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Luc Michel, M.D., Hermes C. Grillo, M.D.,
and Renald A. Malt, M.D.

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ABSTRACT Esophageal perforation can be caused by any instrument, device, or foreign body reaching the hypopharynx. Diagnosis remains difficult. If esophageal perforation is suspected, Gastrinogen (megalacmine diatrizoate) swallow study, eventually followed by barium swallow study, is the most useful diagnostic test. Absolute rules cannot be made about the selection of nonoperative or surgical treatment. If diagnosed early, cervical or thoracic esophageal perforations can sometimes be treated conservatively if there are no signs of systemic sepsis. Recurrent leakage after surgical closure is not unusual. Local tissue flaps can reinforce the closure, particularly after delayed operation, thereby often avoiding the necessity for a reoperation or an esophageal exclusion.

The decision for surgical or nonsurgical management of esophageal perforation remains difficult, especially now that nonoperative management of this condition has been advocated even for forceful, spontaneous postemetic perforations (1-4), which normally have a poor prognosis after delayed surgical treatment. In this review, we discuss the etiology and pathophysiology of esophageal perforation and attempt to clarify the concepts of management.

Etiology and Pathophysiology of Esophageal Perforation

Iatrogenic Perforation

The most common cause of esophageal perforation is instrumentation. Because perforations occur during esophagoscopy, gastroscopy, and esophageal dilation, frequent use of upper gastrointestinal fiberoptic endoscopy has led to an increase in the actual number of perforations. Katz (5) reported a perforation rate of 0.074% with the rigid esophagoscope and 0.093% with the fiberoptic esophagoscope, representing no significant improvement in safety with the use of fiberoptic instruments. The most common area of perforation from an endoscopic examination is the region of the cricopharyngeus muscle. The esophageal intrathorax is the narrowest region of the esophagus, and the cricopharyngeus muscle contributes to this decrease in diameter of the lumen. This anatomic feature led Jackson (6) to call this constricted area the "pass of Bal-eil Mandsri" or the "gate of tears." In a series from the Mayo Clinic (7), esophageal perforation occurred in 0.4% of 8,038 patients who underwent peroral gastrointestinal endoscopic procedures. Similarly, 24 of 31 cases of esophageal perforation reported from the Ochsner Clinic were due to endoscopic examinations (8). The 1974 survey by the American Society for Gastrointestinal Endoscopy (9) of 211,410 esophagogastroduodenoscopic examinations showed a perforation rate of 0.13% (0.03% perforation from esophagogastroduodenoscopy and 0.25% from esophageal dilation).

Dilations of the esophagus carry a risk of perforation because most of them are performed for stenosis (reflux esophagitis, postoperative stricture, achalasia). Perforations generally occur in the diseased thoracic or abdominal portion of the esophagus. Even bougienage under endoscopic control gives a false sense of security, as perforation can occur without the operator's knowledge when the bougie is pushed ahead of the esophogoscope. Early dilation after a caustic burn may also predispose to perforation because a is performed through friable tissues. Forceful hydrostatic or pneumatic dilation for achalasia carries a 4% incidence of esophageal perforation with mediastinal sepsis versus only a 1% incidence after esophagomyotomy (10).

Celestin tubes and similar intraesophageal tubes have also been implicated in iatrogenic perforation of the esophagus (11, 12). The upper
end of the tube can form a sharp edge when grasped for removal, or the tube itself can erode through the esophagus. The risk of erosion has been reduced with the use of polyvinyl esophageal prosthetic tubes placed perorally [13].

Sengstaken-Blakemore tubes and Linton tubes can cause esophageal disruption when they are kept inflated too long or when an agitated patient extracts them while they are still inflated [14]. Furthermore, esophageal perforation occurs when the gastric balloon is inflated in the esophagus. Traction on a Linton tube must not be maintained for more than twenty-four hours; no traction should be applied to a Sengstaken-Blakemore tube.

Traumatic endotracheal intubation can perforate the esophagus just below the cri- opharyngeus muscle, generally in the dorsal midline at an area of weakness where the muscosa is supported only by luscia (Lannier's triangle) [15]. Any type of endotracheal tube inserted with good visualization usually causes no problems. With poor glottic exposure, a blind thrust toward the larynx often causes laceration, hematoma, or perforation of the hypopharynx and the cervical esophagus. Consecutive postmortem examinations were done on 103 patients who had undergone instrumentation either orally or nasally with endoscopes or flexible tubing (nasogastric tubes, oral and nasal suction catheters, and endotracheal tubes); 62 of the patients had specific lesions in the hypopharynx and cervical esophagus directly related to the instrumentation [16]. Only 2% of these injuries were diagnosed ante-mortem. Among the 17 patients in whom only an endotracheal tube was placed, 4 had a hematoma in the cervical esophagus and hypopharynx, and 1 sustained a fatal cervical perforation.

Parasophageal operations such as hiatal hernia repair [17–20], vagotomy [21, 22], and radical pneumonectomy [23] are responsible for esophageal perforation. A hiatal hernia repair in which sutures are placed through the esophageal muscularis as part of the repair can cause a tear or a perforation of the esophageal wall. Perforation can occur also after a Hill repair [17], in which the median arcuate ligament is employed as an anchor, as well as after a Nissen fundoplication [19]. The incidence of esophageal perforation after vagotomy is about 0.5%, with 4 deaths related to perforation in a total of 5,910 patients [21, 22].

'Simple' monitoring devices such as the esophageal stethoscope or the new esophageal obturator airway often included in the cardiopulmonary resuscitation kit have caused esophageal perforation [24, 25].

Spontaneous Perforation
Spontaneous perforation of the esophagus is being reported with greater frequency, most probably because the incidence is actually increasing, but because the condition is more generally recognized. Since the original description of Boerhaave [26, 27], the term spontaneous rupture of the esophagus has been used almost routinely in the literature to include all perforations involving the entirety thickness of the esophageal wall, whenever perforation was associated with forceful or prolonged emesis. Many other causes have been reported: heavy lifting, defecation, seizures and forceful childbirth, blunt trauma, and even forceful swallowing [28–32].

Experimental work has permitted a better understanding of the mechanisms involved in 'spontaneous' rupture. Abbott and colleagues [33] proposed a new classification of atraumatic, so-called spontaneous, rupture of the esophagus. Although their classification may seen to complicate the taxonomy of esophageal perforation, it actually clarifies the factors concerned in spontaneous rupture of the esophagus: (1) increased intraluminal pressure, (2) preexisting esophageal diseases, and (3) neurogenic causes of perforation. Each of these factors can cause spontaneous rupture independent of the other, but they commonly are combined in an individual patient. In a minority of patients, no cause can be found.

Increased Intraumal Pressure. A tremendous and sudden increase in pressure in an apparently normal esophagus is the usual cause of rupture [34–36]. The increase in pressure is usually due to vomiting or retching. Because vomiting is a common phenomenon, an explanation is needed to account for the rarity of
spontaneous esophageal rupture. According to Tidman and John [36], the mechanism of spontaneou s rupture of the esophagus is sudden in- crease of intraabdominal pressure transmitted to a relaxed esophagus (causing a rise in intraabdominal pressure), the inlet of which is obstructed. Obstruction of the inlet results from spasm of the cricopharyngeus muscle in response to vomiting entering the upper airways [36] or from failure of the upper esophagus to relax due to fatigue of the vomiting center or from spasm of the muscles themselves because of repeated vomiting [35, 37].

**Preeexisting Esophageal Disease.** In support of preexisting esophageal disease as an important etiological factor in esophageal perforation, O’Connell [35] mentioned that many patients rupture the esophagus while relaxed, while reading, while watching television, and even during sleep. Distal obstruction of the esophagus is thus probably the most important single factor in rupture of the esophagus in the absence of vomiting and sudden increase of intraluminal pressure. The nature of the obstruction may be a stricture, web, malignancy, esophageogastric ring, or achalasia. Conte [36] explained the sequence of events leading to esophageal perforation when obstruction is present as occurring by a notility disturbance manifested by weak esophageal response. A forceful voluntary swallowing ensues in an ef fort to assist the weak esophageal response, propelling the meal bolus downward. But the inability to pass the bolus because of the distal obstruction results in lateral wall pressure, which, if sudden and elevated, can rupture the esophagus. If the build-up of pressure is slow and moderately elevated, an esophageal diverticulum above the obstruction could result.

**Neurogenic Causes of Esophageal Perforation.** Rupture of the esophagus is a complication liable to develop in patients with disease of the central nervous system. For instance, esophageomalacia found in association with central nervous system disease can predispose to esophageal rupture. Several authors have reported cases of esophageal perforation that were associated with intracranial lesions or followed intracranial operative procedures [36, 39]. According to Cushing [39], the stimulation of the vagal tract in the brain, with resultant local spasm of small blood vessels, can lead to ischemia in the upper gastrointestinal tract, resulting in softening of the esophagus that can then rupture under increased pressure. Kissella and colleagues [40] included “vomiting by a patient with a neurogenic lesion” as one of the recognized circumstances under which rupture of the esophagus can occur. Head injury and cerebral aneurysms have also been implicated in the process [41].

**Esophageal Perforation Caused by Foreign Bodies.** Foreign bodies of the esophagus are a frequent problem. Jackson and Jackson [42] reported a series of 2,730 foreign bodies, 526 of which were bones and 535, coins. The frequency of bones is not surprising as they can be present in food. The frequency of coins is harder to explain, when not found in patients with psychiatric problems. In adults, the wearing of artificial dentures may inhibit feeling an object before swallowing and can be the indirect cause of swallowing or inhaling a foreign body. Swallowing a piece of artificial denture also happens.

Severe perforations can be caused by attempted removal of foreign bodies, either by a poorly trained endoscopist or by one who tries to push the foreign body ahead of the endoscope into the stomach too vigorously. Endoscopic removal of the foreign body should be performed under general anesthesia, especially in the pediatric age group. Whenever possible, a duplicate of the foreign body should be obtained in order to select accurately the best endoscope and forceps to use. The duplicate can also afford an opportunity to study possible presentations of the object in the esophageal lumen [42].

Roentgenograms of the neck (anteroposterior and lateral views) should be made in hyperextension, since the normal position of the clavicular shadow hides the esophageal inlet. This maneuver raises the larynx and the esophageal inlet, making them visible in the latera projection. If the suspected object is not radiopaque (the pull-tab of a cola can is a frequent example in children), a contrast examination with thin barium solution is indicated.
Sometimes the perforation is not caused by the sharp edge of the foreign body penetrating the esophagus, but by pressure necrosis, progressive breakdown of the esophageal wall and migration of the object into the pleural cavity or even into the major vessels [43]. When the impacted object cannot be removed by endoscopy, direct surgical intervention is indicated. A surgical approach is also indicated in free perforation, or when there are signs of mediastinitis.

A foreign body can be extracted by passing a Foley urethral catheter distal to the object, then inflating the balloon of the catheter to dilate the esophagus and dislodge the foreign body [44, 45]. This technique does not require endoscopy and general anesthesia and appears to be safe when used to remove smooth-edged foreign bodies in the esophagus.

There are isolated reports of perforation related to esoteric causes: sword swallowing [46] and self-dilatation by the passage of a length of heavy electric wire into the esophagus [47].

External Trauma

Because of the relatively well protected location of the esophagus, penetrating injuries are rare. In general, they are associated with injuries to other surrounding structures. In a 1979 review [48] of 125 consecutive penetrating wounds of the chest (54% stab wounds and 46% gunshot wounds) esophageal perforation was disclosed at early thoracotomy in only 3 cases. Reference to earlier series reveals that penetrating wounds of the thoracic esophagus carried a mortality of 47 to 57% compared with 11 to 17% for cervical esophageal wounds [4, 48a, 49, 50].

Widening of the superior mediastinum, cervical subcutaneous emphysema in a patient with a neck wound, and increased distance between the trachea and the vertebral column (prevertebral shadow) are the main roentgenographic signs of traumatic cervical esophageal perforation. Widening of the entire mediastinum, mediastinal emphysema, and hydro pneumothorax are the most common findings in thoracic esophageal perforation. Sometimes a mediastinal "crunch" can be heard (Hamman's sign). Symbas and coworkers [50] and Lundy and colleagues [51] recommend that all patients who are seen with hematemesis or bloody drainage from the trachea be studied promptly with Gastrografin (meglumine diatrizoate) esophagography to ascertain presence of esophageal perforation.

All cervical wounds penetrating the platysma muscle should be explored. Esophageal injury and laryngeal, tracheal, or pleural injuries may lie below an apparently minor neck injury in patients who appear to be in stable condition. For example, one case report demonstrated how a knife falling only a few feet onto a 5-year-old boy could penetrate the left internal jugular vein, the esophagus, and the right pleura, yet spare the left carotid artery and left pleura [52].

Ruptures of the esophagus from blunt abdominal trauma are rare. To our knowledge, a total of 31 cases of solitary esophageal perforation from nonpenetrating trauma have been reported [53, 54]. The pathogenesis and the clinical manifestation are similar to those of spontaneous postemetic esophageal rupture. The sudden elevation in the intraesophageal pressure is probably one of the contributing factors in this type of perforation, which occurred in 90% of the ruptures in the thoracic esophagus and in 10% in the cervical portion. Only a single cervicothoracic perforation was caused by a steering wheel trauma [55]. The sudden compression of the thorax, as it occurs in deceleration types of injury, may have an explosive effect on the esophagus without evidence of severe injuries to other structures [55]. The esophageal rupture occurs when the esophagus is stretched beyond the limits of its elasticity. In the neck, the unyielding spine may function as it does in the abdomen, where anterior blunt trauma can cause transection of the pancreas or the small bowel by vertebral.

Accidental discharge of gas under pressure into the oral cavity is another unusual cause of traumatic esophageal rupture. Only 13 cases have been reported [56-58], despite the widespread use of compressed gases in industry. A dramatic variant of pulmonary rupture of the esophagus involved a 6-year-old boy who had bitten the inner tube of a tractor tire that protruded through a defect in the tire [56].
Concepts of Treatment

Surgical closure of esophageal perforation has been advocated [54, 59-64], but the therapeutic decision is not so simple. The approach depends on the site of injury (cervical, thoracic, or abdominal) and the severity of systemic response. For instance, in the early stage after perforation of the thoracic esophagus, it can be difficult to determine whether a tiny transmural perforation will lead to massive pleural contamination with subsequent septic shock and respiratory failure, or will remain contained within the mediastinum.

Criteria for considering nonoperative management of "continued" perforation have been proposed by Cameron and associates [2]: (1) the cavity should be well drained back into the esophagus; (2) minimal symptoms should be present; and (3) there should be minimal evidence of clinical sepsis. In mild cases with few local and systemic symptoms, Meorgoli and Klasson [65] and Lyons and associates [3] also attempted medical treatment, which included massive antibiotic therapy, intravenous hydration, withdrawal of all oral intake, and eventually nasogastric drainage and total parental nutrition. The same approach has been advocated for "forced spontaneous perforation," which usually has a poor prognosis even after early surgical treatment [1, 2, 66].

Absolute indications for emergency operation are presence of a pneumothorax, pleuropneumonia, mediastinal emphysema, systemic sepsis, shock, and respiratory failure. Preoperative confirmatory evidence of perforation should be obtained by gastrografin swallow study, followed eventually by barium study if the gastrografin swallow is negative.

If the perforation is cervical, suture and drainage can be performed through an incision along the sternocleidomastoid muscle. Drainage alone, preferably with wound suction, will sometimes suffice if no distal obstruction exists. For thoracic perforation in the poorest surgical risks (elderly patients with complicating factors, physical status corresponding to classes 4 and 5 in the classification of the American Society of Anesthesiologists), simple pleural drainage or drainage through a paravertebral rib resection is sometimes indicated. Even in those difficult cases, Loep and Groves [23] recommended early thoracotomy and closure of the perforation. Since recurrent leakage is a frequent occurrence, the closure should be supported with a local tissue flap. These flaps include pericardium [67], diaphragm [68], intercostal muscle [69, 70], and stomach wall [71]. Support with a local tissue flap is particularly helpful when surgical treatment has been delayed, because the esophageal wall can present such an inflammatory reaction that it becomes unsuitable for a technically satisfactory suture. Another simple way of dealing with the problem of delayed recognition of esophageal perforation consists of flapping or wrapping a pedicled pleural flap or around the esophagus, suturing it firmly over the area of leakage and around its margin [53]. This procedure has the virtue of permitting closure of the perforation and complete cleansing of the pleural space, with decortication of exudate from the lung surface and avoidance of a second operation. Drainage gastrostomy and sometimes feeding jejunostomy are performed concurrently.

The presence of an obstructing lesion of the esophagus (e.g., cancer) is another critical factor, as relief of the obstruction is one of the pre-requisites of successful treatment of esophageal perforation. In some cases, it is better to restore both the perforation and the original lesions rather than to rely on repair and drainage. Forty-one cases of primary esophagogastrostomy for treatment of perforation of the esophagus, generally after rigid endoscopy, have been reported [60, 72-77]. Johnson and associates [74] suggested that primary esophagogastrostomy should be performed within a few hours after perforation, because with greater delay and the presence of more severe infection, the resection would be made in badly contamined tissues with potential risks to the esophageal anastomosis. For these cases, they recommended exclusion of the thoracic esophagus [78]. However, even when diagnosis was delayed, successful primary resection was performed in the face of established mediastinitis [73, 76].

The principle underlying esophageal exclusion has long been applied to the treatment of
fistulas of other portions of the intestinal tract. It seemed reasonable to apply the same princi-
ple to the treatment of fistula of the thoracic esophagus [78, 79]. This technique consists of
suturing the esophageal perforation, instituting mediastinal and pleural drainage, and inserting an
uniliocidal tape around the esophagus below the perforation and deep to the vagus nerve.
Initially, Johnson and co-workers [78] proposed dividing the stomach at its junction with the
esophagus. The cervical esophagus is also di-
verted in continuity by opening it longitudi-
nally and suturing its muscularis to the sub-
cutaneous fascia and skin; a gastrostomy is
performed for feeding. At a time of election
after healing of the perforation, a thoracotomy is
performed and the esophageal ligature is re-
moved. Closure of the cervical esophagostomy
is effected by dividing the esophageal cutane-
ous suture line and closing the esophagostomy
transversely. Although this radical approach
presents the disadvantages of prolonged dis-
comfort and multiple operations, it is some-
times the only way to control persistent
mediastinal and pleural infection, especially when the initial operation has been delayed or has been unsuccessful.

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