Spontaneous Rupture of the Esophagus*

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Spontaneous rupture of the esophagus is one of the rarer yet more dramatic conditions for which the recent advances of thoracic surgery and antibiotic therapy have offered a means of definite treatment and cure. Although the condition was first described by Boerhaave\textsuperscript{15} in 1724 and since then has been reviewed by Fitz (1877),\textsuperscript{30} McKenzie (1884),\textsuperscript{57} Walker (1914),\textsuperscript{84} Smead (1931),\textsuperscript{77} Girard (1934),\textsuperscript{35} Ridgeway and Duncan (1937),\textsuperscript{70} Terracol (1938),\textsuperscript{78} Barrett (1946),\textsuperscript{6} and Kinsella, Morse and Hertzog (1948),\textsuperscript{47} it is still not widely recognized. It would seem worth while to consider certain aspects of the problem which should lead to its earlier diagnosis for if the condition is suspected its presence can be readily substantiated and prompt surgical therapy instituted.

Definition and Classification

Under the heading of spontaneous rupture of the esophagus should be grouped all patients with rupture or perforation of that organ due to an increase in the intraesophageal pressure greater than the tensile strength of the wall. Excluded are patients in whom there is clinical or gross pathological evidence of preexisting disease (congenital defects, diverticula, tuberculosis, cancer), or of trauma (gunshot and stab wounds, foreign body, instrumentation with esophagoscope, gastroscope, bougie or dilator), at the site of rupture. This broad definition which includes cases with clinically silent inflammations, ulcerations and softening is not acceptable to some authors\textsuperscript{6,28,30,84} who insist on complete normalcy of the esophagus at the time of rupture. Their opinion does not seem valid for it is frequently impossible even at autopsy to be certain that the esophagus was normal\textsuperscript{39,42,53} and further, the clinical problems of diagnosis and therapy are frequently the same in both cases\textsuperscript{20,21,48}.

It is helpful to divide the cases of spontaneous rupture into two groups. In the first are those patients in whom the esophagus is allegedly normal prior to rupture and in whom the increased intraesophageal pressure is the only factor producing the rupture. In the second group the clinically silent, frequently only micro-

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scopic disease predisposes to rupture by weakening the wall. Certain of the patients in the latter group may be suffering from serious extraesophageal disease which alters the clinical picture but in general the syndrome is the same in both groups. With these two groups in mind the reports of spontaneous rupture of the esophagus in many apparently unrelated conditions and the multiple theories advanced to explain its occurrence fall into one general schema, involving the relationship of intraesophageal pressure and esophageal strength.

Pathogenesis of Rupture

In the production of increased intraesophageal pressure the factors of increased intraabdominal pressure, patency of the cardia, distention of the stomach and obstruction of the esophagus all play important roles. Since the esophagus is normally an empty tube any increase in intraesophageal pressure must be secondary to increased intraabdominal pressure with transmission of this pressure by the stomach contents through the cardia into the esophagus. Whether the pressure reached will be sufficient to rupture the esophagus will depend upon the strength of the esophagus and the intensity of the increase in intraesophageal pressure. The latter will depend on the rapidity and magnitude of the increased intraabdominal pressure, the amount of stomach contents and the patency of the cardia and esophagus. Vomiting and retching from any cause are the most important causes of sudden increased intraabdominal pressure but pressure increases great enough to produce rupture of the esophagus have also resulted from the straining associated with defecation, lifting and convulsing and from blunt trauma to the abdomen.

If the cardia does not open, at least in part, the increased pressure will produce multiple circumferentially arranged lacerations of the cardia which occasionally extend into the adjoining portion of the esophagus. Mallory and Weiss\(^{55,89}\) have described the syndrome associated with these lacerations, seen following prolonged vomiting in chronic alcoholism or pregnancy and characterized by recurrent, frequently fatal, massive hematemesis but rarely perforation.

The transmitted force of sudden increased intraabdominal pressure can only produce significantly increased intraesophageal pressure if the contents of the stomach can not be expelled through the esophagus as rapidly as they leave the stomach. This disposition between esophageal filling and emptying can occur either because of distention of the stomach with food or fluid or because of obstruction of the esophagus. Analysis of the reported cases suggests that in some, distention of the stomach may have been
the determining factor, for rupture occurred at a time when the
stomach was full or over distended and not previously when all
the other factors were present. Rupture proximal to mechanical
obstruction of the esophagus from external pressure by mediastinal
tumor, goiter or nodes, or from an impacted bolus of food or
from a stricture with or without impacted food is not un-
known\textsuperscript{30,59,60,88} and shows the role obstruction can play in pre-
venting or delaying the reduction of increased intraesophageal
pressure produced by vomiting and/or retching. However, in the
majority of cases no mechanical obstruction is present. Therefore,
obstruction due to physiological mechanisms is postulated. The
complex anatomical makeup of the esophagus and the highly
complex nature of the vomiting reflex make this concept quite
tenable. Anatomically\textsuperscript{14,23} the esophagus is narrowest at the esoph-
ageopharyngeal junction where it passes through the restricted
space between the cricoid cartilage anteriorly and the sixth cervi-
cal vertebra posteriorly. It is also at this level that the longi-
tudinal bands of muscle fibers fan anteriorly to their tendinous
insertion on the posterior surface of the cricoid cartilage, leaving
the heavier circular muscle fibers the predominate musculature
of the upper portion of the esophagus. These circular fibers fuse
anatomically and functionally with the lower border of the inferior
constrictor muscle of the pharynx forming a muscular sphincter
at this level. The musculature of the pharynx, upper esophagus
and diaphragm is striated and as such has motor innervation
which is solely excitatory in nature whereas the musculature of
the lower esophagus and remainder of gastrointestinal tract is
smooth with both inhibitory and excitatory innervation through
the autonomic nervous system. This difference in innervation and
muscle type predisposes to inherently greater tonicity of the
striated muscles at all times and to actual spasm during any
generalized motor discharge as occurs during the convulsions of
epilepsy or intracranial disease.

Vomiting is a complex reflex act requiring the synchronous
relaxation and/or contraction of many voluntary and involun-
tary muscles.\textsuperscript{11,41} It is under the control of the vomiting center, a group
of cells in the sensory nucleus of the vagus which is located in
the floor of the fourth ventricle. This center may be stimulated
directly by many drugs and toxins, by partial asphyxia, by in-
creased intracranial pressure, as well as by the usual means of
reflex stimulation from various organs of the body, especially the
gastrointestinal tract and organs of special senses. In common
with other nerve cells, it may become fatigued from too continuous
or excessive stimulation, producing incoordination of the vomit-
ing reflex. Incoordination of the vomiting reflex has been seen as a
result of prolonged vomiting\textsuperscript{28} and in acute and chronic alcoholism.\textsuperscript{53} It probably occurs at times with heavy sedation, anesthesia, acidosis, alkalosis, and diseases of the central nervous system either by their direct action on the center or indirectly through production of anoxia or cerebral edema and resulting increased intracranial pressure. Such incoordination could result in physiological obstruction of the esophagus at either its upper or lower portion. If the esophageal hiatus did not widen by a change in position and tone of the diaphragm and the cardia did not relax, the stage would be set for the development of the Mallory-Weiss syndrome whereas if the pharyngeal passage did not widen by the drawing forward of the larynx and the hyoid bone and the circular muscles of the upper esophagus and lower pharynx did not relax, conditions would favor rupture of the esophagus.

While the purely mechanical aspects of esophageal rupture are relatively clearcut and generally accepted the subject of alterations of the wall of the esophagus which produce weakness and so predispose to rupture is filled with theory and disagreement. Esophagitis (acute and chronic)\textsuperscript{8,13,82} simple (peptic) ulcer of the esophagus\textsuperscript{32,46,54,79} and esophagomalacia\textsuperscript{36,72,94} are related conditions with many common etiological factors. In all three a most important factor seems to be the action of the gastric juice upon a tissue which has lost its normal resistance to the enzymic action of the digestive ferments.\textsuperscript{14} In 1869 Pavy\textsuperscript{97} suggested that this loss of resistance was due to interference with the normal circulation of blood through the wall of the gastrointestinal tract. Subsequent experiments and clinical observations have supported this theory. Thus esophagitis and ulcer have been reported as a result of vascular occlusion from arteriosclerosis, syphilis, embolism, thrombosis and spasm as well as from chronic venous congestion from heart failure or portal hypertension. The poor state of the circulation in shock from any cause and in the terminal stages of many diseases is felt to contribute to the esophageal changes frequently found in these conditions.\textsuperscript{68} Prolonged or repeated contact of the gastric juice upon this devitalized tissue is favored by retching, vomiting and relaxation of the cardia.\textsuperscript{5,32} The latter may be due to nausea, gastric dilatation secondary to pyloric obstruction, or from reflex changes secondary to diseases of the central nervous system. Hyperthermia which enhances digestion, surface irritants such as tubes, alcohol, excessive heat or cold, and miscellaneous factors such as foci of infection, deficiency states and aberrant gastric mucosa may be contributing factors in certain cases.\textsuperscript{31,32,46}

Esophagomalacia or intravital softening of the esophagus was
first clearly differentiated from post mortem softening by Rokitansky\textsuperscript{72} in 1849. He pointed out its occurrence either as a part of the terminal cachexia of various diseases or as a complication of acute and chronic diseases of the central nervous system. In the former cases the process is extensive and is rapid in its progress. In these it represents merely the premature onset of post mortem changes and as such has little place in a consideration of spontaneous rupture of the esophagus, for while rupture into the mediastinal and pleural spaces frequently occurs, it is noted as a hardly perceptible decline in an already moribund patient and has no therapeutic possibilities. The esophagomalacia associated with diseases of the central nervous system is frequently focal in nature and gradual in progression. Its presence helps to explain the relatively high incidence of spontaneous rupture of the esophagus in these conditions as emphasized in the past by Cushing,\textsuperscript{24} Masten and Bunts,\textsuperscript{55} Opper and Zimmerman,\textsuperscript{65} and more recently by Fincher and Swanson.\textsuperscript{29} Its presence and the more frequent gastromalacia has been seen associated with different congenital, inflammatory, neoplastic and traumatic diseases of the brain and its coverings. The clinical observations\textsuperscript{24,55} and experimental studies\textsuperscript{9,25,44,50,87} have emphasized the importance of stimulation of the mid brain and hypothalamic nuclei in the development of esophagomalacia and gastromalacia but disease of any portion of the brain can lead to their development\textsuperscript{65} perhaps through influence on these centers. The bulk of experimental work suggests that the theory of spastic ischemic postulated by Zenker and Ziemssen\textsuperscript{94} in 1878 is valid for ischemia, then diapedesis of red blood cells, and finally softening have been observed by different workers\textsuperscript{9,44,50,87} using different methods of central nervous system stimulation. Thus esophagomalacia appears to be a noninflammatory esophageal softening due to the action of gastric juice on a tissue made ischemic by nervous stimulation. Its presence, or the presence of esophagitis or ulceration would greatly weaken the esophageal wall and so predispose to rupture.

**Etiology**

The etiology of spontaneous rupture of the esophagus has been suggested in the discussion of the pathogenesis of the rupture. It is seen chiefly in association with vomiting and/or retching, alcoholism and diseases of the central nervous system. In all of these conditions there are multiple factors present leading to both increased intraesophageal pressure and weakness of the esophageal wall. Table I lists the specific conditions with which spontaneous rupture of the esophagus has been found. Of necessity there is some overlapping of etiology in this table. For convenience
### TABLE I

**SPONTANEOUS RUPTURE ASSOCIATED WITH**

### I. Central Nervous System Disease.

A. Congenital
   1. Internal hydrocephalus

B. Inflammatory
   1. Meningitis
      a. Tuberculosis
      b. Pneumococcus
      c. Meningococcus
      d. Post-operative
   2. Encephalitis
      a. Poliomyelitis
      b. Measles
      c. "Viral"
   3. Syphilis
      a. Paresis
      b. Gumma

C. Tumors
   1. Primary
      a. Malignant
         (1) Cerebellar
      b. Benign
         (1) Craniopharyngioma
         (2) Meningioma
            (a) Frontal
            (b) Sphenoid ridge
   2. Metastatic
      a. Frontal lobe

D. Trauma
   1. Skull fracture
      a. Basal
      b. Fronto-Temporal
   2. Extradural hematoma
   3. Gunshot

E. Vascular
   1. Arteriosclerosis (Case 2)

F. Craniotomy

G. Epilepsy

*Craniotomy.

### II. Vomiting and/or Retching.

A. Overindulgence of food and alcohol
   1. Reflex
   2. Self induced

B. Gastrointestinal tract disease
   1. Impacted food bolus
      1. Stricture
      2. No stricture

D. Metabolic Disease
   1. Addison’s disease
   2. Diabetes mellitus

E. Post-operative
   1. Abdominal
      a. Gastrointestinal
      b. Pelvic
   2. Neurological
   3. Amputation

F. Miscellaneous
   1. Sea Sickness
   2. Shell fish intoxication
III. Miscellaneous.
   A. Lifting\textsuperscript{39,83}
   B. Defecation\textsuperscript{92}
   C. Blunt Trauma
      1. Chest\textsuperscript{63,88,34}
      2. Abdomen\textsuperscript{4,88}
   D. Burns\textsuperscript{59,71}
   E. Minimal
      1. Large meal\textsuperscript{16}
      2. Glass of water\textsuperscript{52}
   F. Nothing\textsuperscript{17,49,47,61}

the postoperative cases are included with the vomiting group although it is true that vomiting was only one of several important factors in these cases. It will be argued that certain of the cases listed in the central nervous system disease group represent terminal esophagomalacia rather than spontaneous rupture of the esophagus for where any doubt existed they were included in the tabulation under rupture.

Pathology

The pathological findings at autopsy or surgery will vary somewhat depending on the presence or absence of preexisting disease of the esophagus, the interval between rupture and observation, and the course followed after the initial rupture of the esophageal wall. The tear is usually single, longitudinal and just above the cardia. Its edges may be clean cut, "as if cut by a knife," or ragged and ulcerated. The rupture is usually on the left posterolateral wall into the left pleural space where the esophagus has the least external support. It is frequently impossible to determine the previous status of the esophagus if death or surgery is delayed for more than several days. These findings agree with the experimental observations of Zenker and Ziemssen,\textsuperscript{94} McKenzie,\textsuperscript{57} Brosch,\textsuperscript{18} Duval,\textsuperscript{27} and Burt\textsuperscript{19} which have recently been reviewed and corroborated by Kinsella, Morse and Hertzog.\textsuperscript{47} All of these workers, using various techniques, found that the lower one third of the esophagus was the weakest portion of the organ, that it was more susceptible to sudden increased pressure than to gradual increased or sustained pressures and that the mucous membrane offered greater resistance to stress than did the muscular coats. Rupture was usually single and longitudinal. Although the exact pressure required to produce rupture varied somewhat, depending on the techniques used, it was within a range obtainable by physiological mechanisms such as vomiting or straining.

Once rupture of the esophagus has occurred the subsequent course of the stomach contents and air that pass through the rent is variable. If there are adhesions between the pleura and
the esophagus at the site of rupture, perforation directly into the pleural space without contamination of the mediastinum will occur. This gives rise to a hydropneumothorax, frequently of a tension type. If the process remains localized in the mediastinum, mediastinal emphysema and subcutaneous emphysema rapidly develop. The mediastinitis, at first chemical and later bacterial, causes the rapid development of sterile or septic pleural effusion and usually in a matter of hours, ruptures into one or both pleural spaces, occasionally at a higher level than the rent in the esophagus. These events give rise to a marked mediastinitis with or without abscess formation and to an empyema with or without necrosis of the underlying lung. The marked collapse and toxicity and the death of at least 70 per cent of these patients within the first 24 hours, is ascribed to the violence of the chemical mediastinitis and/or pleuritis, and to the marked interference with cardio-respiratory function from the mechanical compression of the vascular structures in the mediastinum as a result of the high pressures in the mediastinal and/or pleural space.

**Clinical Aspects**

The clinical picture of spontaneous rupture of the esophagus varies somewhat, depending on whether it occurs in a previously normal person, in a post-operative patient or in a neurosurgical patient. In the former, where the mechanical factors predominate, the picture is quite uniform and usually most violent. The patient, a middle aged male, usually a chronic alcoholic, frequently with chronic dyspepsia, during vomiting and/or retching usually after recent overindulgence in food or alcohol, has sudden onset of pain, collapse, respiratory distress, upper abdominal rigidity, and later subcutaneous emphysema.

The vomiting, either reflex or self induced, is frequently violent and prolonged. The vomitus often contains small amounts of bright red blood or “coffee ground” material but never large amounts of blood. The vomiting usually ceases with the onset of pain. In a few cases there is no antecedent vomiting and in a rare case there is no apparent precipitating factor.

The pain is usually excruciating and continuous, unrelieved by large doses of opiates, but may be minimal or even absent. It usually is substernal or high in the epigastrium, radiating through to the back or lower thoracic area, more frequently the left, or to the shoulder area. The pain may be most severe in the back or renal areas, with little radiation. The pain is frequently so severe that the patient cannot remain quiet, frequently preferring to sit leaning forward with arms clasped about the chest, or to lie on the effected side with the knees drawn up to the chest.
Some patients describe a sensation of "something giving way," or "tearing," or "bursting" in the lower chest at the onset of the pain.

Evidence of collapse is usually an early and prominent finding but may be minimal and late. Most patients have pale, cool, clammy, sweaty skins, frequently with slight cyanosis. The pulse is usually weak and rapid and the blood pressure may be low. Thirst may be marked in a few cases. The temperature is usually normal or subnormal during the initial hours but then begins to rise. This collapse does not respond particularly well to usual shock therapy, suggesting that it may be due chiefly to mechanical interference with cardiac function.

Respiratory findings of rapid, labored, grunting, shallow respiration with slight cyanosis and dyspnea may or may not be prominent at onset, but become progressively more apparent during the initial hours of the disease. Chest signs are frequently not carefully sought for because of the critical condition of the patient and the more obvious abdominal signs, but even if careful physical examination of the chest is made, they may not be apparent in the first few hours. Then rales, changes in percussion note and breath sounds appear, followed by the signs of hydrothorax, hydropneumothorax or tension hydropneumothorax.

The abdominal signs which may predominate the early hours of the disease have quite understandably led to the erroneous diagnosis of perforated peptic ulcer, for there may be marked upper abdominal spasm, rigidity and tenderness. The lower abdomen is normal, as is the rectal examination.

Subcutaneous emphysema, at first deep in the supraclavicular and suprasternal spaces and later more generalized, appears in about two thirds of the patients. It is seen within the first few hours if the process is confined for a time in the mediastinum but is seen later if there is early rupture into the pleural space. In the latter case it will not develop until the pressure of the hydropneumothorax has reached or exceeded atmospheric pressure making it as easy for the gas to pass into the fascial planes of the mediastinum as to escape into the pleural cavity. In a few patients, subcutaneous emphysema is the presenting symptom, the pain, collapse and other symptoms being minimal or absent. It is the one finding which most suggests but does not prove the presence of a ruptured esophagus.

In the post-operative patients and in patients suffering from serious extraesophageal disease the rupture is primarily due to weakness of the esophageal wall. In these patients, the accident is frequently less fulminating because the violent vomiting and resulting high pressure changes in mediastinal and pleural spaces
are often lacking. In these patients, the clinical picture is variable, showing the features of the underlying disease combined with any of the features of the rupture syndrome.

Cushing\textsuperscript{24} and more recently Fincher and Swanson\textsuperscript{29} have clearly described the clinical picture of spontaneous rupture of the esophagus as a complication of craniotomy. They noted that, although the immediate post-craniotomy reaction was satisfactory with the expected but relatively stationary increase in temperature, pulse and respiration, in the first eight to 12 hours as recovery from the anesthetic hypnosis occurred, vomiting of bright red or changed blood began. Consciousness, partly or completely regained by the end of the first day began to lag and the patient lapsed into stupor and then coma over a four to six hour period. This change in consciousness, which was the only new neurological finding, was associated with extreme restlessness, excessive sweating, cyanosis of fingers and toes, dyspnea and increase of pulse and respiratory rate. The immediate temperature reaction to craniotomy which had begun to fall prior to the onset of these symptoms, rose abruptly and continued to rise in spite of antipyretic therapy usually effective in controlling post-craniotomy hyperthermia. Subcutaneous emphysema did not develop and the blood pressure remained normal until just prior to death which usually occurred within 48 hours of craniotomy.

\textit{Differential Diagnosis}

The differential diagnosis of spontaneous rupture of the esophagus is complex, involving a consideration of all intrathoracic and upper abdominal emergencies. These include coronary thrombosis, dissecting aneurysm of the aorta, spontaneous pneumothorax, spontaneous hemopneumothorax, spontaneous mediastinal emphysema, pulmonary embolism, massive collapse of the lung, ruptured peptic ulcer, acute pancreatitis, mesenteric occlusion, splenic infarction, and strangulation of a diaphragmatic hernia. While it may be possible to diagnose spontaneous rupture of the esophagus by careful consideration of the onset, course, and physical findings, definite proof from radiologic and thoracentesis studies is possible and should be used to verify the diagnosis in every case.

\textit{Radiologic Aspects}

Since the radiologic evidence is most important in substantiating the diagnosis it is usually best to make these studies in the radiology department where more complete, technically better and more rapid work can be done.\textsuperscript{2,26} Satisfactory bedside studies can
be made in comatous or moribund patients by the interaesophageal instillation of contrast media through a Levine tube, as suggested by Abbott.\(^1\) The radiologic findings can be either suggestive or pathognomonic of rupture of the esophagus. A recognition of the possible significance of the former is most important for if rupture is suspected its presence can be proved by observing the passage of radio-opaque material from the esophagus into the mediastinal and/or pleural space. The radiologic findings will vary depending on the course taken by the rupture. Usually the earliest finding is mediastinal emphysema characterized by widening of the mediastinum and the presence of air along the fascial planes of the mediastinum and later of the neck and subcutaneous tissues. It frequently can be best seen in the lateral projection, especially of the neck where an increase in the width between the posterior tracheal wall and the anterior surface of the vertebral bodies is sought.\(^2\) Mediastinal emphysema is also seen in the spontaneous mediastinal emphysema and as a rare complication of rupture of either the uterus\(^4\) or of a gastric or duodenal ulcer.\(^5\) Because of this rare association of mediastinal emphysema with perforated duodenal or gastric ulcer all patients with the former should have overexposed upright x-ray films of the upper abdomen made. Although only 70 to 80 per cent of proved cases of ruptured duodenal or gastric ulcer will show subdiaphragmatic air, its absence in the presence of mediastinal emphysema definitely rules out the possibility of a ruptured ulcer as the cause of the mediastinal emphysema. Lynch\(^3\) emphasizes the importance of a retrocardiac air bubble and fluid level frequently present but often overlooked in the upright films taken of the upper abdomen of patients suspected of having ruptured duodenal or gastric ulcers, who ultimately prove to have had spontaneous rupture of the esophagus. These findings in association with mediastinal emphysema, or alone if diaphragmatic hernia can be excluded, are diagnostic of ruptured esophagus. The presence of massive, rapidly increasing hydropneumothorax is most suggestive of spontaneous rupture of the esophagus\(^7,21,64\) although spontaneous hemopneumothorax may present identical clinical and radiologic characteristics.\(^40\) Hydrothorax without air is occasionally seen early as a result of effusion secondary to the intense mediastinitis and is therefore associated with the x-ray findings of mediastinitis. If the latter are not recognized the presence of the fluid will at least prevent abdominal surgery and focus attention on the chest as the source of the patients symptoms. The passage of a flexible tube through the rent in the esophagus into the mediastinum as shown by an aberrant position of the tip of the tube on x-ray is not a suggested procedure but its accidental occurrence may be the first clue to
the patient's difficulty and is diagnostic.\textsuperscript{33,43} Pneumothorax without demonstrable fluid or with slow accumulation of fluid is so rarely seen with spontaneous rupture of the esophagus that its presence would tend to suggest the diagnosis of spontaneous pneumothorax or spontaneous mediastinal emphysema with secondary pneumothorax. Because of the seriousness of the disorder, the complexity of the differential diagnosis, and the magnitude of the surgical procedures required for its treatment, it would seem valid to insist on absolute verification of the diagnosis of spontaneous rupture of the esophagus by use of radio-opaque media before definitive treatment is begun. This is especially true because the contrast media, either iodized oil\textsuperscript{8,85} or barium sulfate suspension\textsuperscript{22,33,62} does no apparent harm and will usually be removed immediately at surgery if extravasation occurs.\textsuperscript{2,26} Iodized oil is easier to remove from the mediastinal and pleural spaces than barium suspension but there is minimal risk of iodism from its use. In the comatous patient Abbott\textsuperscript{1} has successfully used the intraesophageal instillation of iodized oil through a Levine tube passed into the upper esophagus as a bedside procedure.

\textit{Thoracentesis}

A properly performed thoracentesis in which it can be certain that the tip of the needle is not in a dilated herniated stomach, has not passed through the diaphragm into the stomach, or is not in a blood vessel may give valuable information as well as being of therapeutic value.\textsuperscript{4,7,47} If rupture into the pleural space is delayed the pleural effusion which rapidly develops secondary to the mediastinitis will be a typical inflammatory exudate, either sterile or pyogenic.\textsuperscript{20} After rupture into the pleural space the aspirated fluid frequently has the characteristic appearance and odor of recently ingested food or liquid.\textsuperscript{62,85} It may be colored dirty brown from altered blood but never contains much un-changed blood. This is important for in dissecting anerysm of the aorta with rupture into the pleural space and in spontaneous hemopneumothorax the aspirated fluid is nearly pure blood. The fluid may smell of vomitus or have a sour acrid odor. If it is tested with blue litmus paper or titrated with Topper's solution and sodium hydroxide, it will be found to be strongly acid, proving its gastric origin. Culture of the fluid, though rarely necessary, may reveal massive mixed infection including acidophilus bacillus and other saphrophyles.\textsuperscript{85} In a few cases, the diagnosis was suggested by the prompt appearance of some recently ingested colored solution in the pleural cavity or by the appearance of food,\textsuperscript{33} colored solution,\textsuperscript{62} or massive quantities of fluid through an inter-costal drainage tube.\textsuperscript{6}
Treatment

The treatment of spontaneous rupture of the esophagus consists of the surgical repair of the rent and/or the drainage of the mediastinal and pleural spaces, and the supportive treatment of the patient. Successful outcome is now possible in these cases because of the advances in thoracic surgery, anesthesiology, antibiotic therapy and blood bank techniques.

There is no single established method of surgical therapy and probably never will be for the surgery will vary with the course taken by the rupture, the status of the esophagus prior to rupture, the interval between rupture and surgery, the presence or absence of high intrapleural and/or intramediastinal pressures, the extraluminal diseases of the patient and the experiences and limitations of the surgical team. In the usual case with a previously normal esophagus, marked increased intramediastinal pressure and contamination of the pleura, prompt surgical intervention by a transpleural approach with wide drainage of the pleural space after its irrigation and after re-expansion of the lung seems to be the surgical treatment of choice. \( \text{9,47,51,86} \) This opinion is based on theoretical, experimental and clinical considerations. Kinsella, Morse and Hertzog\( ^{47} \) in discussing the prognosis of 53 patients reported prior to 1947, emphasize that 25 per cent died in less than 12 hours, 70 per cent in less than 24 hours and 85 per cent in less than 48 hours, and only 15 per cent lived over 48 hours. These figures strongly suggest that immediate definitive therapy is indicated for the majority of patients will not survive long enough to permit delayed or supportive therapy. Ware and Strieder\( ^{86} \) have made careful experimental studies in dogs to study the influence of various types of therapy on survival time. They found no significant difference in the survival time of the control untreated animals and those given only supportive therapy of parenteral fluids, sulfadiazine and penicillin. The animals in whom the same supportive therapy and delayed surgical repair was carried out lived somewhat longer than the first two groups. The majority of those given supportive therapy and prompt surgical repair survived.

Prompt surgical repair of the esophageal rent by a transpleural route has been reported only six times. The patient of Collis, Humphreys and Bond (1944)\( ^{20} \) died within 24 hours of surgery after a technically successful procedure and temporary improvement of the patient. The patient of Kinsella, Morse and Hertzog (1946)\( ^{47} \) seemed well on the road to recovery when he died suddenly on the ninth postoperative day of a massive pulmonary embolus. Barrett's patient (1946),\( ^{7} \) the first to be successfully
managed by direct surgical repair, had a stormy course. Leakage of the esophageal repair occurred on the eighth postoperative day, possibly because of an attack of bronchial asthma. This latter necessitated the drainage of a mediastinal abscess and the establishment of an enterostomy. A complicating lung abscess drained spontaneously through a bronchus yet ultimately a complete cure ensued. Lynch's second patient (1948) also had leakage of the repaired rent on the eighth postoperative day but ultimately had spontaneous closure of the esophageocutaneous fistula and complete recovery. The patient of Olsen and Clagett (1947) and the third patient of Lynch (1948) had relatively smooth postoperative courses, the latter taking a liquid diet on the third postoperative day.

The interval of time between rupture and surgical treatment after which repair of the esophagus would not seem desirable because of likelihood of breakdown of the repair due to inflammation of the esophagus has not been determined. The experiences of Lynch suggest that 24 to 36 hours might be the dividing line. In those patients in whom an attempt to repair the rent does not seem advisable, prompt adequate drainage of the mediastinal and pleural spaces is indicated. In 1938 Benson and Pemberthy reported the first recovery from this disease following closed catheter drainage of the pleural space. Subsequently Graham (1944), Fink (1947), and Moore and Murphy (1948) have reported recovery following closed drainage of the pleural space after rib resection.

The difficulty in adequately draining the mediastinum by these methods and the marked improvement noted by Lynch in patients after wide drainage of the mediastinum suggest that transpleural drainage of the mediastinum into the already contaminated pleural space and subsequent closed drainage of the pleura after re-expansion of the lung should be attempted as soon as possible in these late cases. Lynch (1949) has used this method successfully in one case of spontaneous rupture of the esophagus and Seybold has used it as the treatment of choice in patients with lower esophageal perforation following dilatation of a cardioesophagus where the therapeutic problems are similar to those in cases of spontaneous rupture of the esophagus without increased mediastinal pressure.

It would seem wiser to allow the resulting esophageocutaneous fistula to close spontaneously although Moore and Murphy were able to successfully complete an esophagagastropasty 40 days after rupture and so to markedly shorten the period of hospitalization and medical supervision of the patient. Abbott suggests that in the post-craniotomy patient with spontaneous rupture of the eso-
phagus surgical drainage of the area by intercostal tube or better by rib resection be done initially, and that this be followed at a later date by a definitive plastic repair of the esophagus by resection of the area of esophagomalacia and the reestablishment of continuity by an esophagogastro-anastomosis.

Posterior mediastinotomy with drainage of the mediastinum is indicated in the rare case in which the process is localized in the mediastinum. Gardner (1949)\textsuperscript{34} reported recovery of such a case. Overholt\textsuperscript{23,66} successfully used a right and then left posterior mediastinotomy in a patient first seen 15 days after rupture with bilateral pleural and mediastinal contamination.

The transabdominal repair of the rent after pulling the esophagus down\textsuperscript{31} would seem to be inadequate and undesirable. It would seem better to close the abdomen and approach the lesion through the thorax if the diagnosis should be made at the time of laparotomy. Gastrostomy,\textsuperscript{2} jejunostomy\textsuperscript{33,51,62} and enterostomy\textsuperscript{7} have been used as an adjunct in patients in whom early surgical repair was either not attempted or was not successful. The gastrostomy would seem least desirable because of greater danger of regurgitation into the esophagus and out through the rent. In spite of theoretical objections,\textsuperscript{20,37} a Levine tube has frequently been successfully used postoperatively,\textsuperscript{10,64} thus eliminating the need of enterostomy as an added surgical procedure.

The supportive measures which are important in the care of these patients, regardless of the surgical treatment used, have been outlined by Kinsella, Morse and Hertzog\textsuperscript{47} and by Ware and Strieder.\textsuperscript{86} Most important is a realization of the importance of immediate reduction of the high intrapleural and/or mediastinal pressures. The former should be decompressed at once by needle aspiration of the pleural cavity followed by intercostal catheter drainage with suction to prevent its recurrence. The value of mediastinal decompression by a simple collar incision and introduction of a finger into the superior mediastinum was apparent in the patient reported by Vander Laan and Maresh,\textsuperscript{61} in whom definite but temporary benefit occurred following this procedure even though the spontaneously ruptured esophagus as the underlying cause of the mediastinal emphysema was not recognized or treated. Ware and Strieder\textsuperscript{86} and Lynch\textsuperscript{51} emphasize the importance of mediastinal decompression in the care of these patients. Other supportive measures include oxygen, preferably by positive pressure mask, parenteral therapy of whole blood, plasma, dextrose and electrolytes, and massive shotgun chemotherapy with parenteral and intrapleural penicillin and dihydrostreptomycin, parenteral sulfonamides if urine output is adequate, and aureomycin by tube or enterostomy. Decompression of the abdomen
should be maintained by suction through a Levine tube or by an enterostomy. Nothing should be given by mouth or by tube for two or three days. Pleural effusion into the uninvolved side as an early complication and hemorrhage, breakdown of the repair, mediastinal and/or lung abscess, pericardial effusion, and pulmonary embolism as late complications should be carefully watched for and treated with appropriate measures.

The value of modern therapeutic procedures is made evident by an analysis of the approximately 100 cases found in the world's literature. Of this number there were only 13 survivors, all reported since 1938, 10 since 1946.

Case 1: G.B.: J.M.H. No. 31561. A 43 year old white male was apparently in excellent health until he awoke from a nap induced by the consumption of a large amount of beer, vomiting coffee ground material. In the next two and one half hours, he had eight episodes of vomiting of blood, a total, as estimated by the patient, of about one pint. Shortly after the onset of vomiting, he developed slight epigastric soreness. While being examined in the emergency room, he was seized with excruciating pain in the upper abdomen and subxyphoid area which radiated through to the back and was not relieved by an opiate. He was unable to lie on his back but remained curled up on his right side.

His past history revealed an asymptomatic hypertension of three years duration with known systolic levels of 190 mm. Hg.; a "moderate" daily intake of alcohol for years. No previous gastrointestinal disturbances; no previous surgery. System review revealed no other complaints.

Physical examination, performed two and one half hours after onset of symptoms showed an obese white male in acute distress, sweating profusely. Temperature 98.6 (oral), pulse 110, respiration 26, blood pressure 120/80. Positive findings were restricted to the epigastrium where there was marked rigidity, tenderness and rebound tenderness. The lower abdomen and rectal examination were normal. Peristalsis was heard only in the lower quadrants. The chest and cardiovascular systems were normal.

Laboratory studies done soon after entry but after the intravenous infusion of glucose in saline had been begun showed a urine with specific gravity of 1.034, a trace of albumin, 3 plus sugar and 3 plus acetone, a sediment containing a few pus cells and a few hyaline casts per high power field. The blood count showed Hgb. 77 per cent (11.8 Gm.), RBC 4,500,000, WBC 5,650, differential neutrophils 91 per cent (segmented forms 85, stab forms 6), lymphocytes 7, monocytes 2. An upright x-ray film of the abdomen was negative for pneumoperitoneum. Kahn negative.

Course: Three hours after entry 800 cc. of bloody beer was aspirated from the stomach and a laparatomy was performed. No evidence of peritonitis, perforation nor abnormality of the gastrointestinal tract was found. He recovered from the anesthetic still complaining of the severe subxyphoid pain which was not relieved by opiates and of dyspnea. A serum amylase was normal (96 units) and the electrocardiogram showed sinus tachycardia of 176, low voltage, slurring of the QRS and poorly deflected T throughout; interpreted as non-specific myocardial impairment with no evidence of coronary thrombosis. Nine hours after surgery, cyanosis and collapse appeared (B.P. 90/60). There was no response to
oxygen and repeated transfusions. At this time, there was marked subcutaneous emphysema over the upper abdomen, anterior chest, neck, face and upper back. There was dullness, diminished breath sounds and rales over the right chest posteriorly. A portable x-ray film of the chest was interpreted as showing right pleural effusion, marked subcutaneous emphysema and probable pneumomediastinum. No surgical treatment was given and the patient died 27½ hours after onset of hematemesis and 24 hours after onset of acute pain.

Autopsy, done immediately after death, revealed a 6 cm. knifelike slit on the right posterolateral wall of the esophagus, beginning 1½ cm. above the cardia. There were four short mucosal tears of the lower esophagus and cardia but no open vessels were noted. The mediastinum was pocketed with air and green bile-stained fluid. Necrotic food particles were found as high as the arch of the aorta. There was free communication of the mediastinum with the right pleural space which contained similar bile-stained necrotic material. The left pleural space contained a small amount of cloudy exudate and fibrinous pleuritis. Microscopic sections of the esophagus showed only inflammatory changes consistent with an acute process of short duration. Additional findings included marked fatty infiltration of the liver and moderate fatty infiltration of the slightly hypertrophied heart.

Comment: This case represents the rare variation of the Mallory-Weiss syndrome in which perforation of the esophagus rather than fatal hematemesis was the mode of death. Except for the hematemesis, which was greater than that usually seen in spontaneous rupture of the esophagus, the entire picture was characteristic of that group of cases in which rupture of a presumably normal esophagus occurs as a result of sudden increased intraesophageal pressure.

Case 2: H.B.: J.M.H. No. 49944. A 68 year old white male was brought in by the police because of moderately severe substernal pain of several hours duration. He was confused and disoriented and remained so until his death 10 days later. The only history available was that he had had some angina pectoris for two years, more severe in the last four months. He had had intermittent ankle edema for several years. Seven months before he had had granted a government pension for heart trouble. The present chest pain had begun several hours before entry, was substernal without radiation, and unassociated with other symptoms.

Physical examination on entry revealed an elderly obese, dirty white male who was lethargic, confused and disoriented but in no distress. Temperature 100 (oral), pulse 90, respiration 18, blood pressure 130:80. The fundi showed moderate arteriosclerosis. Heart enlarged 2 cm. outside the midclavicular line, rate 90, occasional dropped beat. No murmurs or friction rub. Chest clear except for inconstant coarse bilateral basal rales. Abdomen and neurological examination normal except for the mental status.

Laboratory studies on entry revealed the urine to have 4 plus albumin, 7 to 8 red blood cells per high power field in the centrifuged sediment. The blood count: Hemoglobin 85 per cent (13.0 Gm.), RBC 4,300,000, WBC 5,600, Differential—neutrophils 64 per cent (segmented forms 61, stab forms 3), lymphocytes 33 per cent, and monocytes 3 per cent. N.P.N. 48 mgs. per cent. Prothrombin time undiluted 17.2 seconds. Sedimentation rate (corrected Wintrobe) 26 mm. Total serum protein 6.04 Gm., albumin 3.45 Gm., globulin 2.59 Gm. Kahn negative. X-ray film of the chest (Fig.
2a) was interpreted as showing increased markings in both lower lobes but no actual consolidation. An electrocardiogram showed an irregular rate of approximately 130 due to short runs of sinus rhythm with variable A-V response, premature auricular systoles and fusion beats. T1, 2, 3 were biphasic. The changes were interpreted as being nonspecific. Lumbar puncture showed the spinal fluid to be clear and colorless under normal pressure but with a positive Pandy reaction and a protein of 113 mgm. per cent. The remainder of the fluid examination was normal.

Course: The patient was digitalized, placed in oxygen, given penicillin parenterally in a dosage of 50,000 units every three hours, and fluid balance was maintained by the oral, then intravenous route. In spite of this therapy, his condition gradually but steadily deteriorated. His temperature ran a septic course with daily elevations to 102-103 degrees (oral), the pulse and respiratory rates increased and the lethargy and confusion increased. The physical findings of the heart, chest, abdomen and nervous system remained as on entry. Serial electrocardiograms showed no change. The NPN dropped to 26 mgm. per cent, the CO2 combining power was 43 Vol. per cent, the urine showed less albumin, no sugar and a few casts. A blood count done on the ninth day of his illness showed Hgb. of 64 per cent (9.9 Gm.), RBC 3,380,000, WBC 3,250. Differential, neutrophils 78 per cent (segmented 64, stabs 10, juveniles 4), lymphocytes 16 per cent, monocytes 4 per cent, eosinophils 2 per cent. Blood smear revealed the neutrophils to show toxic changes. An x-ray film of the chest (Fig. 2b) taken on the ninth day showed definite widening of the mediastinal shadow but no pleural or pulmonary changes. He died suddenly on the tenth day.

Autopsy done 10 hours after death revealed an extensive gangrenous mediastinitis with extension of the gangrenous process into the adjoining portion of both right and left lower lobes of the lung. The pleural spaces contained no free fluid and an obliterative pleuritis walled off the involved lung areas. A large irregular perforation of the lower one-third of the esophagus communicated freely with the mediastinum. The perforation had rolled edematous edges which showed petechial hemorrhages. It extended upward from just above the cardia on the posterior

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**FIGURE 2a**  
*Figure 2a:* Admission chest film shows only accentuated basal markings.

**FIGURE 2b**  
*Figure 2b:* Ninth day chest film shows marked widening of the mediastinal shadow.
wall. Histological examination of the esophagus showed acute and chronic inflammation and necrosis but no occlusive vascular disease. Grossly the brain was normal but histological examination revealed marked sclerosis of the smaller vessels with multiple small areas of softening throughout the cortical substance. Additional findings included arteriolar nephrosclerosis, coronary sclerosis with myocardial fibrosis and fatty infiltration of the liver.

Comment: This case represents an example of spontaneous rupture due to esophagomalacia secondary to cerebral arteriosclerosis. The correct antemortem diagnosis might have been made had the significance of the progressive widening of the mediastinal shadow been appreciated.

SUMMARY

1) Spontaneous rupture of the esophagus is due to an increase in intraesophageal pressure greater than the tensile strength of the esophagus. Increased intraesophageal pressure is due to the transmission of increased intraabdominal pressure by the stomach contents through the cardia into the esophagus. Rupture is made more likely by distention of the stomach with food or fluid and by obstruction of the esophagus. Obstruction is usually physiological, produced by the complex anatomical makeup of the esophagus and by incoordination of the complex vomiting reflex. Esophagitis, simple ulcer of the esophagus and esophagomalacia weaken the esophagus and so predispose to rupture.

2) Rupture is seen chiefly in association with vomiting and/or retching, alcoholism and diseases of the central nervous system.

3) The linear tear is in the lower third of the esophagus and leads to the rapid development of mediastinitis and/or hydropneumothorax. The 70 per cent mortality in the first 24 hours is due to the violence of the mediastinitis and pleuritis and to the interference with cardio-respiratory function from mechanical compression.

4) In the usual case, a middle aged male, chronic alcoholic, frequently with chronic dyspepsia, during vomiting and/or retching after recent overindulgence in food or alcohol had sudden onset of chest pain, collapse, respiratory distress, upper abdominal rigidity and later subcutaneous emphysema. In the post craniotomy patients vomiting of blood during recovery from anesthesia occurs: consciousness is lost; and there is progressive restlessness, cyanosis, dyspnea with increasing temperature, pulse and respiratory rates.

5) Definite diagnosis is possible by radiologically observing the passage of radio-opaque media through the rent into the mediastinal and pleural spaces or by aspiration of stomach contents from the pleural space. Mediastinal emphysema, rapidly accumulating
hydrothorax, and a retrocardiac air bubble and fluid level are suggestive radiologic findings.

6) In the early cases prompt transpleural repair of the rent is the treatment of choice. In the remainder drainage of the mediastinal and pleural spaces is indicated. The most important of the supportive measures are the decompression of any tension pneumothorax or high pressure mediastinal emphysema, and the use of antibiotic therapy. The various surgical procedures used in the 13 survivors are discussed.

7) Two case reports are given.

RESUMEN

1) La ruptura espontánea del esófago es debida a un aumento de la presión intraesofágica mayor que la que puede soportar el esófago.

Este aumento de presión es debido a la transmisión de presión intrabdominal por intermedio del contenido gástrico hacia el corazón y el esófago. Es más posible que haya ruptura cuando hay distensión del estómago con alimentos o líquidos y por obstrucción del esófago. La obstrucción es generalmente fisiológica, producida por el arreglo anatómico complejo del esófago y por incoordinación del reflejo complicado del vómito. La esofagitis, la úlcera simple del esófago y la esofagomalacia debilitan el esófago y así predisponen a la ruptura.

2) La ruptura se ve principalmente en asociación con vómitos o eructos, alcoholismo y enfermedades del sistema nervioso central.

3) La desgarradura lineal es en el tercio inferior del esófago y conduce al rápido desarrollo de mediastinitis o hidroneumotórax.

La mortalidad de 70 por ciento que se ve en las primeras 24 horas se debe a la violencia de la mediastinitis y pleuritis y a la interferencia con la función cardio-respiratoria que resulta de la compresión.

4) En el caso habitual se trata de un hombre en edad media de la vida, alcohólico crónico con dispepsia crónica a menudo, que durante el vómito o regurgitación después de haber comido en exceso o bebido mucho alcohol tiene de pronto un dolor en el pecho, colapso, dificultad para respirar, rigidez de la parte superior del abdomen y después enfisema subcutáneo. En los enfermos después de craniotomía, que comitan durante la salida de la anestesia, se pierde la conciencia y hay inquietud creciente, sudores, cianosis, disnea y elevación progresiva de la temperatura, aceleración del pulso y de la respiración.

5) El diagnóstico definido es posible mediante la ingestión de líquido radiopaco que pasa a través de la ruptura hacia el mediastino y el espacio pleural. El enfisema mediastínico, hidrotórax que
se acumula rapidamente y burbuja retrocardiaca y nivel líquido son sugestivos radiológicamente.

6) En los casos vistos a tiempo, la reparación por vía transpleural de la ruptura es el mejor tratamiento. En los demás el drenaje de las cavidades mediastinal y pleural están indicados. La medida más importante como auxiliar es la descompresión de cualquier neumotórax a tensión y de cualquier enfisema a alta presión y el uso de antibióticos. Se discuten los diversos procedimientos quirúrgicos usados en 13 supervivientes.

7) Se relatan dos casos.

RESUME


2) La rupture se voit principalement en association avec les vomissements et l’éructation, l’alcoolisme, et les atteintes du système nerveux central.

3) La déchirure linéaire dans la troisième partie inférieure de l’oesophage, conduit au rapide développement d’une médiastinite ou d’un hydropneumothorax. La mortalité de 70% dans les 24 heures est due à la gravité de la médiastinite et de la pleurésie, et au trouble de la fonction cardio-respiratoire qui résulte de la compression mécanique.

4) Dans le cas habituel, un individu moyennant âgé, alcoolique chronique, souvent avec dyspepsie chronique, ressent à l’occasion de vomissements ou d’éructations survenant après un excès de nourriture ou d’alcool, une douleur dans la poitrine. Il présente une tendance au collapsus et on constate une gêne respiratoire, et une rigidité de la partie supérieure de l’abdomen et ensuite de l’emphyème sous-cutané. Chez les malades traités par craniotomie, un vomissement de sang survient pendant le réveil qui suit l’anesthésie; il y a partie de conscience et agitation croissante, cyanose, dyspnée, avec élévation de la température, accélération du pouls et de la respiration.

5) Un diagnostic précis est possible en observant radiologique-
ment le passage du liquide radio-opaque par la rupture dans les espaces médiastinal et pleural, ou par l'aspiration de contenu gastrique dans l'espace pleural. L'émphyème médiastinal, l'hydrothorax s'accumulant rapidement, une bulle d'air rétro-cardiaque et le niveau liquide sont des constatations radiologiques suggestives.

6) Dans les cas précoces, la réparation par voie transpleurale de la rupture est le meilleur traitement. Ensuite le drainage des espaces médiastinal et pleural est indiqué. La plus importante des mesures accessoires est la décompression de la tension du pneumothorax, ou de l'émphyème médiastinal à haute pression, et l'emploi de la thérapeutique antibiotique. L'auteur discute des différents procédés chirurgicaux employés chez les individus qui ont survécu.

7) L'auteur rapporte deux cas.

REFERENCES

1 Abbott, O.: Quoted by Fincher, E. F. and Swanson, H. S.
10 Blackfan, K. D.: Quoted by Cushing H.
15 Boyd, S.: Discussion of Bowles, R. L.
23 Cunningham's Textbook of Anatomy Edited by A. Robinson, New York, 
25 Cushing, H.: "Similarity in Response to Posterior Lobe Extract (Pituitrin) 
and to Pilocarpine when Injected into Cerebral Ventricles," Proc. 
27 Duval: Cited by Kinsella, T. J., Morse, R. W. and Hertzog, A. J. 47
29 Fincher, E. F. and Swanson, H. S.: "Esophageal Rupture Complicating 
Craniootomy-Symptom Complex and Proposed Surgical Treatment," 
30 Fitz, R. H.: "Rupture of the Healthy Esophagus," Am. J. Med. Sc., 73: 
17, 1877.
31 Foggett, K. D.: "A Case of Spontaneous Perforation of the Esophagus," 
32 Friedenwold, J., Feldman, M. and Zeim, W. F.: "Peptic Ulcer of the 
33 Frink, N. W.: "Spontaneous Rupture of the Esophagus, Report of a 
35 Girard, J.: "LaRupture Spontanee de L'Oesophage," Gaz. d. hop., 107: 
1117, 1934.
36 Glass, W. E. and Freeman, Wm.: "Spontaneous Rupture of the 
37 Gott, R. Jr.: "Spontaneous Rupture of the Esophagus with a Report of 
38 Graham, E. A.: "Year Book of General Surgery," Chicago, Year Book 
Publishers, 1944, P. 382.
39 Griffith, R. S.: "Spontaneous Rupture of Esophagus," Penn. M. J., 35: 
639, 1932.
1924.
43 Higgins, J. F. and Clagett, O. T.: "Complete Disruption of the 
44 Hoff, E. C. and Sheehan, D.: "Experimental Gastric Erosions Following 
45 Hoffman, C.: "Uber die Erweichung und den Durchbruch der Spei- 
47 Kinsella, T. J., Morse, R. W. and Hertzog, A. J.: "Spontaneous Rupture 
1935.
50 Light, R. U., Bishop, C. C. and Kendall, L. G.: "The Production of Gastric 
Lesions in Rabbits by Injection of Small Amounts of Pilocarpine 
52 Mallam, P. C., Whiteloch, H. A. B. and Robb Smith, A. H. T.: "Spontane- 
53 Mallory, G. K. and Weiss, S.: "Hemorrhages from Lacerations of the 
Cardiac Orifice of the Stomach due to Vomiting," Am. J. Med. Sc., 
178:506, 1929.
54 Mallory, T. B.: "Case Records of the Massachusetts General Hospital," 
55 Masten, M. G. and Bunts, R. C.: "Neurogenic Erosions and Perfora-
60 Meyer, J.: Cited by Walker, I. J. 64
75 Seybold, W. D.: Personal communication, 1949.

For additional references see Barrett, R. N.,6 Kinsella, T. J., Morse, R. W. and Hertzog, A. J.,47 and Weder, A.88