Spontaneous Perforation of the Normal Esophagus

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Spontaneous perforation of the esophagus is a distinct clinical entity, in which, sudden rupture through all layers occurs in the lower end of a normal esophagus. Such a perforation of the esophagus occurs usually after violent retching or vomiting. Acid gastric contents and food particles are driven into the mediastinum, left pleural cavity and also occasionally the right pleural cavity. The resulting insult in this unique esophageal lesion is devastating to the patient. The features of the disease are distinctive yet easily missed thus the diagnosis has been rarely made during life so that definitive treatment could be instituted.

Perforations of the esophagus complicating endoscopy or due to carcinoma, stricture or other pre-existing pathological conditions are not considered in this discussion. The diagnosis of perforation of the esophagus after endoscopy or dilatation, for example, is relatively easy. Similarly, if the esophagus is the site of a known pathological process, there is little or no diagnostic problem, and treatment may be instituted at once. Perforations due to these conditions are usually less fulminating in character than the lesion under discussion. An empyema or mediastinitis may develop over a period of time and with adequate drainage, chemotherapy and supportive treatment, the patient has a good chance of recovery.

There are several disease syndromes recorded in the literature which are closely related to spontaneous perforation of the esophagus. The lesions in these syndromes are produced by the same fundamental forceful and/or disarranged vomiting mechanism. However, the patient's subsequent clinical course varies and is conditioned by the degree and extent of the resultant esophageal laceration and the possibility of its superimposition on an esophagus already the site of disease.
A) *Hyperemesis Gravidarum.*

The severe retching and vomiting characteristic of this disease syndrome is well known. Acute longitudinal tears in the mucosa at the lower end of the esophagus may result in severe or fatal hematemesis.

B) *Mallory Weiss Syndrome.*

This syndrome presents fatal hematemesis in an alcoholic following a prolonged debauch with vomiting and retching. The lesion is a longitudinal tear in mucosa or muscularis, usually at the cardia of the stomach and the lower end of the esophagus. Such a laceration in a cirrhotic patient with portal hypertension and esophageal varices may understandably be rapidly fatal from acute blood loss.

C) *True Spontaneous Esophageal Perforation.*

The esophagus is lacerated through all layers, in a longitudinal direction, by vomiting. Gastric juice and food particles are propelled into the mediastinum with consequent gross contamination of it and frequently one or both pleural cavities. In addition air may collect in the mediastinum or pleural cavities under tension and hasten the fatal outcome. This disease entity is the most inexorably ruthless of all esophageal perforations and understandably has carried a one hundred per cent fatal prognosis until very recent times.

We wish to review briefly the clinical aspects of these patients, stress the diagnostic features, mention our laboratory experiments, report a case correctly diagnosed but unsuccessfully treated by surgical means and offer our considered views on the proper surgical and supportive treatment necessary in an attempt to save these patients.

**Historical Aspects**

In 1724 Boerhaave's classical description of the first case of spontaneous perforation of the esophagus, occurring in the Grand Admiral of Holland, Baron DeWassenaer, was recorded. This has been quoted by Barrett. Fitz in 1877 made the first review in the American literature and recorded a case. In 1914, in a review, Walker found 22 cases including his own, which was the first case diagnosed during life. Other comprehensive reviews include Smead, 1931; Ridgeway and Duncan in 1937; and Klein and Grossman in 1943, who uncovered only 40 cases. Recent reports are those of Collis, 1944; Barrett, 1946; and Eliason and Welty, 1946.
In one of the cases reported by Eliason and Welty, the diagnosis was made during life. This represents the fifth time to our knowledge that the correct diagnosis has been made antemortem in this condition. Unfortunately no definitive treatment could be given this patient. Barrett reports three cases in one of which the correct diagnosis was made, but no surgical treatment could be instituted. Collis' patient was a healthy 45-year-old soldier on whom a thoracotomy was performed, the esophageal rent closed and the pleural cavity drained, but despite this definitive treatment, the patient succumbed ten hours later.

Recently Barrett has reported the first case known to us of survival of a spontaneous perforation of the esophagus. His patient had a subtotal hysterectomy then vomited violently 13 days postoperatively. Diagnosis was made by thoracentesis and roentgen examination and operation performed ten hours later. The esophageal perforation was closed by a purse-string suture and the chest drained. The immediate course was good. Three weeks later, a mediastinal abscess required drainage. Two months later, an abscess of the right upper lobe was noted, however, this drained spontaneously and the patient was well thereafter. Our colleague, Doctor Joseph P. Lynch, has likewise successfully treated a classical case recently by early operation and suture of the esophageal perforation, as outlined in this study. This case will be reported in detail at a later date by him.

Etiology and Pathogenesis

In spontaneous perforation, the stage is usually well set by a fairly standard set of circumstances. The patient usually has a full stomach at the time he retches violently, gastric contents are ejected exerting great expansile pressure on the upper gastric and lower esophageal wall, with resultant rupture of the distal esophagus. If the esophagus is obstructed even transiently by a bolus of undigested food leading the column of vomitus, the possible mechanism becomes even more apparent.

Rupture occurs in the lower end of the esophagus apparently due to an inherent weakness of this anatomic area. MacKenzie, quoted by Smead, removed the esophagus from 18 specimens at postmortem examination and ruptured them by water pressure. In 17 cases, the tear occurred in the lower third of the esophagus with from 5 to 11 pounds pressure.

A congenital and anatomic weakness of the lower end of the esophagus has been postulated by Menne and Moore on the basis of their case of spontaneous esophageal perforation in a five-month-old infant following repeated vomiting spells. A curd stuck in the infant's esophagus and throat. The bady vomited...
once more following this and died of a typical ruptured esophagus thirty-six hours later. They state that there are three other authentic cases in the literature over a 200-year period occurring in infants and children.

There is a strong alcoholic history in many of these patients. Ridgeway and Duncan\(^5\) reported 18 of 35 cases as having a significant alcoholic history; 7 cases denied indulgence\(^6\) and 10 cases were questionable or not stated. They felt, therefore, that regurgitation of gastric juice with digestion and/or acute ulceration might very well be a confusing etiological factor and make it difficult to evaluate microscopic changes in the esophagus as to their occurrence before or after rupture.

Alcohol may be an important factor in other ways. Excessive amounts often produce retching and vomiting and also tend to produce incoordinated muscle action that may impair critically the complicated vomiting mechanism.

Gluttony may be a factor since the full stomach is perhaps more easily compressed between the diaphragm and abdominal wall.

According the Weiss\(^12\) vomiting is a complex reflex act composed of a number of highly coordinated bodily changes that he describes as follows:

1) During the stage of nausea, the pylorus closes and the cardia and esophagus dilate. (2) Reverse peristalsis carries gastric contents to the cardia and esophagus. (3) At the onset of retching, the diaphragm descends to the position of deep inspiration. (4) Gastric contents are then ejected with great force by this sudden convulsive increase of intrathoracic and intra-abdominal pressure. (5) Repeated attacks of vomiting at short intervals, however, may fatigue the vomiting center with consequent loss of coordination. (6) Gastric contents may then thrust with even greater force against the cardia and lower esophagus if relaxation of the esophagus and descent of the diaphragm do not occur at a properly timed juncture with the forceful retching movement.

**Pathology**

1) Postmortem examination of a patient who has died as a result of spontaneous perforation of the esophagus reveals a remarkably constant and characteristic lesion. The rupture almost invariably occurs in the lower one-fifth of the esophagus, in a longitudinal direction and on the left posterolateral aspect. The tear is usually sharp edged and incised in appearance. It may be several centimeters in length but may be small and difficult to demonstrate. Rarely the rupture has been reported as occurring transversely, on the right, high in the esophagus, or below the diaphragm. The appearance in microscopic sections varies with
the survival time of the patient and the interval between death and postmortem examination. The majority of pathologists seem agreed that rupture occurs in the "true" case in a normal esophagus, that there is no evidence of pre-existing disease, and that all tissue changes are due to the severe chemical and bacterial inflammation.

2) There are, immediately, several possible mechanisms resultant upon a spontaneous rupture of the esophagus: (a) Rupture into the mediastinum and left pleural cavity which is the commonest lesion. (b) Rupture into the mediastinum and both right and left pleural cavities. (c) Rupture into the mediastinum and right pleural cavity only. (d) Rupture into the mediastinum alone or with later perforation into either pleural cavity. (e) Direct rupture into the mediastinum and/or pleural cavity. A devastating, necrotizing chemical insult with rapidly superimposed aerobic and anaerobic infection quickly jeopardizes the patient's life and he succumbs to the gangrenous mediastinitis, pleuritis, pyopneumothorax, and emphysema usually in a matter of several hours to 1 or 2 days. The pyopneumothorax may exhibit signs of tension, varying degrees of atelectasis are present, and mediastinal emphysema may cause thoracic inlet obstruction. The latter impairs inflow to the right heart with consequent peripheral vascular collapse and profound shock. Varying amounts of fluid and gastric juice, often bloody, together with partially digested food particles will be found in the mediastinum and/or pleural cavities. A pericardial effusion may ensue if the patient survives for any length of time.

Symptoms and Signs

The catastrophe of spontaneous esophageal perforation is usually seen in a male between 35 and 55 years who had a history of heavy or excessive alcoholic intake. Finally, one of these indulgences is accompanied by severe retching or vomiting and at this time rupture of the esophagus is particularly apt to occur.

In Walker's series of collected cases, 20 of 22 patients were males. Only 5 of the 35 cases reported by Ridgeway and Duncan were females. According to Menne and Moore 3 authentic cases have been reported in children in addition to their case.

Vinson has reported several cases in which pernicious vomiting of pregnancy caused acute lacerations in the lower end of the esophagus later resulting in a stricture if the patient survived. These cases are undoubtedly counterparts of the cases reported by Mallory-Weiss. In the Mallory-Weiss syndrome, vomiting and retching occurs in chronic alcoholics during a prolonged debauch leading to hematemesis from a long tear through the mucosa or
even into muscularis of the lower esophagus or gastric cardia. Indeed, one of the patients reported by Mallory and Weiss was a hyperemesis gravidarum case with an identical lesion to that observed in alcoholics. The cases of Vinson and Weiss and Mallory may be regarded as incomplete ruptures of the esophagus.

Cases have also been reported as occurring during a convulsive seizure, defecation, seasickness and retching during or following recovery from general anesthesia.

In most instances, the onset of symptoms is sudden, and the patient rapidly becomes acutely and seriously ill. Usually after vomiting or retching, the patient is suddenly seized with violent pain and may volunteer that he feels as though something was "torn inside."

**Pain** is continuous and excruciating, and may be felt low in the chest, over the xiphoid, may radiate through to the back, or may be referred to the shoulder or upper abdomen. Morphine or demerol even in huge doses do not alleviate the patient's discomfort.

**Shock** is invariably present. The patient is anxious and apprehensive. He may be restless or may remain immobile with trunk flexed. There may be slight cyanosis and the respirations are usually rapid, shallow and grunting. The skin is cold and clammy, the blood pressure low, the temperature normal or sub-normal and the pulse fast and thready.

**Thirst** is insatiable, although pain may be intensified as swallowed fluid passes into the mediastinal and pleural spaces.

**Upper abdominal spasm and tenderness** may be prominent before the chest findings are manifest. Exploratory laparotomy has been performed frequently in the past for this reason.

**The Chest findings** include signs of fluid relatively early. Later, atelectasis and pyopneumothorax are present. The pneumothorax may manifest signs of tension by dyspnea, cyanosis and displacement of the mediastinum to the opposite side.

**Subcutaneous Emphysema** is a very important and helpful sign and should lead one to the diagnosis if present. Usually, it is present in two-thirds of the cases, appearing first in the suprasternal notch and supraclavicular fossae, and later spreading so as to involve the face, trunk or even the extremities and dissecting widely in the subcutaneous tissue.

**Thoracentesis** will clinch the diagnosis in the suspected case. Varying amounts of air may be aspirable. The fluid present usually is sour and in appearance often seems tinged by old changed blood, presumably due to the action of gastric ferments. A thin serous fluid may be present in either pleural cavity if
the infection is confined to the mediastinum. A few hours later this fluid likewise will be invaded by organisms.

Roentgenography may be extremely helpful and is important in the differential diagnosis and as a possible means of early diagnosis. The roentgenogram may confirm the signs of fluid in one or both pleural cavities, demonstrate atelectasis or pneumothorax and corroborate mediastinal displacement. Emphysema may be detected in the root of the neck or in the mediastinum. If rupture has occurred only into the mediastinum, a fluid level may be demonstrated. The absence of air under the diaphragm is helpful if there is doubt as to the location of the primary lesion.

Many of the classic findings in perforated esophagus may be evident only shortly before the patient succumbs. However, if the diagnosis is suspected, thoracentesis and suitable roentgenograms will often solve a perplexing differential problem long before emphysema appears. The difficulty now is not really in making the diagnosis but rather in making it early enough to have definitive surgery offer some chance to the afflicted individual.

Differential Diagnosis must include: (1) acute coronary thrombosis, (2) perforated peptic ulcer, (3) acute pancreatitis, (4) dissecting aneurysm of the aorta, (5) acute cholecystitis, (6) spontaneous pneumothorax, (7) pulmonary embolism, (8) mesenteric occlusion, (9) interstitial emphysema, (10) diaphragmatic hernia.

Laboratory Experiments

Following the unsuccessful case herein reported the production in the laboratory of a lesion exactly comparable to a spontaneously ruptured esophagus was attempted. The creation of an esophageal defect by various methods through the esophagoscope was unsatisfactory because a truly comparable lesion could not be produced and no two perforations by such methods were alike; the mediastinum was not widely opened nor even necessarily contaminated and the right pleural cavity was not opened. Such procedures were, therefore, abandoned.

Eventually, in order to produce a standard defect analogous to the human case, a thoracotomy was done and the lesion produced under direct vision in spite of the obvious disadvantages of assessing therapy on an animal who has had a previous thoracotomy. A standard procedure was used in each animal. The animal was fed then anesthetized with intravenous nembutal, shaved and prepped, an endotracheal tube inserted, and the animal placed in the right lateral decubitus position. The chest was opened through a left intercostal incision in the seventh interspace. The esophagus was mobilized up to the arch and the right and left pleural cavities and mediastinum widely opened. A two-inch left
postero-lateral incision through all esophageal layers was then made approximately 1 ½ inches above the esophageal hiatus. Ten milligrams of apomorphine was given then intravenously supplemented by manual compression of the animal's full stomach. Large amounts of gastric juice and previously eaten food were regurgitated with great force into the mediastinum and both pleural cavities. The lungs were expanded under positive pressure, air was aspirated from the right and left pleural cavities while the chest wall was closed in layers. A drainage tube was led out to an underwater seal. In this fashion, a lesion was produced analogous to the human case in its most severe form, namely, with both pleural cavities, as well as the mediastinum, widely involved by gastric juice and food particles.

The animals were divided into three groups: (a) completely untreated, (b) supportive treatment by parenteral fluids, sulfadiazine and penicillin, (c) operative closure of the esophagus at varying intervals and pleural drainage with parenteral fluids, penicillin and sulfadiazine.

<table>
<thead>
<tr>
<th>GROUP I — UNTREATED</th>
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<tbody>
<tr>
<td>Animal</td>
</tr>
<tr>
<td>Dog No. 1</td>
</tr>
<tr>
<td>Dog No. 2</td>
</tr>
<tr>
<td>Dog No. 3</td>
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<tr>
<td>Dog No. 4</td>
</tr>
<tr>
<td>Dog No. 5 (unfed)</td>
</tr>
<tr>
<td>Dog No. 5 (fed)</td>
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</tbody>
</table>

These animals were intended to serve mainly as a control group. An attempt was made to restore their cardio-respiratory physiology to normal as the chest was closed, but no further treatment was given. As might be expected, varying degrees of pneumothorax and mediastinal emphysema resulted. Varying amounts of serosanguinous fluid was found in the pleural cavities. This fluid invariably became thicker and more foul the longer the animal survived. In Dog No. 5 (unfed) there was no gross contamination by food or gastric juice. The edges of the esophageal lesion bled.
freely and there was a large clot in the left pleural cavity as the chest was closed. Persistent leakage was prevented probably by this fact. In any event, the animal survived and 3 months later, the chest was re-opened. The site of previous laceration was firmly repaired but the scar was apparent on careful palpation. There were numerous adhesions, however, and the examination was otherwise not remarkable. This case presumably illustrates the importance of the gastric juice and resultant chemical pleuritis as a factor of this lesion, particularly in view of the low PH values of the dog's gastric juice. When this animal's esophagus was again lacerated, this time on a full stomach, death occurred in the usual fashion 7 hours later.

GROUP II — SUPPORTIVE TREATMENT

<table>
<thead>
<tr>
<th>Animal</th>
<th>Survival</th>
<th>Post Mortem Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dog No. 6</td>
<td>18 hours</td>
<td>Putrid empyema, mediastinitis, minimal mediastinal emphysema, partial atelectasis, left lower lobe.</td>
</tr>
<tr>
<td>Dog No. 7</td>
<td>18 hours</td>
<td></td>
</tr>
<tr>
<td>Dog No. 8</td>
<td>18 hours</td>
<td></td>
</tr>
</tbody>
</table>

These animals all received 400–800 cc. of 10 per cent glucose in saline intravenously according to their weight. Two hundred thousand units of penicillin were placed in the chest before closure. Sodium sulfadiazine (1 grain per pound) was mixed in the intravenous fluids at the conclusion of operation. Penicillin was then given in a dose of 100,000 units intramuscularly every eight hours and sodium sulfadiazine (1 grain per pound per 24 hours) was given subcutaneously every eight hours. The intravenous fluids were repeated 12 hours following operation. Post-mortem examination revealed the findings charted above.

Group III: Operative and Supportive Treatment

A thoracotomy was performed and the usual esophageal laceration produced. Eight hours after perforation (to allow for diagnosis in the human case) fluids, sulfadiazine and penicillin were begun. Animals Nos. 9, 10, 11, and 12 were then re-operated upon at intervals after perforation of 16, 11, 8 and 8 hours respectively, and the perforation closed. In all these animals, the chest was re-opened by subperiosteally resecting the left ninth rib. The pleural cavities were thoroughly irrigated. The esophageal rent was closed by an inverting Connell suture of 1-0 chromic catgut. The muscular layer was closed by multiple interrupted Lembert sutures of 3-0 silk. A drainage tube was led out through the tenth interspace and connected to an underwater seal. The
chest wall was closed in layers. Supportive therapy was then continued by the same schedule outlined for Group II. These animals died, 24, 15, 10, and 10 hours postoperatively, respectively. In animal No. 12 only the left pleural cavity was opened at the time of perforation of the esophagus with no apparent change in the end result.

Animal No. 13: This animal had his esophagus perforated. Eight hours later supportive treatment was instituted and twenty-four hours later, he was re-operated and the esophageal lesion closed in two layers. The animal did very well until the seventh postoperative day when he became distended and died a few hours later. Postmortem examination revealed an essentially normal chest cavity with the esophagus well closed. Abdominal examination revealed generalized peritonitis.

Then in an attempt to evaluate the time element and the trauma of a previous thoracotomy, the esophagus in three additional animals was perforated. The esophageal laceration was sutured immediately. Two of these animals survived.

Animal No. 14 and animal No. 16: The esophagus was perforated and the mediastinum and pleural cavities contaminated forcefully by vomitus, then thoroughly irrigated with saline which was aspirated. The esophageal rent was closed at once in two layers. Chemotherapy was stopped 24 hours later, and the chest drainage tube removed. The subsequent course of these dogs was completely uneventful.

Animal No. 15: The esophagus was perforated in the usual manner and immediately sutured using the same technique as

GROUP III — SUPPORTIVE AND OPERATIVE TREATMENT
Esophageal Perforation Closed at Varying Intervals

<table>
<thead>
<tr>
<th>Animal</th>
<th>Closure</th>
<th>Esophageal Perforation</th>
<th>Survival</th>
<th>Post Mortem Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dog No. 9</td>
<td>16 hours</td>
<td>40 hours</td>
<td></td>
<td>Esophagus well closed. Pleural and mediastinal inflammation. No suppuration.</td>
</tr>
<tr>
<td>Dog No. 10</td>
<td>11 hours</td>
<td>26 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog No. 11</td>
<td>8 hours</td>
<td>18 hours</td>
<td></td>
<td>Peritonitis.</td>
</tr>
<tr>
<td>Dog No. 12</td>
<td>8 hours</td>
<td>18 hours</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog No. 13</td>
<td>24 hours</td>
<td>7 days</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog No. 14</td>
<td>Immediate</td>
<td>Survival</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dog No. 15</td>
<td>Immediate</td>
<td>24 hours</td>
<td></td>
<td>Putrid empyema and mediastinitis.</td>
</tr>
<tr>
<td>Dog No. 16</td>
<td>Immediate</td>
<td>Survival</td>
<td></td>
<td></td>
</tr>
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</table>

Dogs 9–13 inclusive had two thoracotomies.
in the other animals. In spite of the usual postoperative mea-
ures, the animal died twenty-four hours later. Postmortem ex-
amination revealed the esophagus to be well closed but putrid em-
pyema and mediastinitis were present.

Comment

The severest possible form of esophageal perforation was pro-
duced with contamination of the widely opened mediastinal and
pleural cavities. No absolute conclusions can be drawn from such
a small series of dogs, however, the results are suggestive.

The group of animals with no treatment and with supportive
care only show no great variation in time interval between per-
foration and the expected result. The animals, in whom the
laceration was sutured at a subsequent operation, tended on the
whole, to live a few hours longer and one of these animals, with
a 24-hour interval between laceration and suture of the perfora-
tion, lived seven days and then did not die of chest complications.
Three animals had their esophageal perforations closed immedi-
lately. The subsequent course of two of these animals was un-
eventful. The other animal, however, died of continuing medi-
astinal and pleural infection. The prolonged or complete survival
of an animal with so severe a lesion seems to us directly related
to the closure of the rent and the consequent prevention of con-
tinued contamination of the mediastinal and pleural cavities.

In view of the known disastrous course in the human patient
with an untreated esophageal perforation and the uniformly
fatal course in all dogs whose closure was delayed longer than
the immediate termination of the acute experiment, it is difficult
to explain the survival times of dogs nine to thirteen inclusive
since these were in inverse relation to the interval between per-
foration and suture. The obvious explanation would seem to be
the increased interval between the thoracotomies performed first
to create the lesion and then to institute treatment in each animal. These animals surviving longest had the greatest time
interval in which to recover from the primary thoracotomy. The
animals in whom suture was immediately performed are more
strictly comparable to human cases and to Groups I and II inasm-
much as only one thoracotomy was performed. The essential
difference is closure of the esophageal laceration in these com-
parable cases, with a change from 100 per cent mortality to 67
per cent survival. In this group of dogs, Nos. 9-16 inclusive, who
were treated ideally with time intervals approximating the prob-
lem in the human, the survival rate was such (2 out of 8) that
we believe that the results of the experiments suggest that the
rationale of treatment is justified.
CASE REPORT

This 53-year-old laundry worker entered Boston City Hospital for the first time on May 7, 1946.

Chief Complaint: Excruciating epigastric pain for six hours.

Present Illness: Patient had never been seriously ill prior to this admission. At 8:30 P.M. on May 7, he ate lamb stew. The entire family became nauseated and the patient vomited mildly at 7:00 P.M. He then attended a club meeting where he drank two bottles of beer. He returned home at 9:30 P.M. feeling ill, vomited repeatedly, and shortly experienced:

(a) Severe, sharp, high epigastric and low left chest pain. The pain began anteriorly and radiated around to the back and also to the left shoulder.

(b) Sweating with a cold clammy feeling.

(c) Vomiting of clear gastric juice without blood.

At 1:30 A.M. the patient was seen in the emergency ward and given one-quarter of a grain of morphine sulfate on the admitting floor and sent to the ward with a probable diagnosis of acute coronary thrombosis.

Physical Examination: Temperature, 98 degrees F.; pulse, 140; respiration, 32; blood pressure, 130/90. Inspection revealed a very apprehensive, obese, middle aged male who was cyanotic, clammy, and in acute excruciating pain in spite of morphine. Respirations were rapid and painful. The patient was twisting and turning on the litter and complaining of thirst. His pupils were pinpoint. Chest examination was negative on percussion and auscultation except for high diaphragms. The heart not enlarged. Normal sinus rhythm at a rate of 140. The abdomen was boardlike with high epigastric tenderness. No peristalsis was heard. The reflexes were hyperactive.

Laboratory Data: Hgb., 90 per cent; WBC, 20,800; Polys, 96; Lymphs, 4; Platelets and RBC, normal.

Hospital Course: The patient continued to complain of thirst and of excruciating pain. He received one-half grain of morphine, one grain of codeine and one hundred milligrams of demerol in a period of two hours with no relief. The electrocardiogram was of no significance. The urinary diastase was normal. The flat and sitting abdominal roentgenograms revealed no fluid levels or air beneath the diaphragms. A general surgical consultant believed the abdomen was not a surgical problem. Chest roentgenograms revealed elevation of the left diaphragm and a hazy opacity at the left base. There was no fluid level. (Later review of this film revealed emphysema of the mediastinum). The patient was maintained on nasal oxygen, demerol and penicillin. At 8:00 A.M. on May 8, the patient's blood pressure was 100/70 and the abdomen was rigid. Neck swelling with crepitation was present and air was beginning to dissect in the tissue planes of the face. A diagnosis of mediastinal and subcutaneous emphysema was made. A portable chest x-ray film revealed a partial collapse of the left lung, mediastinal shift to the right with fluid obscuring the left lung field, elevation of the left diaphragm and subcutaneous and mediastinal emphysema. The Thoracic Surgery Service was called in consultation and the patient seen by one of us (J.W.S.) and a diagnosis of spontaneous perforation of the esophagus was made. A thoracentesis revealed fluid with food particles and low PH and the diagnosis was thus confirmed.

The patient's condition at this time was extremely precarious and it
was felt that he would tolerate little if any operative manipulation. A whole-blood transfusion was started, then under local one per cent procaine infiltration, a closed intercostal catheter drainage was performed in the seventh left interspace in the posterior axillary line on May 8 at 12:00 A.M. A large amount of extremely foul thin cloudy fluid together with a large amount of air was immediately aspirated on opening the catheter to the underwater seal bottle. The patient was then transferred to the ward in poor condition. Intravenous fluids and blood were given. A positive pressure mask was used to deliver oxygen. Penicillin was given in doses of 30,000 units intramuscularly every two hours. Sodium sulfadiazine was given intravenously once then discontinued because of the poor urinary output. In spite of medication, the patient was obviously in severe pain; he was perspiring profusely; his color was only fair even with positive pressure oxygen. His pulse was weak and thready with a rate of 116 to 140; blood pressure remained at shock levels. Abdominal distention made it even more difficult for the patient to aerate properly. The patient was catheterized, approximately 20 hours after admission, and fifty cubic centimeters of highly concentrated urine was obtained. His non protein nitrogen was 76; total protein, 6.56; carbon dioxide, 39 per cent; red blood count, 4,000,000; and white blood count, 17,500. A right thoracentesis revealed forty cubic centimeters of cloudy serous fluid. Direct smear and culture of this fluid were negative. Penicillin was instilled, however, the patient's condition remained the same throughout May 8, and early May 9, 1946. He was extremely restless, complaining almost constantly of pain and asking for water. His skin and extremities were mottled and cyanotic and the skin was cold and clammy. The patient was disoriented and irrational and difficult to restrain.

At 9:30 A.M. on May 9, 1946, the patient's temperature suddenly rose to 105.6 degrees F. by rectum where as his previous temperature had been 99 degrees F. At this point, it became extremely difficult to count his pulse and his respirations rose to 40-44 and his blood pressure fluctuated slightly but always at shock levels. He was continued on a supportive therapy. An attempt was made to restore his blood chemistry to normal. The patient was obviously going rapidly down-hill from extensive and overwhelming sepsis.

At 2:00 P.M. on May 9, 1946, the patient was taken to the operating room and under local procaine infiltration, a posterior mediastinotomy was done as a desperate measure in a nearly moribund patient in profound shock. A four-inch paravertebral incision was made. The erector spinae muscle group was retracted. Two-inch segments of the 9th and 10th ribs were removed subperiosteally and the posterior mediastinum was entered. There was evidence of extensive mediastinal infection with a large rent present in the left pleural membrane. There was a small similar tear in the right pleural cavity: both of these openings were widely enlarged for free drainage. The actual point of perforation of the esophagus could not be determined. The cavity was then packed with hydrogen peroxide sponges followed by a dry sterile dressing and adhesive strapping. The closed intercostal drainage tube was kept in place. Immediately after this procedure the patient seemed much relieved; he was, of course, returned to the ward with an extremely poor prognosis.

One hour after his return to the ward his condition deteriorated. Respiration were 40; blood pressure was unobtainable, and his pulse
could not be counted. A continuing effort to elevate the blood pressure was made with whole-blood transfusions; this continued to be unavailing and oxygenation by mask was inadequate. Two hours after his return from the operating room there was no response to stimulants and the patient expired at 4:15 P.M. on May 9, 1946.

Three hours later, post mortem examination revealed a longitudinal laceration of the esophagus in its distal portion on the left posterolateral aspect approximately one-half centimeter above the cardia. B. Coli Communis was cultured post mortem from the heart's blood and right and left lower lobes. There was dissection of the esophagus 15 cm. upward above the laceration and air bubbles in the surrounding mediastinal tissues. Pressure on the stomach pushed gastric contents into the left pleura.

Microscopically, the esophagus, at the point of rupture, revealed an acute inflammatory reaction with tissue necrosis and blood vessel thrombosis extending through even the outermost layers and involving the peri-esophageal tissues as well. The laceration was approximately three centimeters in length and associated with (1) intramural and peri-esophageal abscess formation, (2) severe mediastinitis with mediastinal emphysema, (3) bilateral severe pleuritis, (4) subcutaneous emphysema, (5) passive congestion liver, (6) atelectasis left lower lobe, (7) minimal pericardial effusion.

Treatment

Following a spontaneous perforation of the esophagus, the patient's course generally is progressively and rapidly downhill. Time is of the essence in undertaking to treat this condition. The most important single factor is the time interval between the occurrence of the rupture and the placing of the patient in the hands of the Thoracic Surgeon. It is our belief that operative maneuvers such as closed intercostal drainage and posterior mediastinotomy are measures of procrastination and will probably not be successful in saving one of these patients except under very unusual circumstances. A bona fide case of spontaneous perforation of the esophagus with fulminating sequelae will run a rapidly fatal course in spite of measures such as these. These patients almost from the moment they are first seen, are so seriously ill, so obviously prostrated and so nearly moribund that one's first impulse is to consider their prognosis hopeless and to undertake only to make them comfortable insofar as possible. This policy in the past, aside from the problem of diagnosis, has led to a 100 per cent mortality. Regardless of the condition in which the patient is first seen, we believe that it is of the utmost importance to quickly render the patient whatever supportive treatment is possible and undertake definitive treatment at once. No matter how acutely ill the patient appears at that time, he will show no further improvement and within a matter of hours his condition will rapidly deteriorate as signs of sepsis and toxicity develop.
SPONTANEOUS PERFORATION

To our minds then the procedure of choice in a spontaneous perforation of the esophagus is an open thoracotomy with endotracheal anesthesia, adequate oxygenation and positive pressure as indicated. The mediastinum should be widely opened and decompressed into the left pleura. If the right pleura is grossly contaminated this should also be widely opened. The laceration of the esophagus can be rapidly closed with an inverting mucosal suture of catgut followed by interrupted silk sutures, thus obviating further contamination. The anesthetist should then expand the lungs; the right chest should be sucked out during this process and the chest closed tightly with the left pleura drained by an adequate underwater seal drainage system. The cardio-pulmonary physiology is thus restored to as nearly normal a situation as possible and the patient is given his maximum possible chance to combat infection.

Supportive measures as an adjunct to thoracotomy: (1) Oxygen delivered either by nasal catheter or preferably by a positive pressure mask. (2) Adequate fluids parenterally including the liberal use of whole-blood. (3) Massive doses, 200,000 to 500,000 units, of penicillin in both the right and left pleural cavities and in addition 100,000 units of penicillin intramuscularly every three hours should be given. (4) Streptomycin may be given during the acute phase probably in a dose of 0.5 grams every six hours to help combat the mixed infection realizing that later the organisms may develop resistance to the drug. (5) Sulfadiazine therapy may or may not be practical parenterally depending upon the urinary output. (6) The patient should be made as comfortable as possible by adequate doses of demerol although morphine may be necessary. (7) Nothing by mouth for 24 to 36 hours. (8) The mouth and throat should be sprayed with penicillin solution, 10,000 units to the cubic centimeter giving approximately 1 cc. every two hours until the esophageal laceration is closed. At the same time, careful mouth care should be given to the patient. (9) Abdominal distention may be combated by the usual measures, flaxseed poultices or hot-water bottles, prostigmine, and rectal tubes. (10) If the left pleural cavity is drained, the right pleural cavity should be considered as potentially infected even if no tear exists and a right thoracentesis should be done once or twice daily as indicated by the portable chest roentgenogram and intra-pleural antibiotics given. Irrigation of the drainage catheter through a Y tube may be carried out every two hours if necessary. Penicillin and streptomycin diluted in saline should be used as the irrigating fluid. (11) If mediastinal emphysema is severe with paradoxical pulse and obstructed inflow to the right heart, a simple collar incision may be done as the patient lies in bed.
A finger introduced into the anterior superior mediastinum will suffice to effect decompression. (12) *Competent nursing* care is essential.

**SUMMARY**

1) Spontaneous perforation of the esophagus is a distinct clinical entity, in which, sudden rupture through all layers occurs in the lower end of a normal esophagus.

2) The diagnosis has been rarely made during life so treatment could be instituted. The mortality until very recently has been 100 per cent.

3) Rupture occurs usually in a middle aged, alcoholic male.

4) The laceration classically occurs in the lower one-fifth of the esophagus, in a longitudinal direction and on the left posterolateral aspect and is sharp edged and incised in appearance. Mediastinitis, empyema and pneumothorax usually result immediately.

5) Chest roentgenograms, thoracentesis and subcutaneous emphysema will enable one to make a correct diagnosis.

6) A case report of a patient unsuccessfully treated is presented.

7) On the basis of laboratory experiments a rationale of treatment is suggested involving supporting care with early thoracotomy and suture closure of the laceration.

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**RESUMEN**

1) La perforación espontánea del esófago es una entidad clínica clara, en la que la ruptura repentina, a través de todas las capas, ocurre en la extremidad inferior del esófago normal.

2) Sólo con rareza se ha hecho el diagnóstico durante la vida para permitir que se aplicara el tratamiento. Hasta muy recientemente la mortalidad ha sido del 100 por ciento.

3) Generalmente la ruptura ocurre en un hombre alcohólico de edad mediana.

4) Clásicamente, la ruptura ocurre en el quinto inferior del esófago, en una dirección longitudinal y en el aspecto posterolateral izquierdo, y tiene un borde agudo y que parece cortado. Mediastinitis, empiema y neumotórax generalmente resultan inmediatamente.

5) Los roentgenogramas torácicos, la toracentesis y el enfisema subcutáneo facilitan el diagnóstico correcto.

6) Se presenta un informe sobre un caso que fue tratado con mal éxito.
7) A base de experimentos de laboratorio se sugiere un tratamiento que consiste de cuidado sustentante, con toracotomía temprana y sutura de la laceración.

REFERENCES