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DIAGNOSIS AND TREATMENT OF THE PERFORATED PEPTIC ULCER

by

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SENIOR THESIS PRESENTED TO
THE COLLEGE OF MEDICINE
UNIVERSITY OF NEBRASKA
OMAHA , 1941

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INTRODUCTION

My object in presenting this subject is to call the attention of the general practitioner to the symptoms and early diagnosis of the condition which is so imperative. It is not only the surgeon upon whom the responsibility rests but it is also important that the attending physician recognize and establish an early diagnosis so that early operation can be brought about, which is the most important single factor in successful treatment.

Moynihan (59) once said, "The perforation of a gastric or duodenal ulcer is one of the most serious and overwhelming catastrophies that can befall a human being. The onset of the symptoms is sudden, the course rapid. and unless surgical measures are adopted early, the disease hastens to a fatal ending in almost every instance."

The symptoms most relied upon in the diagnosis of this catastrophe are sudden and severe localized epigastric pain,which soon spreads over the entire abdomen and occasionally to the chest, back, or shoulders. There is a marked pallor, the face is drawn, the skin is cold, the temperature is subnormal,respirations are shallow and the abdomen is retracted soon becoming of

board-like rigidity. The board-like rigidity is of such great intensity and of such outstanding nature that it can be classed as the most pronounced sign pointing to an acute perforated ulcer. Unfortunately they form only half of one's actual experience and not every case presents such a typical picture. Collapse is frequently absent and a previous history of gastric ulcer is very often entirely lacking. Many times perforation has been suspected where operations proved there was not even an ulcer, on the other hand patients have reached the operating table before perforation was even thought of and mistaken for some other calamity.

It is for this reason that I wish to point out some of the other affections in various parts of the body which may be confused and cause errors in the diagnosis of perforated peptic ulcer, and attempt to present some of the differential points and methods which help to eliminate the confusion with which an attending physician is confronted with in such an emergency.

He is also confronted with other problems after the diagnosis is made as to treatment, complications, aftercare etc., and to complete the picture I will attempt

to bring in the most common complications and the most commonly accepted forms of treatment.

DEFINITION

The perforated peptic ulcer may be defined as a complication arising from a gastric or duodenal ulcer which has penetrated through the viscus and gained communications between the alimentary tract and the free peritoneal cavity.

CLASSIFICATION

Perforating ulcer of the stomach and duodenum may be classified as acute, subacute and chronic. The subacute has also been described and referred to as the "formes frustes" type by Singer and Voughan (74).

These types are classified mainly as to the time element involved.

The acute type is rapid and perforates directly into the peritoneal cavity with such rapidity that no defense mechanism can be brought into play. Due to this the stomach contents escape unimpeded into the free peritoneal cavity causing acute symptoms. Acute perforations are usually found on the anterior wall.

In the subacute type the perforating ulcer process takes place more slowly so that a defense mechanism, consisting of omentum or connective tissues which be-

come attached to surrounding viscera thus preventing escape of gastric contents into the free peritoneal cavity. In this type the visceral contents are more or less circumscribed, and symptoms less acute.

In the chronic type the process is so slow that the defense mechanism has taken place before the actual perforation occurs so that the base of the ulcer becomes attached to some adjacent organ as pancreas or liver, and usually formes a subphrenic, subhepatic, retrocolic or perigastric abcess. Chronic perforations usually occur on the posterior wall of the viscus.

HISTORY

Acute perforating ulcer of the duodenum was first described by Joseph Penada, of Padua, in 1793. Archibald Mallock (48) has given us a most interesting abstract of the case with a reproduction of Penada's plates. Penada described the perforating ulcer, upon autopsy, as follows:

"Four finger-breadths below the pylorus, i.e., at the commencement of the duodenum, there presented itself to my eye, a very singular oblong hole, resembling an incision made with a knife. It measured eight Parisan lines in length and in breadth about two. The external edge of this cleft, or, one should

rather describe it as a peculiar morbid ulcer, was of considerable thickness. To the touch it was sensibly hard and somewhat indurated, and was turned in upon itself in a wart-like fashion, thus indicating that this peculiar local ulceration of the intestine was not of recent origin. The callous lips of this perforation were surrounded by a zone, or rather a reddened area, which reached out for about an inch around the ulcer, and, shading gradually into a lighter color, extending upwards to the pylorus, and for a less considerable distance below the ulceration."

Scattered cases were reported at autopsy, for example Dickenson (1) of London in 1859 reported a perforation of the anterior surface of the lesser curvature of the stomach. In 1880 (42) of London found multiple perforated ulcers in a women of 22 years who suffered for 30 hours with an attack before death after which autopsy was done. This was the period when the physicians knew that such a condition existed but were at loss to know how to recognize it in time so that treatment could be administered.

The first successful case was reported by Kreige (1) in 1882. In 1888 Sidney Jones (1) did the first operation for perforation of the duodenum, a diagnosis

of peritonitis being made, the perforation being discovered by autopsy. In 1894 Dean's (1) had a successful case, but the patient died two months later of intestinal obstruction. Lennander (44) and Glucksmann's case (1896) lived six months and died from second perforation. In the same year(1896) Dunn operated a case that made a permanent recovery. In America Finney and Weir (1897) were the first successful operators.

The first successful case treated by simple suture upon this continent was reported to the American Medical Association by Finney of Baltimore in 1900. It was performed by Atherton (2) of Fredericton, New Brunswick, Canada, on a young servant girl fifteen hours after perforation of the gastric ulcer. In 1899 Atherton(2) also reports a successful case 10 hours after perforation and his third successful case was in 1903 three and one-half hours after perforation.

M.Martens (49) of Berlin says: "The early fatal ending of perforated gastric ulcer is the reason why in former years cases seldom came under observation or treatment". From 1895 to 1897 he did not have a single case: 1898 to 1903 eleven cases of perforating gastric ulcers and two perforated duodenal ulcers; There were

eight gastric ulcers in 1907. The thousands of graves made from perforated gastric and duodenal ulcers, like those from appendicitis and perforation of the intestines in typhoid, mark the victims of an undeveloped age in this particular branch of the science and art of surgery".

It was not, however until 1900 that information on this lesion began to assume the form of systematized knowledge when Weir (31) presented to the American surgical association a comprehensive analysis of the cases thus far reported in the literature. In 1901 Moynihan (57) collected reports of fourty-nine cases that came to operation following perforation. The resulting mortality rate was 83.6 per cent. Mayo (51) in 1904 reported six of his own cases operated upon with two deaths resulting. From this time on, the reports comprise series of ever increasing numbers and the data given are constantly growing in comprehensive detail.

The remarks made by Atherton (2) in 1904 and others during this early time in regard to what makes for success in diagnosis and treatment of perforated peptic ulcers is early diagnosis and prompt resort to surgical treatment. Their chief reliance as to signs and symptoms

were based on the location and the severity of the pain, the board-like rigidity of the abdominal muscles and a previous history of dyspeptic symptoms.

From this time, up to the present, progress has been made causing a decrease in mortality rate. Variations in types of sutures and surgical technique is noted from time to time but the basis principles are still the same. The appearance and increased use of the X-ray, which came into use as a diagnostic procedure introduced and promoted by Singer and Vaughan(75) in 1925 has solved many problems in differential diagnosis of the subacute type of perforation. However, as time marches on and the progress of the art and science of medicine with it, the diagnosis and treatment of this calamity of the perforated peptic ulcer will also find better means of diagnosis and treatment than it has today.

INCIDENCE AND OCCURRENCE

FREQUENCY OF PERFORATION-According to various authors, the frequency of perforation in gastric ulcer is estimated from 28.5 per cent, (Frenwick) to 6.6 per cent. (Welch)(70) this seems rather high but statistics vary from 2-20 per cent. Perforation is much more frequent in duodenal ulcer than in gastric ulcer.

Deaver (21) says that about 15 per cent. of patients with gastric ulcer die from perforation, whereas in duodenal ulcer one-fourth will develop this complication. In 272 operations for duodenal ulcer Mayo (cited by Deaver) reports 66 for perforation, making over 24 per cent.

AGE AND SEX- Perforation is known to occur rarely in childhood and in extreme old age. It is found most commonly in the third, fourth and fifth decades of life. Gastric perforation is quite common in women under twenty-five. According to W.J.Mayo (50), chronic ulcer occurs in the stomach with equal frequency in the two sexes, but the duodenal, more than three-fpurths are in the male of the slender type.

White (83) in a very large series of cases in the Roosevelt Hospital of New York City, states that less than three per cent. of the perforations occurred in females and it was not uncommon to find a consecutive series of fifty or sixty cases made up, without exception , of males. Although duodenal ulcer of types is considerably more common in males, the very great difference in the frequency of perforation in the two sexes is difficult to explain, and few have attempted it. Finney (30) points to the anatomical fact that the

first part of the duodenum is more transverse in females, and therefore, more constantly bathed in alkaline bile. Others blame"The more active life",of the average man as predisposing to perforation, and also emphasize the excess use of alcohol as an apparently important factor. One is unable to become enthusiastic over any of these attempts at the explanation of a very puzzling fact.

SITE OF PERFORATION-Although ulcer is most frequently found on the posterior wall, perforations occur most frequently on the anterior wall. This is due to the fact that the posterior stomach wall is in contact with adjacent structures or viscera, as the spinal column, pancreas, spleen, lobe of the liver and the duodenum, so that adhesions take place between these tissues and the base of the ulcer, protecting the general peritoneal cavity from the perforation. In acute duodenal perforation the location is most frequently in the first portion of the intestine and on the anterior surface. In 20 per cent of reported cases the perforations were multiple and in a large per cent of perforated ulcers there were other ulcers in the stomach, though not perforated.

The size of a perforation may vary from one the size of a pin point to one several centimeters in

diameter.

ETIOLOGY

Very little has been offered as to the causes of perforation of a peptic ulcer. Whatever has been offered is purely theoretical. Corlette (17) of Australia offers some theoretically possible modes of causing perforation which is summarized as follows:

The physical stresses to which a gastric or duodenal ulcer may be exposed are reviewed. It is shown that by the operation of Boyle's law no increase of general intra-abdominal pressure can cause a strain in an ulcer, and perforation cannot occur from that cause. It is shown that muscular contraction of the walls of the stomach will increase the hydrostatic pressure within that organ without increasing the general intra-abdominal pressure, and if the difference in pressure passes the bursting strain of an ulcer, perforation must occur.

There are some other theoritically possible modes of causing perforation by pressure.

1. The weight of a column of gastric contents resting on the surface of an ulcer might conceivedly rupture it. Such an ulcer would only be on the greater curvature, at least in the erect position.

2. In collision accidents the gastric contents may be

thrown in the direction of an ulcer. The kenitic energy developed is measured as half the product of the mass into the square of the velocity. The "mass" however, is that of a column of unit sectional area in the line of force, not that of the whole gastric contents. If the kenitic energy is great enough it could cause the rupture of an ulcer.

3. In falls from a height, kenitic energy is liberated accordingly to a similar formula, but the mass factor depends on the vertical depth of a column of fluid immediately above the ulcer, not on the weight of the total gastric contents. If there is no food vertically above the ulcer, this mass factor does not exist. With sufficient kenitic energy liberated to do the work, an ulcer coyld be ruptured.

In the last three cases there is a possible additional factor which may have a qualifying influence, for more or less of the gastric contents might lie or be thrown up, or drop on the adjacent gastric walls so as to stretch it and thereby exert tension on the ulcer.

SIGNS AND SYMPTOMS

The first and most characteristic symptom of acute perforation is pain of an intense burning, agonizing nature. If the patient is seen in this condition

the picture is one that cannot be forgotten. There is usually a sudden outcry and the face becomes pinched and pallid and expressive of the most intense suffering. In a typical case the patient may awaken suddenly in the middle of the night and throw himself from the bed and may be found writhing upon the floor. He wishes to be left alone and does not want to be moved. The picture is that of shock and collapse--the extremities become cold and the face covered with cold perspiration. The respiration is costal and rapid, the pulse feeble and markedly increased in rate. Vomiting usually occurs but not persistant, as in intestinal obstruction. The pain is located in the upper abdomen (epigastric, right or left hypochondriac region). Rigidity of the upper abdominal muscles and tenderness upon pressure are important symptoms. If the extravasated material is extremely virulent and rapidly absorbed, the endotoxins set free by destruction of bacteria in the blood is so abundant that the patient is overwhelmed and dies in a few hours without developing rigidity of the abdominal muscles. If, however, the dose of endotoxins is not fatal, there is a peritoneal reaction within one or two hours and muscle rigidity and tenderness become pronounced. If the peritonitis is a chemical one, which is usually caused by the hydrochloric acid of the

stomach contents, there is the typical board-like rigidity present until the acid is neutralized or relieved of its irritating effect on the peritoneum.

Thirst is usually marked, but should not be gratified, as the fluid escapes through the perforation, spreading infection in the peritoneal cavity. If there is a rapid escape of gas in the abdominal cavity, liver dulness may disappear. An examination for absence of liver dulness, if made in the axillary line, is less misleading, according to Manges, (cited by Allaben, (1) as thereby the distended intestinal element is eliminated. Tympany is a late symptom indicative of intestinal paralysis due to spreading peritonitis or due to profound toxemia of the central nervous system. Immediately after the perforation some authorities claim that a fall of temperature occurs; within an hour or two however, there is a rise of temperature and an increased pulse rate. A marked peritonitis may be present without a corresponding rise of temperature.

Chill and a high temperature is indicative rather of cellular or lymphatic infection. Such a temperature is more common on chronic perforation with general sepsis. In these cases, as in all cases of peritoneal sepsis, the pulse rate is a more reliable sign than the temperature.

It is the subacute types wherein the greatest mistakes of diagnosis are made. The importance of recognition of this typw early is stressed by Singer and Vaughan (75) and the symptomatology as described by them is as follows:

The onset of the perforation is preceded in over half of the cases by periodic attacks of chronic ulcer distress usually for a period of one or more years. In the majority of the patients from one to several days prior to actual perforation,prodromal symptoms consisting of pains,vomiting,and epigastric tenderness are noted. The pain is more severe,of different character, and less responsive to alkalis than th ordinary ulcer distress. Vomiting is more persistant than in the usual case and often fails to relieve the pain, The patient is aware of a point of tenderness in the epigastrium,excited by even slight touch. In practically all instances, however,and not infrequently without even the slightest previous abdominal discomfort the onset is extremely abrupt and sudden. In fact up to this point it is practically identical in all respects to the onset of perforation in the typical type case except perhaps in intensity. The pain,which is located usually in the epigastrium, is violent in character and causes the patient

to double up and writhe about in agony. As a rule the pain is not quite so excruciating as in the classical c case, wherein the patient does not want to be moved. The prostration which accompanies the case is not so overwhelming or so striking in the "formes frustes"type; nevertheless the picture the patient presents is usually quite a dramatic one. If the abdomen is examined the first few hours or so after perforation, the same board-like rigidity and upper abdominal tenderness will be elicited as in the classical case.

Within a few hours after the occurrence of the perforation, that is, from two to ten Hours, the initial symptom may practically subside leaving the patient in a state of comparitive comfort. If the patient is seen during this quiescent period, the presence of an abdominal catastrophe may not be suspected. Frequently however, evidences of peritonitis appear and the subsequent course is dependent upon the amount and character of the escaped gastric contents. In those cases in which only a small quantity of relatively sterile duodenal fluid has escaped and merely a mild,.ocal peritoneal reaction has been excited, little discomfort may be felt and this but for a short period of time. Some of the patients with a trifling leakage of gastric contents, feel quite well within a few hours after onset and unles otherwise in-

structed will resume their normal activities. Extravasation of a considerable quantity of food and secretion from the stomach, however, will produce a more or less diffuse peritonitis with more pronounced symptoms. In these cases, pain of a rather severe nature associated with vomiting at times will persist as a rule for several days following perforation. The pain is generally felt in the epigastrium. much more frequently to the right than to the left of the midline.

In cases in which the pain was originally perceived in the upper abdomen, it frequently occurs that at the time the patient comes under observation the pain is felt exclusively in the right lower quadrant.This shifting of the pain sight is due to gravitation of the escaped fluid into the right iliac fossa. Unless sufficient inquiry is made no history of initial upper abdominal pain may be elicted. It is in this typw of ca case particularily that the mistaken diagnosis of appendicitis is often made. When the inflammation extends to the subphrenic region on the right side, the pain may be experienced in the upper right or lateral abdominal region. Hiccough may be the chief subjective manifestation of subphrenic localization. It tends to be troublesome and insistant and may constitute the patients per-

sisting complaint. The fever is usually subfebrile in degree, rarely reaching above 100 degrees F. except in the more severe cases. The leucocyte count ranges between 10,ooo and 20,000 with a relative increase of the polymorphonuclear leucocytes.

Physical examination during the stage of peritoneal reaction discloses the indications of inter-abdominal inflamation which as a rule are more or less diffuse. Tenderness is elicited in the upper abdominal region at the sight of, and adjacent to, the region of perforation and also frequently in the right lower quadrant. In those cases which are mistaken for appendicitis the erroe results from neglect to palpatate the entire abdomen, to percuss the liver dullness, and to listen to the peristaltic sounds, for in all instances, as far as our(Singer and Vaughan) experience goes, The tenderness when present over McBurney's point is not restricted to this one site but can be elicited in other portions of the abdomen also. The rigidity of this second stage is mild as compared with the board-like resistance encountered shortly after the acute onset. The muscular defense which is noted upon palpatation corresponds roughly to the distribution of the tenderness. Peristaltic sounds are usually much diminished. Tympamy is sel-

dom pronounced early but some slight or moderate distention usually appears by the second day. If the escaped fluid reaches the subphrenic space, a peritoneal rub may be heard over the hepatic region synchronous with respiration. This friction rub, together with hiccough may attract ones attention to the possibility of perforated ulcer. Obliteration of liver dulness is only rarely demonstrated in the "formes fruste" type cases presumably because leakage is only slight.

The symptoms of chronic perforations are those peculiar to perigastric and subphrenic abcesses. There is usually a history of long stomach trouble due to ulcer. There may be sharp pain in the upper abdomen, with tenderness and rigidity, but usually the perforation is so gradual that the peritoneal cavity becomes protected by extensive adhesions and it is impossible to estimate the time when perforation actually occurred. Sooner or later an abcess forms, sometimes manifested by a bulging tumor in the epigastric or hypochondriac regions. Accompanying this there are symptoms of general sepsis, chills, elevation of temperature, increase of pulse rate and rapid loss of flesh. An abcess may penetrate the chest cavity, similating empyema, causing sudden death or the lung and the contents of the abcess

be expectorated. According to Basnard, cited by Deaver (21), thoracic signs and symptoms are present in 56 of the 76 cases of subphrenic abcess studied by that author. There may be dulness on percussion with upward displacement of the lung and increased vocal resonance. The apex of the heart may be displaced upward.

Mayo Robson (70) speaks of the coin test used in a case as follows: "On placing the stethoscope over the eighth intercostal space, which region was dull on percussion, and on placing a coin over the stomach and percussing it with another coin, the sound was distinctly conveyed through the whole of the abcess cavity and through the stethoscope to the ear. The diagnosis was confirmed by operation. Gas may be present in the upper portion of the abcess, giving a tympanic sound between the duller sounds elicited by percussing the lung above and the lower portion of the abcess below. An abcess may ulcerate into the stomach and be vomited or into the retroperitoneal tissue and burrow down into the groin or iliac region. Mayo Robson (69) cites two cases of perigastric abcess, one of which was opened in the left groin and the other in the left iliac region. The X-ray should be employed for diagnosing an abcess where it is impossible to do so. The aspirator needle should never be used except when the surgeon is ready to immediately proceed

with the operation of draining the abcess.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

In acute perforations the picture is a typical and dramatic one and is easily recognized by history and the symptomatology as presented heretofore. However the mistakes in diagnosis are made because of the assumption that all perforations are as easily diagnosed as the acute perforations and due to this it is necessary that other means of diagnosis be presented. It is in this field that Singer and Vaughan (75) have put forth a great deal of effort in bringing to light methods and means of differentiating the borderline cases which come under the heading of the "formes frustes" type of perforations. These are the types that demand early diagnosis but due to the fact that they can similate so many other types of affections much time is wasted in differential diagnosis. I therefore wish to present an article written on The Use of X-ray in the Diagnosis of Acute Abdominal Affections by Singer (74). This article clearly presents a valuable diagnostic aid in differential diagnosis and the x-ray plates which are incorporated within this thesis illustrates the escaped air underneath the diaphragm which indicates perforation, no matter how small or how minute the symptoms. These are taken at intervals and demonstrate the absorption of air as the perforation

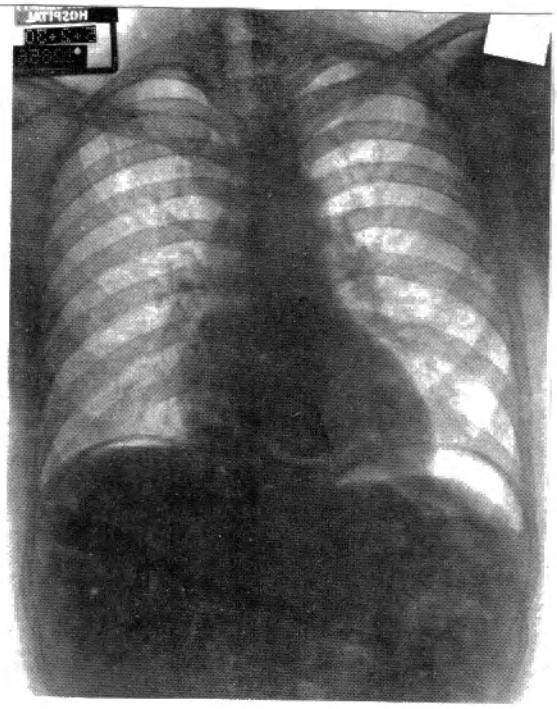

Fig. 1. Case 2. Roentgenogram taken 10 hours after acute perforation. The escaped air has collected beneath both domes of the diaphragm. A fluid level is seen below the gas bubble on the right. At operation the perforation was found to be sealed by fibrinous adhesions with the gall bladder.

Reproductions of X-ray films taken from Surg., Gynec.,and Obst., 54: 945, 1932 illustrating air under the diaghragm as seen by the X-ray after a perforation.

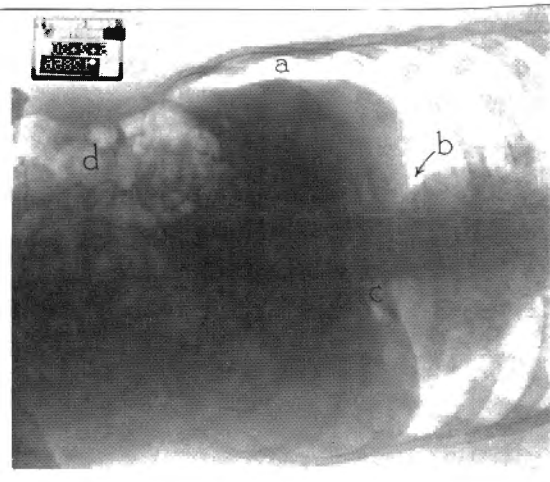

Fig. 2. Case 2. With the patient lying on the left side the free air in Figure 1 shifts to a position between the lower right ribs and the lateral border of the liver. Intraperitoneal air, *a*; air in the lung at the cardiohepatic angle, *b*; intragastric gas bubble, *c*; gas in ascending colon, *d*.

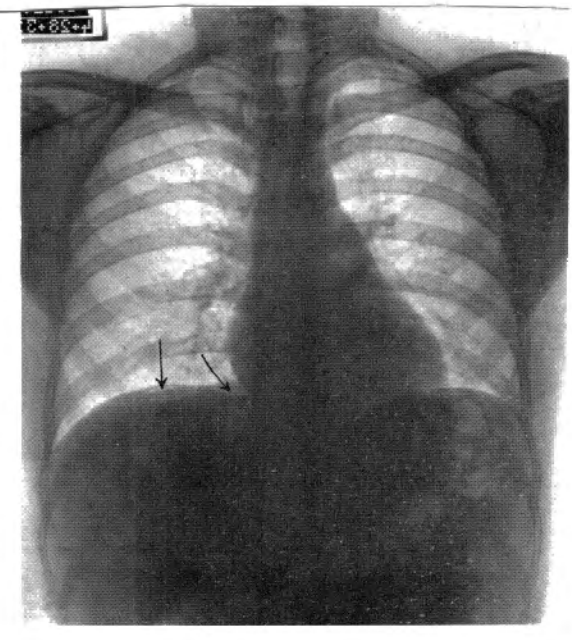

Fig. 3. Case 4. Roentgenogram taken 2½ days after perforation. The thin zone of intraperitoneal air (indicated by arrows) was the only clinical evidence at this time of a perforated viscus.

we anticipate misapplication of the knowledge that spontaneous recovery not infrequently occurs. With the more general use of the X-ray and more frequent resort to surgery in cases with

over

becomes walled off.

According to Singer (74):-Every physician engaged in the practice of medicine or surgery should be well versed in the diagnosis of abdominal affections. Whereas in the general run of frankly medical diseases of an acute nature, i.e.,typical coronary thrombosis, cerebral hemorrhage,etc., the outcome is more or less determined by natural processes of resistance and repair, in cases of acute abdominal pain the result is dependent in a large measure upon the diagnostic skill of the attending physician. Early recognition, coupled of course with timely intervention, is frequently the only means of preventing fatalities in acute abdominal catastrophies such as intestinal obstruction, perforated peptic ulcers, ruptured ectopic pregnancy, etc. A misdiagnosis which leads to a needless operation, in a nonsurgical condition with acute abdominal pain as a typical thrombosis,pneumonia,sepsis,etc., is likely to lead to serious consequences and may even cost the life of a patient.

Granting that the recognition of acute abdominal affections is very important and that the profession as a whole is lacking is diagnostic ability, based solely upon history and physical examination, the obvious need is for some laboratory method which can furnish more or less precise information regarding the presence or abs-

ence of surgical condition in a patient suffering from acute abdominal pain. Although far from being ideal or sufficient, the X-ray offers a method of precision which yields data serving to differentiate a medical from a surgical affection. As with chronic diseases of the abdomen roentgenology in acute disorders is of value only in certain affections, its technic has to be varied according to probable diagnosis and its disclosure requires interpretation in the light of the clinical history and physical examination. Again, as in the case of chronic maladies, the more skilled the clinician is in acute abdominal diseases the less need is found for the X-ray. However, it can be stated without fear of contradiction, that no one can afford to omit roentgenology entirely for it furnishes, at times, information which cannot be obtained by any other preoperative means.

There are two objections which have been raised against the employment of roentgenology both of which are based chiefly upon a lack of knowledge of the simplicity of the technic. The argument that valuable time is consumed by the X-ray study is not a valid one. It requirees no more than five minutes to complete a fluoroscopic examination which is all that is necessary in the general run of cases. There is no need to delay surgical intervention, for fluoroscopy can be done while the operating

room is being "set up". The second objection advanced by some is that the X-ray examination entails excessive manipulation and that the patient is often too ill to withstand the examination. This argument is without foundation since the patient in the course of the floroscopy is not required to move any more than for a thorough physical examination. The patient, on being transported on a hospital cart to the operating room, is taken while en route to the X-ray department. The cart is placed before a vertical screen and the patient merely turns on his left side without being required to do anything further. Often this suffices to establish the diagnosis and concludes the X-ray study. If warrented, the patient can be assisted to the sitting position while still on the cart and floroscopy performed with the chest and abdomen upright. If the patient is too weak to sit up even though supported, he can be lifted directly from the cart to the horizontal floroscope and remain lying on his back during this part of the examination. It can be taken for granted that if the above technic is observed any patient who is too ill to be floroscoped is too ill to be adequately examined physically and far too ill to withstand laparotomy.

It would prove of but little value although perhaps of great interest in enter into detail regarding the X-ray observations in a few selected cases. It will be far more pnofitable to discus the uses and limitations of the X-ray in each of several of the more important abdominal cases. No attempt will be made to include information regarding historic or physical phases of the various affections However, when required reference will be made to clinical data,for as previously stated, the X-ray interpretations must be made in the light of history and physical examinations. In the average case, a tentative ,clinical diagnosis is made which determines the type of floroscopy examination required. If no preliminary diagnostic impression is gained the patient is examined routinely in the following positions, left lateral(lying on the left side) upright (sitting), if feasible, and supine (lying on the horizontal table).

Perforated peptic ulcer- The X-ray finds its greatest field of usefulness in the diagnosis of perforated peptic ulcer. The presence of a pneumoperitoneum is pathognomonic of a ruptured air-comtaining viscus,or an infection with a gas producing organism. It has been determined for pnactical purposes,that in the presence

of a compatible history, the demonstration of free air in the peritoneal cavity is an indication of a perforated peptic ulcer. The air escapes from the gastroduodenal aperture almost simultaneously with the extravasation of the liquid material. The presence of air can therefore be detected extremely early in the course of the illness. Free air is not found in all cases of perforpeptic ulcer. In the early experience with roentgenology in ruptured ulcer Vaughan and Singer observed a pneumoperitoneum in approximately 85 per cent of all the cases examined. In recent years the percentage with free air has fallen to about 70 per cent, chiefly because of the more frequent recognition of perforations with trifling leakage, the so called "formes fruste" type of Singer and Vaughan.

The technic employed is quite simple. The patient is placed on his left side lying on the hospital cart. In this position the free air rises to collect between the lateral abdominal wall and the lateral surface of the liver and can be seen in the vertical fluoroscope as a sickle-shaped area of radiolucence. Furthermore, the left lateral position favors the escape of air from the gastro-duodenal aperture, for in this position the fluid content of the stomach gravitates toward the fun-

dus where the gas accumalates in the region of the pyloris in the region where over 90 per cent of the perforations occur. If, desired, the shifting of the air bubble can be demonstrated by assisting the patient to the sitting position while still on the cart. The air then collects beneath the domes of the diaphragm. Generally the air is found on the right side only but with large amounts of gas accumalations noted beneath both domes of the diaphragm. With the patient in the upright position even minute amounts of air, probably no more than a few cc., can be detected at a glance. In cases where a gas bubble is not demonstrable, where the diagnosis is in doubt and there is no urgent indication for laparatomy, the patient is allowed to remain in the left lateral position for an hour or two to permit air to enter the peritoneal cavity if it will. A second fluoroscopy examination is then undertaken and generally discloses the presence of free air.

The X-ray is of value in the detection of perforated ulcer not only in the earliest stages but also in the later periods of the disease. With progression of the peritonitis the X-ray discloses evidence of the beginning ileus. The presence of a considerable amount of free liquid in the abdominal cavity can also be

visualized through the means of a fluoroscope, however, of greatest importance in the diagnosis of cases seen for the first time late in the illness is the presence of encapsulated air associated often with fluid. In cases of perforations where the symptoms and signs of diffuse peritonitis have abated the diagnosis of an antecedent perforation may be difficult without the aid of a fluoroscope. The gas found in connection with exudate is absorbed very slowly and may be found quite a number of days after perforation. Any air containing subphrenic, subhepatic or other intra-abdominal abcess can be detected very ptomptly with the aid of the fluoroscope and reconsideration of the history will establish the etiology of the perforated peptic ulcers.

Singer (77) also presents points in differential diagnosis which are listed under separate headings as to regions and again subdivided into affections of that region which may be confused with perforated peptic ulcer.

ABDOMINAL AFFECTIONS

APPENDICITIS-Although the resemblence is not a particularly close one, appendicitis is quite frequently mistaken for perforated ulcer. Less frequently the latter is erroneously diagnoses when the lesion is appen-

diceal in nature. A ruptured ulcer may be overlooked and appendicitis diagnosed under three different sets of circumstances,(a)When the perforation is a classical one and the abdominal signs are diffuse,(b) When the rupture is of the "formes frustes"type and the physical signs are localized, and (c) When the perforative or predromal symptoms lead to the assumption that the onset of the illness was gradual.

With regard to group (a) the erroneous diagnosis of appendicitis is generally due to a lack of knowledge of the pathogenesis of appendiceal inflammation. It is most exceptional for peritonitis due to acute appendicitis to manifest itself within a few hours of onset. It usually requires twenty-four hours or more following onset of the primary lesion for appendicitis to reach the stage at which peritoneal complications occur. In other word, within the first twenty-four hours of symptoms, the physical signs are strictly confined to the right lower quadrant. Given a patient who is seen within a few hours of onset and who presents at that early stage, signs of diffuse peritonitis, appendicitis can for practical purposes be eliminated.

A history of onset with annihilating pain of per-

itoneal character is likewise incompatible with the diagnosis of appendicitis. A stated above, perforation of appendicitis requires usually twenty-four hours or more before the phlegmon of the wall leads to the stage of complications. There are instances in which the pain of onset of acute appendicitis is relatively severe but not to be compared with that of ruptured ulcer from the standpoint of intensity. Although the patient with acute appendicitis may assert that his pain of onset was quite violent, he will not behave in the destracted manner of the ulcer victim. The appendiceal patient will not, for instance, fall immediately to the ground, double over, writhe about in agony, yell and grown, or fear to straighten up and take a step. Nor will he arouse others and summon medical aid instantly. It can then be said, that if one takes into account the fact that in appendicitis , signs of diffuse peritonitis do not develope until relatively late and that the onset is not marked by overwhelming pain. There is little excuse for mistaking a ruptured ulcer in the early stages for acute appendicitis. It is well to know that in cases of appendicitis that begin with relatively severe pain the primary lesion is likely to develop rapidly and progress to the complicated stage.

It is in group (b) in which the rupture is of the "formes frustes" type and the physical signs are localized that errors in diagnosis are likely to creep in. If the patient is seen for the first time several hours or more after the onset, the average physician, failing to enter into detail, obtains the history that the patients pain was diffuse, cramp-like at the onset and later localized at the right side. The examiner concentrates upon or confines his attention to the right side of the lower quadrant of the abdomen and elicits a tenderness and muscular defense. The temperature is somewhat elevated, the pulse accelerated and white blood cells moderately increases. He naturally assumes the presence of acute appendicitis. If however, the examiner resorts to a minute by minute history, the following account will be obtained. The pain at onset, which was extremely sudden and violent, rapidly becomes generalized and only after a period of hours did it abate and become confined to the right side. This is in contradistinction to the character of pain in appendicitis which begins mildly and increases in severity.

If the patient is not seen until several hours after the onset the physical signs may resemble those

of appendicitis. However, if the examiner is not blinded by preconceived impressions, he will find that the tenderness and muscular defense are not confined to the right lower quadrant but are present evan to a greater degree in the upper right quadrant. Furthurmore tenderness is likely to be elicited in the cul de sac on rectal examination. The abdomen is usually very quiet in the forme fruste type perforation within a few hours of onset whereas in the early phase of appendicitis normal peristaltic activity is audible. The elicitation of the above physical signs of peritonitis involving more than one quadrant of the abdomen should lead the examiner to doubt the diagnosis of early appendicitis since within the first few hours of inflammation the process is restricted to the appendiceal wall and the peritoneum is practically unaffected.

As stated in the previous article dealing with symptoms of the pre-perforative stage, the predromal symptoms of rupture may last several hours or days. Those patients who fall into group (c) of the above classification may date the onset of their illness a day or more previous to the day of actual rupture. If, for instance,a patient with a picture of diffuse peritonitis states that his abdominal pain began mildly and increased mildly in intensity until two days or more

later when it become excruciating and diffuse an appendicitis complication by rupture and spread would be considered the most likely diagnosis. Careful attention to the history would be the only trustworthy means of detecting the presence of a perforated ulcer for an intra-peritoneal gas bubble may result also from a perforated appendix. History of previous chronic ulcer distress would suggest a complication of ulcer rather than appendix, although the occurance of appendicitis in an ulcer patient is not an infrequent coincidence. The prodromal symptoms of perforated ulcer may consist chiefly of an agravatedulcer distress located in the epigastrium associated at times with sharp, sticking pains passing upward into the chest and posterially into the back. These inaugural symptoms, although sometimes present for a day or more are likely to be intermittent and do not reach into the right lower quadrant at any time. In appendicitis, on the other hand, the pain of onset is likely to be diffuse, cramp-like in nature, more or less continuous, localizing in the region of McBurney's point where the pain changes in character. With the onset of perforation the pain is felt first in the epigastrium and spreads downward. With an appendiceal abcess the pain of rupture begins in the right lower

quadrant and extends upward.

The physical examination frequently fails to aid in the differentiation between perforated ulcer and rupture of an appendiceal abcess. In both, the picture the patient presents is that of diffuse peritonitis, which is characterized by a board-like rigidity. Board-like rigidity is found in appendicitis as well as in perforated peptic ulcer but is much more severe and more board-like in the perforated peptic ulcer. Frequently no actual mass is palpable in the appendiceal region although at times after morphine is administered sufficient relaxation is obtained to permit identification of an exquisitely tender swelling in the lowwer right quadrant. Where the origin of the peritonitis is appendiceal the maximum tenderness is in the lower abdomen. When the inflammation is due to an ulcer the upper quadrants are most tender. If the peritonitis is due to an ruptured appendiceal abcess a rapidly rising fever,at times preceded by a true rigor, may be observed. The presence of a pneumoperitoneum although occasionally encountered in perforated appendicitis is far from common in ruptured ulcers.

GALL BLADDER DISEASE- Second in importance to apendicitis in the consideration of the differential diagnosis of perforated ulcer is gallbladder disease. The mistake

is generally made of diagnosing an acute biliary affection when rupture of an ulcer takes place. As a rule the perforation is of the "formes fruste" type. Less frequently a ruptured gallbladder is confounded with an ulcer that is ruptured. The resemblence between an acute cholecystitis (with or without stones) and a duodenal perforation of the "formes fruste" type is due to the fact that in both there is usually a history of chronic dyspepsia followed by a sudden severe upper quadrant pain and accompanied by signs of a more or less local peritonitis. As a matter of fact the physical signs may be so nearly identical that unless a careful history is taken a distinction is hardly possible.

The following case will serve to illustrate the difficulties in diagnosis. A female patient, who was rather thin, enters the hospital with the complaint of severe pain in the upper right quadrant. There was an anticedent history of chronic dyspepsia. Acute cholecystitis engrafted upon a chronic cholecystitis was diagnosed independently by three attending surgeons. The symptoms subsided after two or three days and ten days after the onset of the acute illness, laparotomy was performed through a gallbladder incision. The operation disclosed the presence of a duodenal ulcer which had recently perforated and had become sealed off

by the gallbladder. Except for the localization of pericholecystitis secondary to the perforation , the gallbladder was free from disease. A review of the history indicated that the anamnesis was not properly evaluated and the diagnosis apparently was based on the preconceived idea that acute pain in the upper right quadrant in a woman meant biliary tract disease.

If one bears in mind certain facts, the distinction between acute cholecystitis and perforated ulcer should not offer great difficulty. Gallbladder disease affects chiefly women, generally of the obese type, whereas ulcer is at the present time quite uncommon in women. When a woman is the victim of an ulcer she is likely to be slender and animated. This is true in the case related above. When gallbladder disease occurs in a man he is likely to be stout. The dyspepsia of gallbladder disease is selective in type, and can be controlled more or less by restrictive diet. The gallbladder patient complains of heaviness and fullness shortly after eating and feels most uncomfortable when the stomach is full. The ulcer patient experiences gnawing, hunger distress when the stomach is empty and is free from discomfort as long as the stomach contains sufficient food. Ulcer pain is periodic and is not

closely related to the quality of food eaten at the previous meal.

The onset in acute cholecystitis is not as sudden as in perforated ulcers. It requires a period of one or several hours before the pain reaches its acme. In perforated ulcer the pain reaches its maximum intensity within a few seconds or at most a few minutes after the onset. In acute cholecystitis the pain is felt more or less exclusively in the upper abdomen, vis., the epigastrium and right upper quadrant, with radiation to the back and shoulders but not spreading downward to the umbilicus. In ruptured duodenal type ulcer, evan "forme fruste", the pain rapidly spreads to involve the right lower as well as the right upper quadrant. At no time does the pain of acute cholecystitis reach the intensity of perforated ulcer and as a result the gallbladder patient is likely to delay or omit summoning medical aid. A hypodermic of one-quarter grain of morphine is generally sufficient to narcotize the gallbladder patient but hardly sufficient to dull the pain of perforated ulcer. Whereas the ulcer patient remains immobile shortly after the onset of the severe pain the individual with biliary tract disease tend to toss about for an indefinite period or to walk in preference

to lying down.

If the patient with the "formes frustes" type rupture is seen early in the post-perforation stage the physical sign of peritonitis, being more or less diffuse, will afford a means of differentiation. However, if the patient, as it generally happens, is not seen until a few hours after perforation has occurred the examiner's attention will likely be drawn to the right upper quadrant where he will elicit tenderness and rigidity. If he will search further he will obtain evidence of peritoneal soiling, perhaps not as marked in the right lower quadrant as in the cul de sac. In gallbladder disease even with pericholecystitis the indications of peritonitis are more or less strictly localized. Jaundice, latent or manifest, is strong evidence in favor of biliary tract disturbance. The presence of a pneumoperitoneum would clarify the diagnosis and invite the necessity of attention to details but unfortunately it is particularly in these "formes frustes" perforations with leakage of only a few drops of liquid that a gas bubble is frequently lacking.

When inflammation of the gallbladder is complicated by perforation the simulation of ruptured ulcer is a very close one. A sudden annihilating upper abdominal pain spreading to the lower abdomen. Associated with the signs

of diffuse peritonitis is common to both affections. Although from the practical standpoint the differentiation is not very important since both requires an incision in the same general location, it is still desirable to make the distinction, if possible, preoperatively. Of particular value are the age, sex, habitus, antecedent chronic and preperforative history. The prodomes of a perforative gallbladder are relatively intense, being those of a severe acute cholecystitis, and therefore are far more violent than one of a perforated ulcer. In ruptured gallbladder, in spite of the tendency toward early localization of the exudates to the upper right quadrant, the patient remains quite ill. With perforated ulcer when the leakage consists of gastro-duodenal secreations with a relatively low bacterial content, early localization is accompanied only by mild evidence of a sepsis and comparitively well being of the patient. The presence of jaundice of a pneumoperitoneum are practically pathognomonic for gallbladder disease and ruptured ulcer respectively and when present are in themselves quite decisive.

PANCREATIC DISEASE- There are a number of acute abdominal diseases which, like ruptured ulcer, begin with intense pain and present early the signs of more

or less diffuse peritonitis. The differentiation preoperatively is often very difficult and at times impossible. However, a careful investigation of the available data not infrequently determines the most favorable type and location of abdominal incision, facilitates finding the lesion after entering the abdomen and occasionally causes the surgeon to refrain from operating when conservative treatment is indicated. An abdominal affection, which is one of the most difficult to differentiate from perforated ulcer, is acute pancreatic disease, fat necrosis, inflamation and apoplexy. The resemblence is a particularily close one since both lesions(perforated ulcer and acute pancreatic disease) the overwhelming pain begins characteristically in the upper abdomen and the maximum tenderness and rigidity are found in the same location.

The following facts generally serve to permit preoperative recognition of the underlying disease. Whereas ulcer affects, as a rule, thin males most frequently under forty years of age, acute pancreatic disease evinces a predilection for obese individuals, usually females, over forty. The chronic symptoms in the pancreatic cases are referable to gallbladder disease, i.e., selective dyspepsia or colics or both. In males

particularly the antecedent is that of excessive indulgence in alcoholic beverages and perhaps food. When pancreatic disease complicates an affection of the biliary tract, the onset of the symptoms and signs of diffuse peritonitis are frequently preceded by manifestations of gallstone colic or biliary tract inflammation or both. Prodomes of perforated ulcer only exceptionally reach any considerable degree of intensity. The pain of onset in acute pancreatic affections generally requires a few minutes or more before it reaches its acme and is therefore not quite as sudden in its appearance as in perforated ulcer. Furthermore the maximum pain of pancreatic origin is frequently felt posteriorly and especially in the left flank and loin, while in ulcer(except in rare cases of rupture into the lesser peritoneal sac) the pain is generally felt anteriorly.

The extreme "shock" and circulatory collapse (rapid pulse of low tension, capillary pulse and ashy cyanosis) generally considered characteristic of acute pancreatic disease occurs only in the vary serious types and of itself is of no great diagnostic value since the same picture is encountered in the cases of perforated ulcer with large holes and rapid profuse

leakage. In these fulminant cases of ruptured ulcer a pueumoperitoneum often associated with an intra-abdominal fluid level is seldom lacking and serves to make the diagnosis clear. The scaphoid, board-like abdomen of ruptured ulcer is usually not encountered in acute pancreatic disease due in part to the obesity of the person and in part to the retroperitoneal location of the pancreas and the escaped secretion. The tenderness in the acute pancreatic disease is much more marked in the upper than the lower abdomen and requires deeper palpation than in the ulcer. What is particularly significant with regard to the tenderness is its presence posteriorly especially on the left side extending into the costovertebral angle. In perforated ulcer the entire abdomen is more or less uniformly tender anteriorly and the loins are generally spared. Although German writers speak of the absense of pronounced meteorism in acute pancreatic disease, in Singer's (77) experience distension of considerable degree appears very much earlier than in peritonitis of ulcer origin. The presence of jaundice and melliturla are greatly in favor of the diagnosis of pancreatic disease but occur only in a small percentage of cases. The same is to be said of acute anemia due to severe pancreatic apoplexy. The special knowledge and the time required for ferment studies on the blood,

urine, etc. render unavailable to the average clinician laboratory data of this nature. Dilation of the pupils following instillation of adrenalin in the eyes (Lowi's) phenomenon which occurs in some cases of acute pancreatic diseases may be of some value when positive.

INTESTINAL OBSTRUCTION-Intestinal obstruction not infrequently simulates perforated ulcer and visa versa. The resemblence is especially close in connection with that type of obstruction which is accompanied by abrupt, gross interference with the circulation. The sudden, excruciating pain and the ensuing manifestations of "shock" which characterize the onset in some cases of strangulated bowel, suggest an acute perforation. When the strangulation is an internal one, difficulty in distinguishing it from ruptured ulcer may be experienced. If the strangulation is an external one, as for instance in inguinal hernia, the presence of a tender mass generally leads to the correct diagnosis. However, it is well to remember that a tender external mass in the region of hernial opening does not necessarily mean that the extruded bowel is deprived of its blood supply. It not infrequently happens in perforate ulcer that due to the increased abdominal pressure a loop of bowel is forced through an old hernial opening and with it irritating gastroduodenal content and exudate. If one is not

critical he may confound cause and effect and operate for strangulated hernia with secondary peritonitis when the cause of the symptoms is perforated ulcer. The error can be avoided if one bears in mind both possibilities and attempts to reduce the hernia. Although the hernial mass is tender, reduction can be accomplished with relatively little pressure particularly if a preliminary hypodermic or morphine is administered. Other facts are of course helpful in the diagnosis. For instance, since diffuse peritonitis is an extremely late complication of strangulated hernia, the presence of signs of widespread peritoneal inflammation immediately following the onset of pain even in the presence of a hernia should lead one to suspect a primary perforation.

In gross strangulation the violent pain of onset described as boring in nature tend to remain localized in the site of involvement and to diminish in intensity as gangrene supervenes. Later a diffuse, paroxysmal, cramp-like pain develops due to attempts of the proximal bowel to overcome the obstruction. In perforated ulcer, on the other hand, the generalization of pain occurs within the first few minutes. Although in both affections the patient doubles up with the severe initial pain, the peritoneal phenomena, i. e., sharp cut pain felt in deep breathing, change of position, especially straightening

of the body jarring, etc. are not experienced by the person in early strangulation. Vomiting is not infrequently absent in the first day or so of a perforated ulcer and rarely occurs repeatedly unless a patient persists to ingest nauseating cathartics as epsom salts, castor oil, etc. After paralytic ileus has advanced, the usual type of overflow of feculent material occurs. In strangulating ileus, however, vomiting is frequent from the inception of the illness and early becomes stercoraceous in character. Although constipation is a rule in both affections, complete obstipation favors the diagosis of obstruction. However, it is necessary to keep in mind that a watery diarrhea, at times associated with an admixture of blood, occasionally occurs in strangulation.

If the patient is seen early, physical examination will reveal that ileus, tenderness and resistence are absent except perhaps over the site of the obstruction. In favorable cases one may pulpate a firm mass which represents the loop of bowel involved in a state of spastic contraction. In a short while, however, the constricted loop becomes paretic and one may succeed in demonstrating a markedly distended loop of bowel which is fixed and devoid of visable or audible peristalsis (von Wakl's sign). It is possible in

favorable cases to visualize this gas filled loop with the roentgen ray. The outspoken picture of diffuse peritonitis in early ruptured ulcer (including the "formes frustes" type) with its board-like rigidity, widespread tenderness and scaphoid abdomen constitutes a striking contrast to the picture of early strangulation ileus. If the patient is seen for the first time several hours or more after the onset, evidence of secondary peritonitis localized to the region of strangulation may be obtained. Due to the obstruction, the paroximal bowel distends rather rapidly and tympanites unassociated with increased peristalsis appears. In high intestinal obstruction the evidence of dehydration and toxemia are likely to be more apparent than in ruptured ulcer unless the perforation is a large one. Up to and through the stage of local peritonitis the physical examination permits a satisfactory differentiation. However, with the spread of the secondary peritonitis to other parts of the abdomen, which occurs late in the course of strangulation ileus occurs. The picture acquires a close resemblence to that of perforated ulcer and the differentiation must be made chiefly on the basis of the history if a pneumoperitoneum is lacking.

There are two types of perforated ulcer which stimulate intestinal obstruction of the mechanical type. One

is rather common, the other is quite rare. The common type is the perforation associated with only moderate leakage and subsequent spontaneous sealing. The rare type is characterized by the selective escape of air into the peritoneal cavity with each respiration until eventually the abdomen assumes the shape of a barrel and the tenseness of a drum. In the form with moderate leakage followed by spontaneous closure of the hole, it is not unusual for the patient to present himself for examination one or more days after the onset of his perforation. At this time the pain is not particularly severe and the obtrusive sign is the abdominal distention. If a cursory physical examination is made, one is struck by the amount of distention and in some cases an increase in audible peristalisis. The ileus is due chiefly to the rather low grade peritonitis which tends to subside and leave in its wake fibrinous adhesions. These in turn cause multiple kinks and angulations with narrowing of the intestinal lumen leading to hyperistalsis. The obstruction is not of surgical significance and disappears spontaneously. If the perforation is securely closed, operation is not indicated and in fact may produce distinct harm by activating and receding peritonitis. It is therefore of more than theoritical importance to distinguish the pseudo-obstruction of peritonitis of

ulcer origin from ileus due to mechanical causes.

In the case of distention following perforation ulcer with spontaneous closure, a careful history is generally sufficient to warn one against making the incorrect diagnosis of mechanical obstruction. In ruptured ulcer the pain, although stated to be cramp-like because it causes its victims to double up, is not intermittent, rhythmic and colocky as it is in intestinal obstruction. The sharp knife-like pain of peritonitis being agrivated by the slightest motion causes the patient to lie as quietly as possible whereas in mechanical obstruction the patient rolls and tosses about for the whole duration of the cramp. Between paroxusms the patient is conversable and relatively comfortable whereas the ulcer is more or less constantly in agony. In addition to the difference to the behavior of the pain, the character of the vomiting is of considerable importance. In ulcer, as stated above, emesis is not a prominent symptom whereas in obstruction it is a cardinal manifestation. Early in mechical ileus vomiting is likely to be frequent, occuring at the height of each painful paroxysm. Later in the course, when the obstruction becomes more or less complete a constant overflow of feculent fluid occurs.

The physical examination discloses the fact that the patient is not as ill as the history of the first twenty-four hours of the disease would indicate if the mechanical obstruction were present. The occurance of fever, of moderate diffuse tenderness and rigidity if looked for will be observed in the presence of a perforation but will be lacking in the mechanical obstruction. In ulcer perforation an enema will often yield results whereas gastris aspiration will not in contrast to mechanical ileus. The roentgen ray is often of paramount importance if a pneumoperitoneum is present. In the absence of free air, the roentgen ray may still serve a useful purpose in distinguishing a paralytic from a mechanical ileus.

The type of perforation in which there is a selective escape of air into the peritoneum cavity with each respiration has been referred to in the literature as gas peritonitis and by German writers as Span-nungspeumoperiteum. On account of the mechanism involved which is anologous to that seen in the chest the author has suggested the term "valvular pneumoperitoneum".(Singer) The amount of distention involved which occurs in valvular pneumopertoneum is at times so extreme that circulatory embarrassment develops, due chiefly to upward displacement of the diaphragm. It

is the marked abdominal distention which leads the patient to seek medical aid and which suggests to the examiner the presence of typanitis and obstruction. However, in these cases of valvular pneumperitoneum of ulcer origin there is a history of severe pain at the onset due to an initial extravasation of fluid which accompanies the first puff of air. The original pain rapidly subsides and the patient is relatively comfortable until the distention becomes marked, when a feeling of fullness is experienced. In contrast to intestional obstruction, there are no cramps, vomiting or obstipation. No audible peristalisis is heard. A glance through the fluoroscopic screen shows the air to be free in the abdominal cavity and not within the intestional coils. As a matter of fact, at operation the loops of the intestinal coils. As a matter of fact, at operation the loops of the intestines are seen to be empty and crowded within a small space against the posterior abdominal wall.

INTESTIØNAL INFARCTION(MESENTERIC OCCLUSION). The greatest difficulty can be experienced in attempting to differentiate intestional infarction, especially due to the occlusion of the mesenteric vessels, from perforated ulcer. From the standpoint of symptoms and physical signs, mesentaric vascular occlusion bears a

close resemblence to strangulation ileus. A great deal of what has been brought out in previous paragraphs concerning strangulation applies to intestional infarction. The onset as in perforated ulcer occurs with extreme suddeness and is characterized by severe, continuous boring pain associated with general manifestations of "shock" or collapse. If the patient is seen within a few hours of onset the differentiation from perforated ulcer can be made more easily than later. As in strangulation the board-like rigidity and diffuse tenderness of perforated ulcer are not present early in mesenteric vascular occlusion. A mass or resistance may be felt, corresponding to the sight of infarction, accompanied by local tenderness. Physical signs of diffuse abdominal inflammation do not appear until later when secondary peritonitis spreads from the infarcted bowel. Of great significance, when present, is the occurrence of thin bloody diarrhea or of gross blood in the content of the lower bowel providing the stool is watery rather than tarry in consistancy. Although it is generally true that bleeding ulcers seldom perforate and visa versa, nevertheless in the presence of a saddle ulcer, it is not rare for the anterior half to perforate and the posterior to erode a blood vessel. Under these circumstances the stool consists of almost

pure blood, rather than a watery fluid tinged with blood. The same difference applies to the type of hematemesia seen in the two affections under discussion.

Of particular value for the diagnosis of infarction, especially in those cases seen for the first time rather late in the course of the illness, is the demonstration of an etiologic factor. The occurrence of thrombosis in the mesenteric artery or its division is likely to occur in the presence of arteriosclerosis or hypertension or both. Embolism of the artery is prone to result from endocarditis and especially from bland thrombi derived from the left auricle in the presence of fibrillation. Some have been most successful in diagnosing mesenteric occlusion when a patient with known auricular fibrillation develops sudden violent abdominal pain. Two such patients were being treated for the cardiac arhythmia when the abdominal catrostrophe occured. The predisposition toward venous occlusion is caused by stasis, especially portal, and by supperative disease in the structures draining into the mesenteric vein, the appendix and intestines in particular.

RUPTURE OF INTESTINAL ULCERS. In addition to the more common affections which were considered above there are a number of infrequent abdominal lesions that produce a clinical picture resembling that of ruptured peptic

ulcers. These lesions merit but brief discussion. Ulcers which are due to causes other than peptic corrosion and which are generally located in the intestine may suddenly rupture and result in manifestations indistinguishable from those of perforated peptic ulcers. When the rupture occurs in a patient who was previously well or apparently so, the error can hardly be avoided. This is true of solitary tuberculous ulcer which often remains silent to the perforation and ruptured ulcers of the ileum in an ambulatory typhoid patient. In typhoid however, a a history of some vague systemic disorder is generally forthcoming. The same is true of colonic perforation as in chronic ulcerative colitis. Other specific types of ulcers which may perforate include Hodgkin's lymphogranulomatosis and syphilis both of extreme rare occurrence.

PERFORATION OF GASTRO-INTESTINAL CARCINOMA AND OF DIVERTICULUM OF SIGMOID- It is often hard to distinguish perforation of a benign ulcer from perforation of a carcinomatous ulcer of the stomach. In both instances the ulcer is due to the peptic activity of the gastric juices. Unless the antecedent history is more suggestive of malignancy than benign ulceration, the error is almost in escapable. Perforation of carcinoma of the gastro-intestional tract other than the malignant ulcer

of the stomach is generally associated with localized peritonitis and subsequent abcess formation. The same is true of diverticulitis . It is the rupture of the abcess associated with the carcinoma or diverticulum which often leads to an erroneous diagnosis of perforated peptic ulcer rather than perforation of the original lesion per se. Since the abcess frequently communicates with the lumen of the bowel, rupture of abcess is often followed by pneumoperitoneum. In spite of the presence of free air some have succeeded in diagnosing correctly the presence of a ruptured abcess rather than a perforated peptic ulcer in cases of carcinoma of the bowel and diverticulitis of the sigmoid. In ruptured abcess with sudden and rather intensive spread, the occurrence of initial pain of onset may be preceded or followed by a severe chill. A true rigor is not encountered at the onset of a perforated peptic ulcer. The general signs are those of a infection with early rise in temperature and acceleration of pulse rate. In ruptured ulcer the pulse tends to remain slow and the temperature to rise very gradually except in fulminant cases in which the pulse becomes quite rapid but the temperature remains normal or falls. In ruptured abcess a mass can frequently be identified and around this area the maximum degree of ten-

derness is elicited. The antecedent of bowel disturbance or pain related to peristaltic activity can be obtained.

AFFECTION OF THE PELVIC ORGANS-Several diseases of the pelvic organs in women begin with sudden, violent cutting pain referred to the epigastrium with radiation to the chest and shoulders. Under such circumstances a perforated ulcer could easily be misdiagnosed. Of particular value in the differentiation is the fact that according to recent statics, perforated ulcer is quite uncommon in the female sex in contrast to the older statics. Given a girl or woman of child-bearing age who develops, out of a clear sky, a picture of perforative peritonitis unassociated with a pneumonoperitoneum the probabilities based on statistics alone are overwhelmingly in favor of pelvis affection. In dealing with a patient in whom the question of pregnancy or pelvic disease is involved it is well not to give too much credence to the history. Unless one is skeptical, he may be led far astray particularly in regard to contact with unmarried girls. A history, which is trustworthy and can be relied on, is of great value.

RUPTURED ECTOPIC PREGNANCY- In ruptured extrauterine pregnancy, a history may be obtained of recent contact followed by menstrual irregularity, especially spotting. Mild cramps in the lower abdomen and back,

sharp stabbing pain in the pelvic region radiating into the thighs or merely of uneasiness in the lower quadrants may usher in the overwhelming pain of onset. The initial pain of ruptured ectopic pregnancy is characterized, as in perforated ulcer, by its suddeness and violence. It is generally lancinating in character and followed by collapse and frequently actual syncope. Fainting in perforated ulcer even in the presence of large apertures is very uncommon. The pain of ruptured ectopic pregnancy even when felt throughout the abdomen generally passes from below upward in contradistinction to perforated peptic ulcer which generally begins in the epigastrium and diffuses from this point. The reverse not infrequently happens, however.

Physical examination in the gravid patient may disclose evidences of anemia if sufficient blood has escaped. Signs of pregnancy are generally not apparent. Even early the pulse tends to be rapid but the temperature is but little if at all raised. The pulse tends to often be remarkably slow in the ordinary case of perforated ulcer. Of particular significance is the noteworthy absence of rigidity in ruptured ectopic pregnancy, the abdomen when palpatated very gently being quite soft even in relatively thin women. Deep pressure on the other hand elicits exquisite tenderness

particularly in the lower half of the abdomen. The striking degree of tenderness in the absence of muscular rigidity is in direct contrast with that observed in perforated ulcer, in which, due to the board-like abdomen, one is practically prevented from elicitating deep tenderness. Pelvic examination may disclose a unilateral mass or merely resistance in both fornices due to jelly like lumps of clotted blood. Pelvic tenderness, although present at times in perforated ulcer due to gravitation extravasated gastroduodenal fluid, is not as constant or as marked as it is in ruptured ectopic pregnancy.

The absence fluoroscopically of a pneumoperitoneum and presence of an opacity in the lower abdomen which assumes a level and which can be made to shift with change of the patient from the left to the right lateral position is strong presumptive evidence for ectopic pregnancy.

PELVIC PERITONITIS- Acute pelvic peritonitis secondary to gonorrheal salpingitis often begins, as does perforated ulcer, with sudden, severe peritonitic pain rapidly becoming diffuse due chiefly to the leakage of the infectuous material from the patient's fallopian tubes. If the pain is referred to the upper abdomen or chest, difficulty in diagnosis may follow, especially

if a vaginal examination is omitted. The history, the presence of a discharge, smears, etc., will serve to establish the correct diagnosis.

AFFECTIONS OF THE THORAC STRUCTURE- Many of the abdominal and pelvic diseases which simulate perforated peptic ulcer and which were discussed in the previous paragraphs were surgical in nature and require urgent operative intervention. A correct preoperative diagnosis, although not exactly vital, is very desirable, even in these surgical cases as it often eliminates unnecessary incisions and facilitates the work of the surgeon. However, to mistake an intrathoracic lesion for an intra-abdominal one or visa versa generally proves quite significant and at times fatal. Of the various diseases of the chest organs that similate perforated peptic ulcer, the one which has attracted most interest recently, and reservedly so, is coronary thrombosis.

The onset in coronary occlusion with sudden overwhelming pain referred to the tip of the xiphoid or even lower, followed by symptoms of "shock" and vasomotor collapse and associated with abdominal rigidity are likely to lead the unwary into assuming the presence of ruptured ulcer. A previous history of post-prandial distress in a patient with coronary disease is likely to add furthur to the diagnostic difficulties. However,

if inquiry is made, it will be found that the pain which occurred following meals was present if the patient was only physically active, for instance, walking shortly after eating. In other words, it required the combination of digestive and physical activity to induce the coronary pain. Furthermore, although the discomfort is frequently described as burning, it is generally subxiphoid rather than epigastric and is relieved by rest and not by foods or alkalies.

The pain of coronary thrombosis is more likely to be constricting, burning or boring rather than knife-like and cutting as in ruptured ulcers. The ulcer patient doubles over with the pain of onset and often moves about in an attempt to obtain relief. The coronary patient is generally rendered prostrate at the first moment of onset and is unable to move because of collapse. The ulcer patient after learning the futility of seeking relief in various positions remains immobile for fear of aggravating the pain. The coronary patient lies motionless chiefly because of the great prostration. In perforated ulcer the epigastric pain rapidly becomes diffuse throughout the abdomen and frequently radiates to one or both shoulders. In coronary thrombosis the pain tends to extend from the epigastrium upward to involve the chest and to radiate down or into the left

upper extremity. Mental anguish and severe dyspnea are more characteristic of coronary disease. Vomiting is more frequent in ruptured ulcer.

The physical examination in the patient with coronary disease will show the pulse and respiratory rates to be disproportionately rapid and the blood pressure to have fallen more than the abdominal change warrents. In a ruptured ulcer with a board-like abdomen the pulse, except in the fulminating cases, remains surprisingly slow (due presumably to vagal stimulation) and full. The respirations in coronary thrombosis are abdominal in type, hurried but not painful as a rule. In perforated ulcer the respirations are chiefly thoracic and the breathing shallow due to pain. Relief of the pain through morphine eliminates the dyspnea of early perforated ulcers. Fever is generally absent for a longer period after coronary occulsion than after perforation. The presence of fever after the first twelve hours or so favors the diagnosis of ulcers. With reference to the heart, the presence of a pericardial rub is of no great diagnostic value. Its absence, however, does not militate against the assumption of coronary thrombosis. Cardiac enlargement, feeble heart tones, anarhythmia and a systolic murmur due to a relative mitral insufficiency are more frequently found in

coronary occlusion than in perforated ulcer. Signs of pulmonary congestion or edema are more characteristic of coronary disease.

Although the abdomen in coronary thrombosis may be held rigidly, the muscular resistance is not board-like or constant as on perforated ulcer but fluctuates from time to time. If the palpating hand is held over the rectus muscles continually during several respiratory cycles, very noticeable degree of relaxation is experienced at the end of each expiration. The abdominal tenderness in coronary thrombosis is generally superficial and often associated with hyperasthesia in the precordial area. The abdominal reflexes are preserved or even increases in coronary thrombosis whereas in the presence of diffuse peritonitis they are not elicitable. In ruptured ulcer the abdomen is much more quiet than in coronary disease. Peristalsis in the later affection remaining active. An electrocardiagram, if available, is of considerable aid in confirming the diagnosis of coronary thrombosis and roentgen-ray examination in disclosing the presence of a pneumoperitoneum makes the diagnosis apparent at a glance. Suggestive fluoroscope evidence for a coronary thrombosis includes a rapid,weak contractions of the heart muscle resembling a quivering,

enlargement of the cardiac shadows and an increase in the lung markings due to passive congestion.

SUDDEN DECOMPENSATION- Of the other coronary affections which may resemble perforation of an ulcer, sudden decompensation leading to an acute hepatic engorgement, acute fibrinous pericarditis and interpericardial rupture of an aortic aneurysm may be mentioned. In sudden decompensation, the acute abdominal pain,due presumably to stretching of the capsule of the liver,may lead the examiner to focus all his attention below the diaphragm. In a case of this type there was tenderness and firm resistance in the upper abdomen, undoubtedly due to the engorged liver but originally interpreted as the tenderness and rigidity of perforated ulcer. The error can readily be avoided if the heart is actually examined. A surgeon operated upon a young man with acute severe abdominal pain expecting to find a perforated ulcer. Much to the suprise and consternation of the operator the peritoneum was pale , thickened and gelatinous but free from any sign of onflammation. There was no indication of an ulcer. The following day the doctor was asked to see the patient who was convalescing from the operation and to explain the peculiar character of the peritoneum. A routine physical examination revealed

the presence of an old mitral stenosis and a brief history disclosed the fact that the patient had engaged in violent exercise(basket ball)just prior to the onset of the pain. The peritoneum was in all prebability the seat of edema due to sudden cardiac decompensation.

ACUTE PERICARITIS-Acute fibrinous pericarditis frequently gives rise to infradiaphragmatic pain of a sharp, cutting character to which event it tends to suggest primary abdominal disease. The error is more likely to occur in children, appendicitis being generally assumed. The pain may precede the appearance of a demonstrable rub which may be discovered some time after laparotomy has been performed. However, if careful attention is paid to the history, which generally indicates a rheumatic infection, and to the lack of clear cut abdominal signs,the mistake of diagnosing perforated ulcer will be avoided. Fluoroscopic examination will be of value even if a pneumoperitoneum is absent, for in questionable cases it sounds a note of caution in suggesting furthur diagnostic studies before undertaking operation. Early the amount of fluid is too limited to produce a recognizable change in the contour of the heart shadow but later in the course of the illness the fluoroscope may be of distinct value.

INTRAPERICARDIAL ANEURYSMAL RUPTURE- Rupture of a supravalvular aortic aneurysm, with hemorrhage into the pericardinal sac may, like coronary thrombosis, announce its occurance with abdominal pain. In a patient who sufferred two distinct attacts of pain and was thought to be sufferring from a perforated ulcer with intermittent leakage, the fluoroscope disclosed an enlarged, somewhat pearshaped heart with extremely week pulsations. Operation was not performed. The autopsy revealed old clots as well as recently clotted and fluid blood. The age of the older clots corresponded with the time which elapsed between the patients first attack of pain and his death. Rupture of an aneurysm of the heart gives a picture practically identical with that of supervalvular aortic rupture. The antecedent of coronary thrombosis in the one and of lues in the other may serve to aid in the differentiation of the two lesions.

LOBAR PNEUMONIA- The simulation of the acute abdomen by pulmonary disease is of constant concern to the abdominal diagnostician. Lobar pneumonia, acute fibrinous pleuritis, especially diaphragmatic, infarction of the lower lobe of the lung and acute pneumothorax enter into consideration. Of these diseases, lobar pneumonia is more important as it quite frequent-

ly begins with a sharp pain referred to the abdomen, extreme prostration, and often circulatory collapse. Cough may be present for many hours following onset. The following facts are particularly worthy of consideration in the differentiation of croupous pneumonia from ruptured peptic ulcers. In pneumonia the antecedent history with reference to the digestive tract is negative. A chill, when it occurs at the onset of an undiagnosed illness, is not ascribable to perforated peptic ulcers, although it may be due to rupture of a perforated intra-abdominal abcess. The pain in pneumonia is not as overwhelming at the onset as in ruptured ulcers and does not cause the patient to double up. The pneumonia pain is related chiefly to respiration rather than to motion. Vomiting is most unusual at the onset of pulmonary affections.

The appearance of a patient with pneumonia is quite distinctive. The malor, flush, anxious expression, inspiratory dilation of the alae nasi, The expiratory grunt, the herpetic lesions, etc., generally permitting a correct diagnosis even in the absence of any physical evidence of pulmonary involvement or cough. The respirations in pneumonia are more rapid and more painful than in ruptured ulcers. In pneumomia the

breathing is of abdominal type and the involved half of the chest seems to lag. The temperature in pneumonia rises much more in degree and in rapidity than in perforated ulcers,as does also the white blood count. In the first twelve hours or so following onset the physical signs of pulmonary involvement are of questionable dependability unless one is virtuose in physical diagnosis. A decrease in breath sounds over the involved lobe is generally the only signs elicitable. In central pneumonia physical signs are generally delayed too long to be of value for differential diagnosis. Tenderness over the abdomen is generally superficial and is often less marked than the tenderness over the chest which is involved. At the onset of pulmonary affections the muscular defense, as in coronary thrombosis, is a pseudorigidity since with respiration the recti relax considerably. Morphine of sufficient quantity to moderate the pain results in subsidence of the rigidity of the abdominal wall. The abdominal reflexes and audible peristalsis are preserved in pneumonia whereas in peritonitis they tend to be abolished. The roentgen ray is often of incalcuable aid in differentiating the two affections under considerations, particularily when a central pneumonia or a pneumo peritoneum is demonstable.

OTHER PULMONARY DISEASES- Many statements made in connection with the discussion on the difference between lobar pneumonia and perforated ulcer can be applied to other pulmonary diseases which simulate an acute abdominal emergency. Here again the roentgen ray renders valuable service. In acute pleurisy a lag of the diaphragm on the affected side and a clouding of the costophrenic angle due to an accumulation of exudate are generally observed quite early. It is well to bear in mind, however, that later in the course of ruptured ulcer the same roentgen signs are detectable due to subphrenic inflammation and extention through the diaphragm to the chest. Pulmonary infarction likewise lead early to changes in the roentgen rays which are helpful in corroborating a clinical diagnosis. Sudden colapse of a lung due to spontaneous pneumothorax may be mistaken for ruptured ulcer particularily when the amount of escaped air is limited. The fluoroscope examination which establishes the diagnosis at a glance is particularily helpful to those whose skill in physical diagnosis is limited.

MEDIASTINUM- Beside the thoric affections involving the heart and lungs there are several others which simulate ruptured peptic ulcers and which are located

in the mediastinum. Of these ruptured or dissecting aneurysm of the thorac aorta, ruptured esophagus(which may occur spontaneously, particularily in alcoholics), acute suppurative mediastinitis are the best known. Strangulation in a diaphragmatic hernia has also been observed. The fluoroscope is almost indispencible if the above affections are to be suspected or diagnoses made preoperatively.

AFFECTIONS OF THE NERVOUS SYSTEM-Tabetic Crisis. Of the disease of the nervous system which may simulate ruptured peptic ulcer, The gastric crises of tabes merit most consideration. The error should seldom occur, however, as the simulation is only a superlative one. With reference to the abdominal symptoms in particular, a history of freedom from digestive disturbances is generally obtained in tabes. If previous attacts occurred, they were characterized by sudden onset and just as sudden spontaneous cessation with perfect health in between. The onset of pain in a tabetic crises is not annihilating as in perforated ulcers and does not cause the patient to double over. The patient with tabes instead of assuming a position of rigid inmobility frequently tosses about like a mad man and goes through a series of contortions and gyrations which stretch his abdominal

muscles rather than contract them. Emesis when present is persistant and often more distressing than the pain. The patient really retches more than vomits, the vomitus being small in amount and bilious in character. Upon examination the abdomen in entirely devoid of any tenderness or rigidity. One is usually able to press the anterior against the posterior wall without provoking any complaint on the part of the patient. The tabetic aspect, including the history of syphilis, of treatment thereof, the pupillary and other neurological signs, the occurrence of lightening pains, bladder disturbances, etc., should not be overlooked. However, ruptured peptic ulcer is not at all uncommon in tabetics and the differential diagnosis must generally be made on the basis of the abdominal symptoms and signs. Noteworthy, also, is the fact that a patient in whom repeated operation failed to relieve attacks of severe pain and vomiting, tabetic crises should be considered before a perforated peritonitis.

AFFECTIONS OF THE RETROPERITONEAL STRUCTURES-

RENAL DISEASE-Of the structures located retroperitoneally that are capable of producing acute severe abdominal pain, the kidneys occupy first place. Although it would seem that renal disease and ruptured ulcer

could hardly be confounded since one produces a picture of diffuse peritonitis and the other spares the peritoneum, yet mistakes are by no means rare. When the perforation is a typical one, for instance,"formes frustes", in type or associated with spontaneous closure after moderate leakage, it may be difficult to establish the correct diagnosis. Jenkinson and Ellis(cited by Singer) reported a puzzling case in which the clinical symptoms pointed to the presence of kidney disease. From the context including the presence poentgenologically of a spontaneous pneumoperitoneum which was not explained satisfactorily, this was almost without question a perforated peptic ulcer with spontaneous closure. The renal affections which require consideration include torsion of a floating kidney on its pedicle(Dietl's crisis)renal artery, pyelitis, pyonephrosis, peri-& and paranephritis.

RUPTURED OR DISECTING ABDOMINAL ANEURYSM-Aneurysm of the abdominal aorta upon rupture gives a picture which suggests perforated ulcer at the onset plus bleeding from the ulcer when the anemia becomes apparent. In a case of this type in which the correct diagnosis of ruptured aortic aneurysm was made ante-mortem, a large mass could be felt to the left of the spine over which a thrill could be felt and a bruit heard. Dissecting aneurysm (luetic or arteriosclerotic) of the abdominal aorta may furnish

a great deal of difficulty. A senile individual with extensive calcification of the aorta(seen in the roentgenogram) suffered from a severe pain in the lower dorsal region. Except for tenderness on deep pressure in the upper epigastrium, the abdominal examination was negative. The patient recovered from initial pain and left the hospital only to return later with a picture of sepsis. At autopsy the cause of sepsis was found to be an abcess in the lesser omental cavity caused by rupture of an ulcer, which had become sealed off spontaneously. The general peritoneal cavity was free from evidence of inflammation. High grade sclerosis but no aneurysm or tear of the aorta was demonstrable.

COMPLICATIONS

ACUTE DIFFUSE PERITONITIS-Acute diffuse peritonitis stands out in bold relief as causing the greatest number of deaths;in fact more than all other causes combined. Of these 253 fatal cases 65 per cent were deaths from this cause. The series presented herein for the first time shows an incidence of 41.1 per cent. In the group presented by Brown (7) all deaths from peritonitis occurred before the eighth day. In another group all deaths from this cause occurred on or before the ninth postoperative day. Four of the seven deaths of peritonitis occurred in patients operated upon less than five hours

after perforation and whose age ranged from 35 to 47 years. Yet this was in a group where only 41.1 per cent of all deaths were due to peritonitis as compared to the average of 61 per cent. Other factors being equal, these patients represented the class that theoritically should have had the best chance for recovery.

In this connection it is interesting to consider the fact that the presence of bacteria in the foreign material in the peritoneal cavity following perforation has not been stressed, and the impressions conveyed by the literature have rather emphasised its innocuousness. Results of cultures seemed to substantiate this impression until Brutt (9)(1926) reported cultures taken from 112 cases at operation. The ultra-abdominal fluid in those operated upon from six to twelve hours following perforation gave 74 per cent positive cultures. By the same technique 93 per cent positive cultures were secured by those coming to operation twelve hours or more following perforation. One must remember that positive cultures prove beyond dispute the presence of bacteria, negative cultures do not necessarily prove their absence. Streptococcus hemolyticus, streptococcus veridans, bacillus coli and staphylococci were the organisms found.

With peritonitis as the chief cause of death and

with pathogenic bacteria so frequently present in the inter-abdominal fluid following perforation, the need of the utmost care in post-operative treatment becomes strikingly apparent. The first eight post-operative days are the most dangerous ones from this complication.

PULMONARY COMPLICATIONS-In a table it is found that thirty-three deaths, or thirteen per cent from the entire group are attributed to pulmonary complications. It is to be regretted that more detailed informmation is lacking.However,two important inferences may be drawn. In one group it will be noticed that some pulmonary deaths are attributed to atelectatic collapse occurring on the third to the fourth post-operative day. Brown (7) notes that three deaths in the series reported by him occurred with pulmonary complications on the third,fourth and eighth post-operative days, and, of seven deaths attributed to pneumonia, three occurred during the first post-operative week. These early pulmonary deaths suggest very strongly lung atelectasis as the primary cause, particularlly since they occurred before the time of our present knowledge of its significance in early post-operative complications. Colp (15) has reported fatality from this cause following operation.

It has been fairly well established by Coryloss(18)

and others that pulmonary complications beginning from within twenty-four hours to fourty-eight hours following abdominal operations are due to atelectasis; When pneumonia developes it is a secondary process. Lahey ((43) has shown that a definite elevation of the diaphragm on the right side occurs following operations on the upper part of the abdomen. He believes that this causes partial atelectasis of the lung immediately above the elevated diaphragm. Churchill (13) found a reduction in vital capacity of from 50 to 75 per cent following operation involving the abdominal muscles.

The remaining pulmonary deaths on which information is available occurred during the second postoperative week. It is significant in this relation to notice that all patients reported as dying from subdiaphragmatic abcess in one series were shown at the autopsy to have empyema and multiple lung abcess as well. The possibility of septic emboli and the probability of direct extension from the subdiaphragmatic infection are factors of importance in the consideration of late pulmonary complications. It is well to recall that the diaphragm is freely supplied by lymphatics which course under the peritoneal covering on both abdominal and pleural surfaces. They anastomose freely. It is pos-

sible for the lung to become attached to the diaphragm and thereby direct extension is facilitated. Graham(35) has emphasised the frequency with which empyema is associated with subdiaphragmatic abcess and the rarity with which subdiaphragmatic infection results from empyema.

SUBDIAPHRAGMATIC ABCESS- Fifteen deaths from a total of 253, or an incidence of 5.09 per cent, are attributed to subdiaphragmatic abcess. The association of late pulmonary complications with this lesion suggests a still higher incidence in fatal cases not subjected to autopsy. Richardson (68) drew attention to the fact that in the series reported by him in 1917, 25 per cent of all fatalities were due to this complication. This is much higher than the average. In the same report he wrote that "Drainage in the neighborhood of the perforation was almost universally used." In the two patients of the entire collected group who were found at autopsy to have perforation of the diaphragm, upper abdominal drains were used.

Subdiaphragmatic abcess occurs most frequently following perforated ulcer between the right lobe of the liver and the right side of the diaphragm posterially and extending to the right kidney. It is limited below by the transverse colon and the great omentum. Next in

frequency is the location anteriorly to this between the liver and the diaphragm, limited laterally by the falciform ligament on the left and the transverse colon and the omentum below, and occassionally anteriorly by the abdominal wall. These locations should be borne in mind in caring for the ultra-abdominal foreign material.

Subdiaphragmatic abcess, from its location, is most inaccessible to the great scavenger of the abdomen, the great omentum. Its treatment in the scope of this paper lies in its prevention. Early recognition and treatment is considered in a later paragraph.

SHOCK- Nineteen deaths from a total of 253, or an incidence of 7.5 per cent are attributed to shock in the reports herein abstracted. We have included under this heading those which failed to react from the anesthetic and operation. These certainly must be attributed to surgical shock. Luff's (47) series shows 28 per cent of the deaths attributed to shock and prostration. Winslow reported 20 per cent in his series. When figures are available as in the series mentioned an higher incidence is noted. It is interesting to note that only one-half the reports from which the statics are taken note shock as a cause of death. In Wilson's (84) series three of the deaths attributed to shock occurred in patients coming

to operation thirty hours or more following perforation, and this fact alone marks them as extreme surgical risks. Moynihan (56) believes that true surgical shock as indicated by low blood pressure, diminished blood volume and rapid pulse is not present after perforation until generalized peritonitis has developed. The primary condition, he believes, to be one of extreme prostration. This distinction should not interfere, however, in the treatment indicated preparitory to operation.

Present methods of closing duodenal perforations are highly efficient. Early operation is of primary importance and has been repeatedly emphasized. The question naturally rises as to how the present mortality rate may be lowered. More accurate knowledge of the commonest causes of death indicates clearly the course for future endeavor. Attention must be directed to the preoperative preparation and the post-operative treatment in keeping with present knowledge. Consideration of these vital factors in the treatment of this lesion is conspicuous by its absence from medical literature. A knowledge of the commonest complications that may prove fatal is essential to their prevention or early recognition and successful treatment. The surgical management of intra-abdominal foreign matter is not being

handled at present with uniform success.

TREATMENT

Surgery offers the only remedy in the handling of this distressing complication. The death rate with non-interference, according to Robson(69), is 95 per cent, according to Deaver (20), per cent. The treatment naturally falls in two divisions:

1. Acute cases, the treatment of which is abdominal intervention at the very earliest moment after perforation repair of the perforated viscus, surgical procedure to overcome or anticipate obstruction or for healing ulcers (gastrojejunostomy), and the application of those principles to conditions of the peritoneum best calculated to limit or prevent septic peritonitis.

2. Subacute and chronic perforations, in the management of which we have to deal, not with the perforation itself, but with conditions resulting from perforation-vis., the various forms of abcess and their ramification, as subhepatic, retrocolic perigastric and subphrenic and the general sepsis resulting therefrom. The principle involved in the treatment is the location of the abcess and providing a safe conduit through which its contents may reach the surface of the body.

In the acute perforation the upper abdomen should

be entered through an incision in the median line through the upper portion of the right rectus muscle. The later incision renders perforations near the pylorus and duodenum most accessible. When the abdomen is usually opened gas usually escaped, and more or less of the stomach contents will be encountered. If the stomach is full at the time of perforation or liquids are given the abdominal cavity may be found flooded . In a very interesting and successful case of gastric perforation operated on by Dr. Arthur C. Roper (71) of London a pint of curded milkywas found about the stomach and the pelvis was filled with milky fluid. In Allaben's (1) case perforation occurred when the stomach was empty and only a small amount of gastric juice and muceous was found about the pyloris.

The intestines should be protected by gauze sponges during the time of suturing. Usually it is not difficult to locate the perforation, for in acute cases it is found on the anterior side of the stomach most frequently in the pyloric region or upon the lesser curvature. The perforation should be closed with linen purse string, interrupted Lembert, or Lembert mattress suture being used. Frequently the base of the ulcer is hardened and friable from induration and will permit but little ten-

sion upon the suture. In such cases the perforation may be more securely sealed by grafting over the sutures a piece of the greater or gastro-heptic omentum. After an ulcer perforates there is a tendency to heal spontaneously, so if leakage can be controlled for a short time, healing occurs readily. In case the location of the perforation is inaccessible, or its edges are friable so that suture is impossible good results have been obtained by tampon or by draining the stomach through temporary gastrostomy.

Lennander (44) thus drained the stomach of a patient with perforation and stenois upon whom he operated 60 hours after perforation. About three and one-half months later gastrojejunostomy was done for pyloric obstruction. Lennander (44) recommends temporary gastrostomy in cases where stenoses is liable to follow operation for perforation and a patients condition will not allow a more prolonged operation of gastrojejunostomy. Gastrojejunostomy is recommended for a second operation when symptoms of stenosis appear. He says temporary gastrostomy is indicated: 1.If the gastric wound cannot be absolutely securely closed on account of the location or quality of the ulcer. 2.If there are symptoms of paralysis of the smaller intestines. Paring the edges of the ulcer is necessary and excising the ulcer before suturing increases the mortal-

ity and renders pyloric stenoises more probable. Von Kautz of Wein excised the ulcer in three cases, the patients all dying.

When the perforating ulcer is near the pylorig, stenosis is liable to occur and the question of doing a gastrojejunostomy at the same time that the perforation is repaired must be considered. The advisability of such a procedure will depend much upon the amount of time elapsing between the perforation and the operation for its repair. If the operation can be done early, two or three hours after perforation, and the patients condition is good, a gastrojejunostomy should be done. This is recommended by Deaver (21), Mayo (51), Robson (69), Lennander (44), Korte, Martins (49) and others. The Mayos who have operated upon thirty cases, perforating into the free peritoneal cavity, have done gastrojejunostomy at the same sitting five times. As a secondary operation two of their cases have demanded this procedure.

Allaben (1) states, "In my case laparotomy and repair of the perforation were done three hours after the accident. The perforation was so near the pyloris and its base so indurated it was certain that pyloris stenosis would follow. The patient was in good condition, yet the fear of prolonging the operation and spreading

infection deterred me from doing a gastrojejunostomy. Three weeks later I was obliged to do this operation for pyloric obstruction. The patient making a good recovery. I now believe, under similar conditions, the two operations under the one sitting is the operation of choice. If there is a question of doubt, however, the patient should be given the benefit and gastrojejunostomy done as a second operation. The posterior no-loop operation as now performed is as satisfactory as any operation in surgery".

The proper toilet of the peritoneum where septic material is present is still a question of some discussion among surgeons. Where undigested food has entered the peritoneal cavity through a perforation it should be removed but the belief is growing stronger that irrigation and mopping away of pus and exudates in septic peritinitis is not only a waste of time but a dangerous procedure calculated to spread septic material toward the diaphragm and open new fields for infection. The experiments of Buxton (10) on the processes of absorbtion from the peritoneal cavity demonstrates that there is practically no other absorbtion by any other part of the peritoneum beside the diaphragm and the omentum. Many bacteria are entangled by exudates on the surface

of the omentum and destroyed by the phagocytes, while some are absorbed by the omental lymphatics. Within five minutes after bacteria are introduced into the peritoneal cavity they will be found in greater numbers in the liver and spleen and in the anterior mediastinal lymph nodes which they reach from the diaphragm. It is evident, therefore,that anything which spreads bacteria toward the diaphragm, as mopping and irrigation, increases the danger for the patient. From this we get our rational treatment for reducing manipulations in the peritoneal cavity to a minum and for maintaining the patient in the Fowler position.

Simple suture was advised by Mikulicz(cited by Allaben (1)) in 1880. Twelve years later the first successful operation for acute perforation was reported by Huessner (cited by Allaben(1)). In spite or forty years of operative experience since then, opinion as to the operation of choice has not yet been cyystallized. Surgeons of skill and experience write very contradictory articles, and these have occassionally appeared in the same number of the same journal. All agree, of course, that the essential factor in mortality in the time allowed to elapse between the time of perforation and the time of operation. The heated difference of opinion bear particularly on the end results that follow conservative procedures

as compared with those that follow the more rapid radical operations.

In 1923, puzzled by the situation, Guthrie (36)sent out a questionnaire to many surgeons all over the country, in an effort to find out their opinions in regard to the procedure of choice in the presence of an acute perforated gastro-duodenal ulcer. He received 152 replies to this, and the answers revealed great differences of opinion among the leaders in the profession. This is rather remarkable when one considers the condition a common abdominal emergency seen not infrequently on all surgical services.

As to the choice of operations there are so many factors to be considered here that it seems wise to list some of the pros and cons of each of the advocated procedures and then to draw what conclusions seem justified by the actual facts that are available.

SIMPLE SUTURE- A great many leading surgeons are confident that simple closure of the perforation is the procedure of choice. The suture material and the exact method of using it may differ slightly, but the principles to which these surgeons point are as follows: They claim:

(1)That it is the quickest and simplest procedure to

meet a grave emergency and that it adequately meets the emergency.

(2)That the operative mortality is lower than any other procedure.

(3)That the postoperative course is smoother.

(4)That the ulcer,in the majority of cases,heals promptly and remains healed.

(5)That subsequent pyloric obstruction is infrequent, and that when it does occur there is an ideal indication for a secondary gastroenterostomy under the most favorable conditions."Two safe operations are always better, and to be desired, than one dangerous one".

(6)That primary gastroenterostomy subjects the patient unnecessarily to the dangers of gastrojejunal ulcer.

(7)That gastrojejunostomy does not prevent the occasional occurance of hemorrhage, reperforation, formation of new ulcers, or the recrudence of old ones.

(8)That a careful evaluation of late results justifies the above impressions.

CLOSURE PLUS IMMEDIATE GASTROENTEROSTOMY-There are many advocates of this procedure, most of whom agree that it should be done only in comparitively early cases. They make the following claims:

(1)That gastroenterostomy does not increase the mor-

tality in the early cases, adding only fifteen minutes to the procedure.

(2)That the danger of spreading infection by the additional procedure has been overemphasised, the exudates in these cases being almost uniformly sterile for many hours after perforation.

(3)That the immediate postoperative course is much smoother,with fluids given orally much sooner than is posible after simple closure.

(4)That the perforation cannot in amny cases be safely and adequately closed without producing a pyloric obstruction of considerable degree.

(5)That the ulcer heals rapidly in the presence of a gastroenterostomy, and that complications such as reperforations are less likely to occur.

(6)That the late results are better than with simple suture.

(7)That a large percentage of simple suture cases require subsequent gastroenterostomy.

PYLOROPLASTY-Such operative procedure as the Heineke Mikulicz, Finney (30), Horsely,and other modifications of pyloroplasty have at times been quite popular in treating acute gastroduodenal perforations. The advocates of such procedure pointed out:

(1)That peptic ulcers which present acute perforations are usually situated just where such an operation can be easily done- on the anterior surface near the pyloris.

(2)That the operation takes only a few minutes longer than simple closure, and does not involve any soiling of the lesser peritoneal cavity, as in gastroenterostomy.

(3)That the operation serves the purpose of avoiding the possibility of future pyloric stenosis, without subjecting the patient to the "unphysiologic"procedure of gastroenterostomy.

(4)That the lesion is directly attacked and actually removed.

RESECTION- Advocates of immediate partial gastrectomy in these cases vary in the operation they refer to ,the extreme being sort of minuture Billroth No.1 operation on one hand, and a subtotal gastrectomy on the other.

They would limit the operations to those cases that reach the surgeon soon after perforation, but claim that in these cases the rather formidable procedure does not add greatly to the operative mortality. They emphasize that patients who have had the more usual and conservative procedure done at the time of acute perforation require subsequent surgery in a rather high percentage of cases. They believe that the doing of a real curative

operation rather than a paliative one, should be the aim of the surgeon even in the presence of an acute perforation.

DISCUSSION-Although the same facts are interpreted differently by the different groups, a review of all available facts bearing on this problem would seem so important as to quite overshadow the theoretical discussion of pros and cons that have been listed. It is quite important to find out what actually happens to t this group of patients.

Gibson(33) who has long been interested in the subject, did simple suture in seventy-five or seventy-six cases of acute perforation. Half of the cases remained symptom free, and in only eight cases did the symptoms indicate the necessity of a second operation. Farr (29) found that ninteen of twenty-one cases remained well after a simple closure, and ten of Benner's(6) cases remained symptom free. Two-thirds of Poole's(66) series of simple closure cases were cured by the procedure. Urritia (81) recently reported a series of fifty-two private patients in whom he had done simple closure of acute duodenal perforation. Of this series 63.6 per cent remained well. Southam (79) reports that a pyloric obstruction eventually necessitates gastro-

enterostomy in only four of thirty-seven cases of simple closure. These rather optomistic reports are bolstered considerably by such men as Finney (36), Pannett (64) and Engelsing (27), who have considered the results of simple closure very satisfactory. Johnston (38) collected results in 568 cases of simple closure, and twenty-five per cent of these had persisting symptoms. Only eleven per cent of these required gastroenterostomy later. Williams (85) and Walsh report that of fifty-eight cases with simple suture, subsequent gastroenterostomy was necessary in only ten. Lewiston (45) classes thirteen of thirty-three cases followed as "failures" from conservative surgical treatment. Mills (55) thought the late results of simple closure were discouraging.

On the basis of available statistics it seems fair to estimate that of every one-hundred cases that leave the hospital following simple closure of an acute perforation, approximately sixty to sixty-five will remain free of gastric symptoms, approximately ten to fifteen will require further surgery, and of the remaining twenty-five those who are resonably careful about diet and general activity will get along satisfactorily.

In addition to clinical evidence, there is pathological evidence to support the statement that many ulcers heal(and remain healed) after simple closure. There are

numerous cases to be found in the literature in which subsequent exploration for some other cause revealed that there was no trace of the old ulcer(Leecene(46), (Basset(4),Benner (6),Pannett(64)). An experimental support for the view that pyloric obstruction does not occur as commonly as would be supposed,in cases where a perforation near the pylorus has been infolded, is to be found in the work of Stewart (80) and Barber on dogs. They perform cautery puncture of the duodenum near the pylorus in a series of dogs, and infolded the opening so completely that the lumen was practically occluded. X-ray and clinical studies subsequently seem to show that it is very difficult to produce pyloric obstruction in this way. The same conclusion was reached by Eliot, who found that the constriction produced by excision of rwo-thirds of the circumference of the duodenum and closure(in cats) ironed out very promptly.

In a larger series of sixty-two cases, reported by a junior author at a staff conference in 1927, it was found that approximately ten per cent of the cases treated by simple suture required a subsequent gastroenterostomy.

The advisability of instituting drainage in these cases finds also a surprising difference of opinion.

Gibson (33) found that cultures from the exudate were almost invariably sterile up until eighteen hours after the perforation, and many observers have pointed out the widespread nature of the soiling, and the futility of attempting to drain all the affected area. The question of drainage in late cases finds all in agreement that it should be done, the only disagreement being as to the details. Nearly all the answers to Guthrie's(36) questionnaire agreed on drainage-many of them advising a suprapubic drain as well as subhepatic drainage. Deaver(26), and Brown(8) urge the use of a suprapubic drain as very important. Finney(30) advises against the use of any drainage except in late cases,and urges multiple thorough drainage if any. White(83)and Patterson state that they have been closing the early cases without drainage, and have been very much pleased with the results, most of the wounds healing per primam.

In 1901, Moynihan(59) suggested that it might be wise to add some procedure,after closure of an acute perforated ulcer near the pylorus,which would guard against the future developement of pyloric stenosis. Since that time it has become more and more common for the surgeon to perform a pyloroplasty with excision of the ulcer or an immediate gastroenterostomy after clos-

ing the perforation. In many hospitals, the later is done as a routine in early cases. The advocates claim that the mortality is actually decreased by the additional procedure. They suggest that a pyloric perforation is often inadequately closed because of fear of producing an obstruction by infolding too much, and that if the perforation is thoroughly closed and infolded an ideal condition for the function of a gastroenterostomy is produced. There appeared in the literature so many statements to the effect that gastroenterostomy lowers the mortality that Cope(16),a careful student of acute abdominal surgery, wrote a protest to the British Medical Journal to give it as his opinion that this was due to the selection of cases. He referred to an analysis of five years at St. Mary's Hospital, London, during which time the mortality of simple suture cases was higher than in those cases in which closure of the perforation was conbined with gastroenterostomy, pointing out, however, that the time elapsing between perforation and operation in the former group was thirteen hours, and in the second group only five and one-half hours. The same observation applies to Dineen's(24) cases. Ten cases were subjected to immediate gastroenterostomy, with no deaths, but the average time elapsing before operation was only three-

quarter hours.

The statistics of the advocates of suture and gastrojejunostomy are not comparable with those of surgeons who advise suture only. Gastrojejunostomy is generally carried out in selected cases only, and the statistics of a series of such cases are, therefore, not comparable with a series where suture only has been done, for the later would include the bad as well as the good operative risks. White(83) and Patterson in their article say, "In many of our cases where, from the general condition of the patient and the duration and the size of the perforation, the case would have been judged fit to withstand a gastrojejunostomy, we have been impressed with the critical condition they have passed through before convalescence has been established. We cannot but feel in many of these cases, the additional strain of a gastrojejunostomy would have turned the scales against them". They also present a table of the mortality rate of the Roosevelt Hospital of New York City in regards to surgery performed on the perforated peptic ulcer with a discussion of the results following:

Mortality rate of the Roosevelt Hospital

Time after operation	Number	Deaths	Per Cent
Under 10 hours	50	3	6
10 to 20 hours	13	6	46
20 to 30 hours	10	4	40
over thirty hours	6	4	66
	79	17	21.5

Of these cases fifty-three were simple suture with ten deaths, an operative mortality of 19 per cent. Twenty-six cases had simple suture plus posterior gastroenterostomy with seven deaths, an operative per cent of 27 mortality.

In spite of these later figures, we feel that gastroenterostomy at an early stage has not, on the average, such a high mortality as we here report. Three of the gastroenterostomies were performed in cases that had been perforated over twenty-four hours and these three patients died. We consider that in these cases poor surgical treatment was used regardless of a conservative or radical attitude.

Seven cases not included in the above table were subjected to immediate excision of the ulcer with pyloroplasty. These were all early cases except one which was perforated late, and this patient was the only one to die.

An important consideration is that of the late results in those cases subjected to immediate gastroenterostomy as compared to those treated with simple suture. Only six of our cases were followed over three years and of these, four were considered cured. Johnston (38) collected a series of 158 cases. Only twelve per eent of these had"severe persisting symptoms",and the percentage that required operation for gastrojejunal ulcer was 1.3. Mills (55) had ten out of fourteen perfectly well.

Cutler and Newton (19) decided, after a careful review of the literature and of their own cases, that it is wiser to do a gastroenterostomy as a routine in these cases. However, Metzger (53) concluded, after a review of late results in nearly a hundred cases, that the late results are about the same whether a gastroenterostomy is added to the closure of the perforation or not.

Lewiston (45), who strongly advocated gastroenterostomy a few years ago, has more recently stated that he considers the likelyhood of gastrojejunal ulcer so great that gastroenterostomy should not be done. In early cases he advocated partial gastrectomy. McCreery (52) urges picking the operation to fit the case, and feels that the best late results will follow simple suture in the

majority of cases, although certain cases, where the perforation is large and the induration is extensive, immediate gastrectomy is indicated. Unlike most European surgeons,Urrutia(81) is very strong for conservatism in these cases. He concluded that,"Gastrectomy does not modify the postoperative course, is an insufficient treatment of the ulcer itself,and may have unfortunate late results". His opinion becomes more valuable when one realizes that he has had a most extensive experience with gastric ulcer surgery, having performed more than five-hundred partial gastrectomies for chronic gastric and duodenal ulcers. In spite of his dexterity and radical tendencies in "elective" gastric surgery, he does not feel that any procedure should be added to a simple closure in the presence of acute perofration.

Noehren (62) quotes McKnight, of the Mayo clinic, that in seven of twenty reported cases, the ulcer had persisted or new ones formed in spite of the presence of gastroenterostomy. Radoievitch (67) collected more than 10,000 cases of gastroenterostomy for a duodenal ulcer, and eighty-one of these cases were performed in spite of the presence of gastroenterostomy. There are frequent reports of reperforation, formation of new ulcers, etc.,after gastroenterostomy in these cases that gives

one the definite impression that there should be a trend away from gastroenterostomy except in cases of extreme pyloric obstruction of considerable degree.

Pyloroplasty, of the Heineke-Mikulicz or Horsely type, has been discussed earlier in this paper as an ideal theoretical solution to the problem of acute perforated peptic ulcers. Unfortunately the late results do not justify this hope. Erdmann(28), who did many of these operations for chronic ulcers some years ago, has come to the conclusion that the late results of this operation are poor. Hinton (37) reports that Horsely type of pylorplasty in twelve cases with one death (the time after perforation varying from four to fourteen hours). Nine of the survivors were traced sixteen months to four years and only two of these were entirely well after two years, seven results being unsatisfactory. Keen's (70) twenty-five cases were well for six years after excision of the ulcer and pyloroplasty, and then had severe reoccurance of pain. Williams and Welsh(85) report five of six pyloroplasty operations that had good end results. White's (83) experience with the follow up results of six cases has not been encouraging. X-ray studies seem to indicate too rapid emptying was responcible, in part at least, for the persisting symptoms.

In his monograph on gastroduodenal ulceration, Pannett (64) refers to the advocacy among European surgeons of partial gastrectomy in the presence of an acute perforation as,"an amazing development". Even more amazing, in view of the fact that subtotal gastrectomy in elective operations carries even in the experts hands a mortality of ten to sixteen per cent.

The theoretical considerations have already been discusseda As to the actual results of gastric resection, these are available, for the mounting reports in the literature nearly all pepresent recent cases. The radical tendancy in European gastric surgery may be illustrated by this experience; He had been entirely symptom free for more than two years following the simple closure for the perforated duodenal ulcer. While on a recent visit to his home in Graz he was advised to have a subtotal gastrectomy at once,on the grounds that anyone who had ever had a duodenal ulcer would never be permanently well until subjected to partial gastrectomy.

Odelberg (63) reports twenty partial gastrectomies in acute perforations with only one death. All were early cases. The result of the cases traced more than one year were good. He believes that in spite of the pain and the emergency atmosphere, most of these early

cases are in better condition to stand a major gastric operation than the average patient who has had a long seige of dieting on account of chronic ulcers. The only death in his series occurred in a case in which culture from the peritoneal exudates showed a haemolytic streptococcus.

Krainik (40) adds to his testimony on the side of resection, and referres to the first successful resection done in France in the treatment of acute perforation in 1919. Kunz (41) reports seven resections without a death, two of the cases having been perforated more than fifteen hours. Radioevitch (67) gives a mortality figuré for resections done within the first twelve hours after perforation as 11.29 per cent. One of Lewisohn's (45) partial gastrectomies (out of four done for persisting symptoms after simple closure) developed a marginal ulcer. One of Lecene's (46) twenty-five cases met the same fate, and recent reports of Balfour(3) at the Mayo clinic warned us that the incidence of gastrojejunal ulcer followed partial gastrectomy is a very definite one.

POST OPERATIVE TREATMENT

SHOCK-Regardless of the thoery as to the procedure of shock to which one subscribes, the pathological chances are quite logically agreed upon. It is generally accepted

that there is fundamentally an increased possibility of the blood capillaries. The fluid portion of the blood exuded into the surrounding tissues and the intervascular blood is increased in viscosity and decreased in volume. Lowered blood pressure results. There is an excess of the blood cells in the capillaries. The nonprotein nitrogenous elements of the blood are increased. The blood tissues, especially those of the central nervous system, suffer from lack of water, food and oxygen. Oxidation is diminished and heat prodution lowered. This may lead to a reduction in the alkali reserve and a state of acidosis.

The indications therefore are for (1)administrations of fluids;(2)relief of pain to provide rest;(3)elevation of blood pressure;(4)elevation and conservation of body temperature;(5)treatment of the cause;(6)prevention of operative shock.

Pain is best cared for by the administration of morphine hyperdermically in moderate dosage. If true shock is established, large doses particularily are contra-indicated. Blood pressure may best be raised by adminustration of fluids. Physiologic solution of sodium chloride by vein or conyinuous hypodermoclysis usualls fulfills the requirments. If true shock exists this may

be suplimented intravenous administration of from 300 to 500 cc. of saline solution to which is added dextrose sufficient to make from 10 to 20 per cent solution. If these measures fail blood transfusion should be performed. Blood temperature should be preserved by ample protection in the form of woolen blankets and external heat.

The prevention of operative shock is concerned with proper selection of an anesthetic and care in its administration. Rapid gentle operating, avoiding exposure of viscera, sponging traction, unnecessary manipulation and hemorrhage is also important in the prevention of shock.

The treatment of shock from this lesion, unlike the treatment of shock from hemorrhage, indicates that the procedure already outlined is to be carried out before direct treatment of the cause is undertaken. Before the abdomen is opened, the position of the patient to facilitate drainage from the subdiaphragmatic space seems to be the only means of influencing the factors at work. The reclinigg position on the right side is better than the Fowler position at this stage. The head should not be lowered even if shock is established. When operation is undertaken, closure of the perforation, removal of the foreign material, and institution of pro-

per drainage further fulfill the requirments. The position of the patient at operation is important. The head, body and extremities should be at the same plane with the head end of the table elevated from 10 to 15 degrees.

What has been said does not imple that all cases of acute perforation need this most careful preoperative preparation. Prostration or shock is the indication.

PULMONARY COMPLICATIONS-Beginning within twenty-four to forty-eight hours after operation pulmonary complications should at once suggest atelectasis and painstaking examination should be made for its detection. The treatment comprises from five to ten per cent carbon dioxide and oxygen for five minutes period every two hours. This lowers the viscosity of the bronchial secretion and facilitates expectoration. It likewise produces deep breathing and thereby aids in expanding the collapsed portion of the lung. Corollos (18) believes that it retards the growth of pneumococci. In addition to this, cough and extertoration should be induced, as suggested by Sante (73), by turning the patient on his sound side several times daily. Clerf (14) believes that in the event that this does not give prompt relief, bronchoscopic drainage should be resorted to. In patients

with alarming symptoms their response to this treatment is often most striking.

FOREIGN MATERIAL-The intra-abdominal foreign material has been variously managed at operations. Irrigation of the abdominal cavity may be viewed as incorrect in principle and disappointing in practice. Upper abdominal drains, from all evidence available, often do actual harm. Klopp (39) believes their use should be reserved for patients coming for operation late and in which localized supuration has occurred. Suction,while correct in principle, is ineffective in practice. The suction tip is almost always clogged by omentum or some portion of the viscera. Care must be exercised to remove as much foreign material as possible ,thereby reducing the dose of bacteria. Present knowledge indicates that the best means available are postural drainage,and absorbtion by carefully placed dry sponges. These sponges should be removed and replaced when they become saturated. Wiping should never be indulged in. Every care is exercised to preserve nature's barrier, the plastic lymph.

Unless the foreign material remains strictly localized in the upper part of the abdomen, suprapubic drainage by a rubber tube into the pelvis is very useful.

If the intra-abdominal fluid is copious and widely disseminated, this drain should be placed before an attempt is made to close the perforation. Drainage therefore greatly facilitates the procedure.

SUBDIAPHRAGMATIC ABCESS- As pointed out, one's first effort aims at prevention . This lesion should always be sought in patients who, following operation show improvement for several days or even weeks, after which they develope a remittent fever with pain in the abdomen, back or lower part of the chest. The diagnosis is not simple in the early stages. Roentgen examination and paracentesis are helpful. The treatment is surgical drains by the transpleural approach, if possible. Abcess pointing anteriorly or low in the abdomen may be drained abdominally. The average mortality rate is approximately 70 per cent without surgical drainage and 30 per cent following its use.

PERITONITIS-A consideration of postoperative treatment necessatites a discussion of the treatment,prophylactic and acute, of acute septic peritonitis in keeping with established principles and modern knowledge. Fleming (31) says:"I am impressed from personal experience and from the evidence already presented, that the safest method of procedure is to consider every case one of generalized peritonitis until proved otherwise. When

it becomes evident beyond reasonable doubt that intra-abdominal localization or resolution has taken place, one's method of treatment may be modified. Sucessful treatment designed to meet requirments call for(1) absolute rest bodily, abdominally and peristaltic;(2)maintainance of fluid food and salt balance;(3)the reduction or the prevention of toxemia, and (4)drainage.

In these principles there are nothing new. Happily, means of fulfilling them are more efficient. Rest should be secured in the Fowler position after postoperative reaction. Morphine may be used as a satisfactory analgesic and because it aids in securing peristalic rest. Its use must be guarded, for, if vomiting is induced thereby, the much needed abdominal and peristalic rest is sacrificed. Complete intestional rest means that nothing be allowed by mouth or bowel.

The administration of fluid by bowel also increase peristalisis, though to a less degree and usually confined to the colon. Under some circumstances such administration may be used with relative safety;for example, when the peritoneal inoculation is confined strictly to the upper part of the abdomen or to the right side of the abdomen lateral to the ascending colon. Physiologic solution of sodium chloride is best for this pur-

pose and usually more effectively given as small retention enemas of from four to six ounces introduced very slowly every three or four hours. When the intra-abdominal inoculation or infection is generalized the rectal route should not be used. The cleanest most accurate and certain method, because of variable absorption from the colon, is by hypodermoclysis. It may be given as a daily single dose or intermittently. From 1,500 to 4,000 cc. is needed in twenty-four hours. The fluid and salt balance may therefore be maintained and toxemia be combated by the resulting increased elimination through the kidneys. The use of enemas designed to empty the bowels either of gas or of fecal contents has no place in the prophylactic or actual treatment of peristalsis. In fact, their use in this condition is pernicious and is exceeded only by the giving of purges in the same condition; The difference is merely one of degree. An enema is usually contraindicted following operation until the pulse and temperature have remained normal or nearly so for fourty-eight Hours or so, provided the abdomen remained soft and free from distention. Unmistakable evidence of intra-abdominal resolution or localization gives one the clue as to when an enema may be safely used. I am so impressed by the error of this practice and its frequently deadly effects that some temperature charts are present-

ed to show its more deadly influence. These are drawn of old charts of patients who died from peritonitis from duodenal perforation and who were treated incorrectly in this respect. The behavior of the temperature and pulse following the administration of enemas as shown on charts is mute evidence of their influence. The treatment of the patient in whom the postoperative course is charted thereon was otherwise correct in principle.

Abdominal distension may be troublesome. It is best treated by methods designed to prevent it. Ample fluid intake with peristalic rest are examples. Continual warmth to the abdomen is helpful. The rectal tube is sometimes bebificial and it does no harm. Morphine for distension is contraindicted. If distension developes and it leads to a regurgitation or vomiting, or if the stomach becomes dialated, it is helpful to perform gastric drainage by means of Rahfuss tube.

The food and fluid requirements of a patient may be taken care of in a most satisfactory way by the intravenous administration of the dextrose solution. It is best given in a five to ten per cent solution in freshly distilled water by the extravenous drip method. From .4 to .8 gm. per kilogram of body weight per hour may be used. Glucosuria should be avoided and is a guide

for the rate of administration. If vomiting occurs and persists until ketonuria results, or if intestional obstruction developes, intravenous dextrose with other treatment as indicated is frequently life saving".

SUMMARY

Acute perforating ulcer was first described by Joseph Penada in 1793. Since that time it was found only at autopsy but no means of treatment was instituted until the last ten years of the nineteenth centurt. It was at this time that they recognized that early diagnosis and prompt treatment was necessary for successful treatment which has remained unchanged up to the present time.

Three types of perforation are recognized, namely the acute, subacute and chronic. Of these the subacute and chronic present problems of diagnosis. The acute type presents a typical picture but must not be confused with appendicitis, acute pancreatitis, intestional obstruction, etc. Much has been done by Singer and Vaughan (75) in the use of the X-ray in the differential diagnosis of the subacute type by the finding of an air bubble under the diaphragm as being pathognomonic of a perforation. Early intervention is important in this type before the walling off process takes place.

Intermittent leakage may take place periodically if left alone but it is advisable not to intervene or disturb the lesion after it has become walled-off, as there is further spread of the gastric contents, which has been localized and taken care of.

Differential points have been presented wherein the perforated ulcer must be differentiated from other abdominal affections, pelvic affections as ectopic pregnancies etc., affections of the thoracic structures as coronary thrombosis, acute pericarditis, aneurysms etc., and affections of the nervous system.

Reasons and causes of death following operations for perforations of peptic ulcers have been presented. Causes of death are as follows:generalized peritonitis, 65 per cent; pulmonary complications, 13 per cent; shock,7.5 per cent; sub-diaphragmatic abcess,5.09 per cent; other causes, 8.06 per cent. These figures are only approximations after review of the literature.

A discussion as to the surgical procedure of choice has been presented, bases somewhat on a resume which resulted from a questionaire sent out by Guthrie(36) in 1926. The recent literature reveals very little change in procedures.

Postoperative treatment has been presented which

reveal a decrease in mortality after its administration.

CONCLUSIONS

Before recognition of this complication an era of very high mortality and almost certain death resulted, which can be compared to the era of acute appendicitis, and may have been mistaken for appendicitis.

A great responsibility rests on the shoulders of the attending physician ,who must be acquainted with points which aid in differential diagnosis of the perforated peptic ulcer and other affections.

The X-ray should be a part of a physicians equipment and offers a great aid in differential diagnosis. Whenever any doubt arises the procedure should be carried out, knowing that an air bubble under the diaphragm is pathagnomonic of a perforation, except in the presence of a gas forming organism, and that approximately 78 per cent of all cases show air under the diaphragm.

Operation in acute perforation of peptic ulcer should be speedily performed as possible. The mortality in such cases when operated on within four or six hours is very low, after six to twelve hours the mortality will run 50 to 60 per cent, and after twenty-four hours practically all die.

Operations in the subacute types should be per-

formed early or as soon as a definite diagnosis can be made. After the perforation has been walled off intervention is not advisable as a spreading peritonitis may occur. Intermittent leakage may be detected from time to time with symptoms of subacute peritonitis.

In the vast majority of cases of acute perforated gastroduodenal ulcer, including those that reach the surgeon soon after perforation, simple closure of the perforation, with or without drainage, is the treatment of choice, in early cases the abdomen should be closed without drainage.

Patients so treated who survive immediate hazard have approximately a 60 per cent chance of remaining permanently quite free of gastric complaints, and a 10 to 15 per cent chance of requiring a gastroenterostomy at a later date on account of pyloric obstructions.

The remaining 25 per cent will be comfortable if careful as to diet and general daily routine. Some of them might best be subjected to partial gastrectomy as a curative measure.

Secondary gastroenterostomy, done on account of subsequent pyloric obstruction after a simple closure, should produce uniformly good results. Primary gastroenterostomy does not produce uniformly good results and should not

be done as a routine.

A few cases of acute perforated duodenal ulcers are seen in which adequate closure cannot be affected without producing a pyloric obstruction on account of a large perforation, unusually extensive surrounding induration or both. These cases may justifiable be subjected to immediate gastroenterostomy. Careful closure with use of omentum reinforcement will reduce the cases in which this is necessary to a minimum.

The late results of the Horsely type pyloroplasty in these cases are only fair.

Partial gastrectomy in the presence of an acute perforation is not justified.

A knowledge of the commonest complications causing death is essential to their prevention or early recognition and successful treatment.

The fact that acute diffuse peritonitis is the commonest fatal complication that has not been sufficiently emphasized.

The use of enema in the treatment of peritonitis is a pernicious practise. This fact also needs emphasis.

Opportunity is offered for a further reduction in mortality rate by:

Better preoperative preparation of patients.

Postoperative treatment rigidly in keeping with established principles for the treatment of acute generalized peritonitis.

Efficient disposal of the intra-abdominal foreign material(not peritoneal exudate).

Prompt and thorough search for atelectasis in patients developing early pulmonary complications with immediate institution of treatment when found.

Early diagnosis and prompt treatment of subdiaphragmatic abcess and empyema as late cpmplications.

Restrictions of the use of upper abdominal drains to patients coming to late operation and in whom localized suppuration has occurred.

BIBLIOGRAPHY

1. Allaben,J.E.: Surgical Treatment of Perforated Gastric and Duodenal Ulcer With Report of a Case of Perforated Gastric Ulcer Recovery, Ill. Med.Jour. 16: 146-155, 1909.

2. Atherton,A.: Report of Third Successful Case, Ann.Surg. 40: 686-688, 1904.

3. Balfour, D.C.: Surgical Treatment of Peptic Ulcer, Surg.Clin.of N.Amer. 7: 1420,(Dec.pt.2)1927.

4. Basset,A.: Les Resultats Eloignes Dans Les Ulcers Perfores de L'estomic et du Duodenum,E.C.:Bull,and Mem.Soc.Nat.de Chir., 1: 224-232, 1924.

5. Brenner,E.C.: Perforated Ulcers of the Duodenum: A Report of Fifteen Consecutive Cases, Surg.,Gynec,. and Obst., 34: 370, 1922.

6. Brenner,E.C.: Perforated Ulcers of the Duodenum: A Study of Twenty-seven Cases, Ann.Surg., 86: 393-400, 1927.

7. Brown,K.P.: The Late Results in asSeries of Forty-nine Cases of Perforated Gastric and Duodenal Ulcers, Edinburgh Med.Jour.,n.n. 32: 207-213, 1925.

8. Brown,H.P.: Perforation of Peptic Ulcer: Observation of One Hundred Cases at Pennsylvania and Presbyterian Hospital, Ann.Surg. 89: 209-217(Feb)1929.

9. Brutt,H.:Bacteriologic Consideration of Excision of Perforated Gastric and Duodenal Ulcers, Beitr.z. kiln.Chir. 138: 601, 1926.

10. Buxton,B.H.: Jour.Med.Research. 15: 3-18, 1906 and 16: 17-24, 1907.

11. Caird,F.M.: Perforated Duodenal Ulcer: Collected Reports of Two Hundred Cases, Edinburgh Med.Jour. 11: 405, 1913

12. Cellan-Jones,C.J.: A Rapid Method of Treatment in Perforated Duodenal Ulcers, Brit.Med.Jour. 11: 405, 1929

13. Churchill,E.D.,and MeNeil,Donald: The Reduction in Vital Capacuty Following Operation, Surg.,Gynec., Obst., 44: 483(April,pt.1) 1927.

14. Clerf,L.C.: Personel Communication to the author (Fleming(31)).

15. Colp,Ralph: Perforated Gastric Duodenal Ulcers,N.Y. State Jour.Med. 29: 13-16(Jan) 1929.

16.Cope,Zachary: Treatment of Perforated Gastric and Duodenal Ulcers,Brit.Med.Jour.3: 139(Jan.)1925.

17. Corlette,C.E.: What Makes a Gastric or Duodenal Ulcer Perforate and What Protects It?.Med.Jour.of Australia 2: 587-590(Nov.) 1931.

18. Coryloss,P.N.: Postoperative Pulmonary Complications and Bronchial Obstruction,Surg.,Gynec.,Obst. 50: 795-827(May) 1930.

19.Cutler,E.C.and Newton,F.C.: Perforated Ulcer of the Stomach and Duodenum, Boston Med.and Surg.Jour. 188: 789, 1923.

20. Deaver,J.B.,and Pheiffer,D.B.: Gastric Enterostomy in Acute Perforation Ulcer of the Stomach and Duodenum, Ann.Surg. 73: 441(April) 1921.

21. Deaver,John,B.and Ashhurst,Astley Paston Cooper: Surgery of the Upper Abdomen, 1: 203, Philadelphia,P.Blakiston's son and Co.,1909-13.

22. Deaver,J.B.,and Pheiffer,D.B.: Gastroenterostomy in Acute Perforating Ulcer of The Stomach and Duodenum, Ann.Surg. 73: 73, 1921.

23. Dickenson,J.T.: Report of Case of Perforated Ulcers Lancet 2: 589, 1859.

24. Dineen,P.A.:Acute Perforated Ulcers of the Stomach and Duodenum, Ann.Surg. 90: 1027, 1927.

25.Dunbar,J.: Acute Perforating Peptic Ulcer:An Alysis of 227 Cases, Glasgow Med.Jour. 106:109-113, 1926.

26. Eisendrath,Daniel,N.: Subphrenic Abscess, Jour.Am. Med.Assn. 50: 751-757(March 7) 1908.

27. Engelsing,H.: Zur Frage der Behandlung der Akuten Magen und Duodenal Perforation Deutsche Ztschr. f.Chir. 186: 25-40, 1924.

28. Erdmann,J.F.:Personnel Communication(Fleming(31))

29. Farr,C.E.: Perforating Gastric and Duodenal Ulcer, Ann.Surg. 72: 591-594, 1920.

30. Finney: The Surgery of Gastric and Duodenal Ulcers, Amer.Jour.Surg.n.s. 1: 323-343, 1926.

31. Fleming,B.L.: Acute Perforation of Duodenal Ulcer, Causes of Death and Consideration of Treatment, Jour.A.M.A. 97: 6-11(July-Sept.) 1931.

32. Gibbon,J.H.: Ultimate Results of Operations for Gastric and Duodenal Ulcer, Ann.Surg. 92: 616-619(Oct.)1930

33. Gibson,C.L.: Acute Perforation of the Stomach and Duodenum, Jour.A.M.A. 91: 1006-1008(Oct.6) 1928.

34. Gibson,C.L.: Acute Perforations of Duodenum and Stomach, Boston Med. and Surg.Jour. 189: 425-433, 1923.

35. Graham,E.A.: Surgical Diagnosis,3: 358 Philadelphia, W.B.Saunders Co. 1930.

36. Guthrie,D.: Should Gastroenterostomy Be Performed In the Presence of Ruptured Duodenal Ulcer? N.Y. State Med.Jour. 23: 66, 1923.

37. Hinton,J.W.: The Horsley Pyloroplasty in Acute Perforated Duodenal Ulcers, Surg.,Gynec.,and Obst. 47: 407, 1928.

38. Johnston,L.B.: Acute Perforation of Gastric and Duodenal Ulcers,Internat. Clinics,36s, 2: 145-156, 1926.

39. Klopp,E.J.: Personal Communication to the Author (Fleming(31)).

40. Krainik,R.: Contribution a L'etude du Traitement de L'ulcere Perfore Gastrique et Duodenal,Paris 1922.

41. Kunz,H.: Ueber das Perforierte Magen-duodenalgeschwur und das Perforierte Ulcus Pepticum Jejuni, Arch. f.klin.Chir. 140: 419-446, 1926.

42. Lacey,John: Autopsy Report, Lancet 1; 643, 1880.

43. Lahey,F.H.: The Management of Some Complications Following Abdominal Operations, Jour.A.M.A. 89: 1736 (Nov. 19) 1927.

44. Lennander,K.G.: Temporary Gastrostomie bei Magen-oder Duodenal-gesch,Besonders bei Perfornerten Geschowinen ut Gleichzel Tiger Rentention, Deutschl Ztschr.f.Chir, 92: 297, 1908.

45. Lewiston,R.: Persistance of Pyloric and Duodenal Ulcers Following Simple Suture of an Acute Perforation, Ann.Surg. 72: 595-599, 1920.

46. Lucene: Resultats Eloignes du Traitement des Ulceres Gastro-duodenaux Perfores, Bull.et Mem.Soc.Nat. de Chir. 1: 275, 1924.

47. Luff,A.P.: Collective Investigation into After-history of Gastro-enterostomy, Brit.Med.Jour. 2: 1125, (Dec.14) 1929.

48. Mallock,Archibald: An Early Record of Perforating Duodenal Ulcers.Contributed to Medical and Biological Research. Dedicated to Sir William Osler. 1: 137-145, 1919.

49. Martens,M.: Zur Technik der Operation des Perforierten Magengeschwurs,Deutsch.Med. Wchnschr. 33: 1857,1907

50. Mayo,W.J.: Chronic Ulcer of the Stomach and Duodenum, St.Paul Med.Jour. June 1908.

51. Mayo,W.J.: Duodenal Ulcer,Ann.Surg. 40:900(Dec.) 1904.

52. McCreery,J.A.: Acute Perforated Ulcer of the Stomach and Duodenum, Ann.Surg. 179: 91-99, 1924.

53. Metzger,A.: Contributionna Letude des Ulceres Gastroduodenauz Perfores en Peritoine Libre, Envisages a Deux Points de Vue.Paris These,No.346, 1925.

54. Meyers,K.A.,and Brams,W.A.: Acute Perforation of Gastric and Duodenal Ulcer,A Study of Sixty-two Consecutive Cases, Amer.Jour.Med.Sc. 121: 510-514, 1926.

55. Mills,G.P.: The Treatment of Perforated Gastric and Duodenal Ulcers,Report on Forty Consecutive Cases, Brit.Med.Jour. 1: 12, 1925.

56. Moynihan,Lord:Perforation of Gastric and Duodenal Ulcers,Practitioneer, 120: 137-174, 1928.

57. Moynihan,Lord: Duodenal Ulcer and its Surgical Treatment,Lancet, 2: 1662, 1901.

58. Moynihan,B.G.A.: Abdominal Operations,1: 238, Philadelphia,W.B.Saunders Co., 1926.

59. Moynihan,B.G.A.: On Duodenal Ulcer and Its Surgical Treatment, Lancet 2: 1556-1663 (Dec.14) 1901.

60. Murphy,J.B.: Proctoclysis in the Treatment of Peritonitis,Jour.A.M.A. 52: 1250,(April17) 1909.

61. Newman,Samuel,E.: Continuous Enteroclysis,Jour.A.M.A. 52: 1251,(April 17) 1909.

62. Noehren,A.H.: A Perforation of Gastric and Duodenal Ulcer, Amer.Jour.Surg. 38: 59-64, 1924.

63. Odelberg,A.: Resection of the Stomach in Pefforating Ulcer,Acta Chir.Scandin.,62: 159-166, 1927.

64. Pannett,C.A.: The Surgery of Gastroduodenal Ulceration, London,Oxford Univ. Press, 1926.

65. Poll,E.H.,and Dineen,P.A.: Late Results of Gastroenterostomy for Gastric and Duodenal Ulcers,Ann. Surg. 76: 457-466(Oct.) 1922.

66. Poole,Eugene,and Dineen,P.A.: Late Results of Gastroenterostomy for Gastric and Duodenal Ulcers,Including Acute Perforated Ulcers,AnnSSurg.76: 457(Oct.) 1922.

67. Radoievitch,S.: Du Meilleur Traitement Chirugical des Ulceres Gastroduodenaux en Peritone Libre. Rev. deChir., 63: 161-212, 1925.

68. Richardson,E.P.: Acute and Subacute Perforation of the Stomach and Duodenum at Massachusetts General Hospital, Boston Med.and Surg.Jour, 176:158-165 (Feb.1) 1917.

69. Robson,Mayo: Keene's Surgery, 3: 868.

70. Robson,Mayo: Keene's Surgery, 3: 860.

71. Roper,Arthur C.: Perforated Gastric Ulcer:Operation Forty-four hours After Perforation, Brit.Med. Jour. 2: 785-786 (Sept.19) 1908.

72. Rowlands and Turner: Operations of Surgery, Seventh Ed. 2: 117, London.

73. Sante,L.R.: Massive(Atelectatic) Collapse of the Lung with Especial Reference to Treatment,Jour.A.M.A. 88: 1539(May 14) 1927.

74. Singer,Harry A.: The Use of X-ray in the Diagnosis of Acute Abdominal Affections, Med.Clin.of N.Amer. 18:(pt 2) 1421-1428, 1935.

75. Singer,Harry andVaughan,Roger: The Formes Frustes Type of Peptic Ulcer,Surg.,Gynec.,and Obst. 50: 12-14, 1930.

76. Singer,Harry,A.and Vaughan,Roger:Treatment of Formes Frustes type of Perforated Peptic Ulcer, Surg. Gynec., and Obst. 54: 945, 1932.

77. Singer,Harry,A.: The Diagnosis of Perforated Peptic Ulcer, Internat.Clinics 3: 45th Series 117-143, Sept. 1935.

78. Smith,M.S.: Perforated Ulcer of the Duodenum,Lancet 1: 895, 1906.

79. Southam,A.H.: The Treatment of Perforated Gastric and Duodenal Ulcer, Brit.Med.Jour. 1: 556 1922.

80. Stewart,G.D.:and Barber,W.H.: Acute Perforating Ulcer of the Stomach or Duodenum, Ann.Surg. 75: 349-355, 1922.

81. Urrutia,L.: Late Results in Perforated Gastroduodenal Ulcers, Ann.Surg. 90: 73, 1929.

82. Wechsler,B.B.: AnApparatus to Keep Enteroclysis Solution Hot, Jour.A.M.A. 52: 1251(April 17) 1909.

83. White,W.C.,and Patterson,H.S.: Late Results of Simple Suture in Acute Perforation of Duodenal Ulcer, Ann.Surg. 94: 242-255, (Aug.) 1931.

84. Wilson,John L.: The Diagnosis and Treatment of Perforated Gastric Ulcers with the Report of Two Cases, New Orleans Med.and Surg. Jour. 74: 473-479, 1921-22.

85. Williams,H. and Walsh,C.H.: Treatment of a Perforated Peptic Ulcer, Lancet 1: 9-12 (Jan. 4) 1930.