Esophageal Perforation: A Continuing Challenge

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Perforation of the esophagus remains a diagnostic and therapeutic challenge. Currently, the most common cause of perforation is instrumentation of the esophagus, and the incidence of esophageal perforations has increased as the use of endoscopic procedures has become more frequent. Diagnosis depends on a high degree of suspicion and recognition of clinical features, and is confirmed by contrast esophagography or endoscopy. Outcome after esophageal perforation is dependent on the cause and location of the injury, the presence of underlying esophageal disease, and the interval between injury and initiation of treatment. Reinforced primary repair of the perforation is the most frequently employed and preferable approach to the surgical management of esophageal perforations. Nonoperative management consisting of antibiotics and parenteral nutrition is particularly successful for limited esophageal injuries meeting proper selection criteria.

Historical Notes

The clinical features of esophageal perforation have long been recognized and have been extensively described in the literature. Although mention is made of an esophago-cutaneous fistula secondary to perforation in the Smith papyrus dating from 2,500 BC [1], the first report of spontaneous esophageal perforation was made by Boerhaave [2] in 1724. In 70 pages of clinical and pathological observations, Boerhaave described the distal esophageal rupture that occurred in the High Admiral Baron de Wessenaer in association with forceful vomiting after overindulgence in food and drink. Although Frink [3] had treated a patient with esophageal rupture by surgical drainage in 1941, it was not until 1944 that the first surgical repair after spontaneous perforation was attempted by Collins and associates [4], laying the foundation for the first reported successful repairs by Barret [5] and by Olsen and Clagett [6] in 1947. In that same year, Brewer and Burford [7] also reported the successful surgical repair of a traumatic esophageal perforation. Five years later, Satinsky and Kron [8] reported the first successful esophagectomy after a perforation. Over the subsequent 40 years, the incidence of esophageal perforation has increased [9], iatrogenic causes of esophageal perforations have become more prevalent than spontaneous ruptures, and different management techniques have evolved.

Etiology and Location

The causes of esophageal perforation are as follows:

I. Instrumentation/iatrogenic
   A. Intraluminal
      1. Esophagoscopy
      2. Bougienage
      3. Pneumatic dilatation
      4. Sclerosis of esophageal varices
      5. Placement of intraesophageal tubes (nasogastric, Sengstaken-Blakemore, prostheses)
      6. Endotracheal tube placement
   B. Extraluminal
      1. Mediastinoscopy
      2. Intraoperative injury
         a. Thyroid resection
         b. Leiomyoma enucleation
         c. Proximal gastric vagotomy
         d. Pneumonectomy
      3. Radiation therapy

II. Traumatic
   A. Blunt
   B. Penetrating
   C. Ingestion of caustic substance
III. Spontaneous (barogenic)
   A. Postemetic
   B. Other

IV. Ingestion of foreign body
V. Tumor
VI. Surrounding infections

Their relative frequency from reported series is depicted in Figure 1, and the relationship of cause to location is shown in Figure 2.

Instrumental esophageal perforation occurs in about 1/10,000 upper gastrointestinal endoscopies. In a 1974 American Society of Gastrointestinal Endoscopy survey [10] of 211,410 procedures, perforation occurred in 0.03% of all simple upper endoscopies and in 0.25% of procedures involving bougienage. With an incidence ranging from 1% to 10% [11, 12], pneumatic dilation for achalasia is associated with a fourfold greater risk of perforation than myotomy [13], and this may be related to patient malnutrition [14] or high-compliance balloons [15]. Endoscopic sclerosis of esophageal varices leads to perforation in 1% to 4% of cases [16–19] and the result may be in part dependent on the type and volume of sclerosing agent employed [18, 19], although this concept is disputed by Korula and colleagues [20]. Esophageal perforation can also occur during placement of nasogastric tubes [21, 22], Sengstaken-Blakemore tubes [23, 24], endoesophageal prostheses [25–27], and endotracheal tubes [28–30].

The area at greatest risk for instrumental injury is the cricopharyngeal region of the cervical esophagus. At Lanner's triangle, formed by the constrictor pharyngeus and the cricopharyngeus muscles at the level of the C-5 and C-6 vertebrae, the posterior esophageal mucosa is covered only by fascia. The risk of perforation in this area is further increased by hyperextension of the neck and the presence of kyphosis or cervical vertebral spurs [31].

The next most common areas of instrumental esophageal perforation are the relatively narrow but fixed portions at the distal esophagus just proximal to the hiatus and near the aortic arch and left main bronchus. Other areas at risk include portions of the esophagus just proximal to obstructing processes, portions with extensive carcinomas, and portions at biopsy sites.

Intraoperative injury to the esophagus can occur in areas where an operative procedure is in close proximity to the esophagus. Intraoperative esophageal injury has been reported after anterior cervical spine operations [32, 33], mediastinoscopy [34], thyroid resection [1], leiomyoma enucleation [1], proximal gastric vagotomy [35–38], hiatal hernia repair [35, 39, 40], and pneumonectomy [41].

Perforations from either blunt or penetrating trauma occur primarily in the neck, whereas perforations from ingested foreign bodies or caustic substances are more likely to be intrathoracic. Spontaneous esophageal perforations are barogenic in origin, resulting from a sudden rapid rise in intraesophageal pressure such as can occur with forceful vomiting, childbirth, weight lifting, or blunt abdominal trauma. Barogenic perforations usually occur on the left wall of the distal supradiaphragmatic esophagus, extending into the pleural cavity in 80% of cases [1, 42, 43]. Other less common causes such as perforations of esophageal carcinomas [44–47] and perforations from surrounding infections, especially in immunocompromised patients [48], can occur anywhere in the intrathoracic esophagus.

The association of underlying esophageal disease with esophageal perforation is depicted in Figure 3. Among 147 patients in seven large series of esophageal perforation from a variety of causes [23, 49–54], no underlying esophageal pathology was present in 40%. Benign strictures and anatomic lesions such as diverticula were most commonly associated with perforations, usually instrumental in origin, whereas tumors or achalasia occurred less frequently.
Clinical Features

Diagnosis of esophageal perforation can be difficult, as the presentation is often nonspecific and is easily confused with other disorders such as peptic ulcer disease, pancreatitis, myocardial infarction, dissecting aortic aneurysm, pneumonia, or spontaneous pneumothorax \[55, 56\]. The signs and symptoms of esophageal perforation depend on the location and cause of the perforation and the interval since the perforation occurred.

Cervical perforations most often occur posteriorly where the esophageal wall is thin. Dissection through the retroesophageal space allows spread of contamination to the mediastinum over time, but attachments of the esophagus to the prevertebral fascia may limit lateral spread. Thus, early signs of cervical esophageal perforation include neck stiffness and a dull neck ache, regurgitation of bloody material, and the finding of cervical subcutaneous emphysema. Inflammatory changes in the neck may not develop for several hours, with signs of systemic sepsis often not occurring for up to 24 hours.

In contrast, perforations of the thoracic esophagus result directly in mediastinal contamination, leading to a more rapid development of pneumomediastinum and mediastinitis than after cervical perforations. The thin mediastinal pleura is usually ruptured by the inflammatory process, producing contamination of the pleural space and a pleural effusion. Gastric contents and fluids are then drawn into the pleural space by the negative intrathoracic pressure \[41\], resulting in further inflammation and fluid sequestration, hypovolemia, and the early appearance of tachycardia and systemic sepsis. Chest pain and subcutaneous emphysema are usually present, and dyspnea is often prominent even in the absence of pneumothorax.

Intraabdominal esophageal perforations occur into the free peritoneal cavity and result in peritonitis. A dull retrosternal ache in association with epigastric pain radiating to the shoulders is characteristic because of the relationship of the intraabdominal esophagus to the diaphragm. Systemic signs such as tachycardia, tachypnea, and fever develop early, with progression to sepsis and shock occurring within hours.

Diagnosis

Prompt diagnosis of esophageal perforations is essential, as the incidence of complications and the rate of mortality are dependent on the interval between the perforation and the initiation of treatment. The presence of neck, chest, or abdominal pain after upper gastrointestinal endoscopy should always raise the suspicion of an esophageal perforation and necessitates further study to eliminate the possibility that perforation has occurred.

Plain film findings vary according to the location and cause of the perforation, as well as the interval between injury and radiographic examination \[57\]. Findings of cervical or mediastinal emphysema, pneumothorax or pneumopericardium, pleural effusion, or subdiaphrag-
matic air are all suggestive of a perforated esophagus and warrant further diagnostic study with contrast esophagograms. The presence of food particles or a pH of less than 6.0 in the pleural fluid aspirate is virtually diagnostic [58].

Noncontrast radiographic studies will be normal in 12% [57] to 33% [59] of cases of esophageal perforation, leading some authors [60] to recommend contrast studies after all endoscopic dilations. Although contrast esophagograms are the standard diagnostic procedures in cases of suspected esophageal perforation, it should be noted that the false-negative rate of these examinations can also exceed 10% [45]. Thus, "negative" studies may not completely eliminate the possibility of a perforation. When positive, contrast studies have the advantage of demonstrating the level of the perforation and the presence of extension into the pleural cavity.

The ideal contrast agent for esophagography for suspected perforation was previously the subject of some debate [61–64]. Water-soluble contrast medium is rapidly absorbed and is purported to be less irritating than barium should substantial extravasation occur [65]. Our preference is thinned barium, which may improve localization of subtle perforations [66], and it should be employed whenever a major risk of aspiration is present or whenever a concomitant respiratory tract injury or fistula is suspected. Foley and co-workers [65] recommend an initial bolus of water-soluble contrast medium and then a repeat of the study with a small amount of barium if no leak is seen.

In the patient in whom esophageal perforation is highly suspected clinically but in whom contrast esophagograms are negative, flexible esophagoscopy and computed tomography can be useful diagnostic adjuncts. Flexible esophagoscopy may currently be underutilized in the diagnosis of esophageal perforation. This method provides highly accurate localization as well as direct visualization of perforations [67]. Furthermore, results of esophagoscopy may directly influence treatment decisions, as small perforations or perforations limited to the mucosa can be expected to heal with nonoperative management in the absence of distal obstruction. In select cases, esophagoscopy can facilitate placement of a tube through the perforation for irrigation and drainage [68].

Computed tomographic findings suggestive of esophageal perforation include air in the soft tissues of the mediastinum surrounding the esophagus, abscess cavities adjacent to the esophagus in either the pleural space or the mediastinum, or the demonstration of an actual communication between the air-filled esophagus and an adjacent mediastinal or paramediastinal air-fluid collection [58]. Computed tomographic scans are also particularly useful in follow-up after initiation of therapy and in the evaluation of patients who fail to improve despite either operative or nonoperative management [69]. Localization of loculated pleural fluid collections can also allow computed tomography-directed drainage catheter placement in the postoperative setting or during nonoperative therapy [70].

Treatment of Esophageal Perforations

Once the diagnosis of esophageal perforation is established, multiple factors must be considered in selecting the appropriate therapy. The cause of the perforation, its location, the presence of underlying esophageal disease, and the interval between perforation and diagnosis are critical factors in the determination of treatment. In addition, the condition of the esophagus and the extent of soilage or injury to adjacent organs and tissues as well as the age and general condition of the patient must also be considered.

Treatment options for esophageal perforations are as follows:

1. Operative management
   a. Primary closure
   b. Reinforced primary closure
   c. Resection
   d. Drainage alone
   e. T-tube drainage
   f. Exclusion and diversion
   g. Intraluminal stent

2. Nonoperative management

The goal of any treatment modality must be to prevent further soilage from the perforation, to eliminate the infection produced by soilage, to restore the integrity and continuity of the gastrointestinal tract, and to restore and maintain adequate nutrition [26, 45, 71–74]. Consequently, the principles of operative management include debridement of all infected and necrotic tissue, secure closure of the perforation with appropriate measures to prevent leakage, correction or elimination of distal obstruction, and drainage of contaminated and infected areas. As recovery from esophageal perforation can be prolonged, provision of a route for enteral nutrition such as a jejunostomy should also be considered during any surgical procedure.

Surgical Approaches

The surgical approach is dependent on the location of the perforation. Cervical esophageal tears are best exposed through an incision parallel to the left sternocleidomas-toid muscle and anterior to the carotid artery and internal jugular vein. Perforations in the upper two-thirds of the thoracic esophagus can be reached through a right posterolateral thoracotomy in the fourth or fifth intercostal space, whereas those in the lower third are best approached through a posterolateral thoracotomy in the left sixth or seventh interspace. Abdominal esophageal injuries usually require an upper midline laparotomy. Once the esophagus is exposed, localization of subtle perforations may require instillation of methylene blue into the esophageal lumen or the insufflation of air into the esophagus immersed in saline solution.

Primary Closure

Primary closure of esophageal perforations was first described nearly 50 years ago [4–6]. Primary closure, with or
without tissue reinforcement, is the most frequently used and preferential approach to the surgical management of esophageal perforations. Successful repair depends on debridement of nonviable tissue and closure of the mucosa and muscle in separate layers. Closure of the mucosa with mechanical stapling devices has also been reported [75, 76]. In most instances, the mucosal layer remains healthy. However, a myotomy may be necessary to visualize the full extent of the mucosal injury and to facilitate its repair [75, 77]. Inadequate exposure and repair of the mucosal defect can predispose to leakage from the closure [77].

Late presentation or delayed diagnosis of the perforation results in further tissue necrosis and edema, and can prevent closure or even identification of a substantial muscular layer, in which case the mucosa alone is closed. The contaminated thoracic cavity should be thoroughly irrigated and debrided, with decortication of the lung if necessary. Wide external drainage consisting of Penrose drains in the neck for cervical injuries and large-bore chest tubes for thoracic esophageal perforations should be established along with nasogastric drainage. Contrast studies are usually obtained 7 to 10 days postoperatively, at which time oral intake can be resumed if no leak is demonstrated and ileus has resolved.

Primary closure is best applied to perforations in an otherwise normal esophagus. In the case of prestenotic esophageal perforation, closure alone without resolution of the obstructing process can be doomed to failure [78, 79]. Moghissi and Pender [67] reported a 100% mortality rate among 9 patients with prestenotic perforations treated by either drainage alone or primary closure. For the 14 other patients in the series in whom the stenosis as well as the perforation was addressed, there was a reduction in the mortality rate to 29%.

Reinforced Closure

The potential for breakdown or leakage from primary closure of esophageal perforations, particularly if the interval between injury and treatment exceeds more than a few hours, has led many surgeons to advocate reinforcement of the closure. In the chest, a variety of tissues have been employed to buttress the esophageal closure. A pleural flap, as suggested by Grillo and Wilkins [80], is particularly useful if local reaction has resulted in a thickened pleura. Other choices include omentum, a pedicled intercostal muscle flap [81, 82] or pericardial fat pad flap [83, 84], elevation of a flap of diaphragm [85, 86], or coverage by inflodded gastric wall as a Thal patch [87, 88] or as part of an antireflux procedure if enlargement of the hiatus is necessary or underlying reflux disease is present [56]. Regardless of the type of material used, secure closure necessitates suturing the reinforcing tissue closely, as for an anastomosis, rather than just simple “tacking” [26].

Esophageal Resection

Excellent results with esophageal resection, particularly for perforations in association with malignant obstruction or massive esophageal necrosis, have been reported [46]. A transhiatal or thoracic approach can be employed, with reconstruction performed immediately or as a second-stage procedure depending on the degree of contamination and the condition of the patient. Matthews and associates [89] advocate the use of near-total esophagectomy if resection is required. They cite the ease of performing the anastomosis in the neck, the decreased necessity to use inflamed tissues in the anastomosis, and better longitudinal lymphatic clearance in support of their view.

Concomitant Treatment of Underlying Esophageal Disease

Perforation in association with functional obstruction, as in achalasia, is best treated by repair with a myotomy performed on the side of the esophagus opposite the perforation. An antireflux procedure is generally not required if the myotomy does not extend onto the gastric cardia [90]. However, a Belsey-type repair may offer the advantage of buttressing the closure of distal perforations [91]. An antireflux procedure should also be performed when perforation is associated with hiatal hernia or major reflux esophagitis.

Thoracotomy and Drainage

Drainage alone, without repair of the perforation, is sometimes deemed necessary in the face of widespread inflammation. This technique is best applied to cervical perforations, where better drainage of the surrounding tissues can be obtained than in the chest, as it does not prevent further soilage. Closure of the perforation around a 6- to 10-mm T tube, as reported by Abbott and coworkers [92], creates a controlled esophagocutaneous fistula “venting” the esophagus. This technique may be useful when the security of an attempted repair is in doubt or adequate tissue to reinforce the closure is not available. Intraluminal tubes or stents have also been used successfully to treat perforations through carcinomas or in association with tracheoesophageal fistulas [93–96].

Exclusion and Diversion

Urschel and others [74, 97–99] have described exclusion/diversion techniques in the treatment of complicated esophageal perforations. Exclusion and diversion involves suture of the perforation, if possible, with wide drainage of contaminated tissues; creation of a loop or end-cervical esophagostomy; division or suture of the gastroesophageal junction; and creation of a gastrostomy. Advocates of these techniques claim that they are an application of principles used successfully in the treatment of perforations of other areas of the gastrointestinal tract such as perforated colonic diverticulitis [97]. Exclusion and diversion, however, commits the patient to a second major operative procedure and can create a stagnant pool in the distal esophagus with bacterial overgrowth. Gouge and associates [50] wrote that the “rationale for [the] procedure is debatable and depends on whether the benefits of preventing reflux from the distal esophagus outweighs the disadvantage of creating an obstruction in the distal esophagus.” Thus, this technique...
Patients are given nothing by mouth for usually more than days. Pleural collections are drained through chest tubes or computed tomography-directed drainage catheters [70]. Nasogastric drainage is widely considered integral to nonoperative therapy, although some authors [102] report using nasogastric tubes only selectively. Nasogastric tubes placed in the region of the perforation can also be used for irrigation with either saline or antibiotic solutions as an adjunct to nonoperative treatment [68]. Failure to show signs of improvement within 24 hours of nonoperative therapy should prompt a consideration for operative intervention [25]. Computed tomographic scanning may be helpful to demonstrate undrained collections in such cases [1, 69].

**Outcome**

Recent series [49, 51, 72] have identified cause, location, delayed treatment, underlying esophageal disease, and type of treatment as significant risk factors for death after esophageal perforation. Examination of reported series of esophageal perforations published from 1980 through 1990 [23, 45-47, 49-53, 67, 68, 71, 111-113], the era of modern antibiotic therapy and parenteral nutrition, allows an estimate of the current risk associated with each of these factors.

Among 450 reported patients [45, 47, 49, 51, 55, 67, 111-113], iatrogenic and instrumental injuries were associated with a mortality rate of 19%, whereas spontaneous ruptures resulted in a mortality rate of 39%. Two individual series [114, 115], however, comparing outcome after treatment for spontaneous and iatrogenic perforations failed to demonstrate any significant difference in associated mortality between these two causes. Traumatic esophageal perforations in the collected series [45, 47, 49, 51, 55, 67, 111-113] had the lowest mortality rate, 9%, perhaps because of a decreased interval between perforation and treatment in the setting of multiple injury [113]. In 439 patients [7, 49, 51, 55, 67, 113], cervical tears resulted in an overall mortality rate of 6%, whereas thoracic and abdominal perforations resulted in death in 34% and 29% of patients, respectively. The diminished risk associated with perforation of the esophagus in the neck is attributed to anatomic tissue planes in this area, which prevent the rapid spread of contamination or infection [1], and to the absence of reflux of gastric contents through cervical perforations [116].

Prompt recognition and initiation of treatment of esophageal perforations has long been recognized as essential to a favorable outcome [117]. Delay in treatment of greater than 24 hours after perforation has occurred has been associated with higher rates of both complications [43] and mortality [43, 49, 51, 55, 67, 72]. Bladergren and associates [49] reported that survival decreased from 79% to 67% and Attar and colleagues [51], from 87% to 55% if treatment was instituted more than 24 hours after the onset of symptoms. Basing a treatment plan on the interval between perforation and presentation has also been associated with reduction in mortality [72]. However, the influence of treatment delay may have dimin-
ished slightly. Nesbitt and Sawyers [55] noted that if treatment was initiated within 24 hours, the mortality rate decreased from 56% to 13% during the years 1935 through 1974 but only decreased from 26% to 11.4% in the years 1975 through 1984. Decreases in mortality in patients with delayed diagnosis and treatment after esophageal perforation may be attributable to improvements in critical care, antibiotic therapy, and parenteral nutrition.

Patients with cancer of the esophagus regardless of the site of perforation appear to have increased mortality compared with patients with perforations associated with benign disease processes or no underlying esophageal disease [26, 49, 72, 114]. Further, Saabye and co-workers [118] studied the long-term results in patients with esophageal perforations treated by repair without further surgical treatment of any underlying benign esophageal disease. They found that all patients without underlying esophageal disease remained asymptomatic up to 22 years after repair of instrumental perforations, whereas 35% of those with underlying disease had symptoms requiring reinstrumentation or reoperation.

Table 1 depicts the outcome after treatment of esophageal perforation in 13 series published between 1980 and 1990 [23, 45, 47, 50, 52, 53, 67, 71, 112-114] in which the mortality for individual procedures is reported. Among 325 patients treated with various types of primary repairs, the mortality rate ranged from 0% to 54% and averaged 15%. Gouge and associates [50] reported a 25% mortality in 158 personal and reported patients treated with primary repair versus a 6% mortality in 99 patients treated with reinforced repair. The rate of fistula formation or serious leak also decreased from 39% to 13% when the esophageal closure was reinforced. Other forms of operative management are associated with higher mortality rates in these series: exclusion and diversion, 39%; drainage only, 34%; and resection, 29%, although these increased mortality rates may also be a reflection of more complicated disease.

Nonoperative therapy resulted in a 22% mortality rate in these series (see Table 1). Previous reviews of series of esophageal perforations have reported a 25% mortality rate among 900 patients [112] in 1982 and a 38% rate among 428 patients in 1983 [23]. However, it is difficult among these cases to distinguish between election of nonoperative management as "best therapy" and nonoperative treatment because of inability to tolerate or refusal to undergo operative therapy, as multiple anecdotal reports and small series of instrumental and pediatric perforations have survival rates approaching 100% [68, 70, 103-108]. Erwall and associates [119] reported similar mortality rates between patients treated nonoperatively (12%) and those treated operatively (16%) in a series of 48 patients in whom the presence of shock or systemic symptoms, large perforations, retained foreign bodies, or esophageal malignancy were considered operative indications. Thus, patient selection according to the strict criteria listed in the Outcome section would appear to be necessary for nonoperative treatment to be successful.

Table 1. Outcome After Treatment of Esophageal Perforation in Series Published Between 1980 and 1990

<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Primary Repair</th>
<th>Drainage</th>
<th>Resection</th>
<th>Exclusion and Diversion</th>
<th>Nonoperative</th>
<th>Overall Mortality (%)</th>
</tr>
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<tbody>
<tr>
<td>Goldstein and Thompson [112]</td>
<td>1982</td>
<td>4/23</td>
<td>...</td>
<td>...</td>
<td>6/9</td>
<td>4/12</td>
<td>14/44 (32)</td>
</tr>
<tr>
<td>Sarr et al [45]</td>
<td>1982</td>
<td>0/15</td>
<td>0/13</td>
<td>3/8</td>
<td>0/3</td>
<td>1/8</td>
<td>4/47 (9)</td>
</tr>
<tr>
<td>Larsen et al [23]</td>
<td>1983</td>
<td>10/47</td>
<td>4/8</td>
<td>...</td>
<td>0/2</td>
<td>14/57 (25)</td>
<td>5/33 (15)</td>
</tr>
<tr>
<td>Ajalat and Mulder [111]</td>
<td>1984</td>
<td>0/12</td>
<td>1/5</td>
<td>0/1</td>
<td>0/1</td>
<td>3/13</td>
<td>19/39 (49)</td>
</tr>
<tr>
<td>Borgeskov et al [53]</td>
<td>1984</td>
<td>9/22</td>
<td>3/7</td>
<td>2/2</td>
<td>...</td>
<td>5/8</td>
<td>7/38 (18)</td>
</tr>
<tr>
<td>Radmark et al [52]</td>
<td>1986</td>
<td>1/174</td>
<td>0/2</td>
<td>1/2</td>
<td>0/2</td>
<td>5/17</td>
<td>9/78 (12)</td>
</tr>
<tr>
<td>Brewer et al [71]</td>
<td>1986</td>
<td>9/83</td>
<td>0/6</td>
<td>0/2</td>
<td>0/2</td>
<td>1/2</td>
<td>8/34 (24)</td>
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<tr>
<td>Moghissi and Fender [67]</td>
<td>1988</td>
<td>7/139</td>
<td>4/5</td>
<td>2/11</td>
<td>...</td>
<td>13/29 (45)</td>
<td>5/67 (7)</td>
</tr>
<tr>
<td>Flynn et al [113]</td>
<td>1989</td>
<td>1/44</td>
<td>2/9</td>
<td>1/4</td>
<td>0/8</td>
<td>3/18 (17)</td>
<td>4/5 (85)</td>
</tr>
<tr>
<td>Gouge et al [50]</td>
<td>1989</td>
<td>0/14</td>
<td>0/1</td>
<td>1/1</td>
<td>0/2</td>
<td>0/3</td>
<td>18/61 (30)</td>
</tr>
<tr>
<td>Attar et al [51]</td>
<td>1990</td>
<td>5/30</td>
<td>7/17</td>
<td>2/9</td>
<td>4/5</td>
<td>...</td>
<td>128/589</td>
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<tr>
<td>Total</td>
<td>1980-1990</td>
<td>49/325</td>
<td>28/83</td>
<td>14/49</td>
<td>18/46</td>
<td>19/85</td>
<td>128/589</td>
</tr>
<tr>
<td>Mortality (%)</td>
<td></td>
<td>15</td>
<td>34</td>
<td>29</td>
<td>39</td>
<td>22</td>
<td>22</td>
</tr>
</tbody>
</table>

* Data are presented as number of deaths/number of patients undergoing procedure.  
  * Numbers in parentheses are percentages.  
  * This includes 2 patients who also underwent an antireflux procedure.  
  * This includes 2 patients who also underwent myotomy.  
  * This includes 5 patients who also underwent fundoplication.

Conclusion

Esophageal perforation is a potentially lethal condition resulting most frequently from instrumentation. Early diagnosis and intervention are necessary to prevent morbidity and mortality. Esophageal perforation is best treated with definitive repair including tissue reinforcement of the closure and elimination of distal obstruction.
Nonoperative therapy consisting of antibiotics and parenteral nutrition may also be appropriate in select patients.

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