CHAPTER X.

DISEASES OF THE PERITONEUM.

INFLAMMATIONS OF THE PERITONEUM.

Inflammations of the peritoneum are the most important lesions which can affect it, and which, on account of its great extent and capacity for absorption, are dangerous to the life of the individual. The various inflammations may be classified according to their pathological appearances, as fibrinous, serous, suppurative, and gangrenous peritonitis; or they may be classified according to their origin, as infectious and non-infectious or idiopathic peritonitis. They may also be separated into acute and chronic, circumscribed and diffuse, or general peritonitis. This last distinction, according to the extent of the inflammation, is of practical importance with reference to prognosis and treatment, for a patient suffering from a well-defined limited peritonitis is in quite another condition from one who is suffering from an inflammation already involving a considerable part of the peritoneum and rapidly extending further. In the first case the local symptoms are prominent, while in the second they are quite obscured by the severer general symptoms. The term general peritonitis or diffuse peritonitis is somewhat misleading. Repeated post-mortem examinations have shown that certain portions of the abdominal cavity are usually protected by adhesions from even a widespread peritonitis. Hence it is better to discard the term general peritonitis, and to use instead the term diffuse or extending peritonitis, meaning thereby that the inflammation has already affected a considerable portion of the peritoneal cavity and has a tendency to spread still further. Just how far inflammation has spread in any particular case it is not easy to determine. Mikulicz recognizes three forms of diffuse peritonitis: a diffuse septic peritonitis, a gangrenous peritonitis, and a progressive fibrinopurulent peritonitis.

In most cases of peritonitis inflammation extends to the peritoneum from some adjacent organ. A primary idiopathic peritonitis in the strict sense is of the rarest occurrence; indeed, a careful examination of the autopsy records of the cases which formerly were diagnosticated as acute primary will oftentimes show the starting-point of the inflammation to have been in the disease of some abdominal organ.

Many writers still retain the term idiopathic as applied to cases of chronic peritonitis in which no starting-point for the inflammation can be found in disease of any abdominal organ, but even here the term should be used with the distinct understanding that failure to find a

starting-point for the inflammation outside of the peritoneum does not prove that the trouble actually started in the peritoneum itself.

Diffuse Peritonitis.—Pathological Anatomy.—The pathological appearances of acute peritonitis vary in different patients, and even in the same patient, so that one portion of the abdominal cavity may differ from another. Inflammation of the serous membrane manifests itself by injection of the vessels and a fibrinous exudate upon its surface. The shiny appearance of the membrane is lost, and instead it looks cloudy and rough. If a migration of leucocytes is added, the fibrinous exudate takes on a purulent character. If the inflammation is of a mild degree, the tissue-cells of the serosa are stimulated to growth, and there will result a new formation of bloodvessels, adhesions and thickening of the peritoneum. In some cases, both mild and severe, the fibrinous exudate may so stick together opposing surfaces as completely to shut in the inflamed area. In other cases the process spreads through the fibrinous exudate and involves extensive areas of the peritoneal surface. The subserous tissue becomes strongly œdematous, and therefore the intestinal walls, the omentum, mesentery, etc., appear swollen.

The fibrinous exudate is usually accompanied by a serous exudate which remains clear if the inflammation is an aseptic one, but is clouded by migrated leucocytes as soon as bacteria begin to grow in it. The serous exudate may be stained by an admixture of blood. If the intestinal bacteria and other bacteria are added to the exudate, it will undergo putrefaction, possibly even to the extent that putrefactive gases are formed. Suppurative inflammation of the peritoneum may involve in a short time the greater portion of the membrane. In other cases it advances step by step, being more or less limited by adhesions. In this manner the process may be stopped altogether and take on a chronic form (abdominal empyema). Sometimes the inflammation in different portions of the abdomen will take on a different character. Thus one pouch may contain pus and another one more or less serum, being separated by fibrinous adhesions.

Etiology.—Peritonitis may be infectious—that is, produced by the influence of pathogenic bacteria and other toxins; or it may be aseptic, brought about by mechanical or chemical lesions. The first type is more frequent and far more dangerous. It is usually due to one of the following named kinds of bacteria: streptococcus and staphylococcus, bacterium coli, and, more rarely, gonococcus, pneumococcus, and others. The infection is usually a mixed one—that is, due to more than one bacterial species.

Wegner, Grawitz, and others have shown that a healthy peritoneum can endure without injury a certain quantity of fluid containing pathogenic organisms. If the surface-cells of the peritoneum are injured and its power of absorption is thereby limited, the same quantities of bacterial fluid will set up suppurative peritonitis. Mechanical and chemical injuries, the presence of a great quantity of toxins or of foreign bodies, such as fecal matter or blood-clots, enable the pathogenic

bacteria to multiply in spite of the protecting action of the peritoneum. These principles have been established by experiments upon animals and are strictly in accord with clinical observations upon man. For example, the results of abdominal operations are far better than they were at the time when operators used strong antiseptic solutions in the abdominal cavity, and were not so careful as they are now to avoid the mechanical irritation of sponging, etc.

Experience has shown that chronic inflammation of the peritoneum, such as occurs about the female pelvic organs and in the neighborhood of the cæcum, reduces the resorbtive capability of the peritoneum.

Infected organisms may enter the abdominal cavity in a variety of ways. They may be introduced through a wound. In women they may pass through the open ends of the Fallopian tubes. They may reach the peritoneal cavity or any organ which is covered with peritoneum. The intestine always contains a great number of bacteria, ferments, and toxins, which in disease may extend to the liver, spleen, and pancreas. Traumatism or an ulcerative process may be the means of transplanting these bacteria into the abdominal cavity, or the bacteria themselves may pass through the diseased intestinal wall without actual perforation. This is impossible as long as the intestinal wall is healthy. These principles hold true not only for the intestinal wall, but also for the walls of the gall-bladder and the Fallopian tubes. Whether ferments and toxins can also pass through the diseased or necrotic intestinal wall has not been determined. Neither is it known whether microbes which are circulating in the blood can set up an infectious peritonitis without previous disease of the intestine.

Infectious peritonitis is usually of a purulo-gangrenous character. If the disease exists in a very acute form, to which Wegner gives the name peritoneal sepsis, death may occur very early on account of overloading of the blood with septic material. Under such circumstances the serosa shows only a slight injection, while its surface is clouded by a delicate deposition of fibrin and the peritoneal cavity contains only a small amount of cloudy or hemorrhagic exudate in which there are great numbers of bacteria.

Aseptic Peritonitis.—Chemical or mechanical irritation may produce an aseptic peritonitis of the fibrinous, serous, or hemorrhagic type. Such inflammation tends to heal with the formation of adhesions, and only becomes suppurative in case pathogenic organisms enter the peritoneal cavity.

A good illustration of chemical peritonitis is that produced by the rupture of an aseptic ovarian, echinococcus, or other cyst, or by the discharge of aseptic bile or urine into the peritoneal cavity. Strong antiseptics, such as solutions of carbolic acid or mercuric chloride, and irritants, such as croton oil, turpentine, or tincture of iodine, will also inflame the peritoneum. A discharge of blood into the peritoneal cavity acts as an irritant just as it does in the cavity of a joint, and sets up an aseptic inflammation resulting in adhesions. Repeated injections of air and the introduction of aseptic foreign bodies set up

a fibrinous peritonitis in animals, while the presence of ligatures, cauterized areas, etc., give rise to a chronic adhesive peritonitis in man.

An aseptic peritonitis may also be caused by a contusion of the serosa without other injury; also by chronic obstruction of the circulation, as caused by twisting the pedicle of a tumor, or twisting or other obstruction of an intestinal loop, provided that the injury does not make the intestinal wall pervious to the bacteria within the intestine. Under such circumstances the serosa is reddened and covered with a layer of fibrin, and the peritoneal cavity contains serous or serohemorrhagic fluid without bacteria. The tendency of the disease is toward recovery as long as bacteria are absent. If they are introduced, they find in the exudate a most favorable soil for their rapid development.

Sources of Peritoneal Infection.—Any organ which is partially covered by peritoneum may give rise to peritonitis, but the two chief sources of this inflammation are the alimentary canal and the internal female genital organs.

Any part of the alimentary canal from the cardiac end of the stomach to the extraperitoneal portion of the rectum may be the starting-point for a peritonitis. Peritonitis which starts from the intestine is especially important on account of its frequency and on account of its virulence. This is due to the number of germs within the intestine, and especially to the different forms of bacterium coli, as well as to the presence of ferments and toxins in abundance. The experiments of Cushing and Livingood have shown that in the upper portion of the intestinal canal bacteria are relatively scanty, and that fasting will often produce a sterile or nearly sterile condition of these parts. This fact helps to explain the difference in virulence of different attacks of peritonitis.

Perforation may take place in typhoid, dysenteric, tuberculous, carcinomatous, or syphilitic ulcer. The more rapid the ulceration the greater the risk of diffuse peritonitis. If the ulceration goes on slowly, an adhesive inflammation may be set up which will either prevent rupture into the peritoneal cavity or will be able to encapsulate the discharge.

Traumatic rupture of the alimentary canal is next in importance to ulceration. The effect of the discharge of gastric or intestinal contents into the peritoneal cavity is a violent inflammation with general symptoms. A traumatic perforation is somewhat more favorable in this regard than an ulcerative one, since contraction of the muscles of the intestine may block or narrow the opening in such a manner as to prevent or limit escape of the intestinal contents. This is, of course, only possible in case of a small perforation. Small lesions of the large intestine are less dangerous than those of the small intestine or stomach, because the thicker fecal contents of the former are less likely to escape.

Even when no perforation exists, an inflammatory or ulcerative process of the gastric or intestinal wall may give rise to peritonitis,

since microbes may develop in the diseased tissue until they reach the serosa and spread upon its surface, even though no demonstrable perforation exists.

The commonest cause of peritonitis is disease of the vermiform appendix. In the majority of cases the inflammation is limited by adhesions which are formed around the diseased organs. In other instances perforation is followed by such a violent inflammation that the whole peritoneum becomes infected. An encapsulated appendical abscess may subsequently rupture and set up diffuse peritonitis. Sometimes the transverse colon and omentum oppose a barrier which prevents extension of the inflammation to the upper portion of the abdomen.

The next most common cause of peritonitis is perforation of a gastric or duodenal ulcer. Such an ulcer is usually of the simple type. It may, however, be carcinomatous, or the peritonitis may be due to diffuse inflammation of the gastric wall. Typhoid and tubercular ulcers of the small intestine and tubercular and syphilitic ulcers of the large intestine may also lead to perforation. Foreign bodies which have been swallowed may perforate the wall of the stomach or intestine. Congenital and acquired diverticula of the intestine may become inflamed and give rise to peritonitis. Constrictions of the intestine, by producing dilatation and ulceration of the proximal mucous membrane, may lead indirectly to peritonitis.

Any obstruction of the intestinal canal by which the intestinal wall is injured and the circulation through it shut off may easily bring about perforative peritonitis. This is true of external and internal hernia, obstruction by bands, twists, and invaginations. Within a certain time after such an accident a sero-hemorrhagic exudate results, and a little later bacteria will be able to pass through the intestinal wall even though it is not completely gangrenous. Closure of the mesenteric vessels by emboli, thrombi, or injury will produce intestinal gangrene and peritonitis.

The uterus, ovaries, and tubes are frequent sources of peritonitis. The affections which may extend in this manner are puerperal infection, and catarrhal and suppurative inflammations of the mucous membrane of the uterus and tubes, especially when such inflammation is due to a gonococcus. In puerperal peritonitis the infecting germ is usually a streptococcus. Traumatic or spontaneous rupture of the uterus or the vagina during birth, perforation of the uterus by instruments, and the injection of fluid into the uterus under conditions which allow it to pass through the tubes into the abdominal cavity, are other causes of peritonitis.

Peritonitis may develop from the liver secondary to such infections as cholecystitis, cholangitis, or hepatic abscess or echinococcus, although if the echinococcus cyst is not suppurating the peritonitis will in that case be an aseptic one.

Inflammation and suppuration of the pancreas may give rise to peritonitis. The disseminated fat necrosis so often spoken of in connection

with pancreatitis is probably the result of the digestive power of the pancreatic juice.

In malarial and typhoid fever infectious material may reach the peritoneum through the spleen. Embolic abscesses of the spleen, and suppurating echinococcus cysts of this organ may also cause peritonitis. Twisting of the pedicle of a wandering spleen may cause peritonitis, but more often causes a chronic inflammation of the serosa of the spleen itself—a perisplenitis.

The lymph-glands of the mesentery may be a source of infection; for instance, if they suppurate as a result of absorbed poison in typhoid fever. Suppurating thrombi in the bloodvessels (pylephlebitis) may also cause peritonitis. Infection of the umbilical wound in a newborn child may spread to the peritoneum or peritonitis may follow a suppurating inflammation of the umbilical artery. Suppuration proceeding from the kidney usually affects the retroperitoneal tissue and may secondarily involve the peritoneum. Traumatic and ulcerative perforation of the bladder may cause peritonitis, and abscess of the prostate or of the seminal vesicle; and cellulitis due to urethral stricture, etc., may extend into the subperitoneal tissue and thus affect the peritoneum. In rare cases gonorrhœa in the male is followed by peritonitis, but it is not known by what channel the gonococci reach the peritoneum. Equally rare is an extension of inflammation from the testicle along the vas deferens to the retroperitoneal tissue and the peritoneum. If the tunica vaginalis communicates with the peritoneal cavity, irritating injections into the former may set up inflammation in the latter. Suppuration in the pleura or pericardium may extend to the peritoneum just as suppuration in the peritoneum may extend to these thoracic serous cavities. Erysipelas and cellulitis of the abdominal wall, and suppuration of the spine or ribs or pelvic bones, are lesions which may produce inflammation of the peritoneum.

In the majority of cases peritonitis is due to an extension of infection from some neighboring tissue, or the infection may reach the peritoneum through the bloodvessels. Thus there is a hæmatogenous peritonitis occurring in connection with acute articular rheumatism and nephritis. The occurrence of peritonitis in connection with acute infectious diseases has occasionally been mentioned, but the accuracy of such observations is doubtful.

Benda, who made autopsies in 446 cases of acute diffuse peritonitis, assigned as the starting-point:

Appendix	115 times.
Stomach and duodenum	68 "
The rest of the intestine	118 "
Female genitals	81 "
Gall-bladder	10 "
Kidney and urinary bladder	10 "
Pancreas	2 "
Spleen	1 time.
Unknown	35 times.
Postoperative	4 "
Hæmatogenous origin (nephritis and acute articular rheumatism)	2 "

Symptoms.—Although the causes of peritonitis are so various the clinical picture of the disease is uniform. Peritonitis is almost always secondary to some injury or affection which has already given rise to symptoms before the peritonitis develops. These symptoms may gradually increase in intensity as the inflammation extends to the peritoneum, or the symptoms due to the peritonitis may appear suddenly in a most striking manner as contrasted with the previous mild symptoms. Indeed the symptoms of the primary affection may be slight or altogether absent, so that the patient has not considered himself sick until the onset of the peritonitis. An example of this is the perforation of some intestinal organ which up to that time was apparently in a healthy state.

The peritonitic symptoms vary in degree according to the intensity of the inflammation, and according to the amount of resorption of poisonous material (toxæmia, septicæmia). The severity of the clinical symptoms by no means corresponds to the amount of pathological change of the peritoneum; thus the evidences of inflammation seen after death may be slight although the patient has suffered from the severest symptoms, while in other cases it will happen that the whole peritoneal cavity becomes filled with pus although the symptoms are of mild character. It is not always possible to determine from the clinical symptoms what the nature or extent of the peritoneal inflammation may be.

A patient with peritonitis usually feels very sick. The disease often begins with a chill. The patient cannot keep himself upright. His appearance is anxious, he is restless, and as the disease progresses the skin of the face becomes wrinkled and the nose more pointed, while the eyes sink in. This is in consequence of the decrease of fluid in the body. Still later the face and extremities become cyanotic on account of the sluggish circulation and insufficient aëration of the blood. The patient lies upon his back and avoids any motion which disturbs the abdomen. The knees are often drawn up in order to relax the abdominal muscles. The head and arms are tossed restlessly about.

Consciousness is at first not affected. Later it may be clouded by toxæmia or the patient may become delirious; frequently just before death the state of mind of the patient is a happy one—a most unfavorable symptom. He expresses himself as free from pain and feeling better, although these statements are in sharp contrast to the sunken face, cyanosis, coldness of the extremities, difficult respiration, and the thread-like pulse which shows that the end is near.

The voice is light in the beginning of the disease, as the patient avoids deep respiratory movement; later the voice may become hoarse. Respiration is from the beginning superficial, being costal rather than diaphragmatic, and consequently the rate of respiration is much increased. The character of the respiration is at first determined by the abdominal pain, and later by meteorism, which presses up the diaphragm and makes easy respiration impossible. At

the close of the disease the respiration becomes still more superficial and of a spasmodic, snapping character, which is one of the worst symptoms of peritonitis.

In the stormy onset of peritonitis the pulse is small and depressed on account of the shock; later its rate is increased until it is difficult or impossible to count it. The tension is low and is only slightly increased by the pulse-waves. The inflammation of the serosa stimulates the vagus, the meteorism opposes a mechanical obstacle to the blood circulation, and the poisons with which the blood is surcharged depress the heart muscle. Sometimes the disease progresses to a certain distance before the characteristic rapid pulse is manifest, but usually the pulse-rate is increased from the first. If at the same time the temperature is low, so that the lines indicating pulse and temperature cross upon the chart, a clear indication of peritonitis is present.

The temperature is variable. In those cases which develop in the course of septic disease (puerperal fever) the temperature is usually high. Fever is often absent in severe inflammation with a purulent and gangrenous condition of the peritoneal cavity; for example, after perforative peritonitis. Hence the absence of fever is no proof of the non-existence of peritonitis. Lennander and others have shown that the difference between the axillary and rectal temperature is greater in peritonitis than it is in other conditions.

The activity of the alimentary canal is suspended. The patient has no desire for food, but suffers often from a burning thirst while the stomach rejects even a few drops of fluid. The tongue is heavily coated, and in septic cases is brown and dry.

The excretion of urine is diminished, since much fluid is lost by vomiting and little or none is absorbed through the stomach. In consequence the urine is scanty, concentrated, and often contains traces of albumin and indican.

In addition to these general symptoms there are local symptoms which are more or less directly dependent upon the inflammation of the serous membrane. While the healthy serous membrane which covers the viscera has little sensitiveness, as can be demonstrated at abdominal operations when local anæsthesia is employed, it becomes very sensitive when inflamed; consequently abdominal pain is an almost constant symptom of peritonitis. Indeed, this pain, which may be so great as to produce a condition of extreme shock, is usually the first alarming symptom. It is most marked in the affected region; thus, in case of gastric perforation it is found in the epigastrium or left hypochondrium. In perforation of the appendix it is found in the right iliac fossa, and in the case of pelvic inflammation it is situated in the pelvis. As the peritonitis develops the pain spreads over the whole abdomen, although it is frequently most intense in the seat of the original trouble, and is most increased when pressure is made upon that region. Sometimes a patient is unable to localize the pain, which he describes as being situated around the umbilicus. In the beginning of the trouble this localization of the pain is a fairly certain guide to the

affected organ ; later in the disease such an inference is less valuable. The intensity of the pain varies according to circumstances. In rare cases it is altogether wanting, so that the diagnosis of peritonitis is made with a good deal of doubt until a purulent exudate makes it a positive one. The pain is usually constant, or it can be produced by the lightest touch upon the abdomen, by the slightest jarring or motion of the body, and especially by any intestinal movement. Palpation of the abdomen in diffuse peritonitis is everywhere painful. The intenssity of the pain is often much diminished when an abundant exudate has formed. As the patient becomes more and more intoxicated by the poison of the disease the pain, whether spontaneous or caused by pressure, is noticed less and less, and often not at all.

The second local symptom of inflammation of the serosa is the formation of an exudate whose quantity and character vary greatly. In general it may be said that the more violent the attack of peritonitis and the more marked the symptoms of general sepsis, the less will be the local reaction of the serosa. Under such circumstances the membrane is injected and cloudy and covered with a thin coating of fibrin, while the peritoneal cavity contains only a few spoonfuls of cloudy grayish-red fluid situated in Douglas's pouch. Such are the conditions found in the very worst cases of septic peritonitis in which the peritoneum does not have time to reach a more marked condition of inflammation. If the acute attack is less violent, an exudate will be formed which is at first purulent and later may become gangrenous. The intestinal coils become matted together with the abundant deposit of fibrin. There are also cases of acute infectious peritonitis in which the exudate has a serous character. A non-infectious chemical or mechanical peritonitis is accompanied by a serofibrinous exudate which may have a hemorrhagic character.

The quantity of exudate may be very small, or it may be very large—several litres (quarts). On account of the meteorism which is usually present, it is difficult to demonstrate the presence of small quantities of fluid, and even larger quantities may be overlooked since the distended and adherent intestinal coils float upon the fluid. The presence of fluid is shown by dulness on percussion over the most dependent portion of the abdomen, the dull area shifting its location with changes in the position of the patient. It should be borne in mind that coils of intestine which are full of fluid will sink to the lowest part of the abdomen, while coils which are distended by air will naturally rise above them. On this account it is not always possible to say whether dulness on percussion is due to free fluid or to fluid in the intestinal coils. If the dull area shifts readily as the position of the patient is changed, it is probably due to free fluid. If pressure with the fingers upon the dull area produces a gurgling sound, the dulness is probably due to fluid in the intestine. But here again one must remember that there may be free gas as well as free fluid in the abdomen after perforation. Fluctuation is a sign not usually obtained in the acute stage of peritonitis on account of the rigidity of

the abdominal muscles. In a chronic stage of fibrinopurulent peritonitis, when large abscess cavities have formed between adherent coils of intestine, fluctuation is not uncommon. A finger placed in the rectum or in the vagina will often demonstrate the presence of a deeply placed collection of fluid.

Aspiration for the purpose of diagnosis is dangerous because a puncture may be made in the distended and inflamed intestinal loop which is not readily closed. Furthermore, if the aspirated fluid is straw colored, thin, and has a strong fecal odor, it is often difficult to say whether it is simply an exudate or an exudate mixed with the contents of the small intestine. If under the microscope it is seen to contain a great number of pus-cells, it is probably an exudate ; whereas if it contains portions of food, a gastric or intestinal perforation must exist.

Sometimes a fibrinous exudate manifests itself by a soft crepitus which may be felt or heard over the surface of the liver, or the surface of the cyst, or possibly elsewhere. This crepitus is similar to that produced in a dry pleurisy.

Gas may enter the peritoneal cavity from the stomach or intestine in the case of perforation ; or in rare cases as a result of fermentation in the exudate. The gas-bubbles have a tendency to rise to the apex of the abdomen, but it is extremely difficult to say before the peritoneal cavity is opened whether the resonant area which shifts with changes in the position of the patient is due to free gas or to gas in the intestinal coils.

While pain and the formation of an exudate are the direct local results of inflammation of the serosa, vomiting and intestinal paralysis may be described as its indirect local results.

Vomiting is a cardinal symptom of peritonitis which is rarely absent. Nausea, eructations, and vomiting form a group of symptoms which are usually present at the very beginning of the inflammation. However, there are cases of acute perforation of the stomach in which vomiting is absent. The first vomitus consists of the food in the stomach and later of mucus and bile which is brought up after a terrible retching. Every attempt to take food, or even a small quantity of fluid, excites renewed vomiting. In other cases vomiting occurs at long intervals, so that whatever has been taken into the stomach becomes mixed with mucus and bile and the whole is thrown violently out in one act of vomiting. Absorption from the stomach is suspended, so that medicines which are administered are without effect. If the intestine is also paralyzed, vomiting may be of a fecal character, since the contents of the upper coils of small intestine may be forced back into the stomach. Between the attacks of vomiting the patient is often disturbed by a spasmodic hiccough.

The vomiting which occurs in peritonitis is of a reflex character, being caused by the inflamed condition of the serosa ; consequently it is just as prominent a symptom when the inflammation is situated in some portion of the abdomen other than the epigastrium.

Hiccough is another reflex symptom, and is explained by the fact

that sensory fibres of the phrenic nerve are distributed to the peritoneum, and that through them the motor fibres of the diaphragm are excited to action. Inflammation of the peritoneum affects the whole intestinal tract as well as the stomach. The intestinal muscles are paralyzed and peristalsis stops. It is true that in the beginning of the disease there is still some intestinal action, so that a patient often complains of pain caused by the passage of gas from one intestinal loop to another. Even when the peritonitis is well developed auscultation may show that there is some movement of gas and feces, but such slight motions being limited to small portions of the intestine are of no assistance in moving the whole fecal stream downward. At a later period of the disease all peristalsis ceases.

The failure of the passage of gas and fecal matter through the anus is an important early symptom of acute peritonitis. Perhaps in the beginning of the disease matters which have collected in the rectum may be ejected, but afterward there is no movement of the bowels. Cathartics increase the pain but have no other result. In some forms of peritonitis, especially in the puerperal septic form, the patient is troubled with diarrhœa. Stoppage of the fecal stream brings about abnormal fermentation and gas formation, so that the paralyzed intestine is greatly distended. This meteorism stretches the abdominal wall and presses the diaphragm upward, and by these means greatly interferes with respiration and blood circulation. The liver is pushed upward until its sharp lower edge is directed forward and the area of hepatic dulness is greatly decreased. The distended intestine may be forced into an old hernial sac, if such exists, and this has been known to mislead the surgeon into thinking that the peritonitis was due to strangulated hernia. Examination will show that such a hernia is easily reduced, while reduction of a strangulated hernia is, of course, impossible. Intestinal paralysis and meteorism are not always present in the beginning of peritonitis, but the cases are few in which they do not later develop and become marked symptoms. There are, however, well-authenticated instances in which peristaltic action has been preserved throughout the disease. The intestinal paralysis is in part due to reflex action, and is in part the direct effect of the inflammatory swelling of the intestinal wall.

The function of the bladder is often interfered with in case the peritoneum which partly covers its fundus is inflamed. Micturition may be painful or it may be quite impossible.

The anterior abdominal muscles are strongly contracted. This again is a reflex contraction due to the pain arising from the inflamed serosa. In the beginning of a peritonitis, especially in cases of perforation, the abdominal muscles are so firmly contracted that they seem to be drawn inward. This contraction continues even after the abdomen has become distended, and it is not in the power of the patient to relax his abdominal muscles.

Symptoms of Special Forms.—The symptoms given above are the typical symptoms of a diffuse extending peritonitis. They vary more

or less according to the origin and the intensity of the process. If the inflammation is accompanied by general sepsis, as is usually the case in puerperal peritonitis and in peritonitis following operation, the symptoms of a general intoxication overshadow those of the peritonitis itself. The patient is stupid, the heart action is extremely rapid and weak, respiration is snappy, and the tongue is dry—all symptoms of a severe septic poisoning. The local signs of peritonitis—pain, exudate, and vomiting—are less marked. Intestinal paralysis is usually present, though there may be diarrhœa, a result of the poisonous toxins.

Peritonitis which is due to the sudden rupture or traumatic opening of some hollow abdominal organ begins with an extremely painful attack. The patient has the feeling that something within the abdomen is tearing apart. Shock is a marked symptom. The abdominal muscles are firmly contracted, the abdomen is sunken, the pulse is small and depressed. As the symptoms of shock pass off those of beginning peritonitis show themselves, and there are vomiting, general abdominal pain, meteorism, etc. If the stomach or intestine is perforated, free gas may accumulate in the peritoneal cavity. Vomiting may be absent after perforation of the stomach.

In counterdistinction to an acute attack described above, the inflammation may spread slowly and gradually over the whole peritoneum or a greater part of it. This is the progressing fibrino-purulent peritonitis described by Mikulicz. In this form of disease there will at first be more or less local pain, which will spread until the whole abdomen is extremely sensitive. Such is the case if an encapsulated abscess breaks through the protecting adhesions and the inflammation develops in an increased area of the abdomen. Sometimes inflammation situated below the umbilicus will be shut off by adhesions at the level of the transverse colon. Such an inflammation will produce the usual signs of diffuse peritonitis, because nearly all of the small intestine is involved. If, however, the inflammation is limited to the upper part of the abdomen, general symptoms may be wanting for the reason that so little of the small intestine is affected.

An acute purulent inflammation may pass into a chronic form. If such change occurs, the violence of the symptoms subsides and pain and vomiting become less or occur only at longer intervals. The local signs of a fluid exudate become more and more marked, and the patient often shows in the remittent type of fever the fact that there is an encapsulated abscess in the abdomen.

Well-localized inflammation in the peritoneum, such as occurs in connection with appendicitis, perimetritis, etc., is marked by diffuse pain, vomiting, meteorism, and rapid pulse. These general symptoms subside more or less in a day or so, and leave only such symptoms as are due to localized inflammation in the neighborhood of the diseased organ. An early operation in such cases will reveal an encapsulated abscess, while the rest of the peritoneum may contain an inflammatory serous exudate which is possibly sterile and hence may be resorbed.