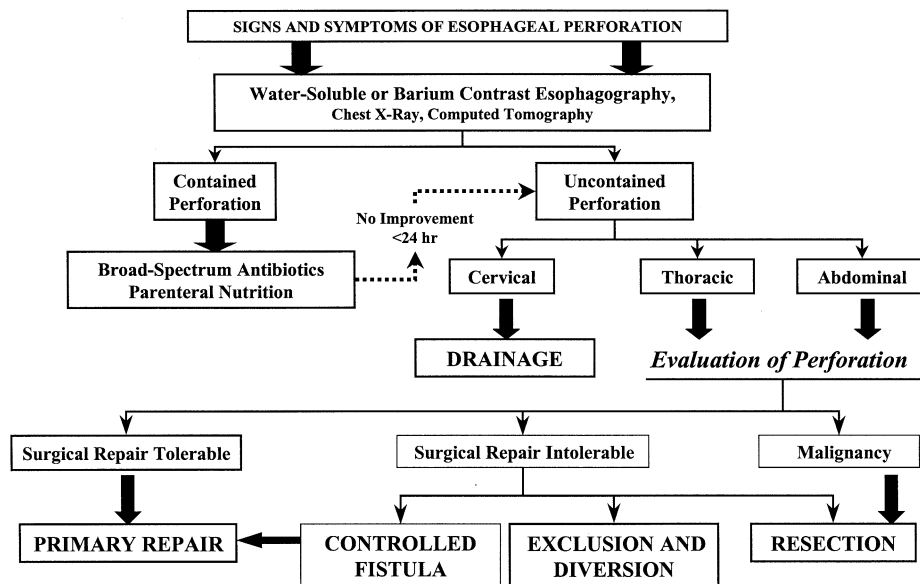
can cause severe, necrotizing pneumonitis due to its hypertonicity [46]. **If no perforation is detected initially with a water-soluble agent, serial barium contrast esophagography should follow** [47]. Dilute barium sulfate should be used exclusively if the patient is at high risk for aspiration or whenever esophageal fistula or respiratory tract perforation is suspected [46]. **Barium** has been shown experimentally [48] and clinically [47] to have **no additional adverse effect** in the **mediastinum**. In addition, barium clearly demonstrates small primary perforations and unsuspected secondary perforations, and will detect 60% of cervical and 90% of surgically confirmed intrathoracic perforations [40, 49]. A positive result clearly indicates the level of perforation as well as the extent of contamination in the pleural space. **Contrast studies have an overall false-negative rate of 10%**, however, and a negative result with persistent suspicion of perforation requires serial repetition of barium contrast esophagography beginning several hours after the first attempt [5, 47]. Because a negative result cannot rule out the possibility of perforation, **an accurate diagnosis may require computed tomography, flexible esophagoscopy, or both.**

Computed tomography is useful when perforations are difficult to locate or diagnose, or when contrast esophagography cannot be performed. Abnormal findings suggestive of esophageal perforation include **extraluminal air in the soft tissues** of the **mediastinum, esophageal thickening**, perceptible communication of the air-filled esophagus with a contiguous mediastinal or paramediastinal air-fluid collection, or abscess cavities adjacent to the esophagus in the pleural space or mediastinum [50, 51]. **Left-sided pleural effusion** strengthens **suspicion** of perforation. In patients who fail to improve after initial treatment, computed tomography is useful in localizing pleural fluid collections and guiding drainage catheter placement [50, 51].

Flexible esophagoscopy provides direct visualization of the perforation and is especially valuable in assessing perforations secondary to external penetrating trauma. In the emergent evaluation of the traumatically perforated esophagus, flexible esophagoscopy is associated with a sensitivity of 100% and a specificity of 83% [52]. The role of esophagoscopy in the evaluation of acute, nonpenetrating esophageal perforations has not been established and is highly questionable [53]. Air insufflation, a requirement during flexible esophagoscopy, is contraindicated when small mucosal or submucosal tears are suspected. In this situation, air may dissect intramurally within the esophagus and cause cervical subcutaneous emphysema, giving the impression of a serious perforation and leading to operative intervention when, in reality, conservative treatment is appropriate [53].

In **pleural fluid collected by thoracentesis**, the presence of undigested **food**, a **pH** of **less than 6.0** [51], or an elevated **salivary amylase** level confirms the diagnosis of esophageal perforation [6].

Fig 2. Algorithm for management of esophageal perforation.



Treatment

The critical determinants of therapy for esophageal perforation are the **cause**, the **location**, and the **severity** of the perforation, as well as the **interval** between perforation and treatment. In addition to the age and general health of the patient, the damage to surrounding tissues and the presence of concomitant esophageal pathology or injury must be considered before initiating therapy [5]. The objectives of treatment include prevention of further contamination from the perforation, elimination of infection, restoration of the integrity of the gastrointestinal tract, and establishment of nutritional support [7]. Therefore, debridement of infected and necrotic tissue, meticulous closure of the perforation, total elimination of distal obstruction, and drainage of contamination are essential to successful management. Complete therapy for esophageal perforation should also include establishment of enteral nutrition and initiation of systemic antibiotic therapy. Treatment options include operative and nonoperative management. Because **patients managed nonoperatively may deteriorate during the course of therapy, all cases of esophageal perforation merit preliminary surgical consultation.** Urgent surgical evaluation is required by evidence of sepsis, shock, respiratory failure, pneumothorax, pneumoperitoneum, or extensive mediastinal emphysema. Furthermore, the development of mediastinal abscess or empyema in the nonoperatively managed patient warrants immediate surgical intervention [8]. An algorithm of current management strategies for esophageal perforation appears in Figure 2.

Operative Management

Surgical options include **primary** or reinforced primary closure, esophageal **resection**, **drainage** alone, **T-tube** drainage, and **exclusion** and **diversion**. Selection of the proper surgical approach depends on the location of the perforation. Small or well-contained cervical esophageal

perforations **down to the level of the carina can usually be managed with drainage alone by cervical incision** [7]. The best surgical approach to perforations in the **middle third** of the esophagus is through a **right thoracotomy** in the sixth intercostal space, and perforations in the **lower third** are best approached through a **left thoracotomy** in the seventh intercostal space. An **upper midline** laparotomy is used to reach the **abdominal** esophagus.

Barrett [2], and Olson and Clagett [3] pioneered the use of **primary repair** for esophageal perforation in the 1940s. Primary repair is the **surgical treatment of choice** in an otherwise **healthy** esophagus, and successful closure requires **debridement of necrotic tissue, vertical esophagomyotomy to fully expose the damaged mucosa, secure closure of the mucosa, and irrigation and drainage** of the contaminated area [9]. Incomplete exposure or repair of the mucosa at either edge of the perforation leads to leakage, and, consequently, increased morbidity and recovery time [54, 55]. With delayed diagnosis or presentation, thorough evaluation of the rupture size, the amount of contamination, and the extent of necrosis and edema are essential in determining a therapeutic course [56]. If the surrounding musculature cannot hold sutures, the mucosal layer should be secured, and the necrotic tissue flanking the wound widely debrided [7].

The persistent **problem** of **leakage** from and deterioration of the **primary** repair site led to the development of reinforced primary repair, in which **tissue grafts are implemented to bolster the repair site.** Grillo and Wilkins [57] first described the successful use of a thickened, inflamed **pleural flap** to buttress an adjacent intrathoracic primary closure. **Other tissues** utilized for reinforced primary repair include an elevated diaphragmatic pedicle graft [58], **omentum** onlay graft [59], rhomboid and **latissimus dorsi muscles** [60], and, for traumatic cervical perforation, sternohyoid, sternothyroid, and sternocleidomastoid muscles [31]. Meticulous exposure and repair

of the mucosa are essential irrespective of which graft is chosen, because leak rates of 25% to 50% have been observed even after reinforced primary repair [10, 55, 60, 61].

Perforation in patients who present with underlying esophageal pathology, such as stricture, achalasia, or severe gastrointestinal reflux, require additional evaluation. Distal esophageal obstruction requires correction at the time of repair, and adequate dilation of stricture should be accomplished intraoperatively [62]. In a report by Moghissi and Pender [63], repair without treatment of distal obstruction resulted in a mortality of 100%, whereas treatment of both perforation and obstruction reduced mortality to 29%. Successful management of perforation due to pneumatic dilation for achalasia consists of primary or primary reinforced repair of the rupture with esophagomyotomy opposite the site of perforation [62]. When perforation occurs in the presence of severe gastroesophageal reflux, an antireflux procedure should be performed and used to bolster the esophageal repair. A Belsey Mark IV repair is used for thoracic perforation, and Nissen fundoplication is recommended for abdominal perforation. Perforation in association with distal malignant obstruction requires resection [64]. Widely disseminated carcinoma may preclude resection and necessitate placement of an intraluminal endoprosthesis following perforation [36, 65, 66].

Esophagectomy has been very successful in the treatment of perforation associated with severe esophageal necrosis or with pathologic obstruction [64, 67, 68]. In addition to maintaining gastrointestinal integrity, this technique definitively eliminates the perforation as the source of sepsis and removes any underlying esophageal pathology [11, 12]. Whether to employ a transhiatal or transthoracic approach depends on the interval from perforation to diagnosis, the severity of mediastinal inflammation, and the extent of pleural contamination [12, 64]. Transhiatal esophagectomy with immediate reconstruction is recommended with early diagnosis, when the perforation is confined to the mediastinum, or when pleural contamination is minimal. The transthoracic approach with staged reconstruction is favored with increased delay from perforation to diagnosis or with extensive mediastinal or pleural contamination. The latter technique allows decortication of the lung and irrigation of the thoracic cavity at the time of resection [12]. The decision to restore gastrointestinal continuity in a single stage must be made on an individual basis. Orringer and Sterling [64] and Matthews and colleagues [69] recommend cervical esophagogastric anastomosis during the primary operation in select patients to restore gastrointestinal integrity. With this approach, the esophagogastric anastomosis is performed outside the soiled mediastinal field. Furthermore, should an anastomotic leak occur, it can be treated simply by opening the cervical incision. Altorjay and associates [12] performed primary intrathoracic anastomosis to the stomach or jejunum in a group of 14 patients with no leaks following intrathoracic esophageal perforation.

The severe contamination and inflammation that result

from delayed treatment of esophageal perforation may preclude primary repair at the time of diagnosis. Drainage alone in this situation, without repair of the perforation, is acceptable only for cervical perforations. Drainage alone is contraindicated with thoracic and abdominal perforations because contamination continues to disseminate in these situations, making containment of infection and drainage of the thorax or abdomen impossible. With injuries that are beyond repair, successful management with an esophageal T-tube has been advocated [7, 70]. The T-tube creates a controlled esophageal fistula allowing drainage of the esophagus and time for surrounding tissues to heal in injuries that cannot be repaired at the time of diagnosis. Although chronic fistula formation has been reported in the past [71], more recent series [7, 70, 72] describe successful management with the T-tube.

Several exclusion and diversion techniques have been reported for the treatment of esophageal perforation following late diagnosis and development of extensive contamination [73–75]. Conventional techniques involve closure of the perforation with wide drainage of contaminated tissues, proximal and distal diversion of the esophagus with exclusion of the perforated segment to prevent further contamination, creation of an end or side cervical esophagostomy, and creation of a gastrostomy. Traditional exclusion and diversion requires a second operation to restore gastrointestinal continuity, and the predisposition to complication with this procedure has led some to question its rationale when other approaches are feasible [7, 54]. Modified, single-stage techniques with improved results have been reported in which esophageal ligation is performed with absorbable sutures, eliminating the need for a second operation [76, 77].

The use of thoracoscopy in the management of esophageal perforation is limited [78]. Closure of esophageal perforation by primary repair with thoracoscopic assistance has been successful in 3 patients following instrumental perforation [78, 79] and in 1 patient following spontaneous perforation [80].

Nonoperative Management

Nonoperative management of esophageal perforation is appropriate in select patients with well-contained perforations and minimal mediastinal and pleural contamination. Mengold and Klassen [81] described the first successful nonoperative management of esophageal perforation in 1965, and reported only one death out of 18 treated within 24 hours for instrumental perforation of the thoracic esophagus. In 1975, Larrieu and Kieffer [82] first reported successful medical management of spontaneous perforation. Based on more than 30 years of clinical evidence [8, 13, 81, 83], nonoperative therapy is most successful when the perforation is instrumental or in the cervical esophagus, when the injury is detected early, and with small, well-contained perforations resulting from dilation of strictures or from sclerotherapy for esophageal varices. Nonoperative management is applicable to patients associated with delayed diagnosis provided only minimal symptoms are present. Cameron and

Table 1. Outcome After Treatment of Esophageal Perforation in Series Published Between 1990 and 2003

Treatment	Number of Patients	Number of Deaths	Mortality (%) Mean (Range)	References
Primary repair	322	40	12 (0-31)	5-7, 10, 14, 16, 17, 42, 55, 90-2, 95
Resection	129	22	17 (0-43)	5-7, 12, 16, 42, 64, 90-2
Drainage	88	32	36 (0-47)	5-7, 16, 17, 42, 91
Exclusion and	33	8	24 (0-80)	5-7, 17, 76, 77, 96
Nonoperative	154	26	17 (0-33)	7, 8, 13, 14, 42, 88, 90-2
Total	726	128	18 (0-80)	

associates [83] established the criteria for nonoperative treatment of esophageal perforation in 1979. Based on these standards and published evidence since then, Altorjay and colleagues [8] and others [13, 49] suggest the following criteria for selection of nonoperative treatment:

1. Early diagnosis or leak contained if diagnosis delayed;
2. Leak contained within neck or mediastinum, or between mediastinum and visceral lung pleura;
3. Drainage into esophageal lumen as evidenced by contrast imaging;
4. Injury not in neoplastic tissue, not in abdomen, not proximal to obstruction;
5. Symptoms and signs of septicemia absent; and
6. Contrast imaging and experienced thoracic surgeon available.

Nonoperative management includes nothing by mouth for at least 48 to 72 hours, at which time clear liquids can be initiated if the patient demonstrates clinical improvement. In addition, nonoperative therapy should include administration of broad-spectrum antibiotics for at least 7 to 14 days and total parenteral nutrition. Mediastinal or pleural fluid collections are drained with chest tubes, computed tomography-guided drainage catheters [50], or with suction guided by transluminal endoscopy [84]. Although some consider nasogastric drainage essential, misplacement of the nasogastric tube into the stomach promotes gastroesophageal reflux and consequent mediastinal contamination [85]. The successful, nonoperative management of malignant [36] and nonmalignant [86, 87] esophageal perforation with an endoscopically guided coated stent has been reported, but the potential role of endoscopy and the use of stents in the treatment of esophageal perforation is not yet well defined. These procedures require the approximation of a foreign body to a wound in a contaminated field and should be reserved for patients who cannot tolerate surgery [87].

Results

The overall mortality associated with esophageal perforation in 726 patients from series between 1990 and 2003 was 18% (Table 1). The cause and location of the injury, the presence of underlying esophageal pathology, the delay in diagnosis, and the method of treatment determine the rate of morbidity and mortality [5, 7, 8, 42]. A

comprehensive review of published evidence from 1990 to the present [6-8, 11, 12, 14-17, 42, 55, 88-92] allows an accurate evaluation of common risk factors and a current assessment of therapeutic management for esophageal perforation.

The etiology of the perforation affects outcome. In 431 patients from 9 recent series [5-8, 14, 17, 42, 90, 93], spontaneous esophageal perforation was associated with a mortality of 36% (0% to 72%), iatrogenic perforation with a mortality of 19% (7% to 33%), and traumatic perforation with a mortality of 7% (0% to 33%). The presentation of spontaneous rupture is ambiguous and often confused with that of other disorders, and the consequent delay in treatment and rapid development of systemic sepsis account for the increased mortality associated with spontaneous perforation. Iatrogenic perforations usually follow instrumentation in the clinical setting, and, therefore, are less difficult to diagnose and manage. Traumatic perforations are often confined to the cervical esophagus with limited dissemination of contamination, and mortality usually results from associated injuries.

Anatomic location affects the mortality associated with esophageal perforation. In 397 patients from 7 recent series [5, 6, 8, 13-15, 88] cervical esophageal perforations were associated with a mortality of 6% (0% to 16%), whereas thoracic and abdominal perforations were associated with a mortality of 27% (0% to 44%), and 21% (0% to 43%), respectively. This difference in mortality results from the containment of contamination by the fascial planes of the neck following cervical perforation. By contrast, contamination secondary to intrathoracic or intraabdominal esophageal perforation is poorly contained and rapidly results in systemic sepsis if treatment is delayed.

The interval from perforation to initiation of treatment is a crucial determinant of outcome after esophageal perforation. Although advances in antibiotic therapy and critical care have reduced the complications following delayed diagnosis, a delay in treatment of greater than 24 hours is still associated with an increase in morbidity and mortality (Fig 3) [6, 15]. White and Morris [5] reported that mortality increased from 13% to 31% among 52 patients, and Wright and associates [61] from 0% to 31% among 28 patients if treatment was delayed for more than 24 hours after esophageal perforation. In 390 patients from 11 recent series, the overall reported mortality of esophageal perforation with treatment delayed by 24

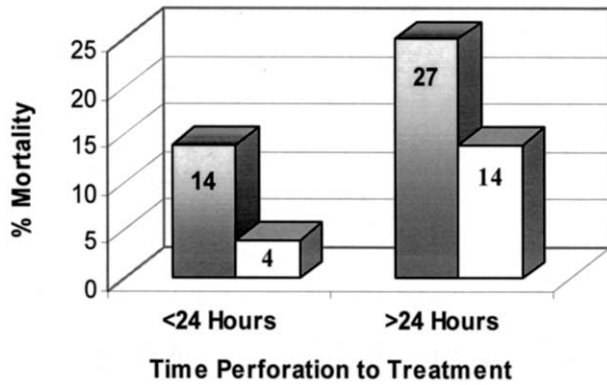


Fig 3. Overall effect of delayed treatment on mortality following esophageal perforation compared with effect of delayed treatment on mortality when perforation is treated by primary repair [5-7, 9, 10, 14, 16, 55, 61, 89, 90, 92, 95]. ■ = all therapy; □ = primary repair.

hours or more was 27% (0% to 46%) compared to 14% (0% to 28%) when treatment was initiated within 24 hours (Fig 3). The wide spectrum of complications that may develop following delayed diagnosis of esophageal perforation exemplifies the need for individualized treatment.

Table 1 displays the combined mortality associated with different modes of therapy for esophageal perforation in reports published between 1990 and 2003. The mortality rate of 18% following esophageal perforation in these series remains high, and has not changed significantly compared to a mortality of 22% reported in a similar review of case series between 1980 and 1990 by Jones and Ginsberg [94].

Operative Management

In 322 patients receiving primary or primary reinforced repair in these series, the mortality ranged from 0% to 31% and averaged 12% (Table 1). Some advocate reinforced repair to prevent leakage and reduce morbidity following surgery [54, 61]. Recent reports [7, 9, 10, 16, 55, 95], however, suggest that both primary repair and reinforced primary repair achieve similar results. Successful reinforced primary repair requires meticulous suturing to avoid postoperative esophageal leaks [54, 61]. Irrespective of reinforcement, the critical determinants of outcome following primary repair are the complete exposure and closure of the ruptured esophageal mucosa and the elimination of distal obstruction [9, 10, 43, 61].

Historically, the use of primary repair following a delay in diagnosis of greater than 24 hours was associated with increased morbidity and mortality [15, 49, 89]. Recent evidence, however, indicates that primary repair offers the highest probability of survival regardless of the interval between esophageal perforation and treatment (Fig 3). Wright and associates [61] reported an overall mortality of 14% following reinforced repair with no deaths resulting from failure of the repair site in a group of 28 patients that included 46% with a delay in diagnosis of greater than 24 hours. Whyte and colleagues [9]

reported outstanding results in a group of 22 patients who underwent primary repair without reinforcement. Although surgery was delayed by more than 24 hours in 41% of these patients, the overall mortality was only 5%. Primary repair thus remains the treatment of choice for esophageal perforation in all patients without esophageal malignancy or diffuse mediastinal necrosis, including those seen more than 24 hours after perforation.

Esophageal resection resulted in a mortality of 17% in these series. Esophagectomy provides the best treatment option when concomitant obstructive esophageal carcinoma or stricture is present, or when attempted drainage, closure, or exclusion has failed to control sepsis [11, 12, 64, 91].

Other surgical therapies are associated with a higher mortality rate in these reports. A mortality of 24% was observed with various exclusion and diversion procedures, and drainage alone was associated with a mortality of 37%. The wide range of mortalities experienced with exclusion and diversion may reflect a lack of technical experience represented in these series. In addition, this technique is often used in patients with severe mediastinal contamination and a high degree of associated morbidity. The best results were achieved when primary closure was included with exclusion and diversion [96].

Nonoperative Management

Nonoperative treatment of esophageal perforation resulted in a mortality of 18% (0% to 33%) in 154 patients from these series (Table 1). As outlined above, each individual patient must be evaluated carefully and meet certain criteria before nonoperative management is used. Survival rates of close to 100% have been reported [7, 8] when the established guidelines were observed. Even with strict adherence to these criteria for nonoperative treatment, however, up to 20% of patients managed nonoperatively develop multiple complications within 24 hours and require surgical intervention [8].

Comment

Esophageal perforation is a serious disorder that is difficult to diagnose and manage. The majority of cases are caused by instrumentation, and mortality remains close to 20%. Early diagnosis and treatment are essential and reduce mortality by at least 50%. Optimal therapy includes primary repair of the perforation site and elimination of distal obstruction. Nonoperative therapy is appropriate in certain well-defined situations. An immediate and individualized approach is required with each case.

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