

Esophageal Perforation: *

Diagnosis and Treatment

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THE INCIDENCE of esophageal perforations appears to be increasing and the problems associated with recognition and treatment of this often catastrophic occurrence remain a challenge. Spontaneous rupture of the esophagus, classically the most explosive and most frequently lethal form, was first described by Boerhaave in 1724 and has since been the subject of many reports, including Barrett's of the first successful repair in 1947.^{2, 4} Only recently have the more frequent iatrogenic instrumental perforations been documented.^{3, 6, 8, 15} Esophageal perforations due to penetrating or blunt trauma are also being encountered more often.

Perforated esophagus is a surgical emergency; it is the most serious and frequently the most rapidly lethal perforation of the gastro-intestinal tract. Untreated it is usually fatal. Contamination of the mediastinum, and often a pleural cavity, with corrosive fluids, food matter and bacteria leads to cardiorespiratory embarrassment, shock, major fluid losses and fulminating infection. With prompt, aggressive surgical treatment survival can be expected in most cases. In 1943 Neuhof and Jemerin⁷ demonstrated that surgical drainage alone resulted in a 60 per cent survival rate whereas conservative nonoperative treatment produced an 84 per cent mortality rate. A decade later Samson,¹⁰ Weisel¹⁵ and Over-

street and Ochsner⁸ reported greatly decreased mortality with early suture closure of esophageal perforations. Yet in recent series of esophageal perforations mortality rates between 15 and 30 per cent are still reported.^{1, 5, 6, 14}

Review of 42 Cases

In the records of the Vanderbilt University Hospital and the affiliated Nashville General and Veterans Administration Hospitals there have been 42 patients with esophageal perforations in the 30-year-period from 1935-1964. Diagnosis was established by means of chest roentgenogram, esophagram, surgical exploration or post-mortem examination. There were 13 females and 29 males ranging in age from 11 months to 73 years; 25 patients were between the ages of 40 and 70. The incidence of esophageal perforation, by 5-year periods, is shown in Figure 1. In the period 1950-54 there was one case per 20,000 admissions and in 1960-64, one case per 8,000 admissions.

Etiology

The various causes of perforations are shown in Tables 1 and 2. Of seven perforations secondary to para-esophageal surgery, five were associated with vagotomy and two occurred during repair of an esophageal hiatus hernia. If these seven cases are added to the instrumental perforation group the number of iatrogenic perforations totals

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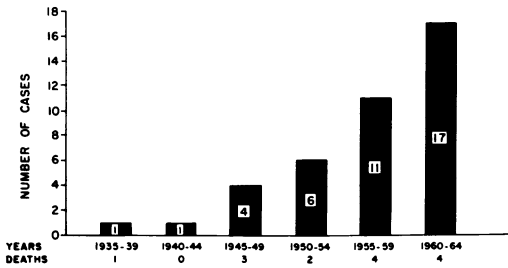


FIG. 1. Incidence of esophageal perforation by 5-year periods from 1935-1964. There was one perforation per 20,000 admissions in the period 1950-54 and one perforation per 8000 admissions in the period 1960-64.

31 cases or 74 per cent of the series. The three cases caused by nasogastric tubes include two Sengstaken tube ruptures of the esophagus and one which occurred when a patient forcibly removed a Cantor tube. Only three of the ten diagnostic endoscopies resulting in perforation included an esophageal biopsy; the other seven examinations were prompted by evidence of upper gastro-intestinal bleeding, foreign body, achalasia or hiatus hernia. Eleven of the perforations occurred in association with dilation procedures, five for caustic strictures and six for achalasia. Each of the caustic stricture dilations resulting in perforation involved antegrade dilation either blindly or via the esophagoscope; none resulted from retrograde or guided bouginage. Three of the perforations in patients with achalasia were secondary to pneumatic dilation (Browne-McHardy dilator) while

TABLE 1. Etiology of Esophageal Perforation

Cause	No.
Instrumentation	24
Gastrosocopy	2
Esophagoscopy & dilatation	6
Dilatation alone	5
Nasogastric tubes	3
Para-esophageal surgery	7
Spontaneous	5
Penetrating trauma	2
Blunt trauma	2
Foreign body	2
Total	42

} 74%

the other three resulted from esophagoscopy and bouginage.

Figure 2 indicates the location of the perforations and the incidence of iatrogenic perforations at each location. General anesthesia was utilized in two of the 21 cases of perforation caused by endoscopy or dilation; in the remaining 19 cases local anesthesia or no anesthesia was employed.

Diagnosis

The incidence of the common signs and symptoms of esophageal perforations are listed in Table 3. The most consistent symptom of thoracic perforation was chest pain. It usually occurred at the time of the perforation, was frequently substernal and quite severe. A significant temperature elevation was the next most frequent manifestation and usually occurred within a few hours; in a few patients fever did not occur until after 24 hours. Of perhaps more importance was the frequency of abdominal pain, tenderness and epigastric muscle spasm which often seemed to confuse the diagnosis. Subcutaneous emphysema manifested by crepitus in the neck, face and chest

TABLE 2. Causes of Esophageal Perforation

Cervical (7)	
Endoscopy	3
Endoscopy & dilation	1
Gunshot wounds	2
Dental prosthesis impacted in circopharyngeus	1
Upper Thoracic (7)	
Blunt trauma	2
Esophagoscopy with biopsy of carcinoma	3
Dilation of caustic structure	2
Lower Thoracic (19)	
Spontaneous rupture	3
Sangstaken tube	2
Diagnostic endoscopy	4
Endoscopy and dilation	5
Dilation alone	3
Open safety pin	1
Transthoracic vagotomy	1
Abdominal (9)	
Para-esophageal surgery	6
Cantor tube	1
Spontaneous rupture	2

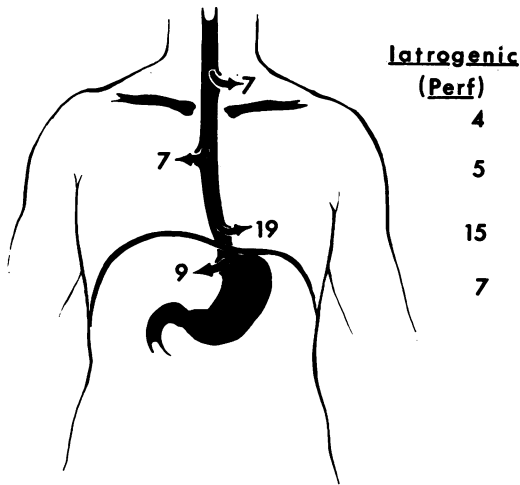


FIG. 2. Site of 42 various esophageal perforations and incidence of iatrogenic perforations.

wall was common. It was noted in the records of about one third of the thoracic perforations, in six of the seven cervical perforations and in none of the abdominal perforations. The diagnosis of a thoracic perforation was established by clinical findings when the triad of fever, chest pain and crepitus was present; following instrumentation the presence of chest or abdominal pain and crepitus was sufficient evidence to establish the diagnosis. The signs and symptoms of cervical perforations were less fulminating in onset and included a painful, tender, swollen neck, usually with crepitus, fever and dysphagia.

Roentgenographic studies were extremely helpful in confirming the clinical diagnosis or establishing the diagnosis in question-

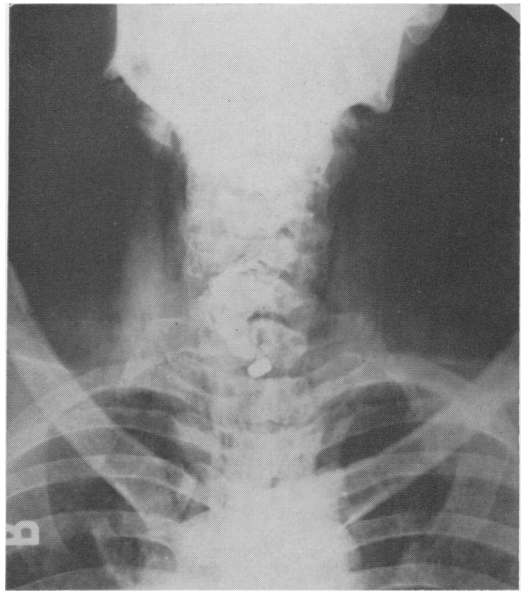


FIG. 3. Esophagram in a patient with a gunshot wound of cervical esophagus; subcutaneous and mediastinal emphysema are apparent as is the pooling of extravasated contrast medium.

able cases. Six of the seven cervical perforations had subcutaneous emphysema demonstrated by x-ray examination (Fig. 3). Twenty-two of the 26 patients with thoracic perforations had chest roentgenograms; of the other four patients the diagnosis was established on clinical findings in two and at autopsy in two. Of the 22 chest roentgenograms two were interpreted as normal and 20 were abnormal. The highly suspect finding of hydrothorax, pneumothorax or pathognomic finding of mediastinal emphysema was present in 17 cases (Fig. 4). Pneumopericardium was demonstrated in

TABLE 3. Signs and Symptoms of Esophageal Perforation

Indications	Location of Perforation			Total
	Thoracic (26)	Cervical (7)	Abdominal (9)	
Chest pain	20	1	1	22
Upper abdominal pain	8	0	4	12
Cervical pain	0	5	0	5
Dyspnea	7	1	1	9
Temp. elevation	18	2	6	26
Crepitus	7	6	0	13

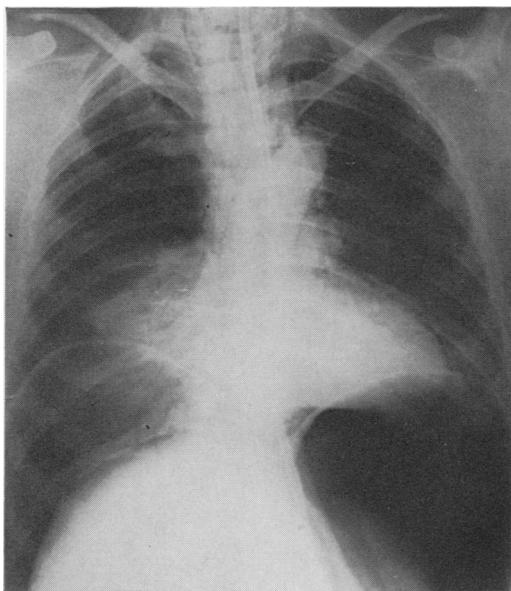


FIG. 4. Mediastinal emphysema, right pneumothorax and pneumoperitoneum are demonstrated in this patient with gastroscopic perforation of the esophagus.

two cases, pneumoperitoneum in one and pneumonitis or pleuritis in three.

An esophagram was done in 21 of the cases. The esophagram demonstrated and localized the defect in the esophagus in 18 of the 21 examinations (Fig. 3, 5, 6).

Diagnosis of esophageal perforation was established on the basis of the clinical findings, chest roentgenogram, or esophagram or was recognized at the time perforation in 39 of the 42 cases. In the other three instances the esophageal perforation was found at autopsy and had not been suspected antemortem. One of these was a patient terminally ill with heart disease who sustained a spontaneous postmicturition perforation and died in shock in 6 hours later. The second was a patient actively bleeding from esophageal varices whose perforation was caused by a Sengstaken tube and who died 2 hours later with continued bleeding. The final patient sustained

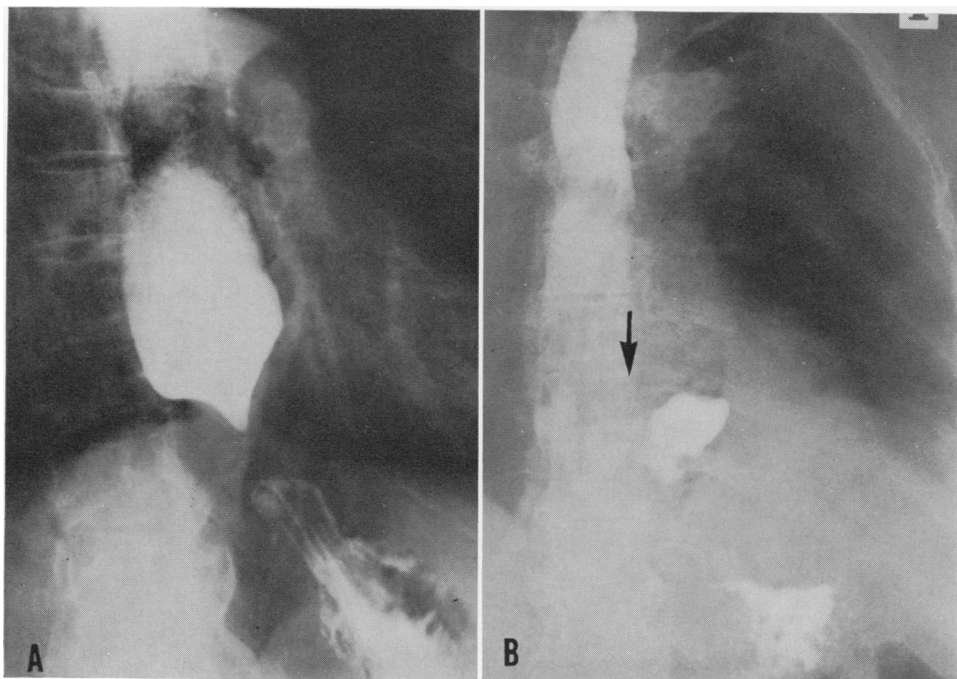


FIG. 5. A. Patient with achalasia which is demonstrated by esophagography. B. Perforation secondary to pneumatic bag dilation demonstrated on esophagram.

a perforation at the time of an abdominal vagotomy for a marginal ulcer, the perforation was not recognized and he died in shock 48 hours later.

Treatment and Results

Cervical Esophagus

There were seven patients with perforation of the cervical esophagus. In five patients suture of the perforation and drainage was done within the first 18 hours; four made a prompt and uneventful recovery and the fifth, with two perforations due to a gunshot wound, developed a fistula which required 8 weeks to close. The sixth patient, with a stricture secondary to laryngectomy and neck dissection, sustained a perforation during dilation; antibiotic therapy was started immediately and an abscess developed which required incision and drainage on the 13th day. A chronic fistula resulted. The seventh patient received the perforation at esophagoscopy; antibiotic therapy was instituted at 36 hours when his neck was painful and swollen, and he died after 96 hours because of laryngeal edema and extensive cellulitis. Treatment of these patients and the results are summarized in Table 4.

Thoracic and Abdominal Esophagus

There were 35 patients with perforation of the thoracic or abdominal esophagus (Table 5). Fifteen patients had early suture closure of the perforation and drainage of the mediastinum; of these 14 survived and one died. The one death was in a patient with Sengstaken balloon rupture of the esophagus; closure was promptly effected but the patient died a few hours later of

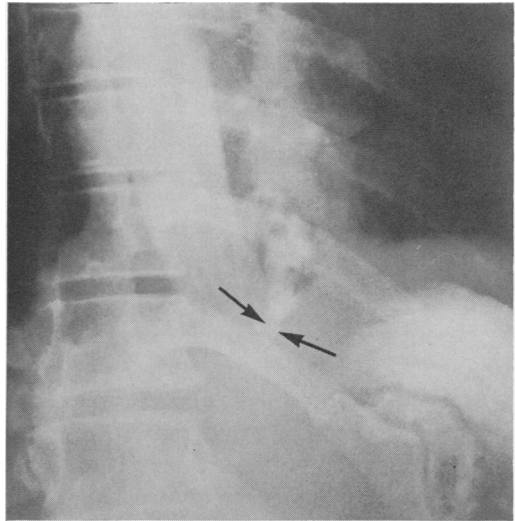


FIG. 6. Perforation in this patient was thought to be "small" as determined by esophagography, but esophageal rent was 8 cm. in length.

persistent shock, peritonitis and mediastinitis. In one case the suture closure of the perforation broke down; adequate drainage was provided and the resultant fistula closed three weeks later. Of the 15 primary closures 12 were effected in less than 24 hours; closure was successful in the other three at 30, 48 and 72 hours. In three patients with achalasia, a Heller cardio-esophageal myotomy was performed at the time of primary closure and in a fourth patient a short stricture was successfully excised and an end-to-end anastomosis performed.

Suture of a surgically created perforation of the abdominal esophagus without drainage was performed in three patients: two

TABLE 4. Cervical Perforations—Treatment and Results

Treatment	Lived	Died
Suture & drainage	5	0
Drainage	1	0
Nonoperative treatment	0	1

TABLE 5. Results with Thoracic and Abdominal Esophageal Perforations

	Lived	Died	Mortality
Suture closure & drainage	14	1	7%
Suture closure without drainage	2	1	33%
Drainage alone	3	2	40%
Nonoperative treatment	3	6	66%

TABLE 6. *Results of Surgical Treatment
Relative to Time*

Delay in Treatment	Lived	Died	Mortality
<24 hours	19	3	13%
>24 hours	6	5	45%

made an uneventful recovery but the third died 2 months later, as a result of early suture line breakdown, after prolonged morbidity and subphrenic abscess, peritonitis and sepsis.

Drainage of the mediastinum without attempting repair of the perforation was done five times; three patients survived and two died. One death occurred in a 6-year-old child who sustained perforation during dilation of a caustic stricture; the child was moribund 16 hours later when transpleural mediastinal drainage was instituted. The other death was in a 71-year-old man with a spontaneous rupture of the abdominal esophagus. At laparotomy 4 days later the perforation was found, omentum was sutured into the area of the defect and a drain placed below the sutured omentum. At postmortem examination extensive infection was found to have extended into the superior mediastinum.

Nine patients had nonoperative treatment consisting of antibiotics, nasogastric suction and intravenous fluids; in three prompt healing occurred—twice after esophagoscopic biopsy of a carcinoma and once after esophagoscopy for hiatus hernia. Each procedure had resulted in chest pain and evidence of free air in the mediastinum on chest roentgenogram, but on esophagram leakage could not be demonstrated. Six patients died with nonoperative treatment; four had terminal drainage procedures performed 5, 8, 10 and 12 days after the perforation but all died in the ensuing 24 to 48 hours.

The three patients who died without treatment of any kind and who were found to have an unsuspected esophageal perforation at autopsy are omitted from the results of treatment analysis.

Table 6 shows the influence of the time between perforation and surgical treatment on the results of treatment, including cervical and thoracic and abdominal esophageal perforations. The mortality rate was more than three times as great when treatment was delayed for more than 24 hours. In the entire series 10 patients had nonoperative, conservative treatment of an esophageal perforation and three survived while seven died.

Five patients in the study had spontaneous postemetic perforations of the esophagus; three were successfully treated by suture closure and drainage within the first 24 hours, in one the perforation was not recognized prior to death and the fifth patient died following surgical treatment 96 hours after perforation.

Discussion

The incidence of esophageal perforations in our experience is increasing and others have noted a similar increase.⁵ With increasing utilization of endoscopy to diagnose and treat lesions of the esophagus and stomach the incidence of perforations may continue to increase. Most of the perforations are iatrogenic but do not seem to be related to inexperience of the endoscopist; over 80 per cent of the instrumental perforations reported here involved experienced endoscopists. Actually the incidence of instrumental perforations is very small—less than one in every 200–250 endoscopies.^{1, 12} For this reason the lesion may at times be more difficult to recognize.

Instrumental perforations either due to endoscopy or dilation should be the easiest to recognize. Chest or abdominal pain which persists for longer than an hour after the procedure is the most consistent clue. If the temperature rises and subcutaneous crepitus is present the diagnosis is then established. We have noted crepitus in only about 50 per cent of the cases and temperature elevation may not appear for several hours. Other signs and symptoms which may help in recognizing a perforation are

dyspnea, dysphagia and a mediastinal auscultatory crunch. However the patient with pain and in whom an esophageal perforation is suspected should not be observed until further signs and symptoms develop; delays in diagnosis mean increased morbidity and mortality. Chest roentgenogram followed by an esophagram with a water soluble agent should be obtained without delay. If these studies reveal no abnormality, then and only then is a period of close observation indicated.

A most serious cause of delay in diagnosis of an instrumental perforation may be the reticence of the endoscopist to believe that he has injured the esophagus. Analysis of the endoscopic perforation in this series convinces us that frequently the endoscopist may have difficulty in recognizing the perforation which he has created. This is illustrated by the case of an experienced gastroscopist whose instrument passed through the wall of the cervical esophagus and on down through the mediastinum into the peritoneal cavity and who, following insufflation, described a view of the gastric folds. The ensuing pneumoperitoneum, pneumothorax and massive cervical subcutaneous emphysema presented a diagnostic and therapeutic challenge (Fig. 4). Forceful introduction of the endoscope or an uncooperative patient would seem to create the most likely situation for perforation. This is rarely the situation with an endoscopic esophageal perforation; far more frequently the examination has been apparently completely satisfactory.¹ The frequency of perforation attending esophageal dilation for achalasia and blind or unguided antegrade dilations of strictures appear to be another matter. We have serious misgivings about these procedures, as have others.³

The frequency of abdominal pain, tenderness and muscle spasm in one third of the patients with lower thoracic esophageal perforations deserves special emphasis. These findings often served to confuse the diagnosis and direct attention away from

the esophagus. A high index of suspicion and detection of early signs or symptoms of pathologic conditions in the left pleural cavity would seem the best hope of avoiding delay in diagnosis.

Roentgenographic findings will depend largely on two factors: the extent of perforation and the time interval after perforation. Perforations which breach the mediastinal pleura will result in the more dramatic signs and symptoms as well as more definitive changes on an early roentgenogram of the chest. A perforation which does not penetrate the pleura is more likely to result in mediastinal and subcutaneous emphysema. However these are the usual but not invariable sequences. A large perforation may result in no detectable mediastinal air or pleural changes for several hours, yet perforations which do not penetrate the pleura may result in a large hydrothorax in just a few hours. Most of the definitive changes encountered on chest roentgenogram in this series were noted 18 to 24 hours after perforation. Early changes were often subtle or absent. These facts emphasize the importance of the esophagram in any suspected case of esophageal perforation; a water soluble contrast medium should be used because it will pass more easily through the perforation and does not cause subsequent problems, as may barium. In this study esophagraphy demonstrated the perforation in 18 of 21 examinations; the three failures must be designated as small perforations, since two responded to nonoperative treatment and the third was a pin hole through the esophageal wall.

The recognition of cervical esophageal perforations is ordinarily not difficult; the painful, tender, swollen neck—usually with crepitus—establishes the diagnosis. The plain roentgenogram also demonstrates the subcutaneous emphysema but the esophagram is more valuable in localizing the rent and guiding the surgeon.

Spontaneous perforations of the esophagus pose the most difficult diagnostic prob-

lems, an excellent report on which has been published recently by Sealy.¹¹ The more dramatic cases with the perforation extending into the pleural cavities present with sequential vomiting, sudden onset of severe pain, dyspnea, hoarseness, cyanosis and shock. Severe pain following an episode of vomiting is the most important clue. Pain may be in the lower chest or the upper part of the abdomen and often mimics the more frequently occurring pain of coronary occlusion or acute abdominal conditions such as pancreatitis or perforated duodenal ulcer. If crepitus is present in the neck the diagnosis is established; if not the chest roentgenogram and the esophagram are the procedures most likely to establish the diagnosis.

Perforations of the esophagus should be treated surgically and treatment should be instituted promptly; the only delay should be for emergency resuscitative procedures. Inevitably the availability of antibiotics induced the use of conservative supportive measures and antibiotic therapy for *small* perforations and this misleading concept remains.^{5, 6, 13} Many patients treated in this manner are ultimately recorded as failures of surgical treatment when, 3 to 5 days after institution of antibiotic therapy, deterioration of the patient prompts belated surgical intervention. The fact that some small perforations often can be treated successfully by conservative management is not questioned; the difficulty in classifying a perforation as small or of predicting the ultimate effects of a small perforation presents the real problems. Early changes on chest roentgenogram or the size of the rent as determined by esophagraphy provide an unreliable index to the future course of events. Figure 6 demonstrates the difficulty in selecting a patient with a small perforation for nonoperative treatment; at thoracotomy a perforation 8 cm. in length was found.

There is no specific time interval following perforation which precludes suture

closure. Our most belated successful closure of a perforation was at 72 hours; others have reported successful closure as late as 27 days after perforation.⁹ Obviously the judgement of the surgeon becomes important in handling a perforation of long standing. There is little advantage in placing sutures in an esophageal wall which obviously will not hold the sutures; in such a case establishment of adequate drainage will usually result in healing although the convalescent period will be extended. In our series a total of 20 patients underwent suture closure of the perforation. The earliest suture closures were the four surgically created perforations of the abdominal esophagus which were recognized immediately and closed; three were successful while the fourth broke down and ultimately caused death because drainage had not been provided. Thirteen suture closures were performed from 2 to 24 hours after perforation; 11 were successful while two broke down, but adequate drainage allowed slow healing and a favorable outcome in both cases. Three suture closures were performed 30 to 72 hours after perforation and each was successful. One patient had suture closure 8 days after nonoperative treatment but died of empyema and bronchopneumonia; at autopsy the perforation was seen to have reopened.

A note should be added with respect to the time between perforation and surgical treatment. The 24-hour period which may seem so critical regarding success of surgical treatment is extremely arbitrary. In our series there were 5 patients who died between 2 and 22 hours after esophageal perforation. Any period of waiting prior to definitive treatment is hazardous.

We find little information in the literature on perforations of the esophagus caused by surgical procedures involving para-esophageal tissues. Of the seven instances in our series five were associated with vagotomy and two with hiatus hernia repair. In four patients the perforation was

recognized at operation and repaired; in three the perforation became apparent 2 to 10 days later and may have been the result of ischemic necrosis of the esophageal wall progressing to perforation. Rarely such a perforation may be unavoidable; the important consideration is its immediate recognition, followed by closure and adequate drainage. The avoidance of excessive surgical trauma and injury of the esophageal wall is obviously important. Finally, awareness of the possibility of esophageal wall perforation several days after para-esophageal surgery may lead to its recognition and appropriate treatment.

The perforated esophagus is a grave surgical emergency. When suspected, antibiotic therapy should be initiated. Nasogastric suction should be instituted to minimize pleural or mediastinal contamination; the nasogastric tube may best be passed under fluoroscopic control, but these measures should not cause undue delay. Diagnosis should be established promptly, and after any resuscitative measures necessary to stabilize cardiovascular and respiratory function, immediate surgical treatment consisting of the closure and adequate drainage should be instituted. With early suture closure and drainage it sometimes may be quite advantageous to perform definitive operation for the underlying esophageal abnormality. For example patients with achalasia who sustain an instrumental perforation may be best treated by cardio-esophageal myotomy in addition to early closure and drainage.

Summary

Perforation of the esophagus is a surgical emergency. With prompt and aggressive surgical treatment survival can be expected in most cases. The rather infrequent presentation of such cases combined with the often nonspecific signs and symptoms makes recognition difficult. A high index of suspicion is the first requisite if early diagnosis and treatment is to be realized. Instru-

mental or iatrogenic perforations are the most frequent. Any patient with persistent chest or upper abdominal pain following endoscopy, esophageal dilation or para-esophageal surgery must be suspected of having a perforation. Cervical esophageal perforations present in a more direct fashion. Conservative or nonoperative treatment of esophageal perforations is attended by a high morbidity and mortality. An aggressive approach to both diagnosis and treatment is indicated.

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