local or *circumscribed*, *general* or *diffuse*. A localization of the inflammation in the pelvis is generally spoken of as a *pelvic* peritonitis, in the neighborhood of the appendix as *peri-appendiceal*, over the spleen and liver as *perisplenitis*, *perihepatitis*, etc. According to the nature of the exudate there may be distinguished a *serous*, *fibrinous*, *purulent*, *hemorrhagic*, and a *putrid* or *gangrenous* peritonitis. Besides these pure types a variety of combinations, such as sero-fibrinous, sero-purulent, fibrino-purulent, sero-hemorrhagic, etc., occur.

Within certain limits the anatomical picture presented in all cases of peritonitis is essentially the same, but the appearances seen at autopsy or operation may vary greatly according to the chronicity, severity, and extent of the inflammation, the amount and character of the exudate, etc. The picture of a severe *acute general peritonitis* is, however, very characteristic. The abdomen is distended and tympanitic over the dome and dull over the flanks. When the cavity is opened the distended coils of intestine rise up through the opening, and an escape of gas may take place, even in non-perforative peritonitis. The serous surfaces are cloudy, in slight cases having the appearance of glass which has been breathed upon. Both the parietal peritoneum and the outer surface of the coils may be more or less reddened, injected, or even show ecchymoses. When the coils are lifted up and separated the reddened areas appear to be arranged in bands along the intestine. As a rule these reddened bands correspond to the spaces between the coils at those places at which the sides of the coils do not touch. The vessels of the serosa of these spaces are usually markedly hyperæmic, while at those points at which the coils do touch the vessels are less congested. According to Wilks and Moxon these spaces, which are triangular on cross section, form a system of communicating tubes through which the exudate may spread; and because of the lower resistance over these free surfaces absorption of the exudate is carried on here to a greater extent than elsewhere over the peritoneal surface. The loss of lustre and the cloudiness of the serous surfaces are due partly to the degeneration and desquamation of the endothelium, and partly to a layer of fibrin which is deposited over the surface. The latter may vary greatly in amount; in some cases the fibrin film may be so thin that it can be seen only as a slight cloudiness when examined by oblique light, or it may be made evident by rubbing off the delicate coating with a damp cloth or by scratching the serosa with the edge of the knife. In other cases the serosa may be covered with a thick, grayish, yellowish, yellowish-gray or reddish-gray exudate which may extend over the coils in a sheet, or in strings or bands of varying size, or appear as flakes scattered over the dull, lustreless surface. The fibrin may bind the coils more or less firmly together; in acute cases such adhesions are easily separated by the fingers, but in older cases the adhesions become organized to a greater or less extent, and so firm that they cannot be torn. The spleen and liver may become entirely covered by fibrinous sheets, or tags and flakes of fibrin may be scattered over their capsules. The amount of fibrin may be much greater in some parts of the cavity than in others, and by causing adhesions between neighboring structures may shut off portions of the cavity and confine the fluid exudate to certain regions.

Free fluid is usually present in the dependent portions of the cavity, in the flanks, pelvis, and the pockets between the coils. The amount varies greatly, sometimes many litres (20–40) causing a great distention of the abdomen, while at other times only a small amount is present. The fluid is always more or less cloudy or opaque, but varies in character according to the nature of the exudate, the character of the infection, the occurrence of perforation, etc. It may be serous, purulent, hemorrhagic, fibrinous, putrid, etc. In the great majority of cases it is fibrino-purulent, a pure purulent character rarely occurring, although a purulent character usually predominates. Rarely is it serous or sero-purulent. It is very often putrid (perforative peritonitis), and is then sanious, greenish, or grayish, having a gangrenous odor, and may contain gas bubbles. Except in the case of tuberculosis or carcinoma a hemorrhagic exudate is very rare. The distribution of the exudate is generally very irregular owing to fibrinous adhesions. It usually collects in greatest amounts in the pelvis, flanks, splenic and hepatic regions, and in the deeper parts of the two hypochondriac regions. It often becomes encapsulated by fibrinous adhesions between the coils themselves, or between these and the abdominal wall or some one of the abdominal organs. The omentum often plays an important part in the encapsulation of exudates of small size in the appendix region and in the neighborhood of perforating ulcers of the intestine. Large collections of exudate are often found between the liver and the diaphragm; when of a purulent character and encapsulated from the other portions of the cavity the condition is often spoken of as a *subdiaphragmatic abscess*. Gas is particularly likely to collect in this region. In some cases the liver may be pressed tightly against the diaphragm, its diaphragmatic surface flattened and dry. The exudate often follows along the tubular spaces between the coils, so that the exudate from a pelvic peritonitis may pass under the lower coils of the ileum into the cæcal region and upward along the ascending colon to the right hypochondrium, then along the lesser curvature of the stomach to the cardiac end. In these regions the exudate may collect in large amounts, while elsewhere in the cavity there may be scarcely a trace of peritonitis. Not infrequently the exudate may become completely shut off from the cavity by fibrinous adhesions, which, becoming organized, finally form a fibrous connective-tissue capsule enclosing the exudate, which is either absorbed, organized, or undergoes inspissation or calcification. As mentioned above, the omentum plays an important rôle in such encapsulations, which are most often seen in the neighborhood of the appendix and the female pelvic organs. In the case of a perforative peritonitis the exudate may contain, in addition to the purely inflammatory products, also stomach or intestinal contents, bile, urine, etc.

In the more severe cases the entire intestinal wall may be involved, becoming swollen, œdematous, and easily torn. Even in the light cases the subserosa is œdematously swollen and may be easily stripped off from the muscularis. The muscle coats may be completely paralyzed; hence the occurrence of marked tympanites, general or local. On opening the stomach and intestines the mucosa is usually found to be pale, swollen, and œdematous. Likewise the superficial portions of the liver, spleen, gall bladder, etc., are œdematous and swollen, often appearing as if macerated. The gall bladder and the lower edge of the liver in particular may show an extreme œdema. The tissues of the mesentery are swollen, moist, and more easily torn than normally. The mesenteric and retroperitoneal glands are usually pale, soft, and enlarged. Even the muscles of the abdominal wall may be pale and œdematous, and the parietal peritoneum can be easily stripped from its attachments. The omentum is swollen, injected, and œdematous. It is very frequently rolled up into a thick mass, but in the case of surgical or perforative peritonitis it is usually more or less adherent to the site of injury. It may be covered with a thick layer of fibrin and may aid in the encapsulation of the exudate. The diaphragm is usually pushed up to the third or even second rib; the lungs show partial atelectasis, particularly the lower lobes; and the heart is pushed upward and more or less to one side.

Although the changes just described apply to the great majority of cases of peritonitis, yet the limit of variation is great, in so far as the appearances presented by individual cases are concerned. A pure *serous peritonitis* (*peritonitis serosa*) is relatively infrequent. Some fibrin is almost always present, so that the exudate should be more properly termed sero-fibrinous. The fluid exudate is yellowish, slightly cloudy, and contains but few cells, but fibrin flakes are found floating in it. Over the peritoneum there is usually a delicate film of fibrin. A pure *fibrinous peritonitis* is not common as a general process,

but is often seen as a local peritonitis. The serosa is cloudy, dry (*peritonitis sicca*) and covered with sticky fibrin.

When the number of cells in the exudate is so great as to cause a turbidity, the peritonitis may be styled *purulent* (*peritonitis purulenta*). A pure purulent peritonitis is also rare, the character of the exudate being usually that of a *fibrino-purulent* inflammation. The coils are usually fastened together by masses of fibrin, while in the pockets between them a relatively pure purulent exudate is found, usually in large amounts (20–30 litres). The so-called *peritonitis ulcerosa* is a misnomer, the appearances of ulceration over the surfaces of the abdominal organs and the parietal layer of the peritoneum being due to a loss of substance through erosion and pressure. In such cases the purulent exudate may perforate into the stomach, intestine, ureter, bladder, uterus, or through the diaphragm or abdominal wall (*secondary perforative peritonitis*). In the last-named case the perforation is usually located at the navel. This event happens more frequently in children than in adults.

In the case of *putrid peritonitis* the exudate has a foul odor and is irritating to the skin of the operator or prosector. In color it varies from dirty-green to brownish. The fibrin of the exudate and the serosa itself may be likewise dark-colored. Gas may be present in the cavity, and the odor of hydrogen sulphide is often very pronounced even when no perforation has occurred. In the case of a perforation of stomach or intestine food remains or fæces may be found in the exudate. Brownish masses are also sometimes present; on microscopical examination they are found to consist of colonies of bacteria.

An acute *hemorrhagic peritonitis* is rare. It is usually associated with carcinoma or tuberculosis, but occurs also in morbus maculosus Werlhofii and in scurvy. Through the presence of hydrogen sulphide in the exudate the blood pigment may become converted into a hydrogen-sulphide compound, giving a gray or slate color to the exudate and serosa (*pseudomelanosis*). Friedreich has described an especial form of hemorrhagic peritonitis under the name of *hæmatoma peritonei*. In such cases the peritoneal surface is covered with layers of blood clot which are becoming organized, the condition being analogous to the subdural hæmatoma.

Circumscribed peritonitis presents practically the same appearances as the general form, except in being localized through the rapid formation of firm adhesions which prevent the inflammation from spreading (*peritonitis adhæsiva*). These adhesions may quickly become organized. Circumscribed peritonitis occurs most often as a perityphlitis (peri-appendiceal) dependent upon perforation of the appendix, and as a pelvic peritonitis (pelveo-peritonitis) due to diseased conditions of the uterus and its appendages. It may be caused also by perforative processes (ulcer, new growths, etc.) of stomach, intestine, gall bladder, etc., by splenic infarction, hepatic abscess, and other local inflammations of the intraperitoneal organs. The so-called subdiaphragmatic abscess is an encapsulated purulent peritonitis. Similar encysted collections of exudate may be found in any part of the peritoneal cavity, but are more frequent in the regions named. Gonococcic peritonitis is usually circumscribed in the pelvis. The part which the omentum plays in the encapsulation of a local peritonitis has been mentioned above. Through its aid many cases of perforation peritonitis, particularly in the case of the appendix, are restricted to a narrow area. The frequency with which omental adhesions are formed about the appendix, tubes, perforating ulcers, new growths, etc., is evidence of its very great service in limiting peritonitis.

Complications.—The complications of acute peritonitis are many and important. Perforation of the peritoneal exudate into some viscus or through the abdominal walls has been mentioned. Occasionally the pus may break through into two neighboring coils of intestine at the same time, giving rise to the so-called *fistulæ bimucosæ*. Fatal hemorrhage may follow perforation into an abdominal blood-vessel. Metastasis may occur and a general pyæmia may result. It is not uncommon in the case of a severe acute general peritonitis to find at autopsy a beginning pleuritis, pericarditis, or meningitis. Sometimes all the serous membranes may be equally involved (panserositis). Such pathological findings are seen most frequently in puerperal peritonitis.

Microscopical Appearances.—Sections through an acutely inflamed peritoneum present changes which are analogous to those of inflammations of other serous membranes. The surface of the membrane is covered with a more or less thick layer of fibrin containing a varying number of leucocytes. The latter usually show karyorrhexis, the diffusion of the chromatin throughout the fibrin causing the latter to stain bluish. Beneath the exudate the endothelium is degenerated, necrotic, or desquamated. The connective tissue of the basement membrane is œdematous and infiltrated with leucocytes. The lymphatics are greatly dilated and contain fibrin and large numbers of leucocytes. The blood-vessels are also greatly congested and filled with leucocytes. The endothelial cells of both lymph- and blood-vessels are usually somewhat swollen. Scattered hemorrhages of small size may also be found. The subserosa is usually actively involved, the connective tissue being œdematous and infiltrated with leucocytes; the vessels are congested and small hemorrhages may occur. Fibrin threads may be found in the intercellular spaces of both serosa and subserosa. The muscularis is involved to a much less extent. Its vessels are congested and the intermuscular connective tissue is more or less œdematous and contains an increased number of leucocytes. In severe cases the nerve cells of the intestinal wall may present various changes, such as vacuolation, hydropic degeneration, chromatolysis, etc. In the later stages of the inflammation evidences of proliferation are found in the endothelial cells of the blood- and lymph-vessels and in the connective-tissue cells of the serosa and subserosa. Fibroblasts wander out into the fibrin of the exudate and a new formation of capillaries takes place. Even in attacks of peritonitis lasting but five to seven days the organization of the exudate may have advanced considerably. In the further progress of the disease connective-tissue adhesions and false membranes may be formed, or marked thickenings of the peritoneal surfaces may result. Should complete healing take place the newly formed connective tissue becomes scar-like and hyaline, and marked retractions of the omentum and mesentery may be caused. The remains of old organized fibrinous exudates are most frequently seen in the form of tendinous patches or small pearly nodules over the surface of the spleen, liver, or intestine. Extensive thickening of the pelvic peritoneum may result from the healing of circumscribed peritonitis in this region. During the stage of active fibroblastic proliferation such a large mass of granulation tissue may be formed that in the case of a circumscribed peri-appendiceal peritonitis a tumor may be produced which may be mistaken clinically for a malignant neoplasm, and even when examined microscopically might be mistaken for a sarcoma. Such tumor-like masses may also be found in the omentum. Should healing take place the tumor may gradually contract and finally entirely disappear ("disappearing tumor"). The presence of exudate not yet organized, the large number of polymorphonuclear leucocytes showing karyorrhexis, the hypertrophic character of the endothelium of the numerous capillaries, the presence of many plasma cells and phagocytes, the development in areas of a fibrous intercellular substance, etc., are among the points to be considered in making a microscopical diagnosis of tissue removed from such tumor-like formations.

Subacute Peritonitis.—In cases running a slower course the amount of exudate is usually less than in the acute forms, although in some cases in which it is more serous in character the amount may be very large. The adhesions are firmer, organization having progressed to a greater extent. The serosa is thickened, roughened, and wholly lustreless, and is often grayish or slate-colored. The injection of the blood-vessels is much less, and there

is also less œdema of the subserosa. Exacerbations, which are very likely to occur, give the appearances of an acute inflammation. Microscopically the exudate covering the serosa is found to be partly organized, and there is a fibroblastic increase of tissue. The cells of the exudate may show fatty or hydropic degeneration.

Chronic Peritonitis.—Chronic peritonitis may follow an acute process or it may develop insidiously. The changes seen in chronic peritonitis are essentially the same as in the acute process, the chief difference being in the more advanced organization of the exudate with resulting firm adhesions and formation of false membranes, tendinous patches, etc. When numerous adhesions are formed the condition is termed *peritonitis chronica adhæsiva*. The capsule of the liver and spleen may be greatly thickened and hyaline (chronic perihepatitis, chronic perisplenitis). Scar-like thickenings and retractions of the omentum and mesentery may be found (*peritonitis chronica retrahens*), and in the former there may be produced tumor-like masses of granulation tissue. It is usually rolled up above the level of the umbilicus. Marked deformities of the intestine and intraperitoneal organs may be produced by the contraction of the new-formed connective tissue (*peritonitis deformans*). Partial or complete stenosis of the intestine, common duct, ureters, tubes, etc., may result from the contraction or pull of band-like adhesions. Incarceration of portions of the intestinal coils in hernia-like sacs formed by adhesions may also occur. Inasmuch as exacerbations are not infrequently seen in chronic peritonitis the picture of a recent exudation may be added to that of an older process. The *hæmatoma peritonei* of Friedreich mentioned above is associated particularly with a rare form of chronic hemorrhagic peritonitis. Chronic peritonitis may be local or general; the former is the more common. The inflammation may be dry, or a sero-fibrinous exudate may be present; less frequently the exudate is hemorrhagic or purulent.

Local chronic peritonitis is seen most often in the hepatic region (cirrhosis, gall-stones, syphilis, etc.), in the splenic region (perisplenitis), and in the pelvis of the female. In prostitutes a local chronic pelveo-peritonitis is nearly always present in the form of adhesions. The appendix region, the neighborhood of gastric and intestinal ulcers, hernial sacs, etc., are also frequent seats of chronic peritonitis.

Attention has recently been directed to the condition designated "*chronic multiple serositis*" or "*multiple progressive hyaloserositis*" which is characterized by a slowly progressive hyperplasia of the serous membranes with secondary hyaline changes. In the case of the peritoneum the condition is usually most marked over the capsule of the liver or spleen (chronic perihepatitis, chronic perisplenitis). The thickened and hyaline capsule presents an appearance resembling the icing of a cake ("Zuckergussleber" of Curschmann, "iced liver"). An obliterative pericarditis or pleuritis may or may not be coincident with the peritoneal condition. The capsules of the liver and spleen may be involved at the same time or either one may alone show the change, or the condition may be diffuse throughout the entire extent of the peritoneum, or it may occur as a part of a multiple affection of the serous membranes. When affecting chiefly the liver capsule the condition may be the result of a local chronic peritonitis, an acute or subacute hepatitis, chronic mediastinopericarditis, or a chronic obliterative pleuritis. It may also be a part of a Glissonian cirrhosis, perhaps associated with syphilis or tuberculosis. The affection of the splenic capsule is usually the result of a local chronic peritonitis, or is associated with syphilis or tuberculosis. Chronic hyaloserositis is essentially a hyperplastic inflammation, and is probably due to an infection with micro-organisms of a low virulence. In the majority of cases it is probably tuberculous. Microscopically the thickened splenic or hepatic capsule consists of laminated hyaline connective tissue, usually poor in cells, but occasionally containing groups of leucocytes or areas of unorganized fibrin. In some cases, at least, the organization of a fibrinous exudate with subsequent hyaline change of the new connective tissue plays an important, if not the chief, part in the production of the condition.

Peritonitis Carcinomatosa.—Scirrhous carcinoma of the stomach or gall bladder may give rise to secondaries scattered diffusely throughout the peritoneum. The membrane at the same time presents the appearance of a chronic hyaloperitonitis, the entire serosa, intestinal and parietal, as well as the hepatic and splenic capsules, being greatly thickened, dense, and hyaline. Such a condition may very easily be mistaken for a simple chronic peritonitis, inasmuch as appearances suggesting malignancy may not be visible to the naked eye. The diagnosis in some cases can be made only on microscopical examination, since the primary growth may consist only of a dense thickening of the pyloric end of the stomach or of the gall-bladder wall and may not be recognized from the gross appearances. Microscopically the hyaline connective tissue of the thickened peritoneum is seen to contain small nests and cords of epithelial cells, corresponding to those of the carcinomatous infiltration at the primary seat. It is well to bear in mind the fact that the mesenteric and retroperitoneal lymph glands may contain large nests of cancer cells, so that the diagnosis of scirrhous carcinoma may be more easily arrived at by the examination of these glands. The peritoneal condition may therefore be regarded as of the nature of a diffuse carcinomatosis with secondary inflammatory changes. More or less fibrinous exudate may be present over the surface of the thickened membrane, and some free fluid may be present in the cavity.

Experimental Peritonitis.—A number of important experimental studies of peritonitis have been carried out by different observers, particularly with reference to the etiological factors of peritonitis. The part played by various bacteria and by such predisposing factors as chemical irritation, cold, etc., has been studied with the gain of much important knowledge. Other writers have recently studied the character of the cells found in the peritoneal exudate. Beattie has made experimental investigations along this line. After the injection of bacteria into the peritoneal cavity various cells appear in the exudate. Polymorphonuclear leucocytes appear in great numbers on the peritoneal surface, and are found abundantly in from six to fifty-four hours after the injection. In fatal cases they increase up to the time of death of the animal, but in non-fatal cases they diminish in from forty-eight to sixty hours. They act as the chief bacterial phago cytes. Mononuclear phagocytes are also found at all stages, but are most abundant from thirty-six hours onward. They are derived from the endothelium of the serous membrane, of blood-vessels, lymph vessels, lymph spaces, etc. They possess amœboid motion, and are especially phagocytic to other cells, but may also take up bacteria. Great numbers of these cells are always found on the omentum, and Beattie regards them as the most important cells of the peritoneal exudate. The presence of large numbers of these cells, if they give evidences of active function, is a favorable sign. Since the omentum furnishes large numbers of the mononuclear cells in peritonitis this organ must be regarded as an important agent in protecting the body from infection by way of the peritoneum. Both the polymorphonuclear and mononuclear phagocytes are destroyed in the peritoneal sac, the former being largely ingested by the latter. In the case of peritonitis associated with secondary carcinoma of the peritoneum tumor cells may be found in the exudate in addition to the polymorphonuclear and mononuclear phagocytes. As a rule the cancer cells cannot be distinguished from the latter; but if numerous cell-division figures are present, especially atypical forms, it is very likely that such cells come from a new growth.

Symptoms.—*Acute General Peritonitis.*—Peritonitis in the great majority of cases being a secondary disease the symptoms are modified to a greater or less extent by the original trouble. The onset in particular is modified.

Perforative peritonitis may occur suddenly in a person apparently in perfect health. Not infrequently the first intimation of the existence of a gastric or intestinal ulcer is the peritonitis following a perforation. In the case of perforative appendicitis the onset may be equally sudden. On the other hand, the symptoms of perforative peritonitis may be obscured in the case of intestinal tuberculosis, typhoid fever, intestinal obstruction, etc., by the severe local or general symptoms. A circumscribed peritonitis may also become gradually changed into a general condition, the change in symptoms not being so pronounced as to be noted. For example, a localized purulent perityphlitis or a purulent puerperal pelveoperitonitis may become generalized. A pneumococcic peritonitis occurring in association with pneumonia or meningitis may have its onset obscured by the general symptoms.

In spite of these exceptions nearly every case of acute general peritonitis runs a clinical course so typical and characteristic that the recognition of the condition becomes a matter attended by little or no difficulty.

Prodromal symptoms may or may not occur. In the former case they consist of chills, prolonged and repeated chilly sensations, fever, loss of appetite, nausea, vomiting, thirst, diarrhœa or constipation, flatulence, etc.

Pain is the earliest definite symptom; and few cases of acute peritonitis exist without it. In patients who are extremely weak or whose minds are clouded, no complaint of pain may be made, and the condition may in this way escape notice. In nearly every case, however, the pain becomes the most marked and important symptom. It may be local, often referred to the region of the umbilicus, but later usually extends over the entire abdomen. The localization of the pain at the onset of the disease may have some diagnostic value in indicating the possible source of the inflammation, as, for example, in the case of gastric ulcer or a perforating appendicitis. The pain is usually excruciating in character, and may be constant, or it may occur with short remissions followed by exacerbations. Colic-like paroxysms sometimes occur. The abdomen is extremely sensitive, so that even gentle palpation, or even the weight of the bed clothes can hardly be borne. The patient assumes a position which relieves the tension of the abdominal muscles, lying on the back with thighs drawn up and shoulders elevated. Such a position may be maintained for days and weeks as long as the condition lasts. The pain is increased by deep inspirations, voluntary movements, and probably also by intestinal peristalsis. The patient fears particularly such movements as vomiting, coughing, sneezing, defecation and urination, etc. The character of the pain is sometimes described as burning, boring, tearing, lancinating, etc., without much light being thrown upon the conditions causing the especial character. The passage of gas through the coils probably plays an important part in producing the painful attacks. Though in the majority of cases the pain is most severe below the umbilicus, in cases of perforation of the stomach the pain may be referred to the shoulders, back, or chest. In the earlier stages of peritonitis following appendicitis the pain may sometimes be referred to the testicle or the penis. Mackenzie believes that the pain which is caused by pressure over the abdomen is entirely due to muscular and cutaneous hyperæsthesia, the peritoneum itself being devoid of sensory nerves. The board-like hardness of the abdominal muscles in peritonitis he holds to be the result of a violent stimulation passing from the affected organs of the peritoneal cavity to the spinal cord, where the irritation spreads, affecting not only the centres of the sensory nerves, but also those of the muscular nerves. When the patient is at perfect rest the pain may be slight or may even entirely disappear.

The *respiration* is very superficial, rapid, and wholly costal in type. The speech usually becomes an almost imperceptible whisper, occasionally hoarse and high-pitched, resembling the so-called vox cholerica. When the attack is fully established the patient presents a very characteristic appearance, the expression is anxious, the face pinched and the eyes are sunken, the Hippocratic facies being seen in this affection more often than in any other disease except cholera. The severity of the pain is usually expressed by the patient's face; in severe cases the eyes are glassy and staring. Consciousness remains preserved usually until the last; rarely there is delirium, coma, or convulsion at the end.

The abdomen quickly becomes distended, the distention becoming gradually more and more marked, and sometimes reaches such an extent that it seems that the abdominal wall must give way. The skin over the abdomen is smooth, shining, thinned, the superficial veins appearing as blue lines. Fresh lineæ albicantiæ may be seen over the surface. The abdominal muscles are stretched and often of a board-like hardness. The distention is due to the intestinal tympanites, which is sometimes so great that complete paralysis of the intestinal musculature results. The distended coils of intestine can sometimes be traced through the abdominal wall. The more lax the abdominal wall before the beginning of the peritonitis the greater will be the distention; hence the distention is most marked in the puerperal cases. When the abdominal muscles are well developed and the abdominal wall is tense, the convexity may not be great. In some cases the wall may be as hard as a board and flat or even somewhat concave. In the latter case the diagnosis may be very difficult. In the later stages the abdominal distention is increased by the fluid exudate.

Vomiting is an almost constant symptom in peritonitis. It usually begins early in the disease and causes great pain. At first the vomitus consists of food remains, later it consists chiefly of a yellowish bile-stained fluid, and finally becomes greenish. Occasionally the vomitus is brownish-black and possesses a slight fæcal odor. The attacks may be spontaneous or follow the taking of food. In the former case the vomitus consists usually of greenish mucus. Eructations of gas usually accompany the vomiting. The frequency and intensity of the vomiting vary. In some cases nothing can be retained upon the stomach, and the patient's strength is very quickly lost. The cause of the vomiting is not clear; it is probably partly reflex and partly due to pressure upon the stomach. In those cases in which the vomitus has a fæcal character the autopsy usually discloses no intestinal obstruction. It is therefore probable that the condition is due to the paralysis of the intestinal musculature and to the pressure upon the intestine. Toward the close of the attack the vomiting may be replaced by painful *hiccoughs*, which are a source of great torture to the patient. It is probable that this is due to the involvement of the peritoneal surface of the diaphragm. In those cases in which the vomiting is kept up to the end some of the vomitus usually enters the respiratory tract and may give rise to an aspiration pneumonia or gangrene of the lung.

Peritonitis not infrequently begins with *diarrhœa*, which in some cases is associated with tenesmus, or assumes a dysenteric character. The paralysis of the intestinal musculature soon leads to *constipation*, so that this condition becomes an almost constant feature of the disease.

The *tongue* is usually coated, grayish-white or brown; in cases of obstinate vomiting it may be clean and red or fissured. A very disagreeable fœtor is usually present, sometimes almost fæcal in character.

After the chill the body temperature usually rises, reaching 104°–105° F. or higher, but later usually falls. The type of the *fever* is very varied; it may be continuous, intermittent, or remittent. The skin may be cool, while the rectal temperature is high. In the case of collapse the temperature may fall even to subnormal, and the body may be covered with an abundant cold perspiration. Just before death the temperature may rise again and reach a very high point.

The *pulse* is usually very frequent, the increase in rate being out of proportion to the fever, 120–140 per minute not being uncommon. The pulse is at the same time

small and soft, and in very severe cases may become imperceptible.

The number of *respirations* is increased, 30–40 per minute. The increased rate is due partly to the fever, partly to the high position of the diaphragm, and partly to the pain attending abdominal breathing. As mentioned above, the type is costal and the breathing superficial. The cessation of movement of the diaphragm is regarded as a bad sign, some writers considering the prognosis hopeful as long as an inspiratory excursion of the diaphragm can be felt, this being taken as a sign that the peritonitis is not wholly diffuse.

The *subjective* symptoms are chiefly pain, uncontrollable thirst, shortness of breath, and anxiety.

Percussion gives a tympanitic tone over the distended intestine. Dulness over the dependent portions is usually not made out until after considerable exudate has collected. When the tympanites is very marked a large amount of effusion may be present without its being detected on percussion. A change of level of the dulness with change of position does not always take place; the adhesions between the coils preventing the movement of the exudate. Moreover, there is usually too much pain to permit of a careful examination. The liver dulness is diminished, the upper border being pushed upward to the fifth or fourth rib. The splenic dulness cannot usually be made out. The heart is pushed upward, the apex beat dislocated upward and to the left. The movements of the heart in the intercostal spaces are more prominent than normal.

Auscultation of the abdomen does not throw much light upon the condition. Gurgling and splashing sounds may be heard in the intestines. Occasionally friction sounds may be heard, but these are not so common as in pleuritis or pericarditis. In pneumoperitonitis splashing sounds may be produced by shaking the patient. The heart sounds may have a metallic resonance in the case of extreme tympanites. The pulmonary second sound is usually accentuated.

The *urine* is usually diminished in amount, dark, strongly acid, of high specific gravity, and sometimes contains a small amount of albumin. The amount of indican present may be much greater than usual.

Certain writers claim that there is a *local increase of temperature* in the abdominal wall, and that this fact constitutes an important diagnostic factor. The normal temperature of the surface of the abdomen is about 35.5° C.

In the case of *pneumoperitoneum* due to perforation or to formation of gas within the cavity the hepatic dulness is obliterated. The liver dulness may, however, disappear when there is no gas in the cavity; but if the patient is placed upon his left side a clear percussion tone will be heard at the seventh or eighth rib in the right axillary line in the case of pneumoperitoneum. In other cases dulness will be found at this region. The disappearance of the liver dulness anteriorly when pneumoperitoneum does not exist is due to the tilting backward of the organ and the presence between it and the abdominal wall of a loop of distended intestine.

The *course* of an acute general peritonitis is usually very rapid. The most severe cases may terminate fatally within thirty-six to forty-eight hours, while the average case usually lasts five or six days. Since the etiological factors vary so greatly no absolute statements can be made regarding the clinical course. The peritonitis occurring as the result of gastric or intestinal perforation is usually quickly fatal, but much is to be hoped from the surgical treatment of these cases. Puerperal septic peritonitis is likewise usually fatal. Pneumococcic peritonitis is also likely to terminate more or less quickly in the death of the patient. The peritonitis which occurs in connection with arthritis, as well as in all other cases in which the exudate is serous or sero-fibrinous, is much more likely to end in recovery.

In the fatal cases *death* may occur suddenly as if from shock; or in the case of extreme tympanites the high position of the diaphragm may cause death from suffocation. In some cases the final picture is that of a general sepsis or pyæmia. In the great majority of cases the pulse becomes more rapid, the vomiting persists, the bodily powers are quickly exhausted, and the patient dies with symptoms of collapse. In such cases the temperature usually becomes subnormal as the end approaches.

In other cases of less frequent occurrence the disease may drag itself out over weeks and months, assuming the characteristics of a subacute or chronic process. Various complications may occur during such a prolonged course. Rupture of the exudate into the gastro-intestinal tract or through the abdominal wall, etc., is most likely to occur in such cases. In the event of such perforation into the stomach, pus will be found in the vomitus; when into the intestine the pus will appear in the stools; when into the urinary tract the urine will show a sediment of pus; when into the respiratory tract the sputum will be abundant and purulent. In the event of a rupture through the abdominal wall redness, swelling, and œdema will first appear at the affected spot, the skin becomes thinned, fluctuation occurs, and pus finally seeps through small fissures, or an opening of large size may be formed, through which the pus may be forced in streams during coughing, etc. The navel is the most common seat of perforation. In some cases the pus burrows beneath the skin for some distance before it finally breaks through. In other cases some of the exudate may be absorbed or encapsulation may take place. Exacerbations are frequent and death may take place finally from general marasmus. General dropsy, bedsores, albuminuria, etc., characterize the course of such cases.

In the mild cases of general peritonitis following labor, abortion, or menstruation recovery may occur. Even in those cases in which the disease runs a rapid and favorable course disturbances of the digestive tract usually develop, and the patient may die later from intestinal obstruction caused by the contraction of adhesions. A favorable prognosis should not be too hastily given in such cases. Nevertheless, complete recovery may result after a year or longer, even in very severe cases.

Acute Circumscribed Peritonitis.—The local symptoms of this form are similar to those of the general process, but are more limited. The vomiting is not so severe or so persistent as in the general inflammation. Though the general weakness is marked the symptoms of collapse do not occur to the same extent as in general peritonitis. An irregular fever is usually present, running a course suggesting that of a pyæmic affection. The condition is usually very chronic. Many patients die from general debility. Spontaneous evacuation of the encapsulated exudate may take place through perforation into the gastro-intestinal tract, pleura and lungs, abdominal wall, etc. Should the perforation take place into the remaining portion of the peritoneal cavity the peritonitis may become diffuse. According to the location of the inflammation (perisplenitis, perihepatitis, perityphlitis, perimetritis, peripancreatitis, perigastritis, epiploitis, mesenteritis, etc.) various symptoms may arise through the disturbance of function of the affected organ. Pain is usually felt in the affected region. The physical signs are local tumor, dulness, fluctuation, peritoneal friction, etc.

Of the various forms of acute circumscribed peritonitis the condition known as *subphrenic abscess* deserves especial attention. It is due to an encapsulated collection of purulent exudate between the upper surface of the liver, stomach, or spleen, and the diaphragm. Owing to the paralysis of the diaphragm the tumor may rise high into the thorax and may be mistaken for a pleural effusion, especially since a secondary pleuritis is usually present. Hepatic abscess, splenic infarction, splenic abscess, rupture of echinococcus cysts, and gall stones are the more common causes of the condition, but it is not a rare complication of perforating appendicitis.

Perforation Peritonitis.—The peritonitis due to perforation may be local or general. It may run a very

acute course, but when circumscribed is more likely to be chronic. In the case of the sudden perforation, as, for example, of the stomach, intestine, cystic tumor, etc., the patient may feel a sharp tearing sensation, as if something had given way within the body. The severest symptoms of collapse may immediately result. The abdomen may be contracted, hard as a board, and even scaphoid. The slightest touch causes extreme pain. Death may take place within a few hours. In other cases the entrance into the peritoneal cavity of stomach contents, fæces, bile, etc., excites a general peritonitis with symptoms as described above. In the case of perforation of the gastro-intestinal tract gas may also enter the cavity, and the picture of a pneumoperitonitis be presented. The liver and splenic areas of dulness are obliterated unless these organs have become attached to the anterior abdominal wall by means of adhesions. Succussion sounds may be produced, and the respiratory sounds may acquire an amphoric quality.

Puerperal Peritonitis.—This dreaded complication usually appears at from the third to the fifth day after labor. Safety from the condition is usually assumed if the woman reaches the end of the second week without its occurrence. The pain is usually much less intense than in the other forms of general peritonitis, and the tympanites more marked. Chills occur more frequently than in other forms and diarrhœa is almost always present, the discharges often becoming bloody and of the character of a dysentery. The vomiting is especially obstinate. The lochial discharges often become offensive. The peritoneal exudate is usually more abundant and more purulent in character than in other forms, and not infrequently becomes putrid. The mortality is great, death taking place with symptoms of general sepsis usually between the fifth and twelfth days.

Infantile Peritonitis.—*Fœtal* peritonitis occurs between the seventh and ninth months of intra-uterine life. Syphilis is the chief etiological factor. Infection of the umbilicus may lead to a *peritonitis neonatorum*. The children of mothers suffering from puerperal sepsis are especially likely to become infected. In addition to the local symptoms of distention, pain, etc., there is usually a picture of general sepsis or pyæmia. The condition is almost always fatal. A peritonitis may also develop in infants and young children (*peritonitis infantium*) as the result of syphilis, or of infection with pyogenic organisms, the pneumococcus, gonococcus, etc. In the great majority of cases peritonitis in young children is the result of appendicitis. In peritonitis which occurs in young girls the possibility of a gonococcic infection should always be borne in mind and the genital passages examined for the existence of such disease. Non-gonococcal vulvo-vaginitis rarely spreads to the peritoneum. Pneumococcic peritonitis is more common in girl than in boy infants. The infection is probably through the genital tract. The onset is usually very sudden, with abdominal pain, fever, and vomiting. The course of the affection is very similar to that of pneumonia, the active symptoms subsiding in from seven to ten days, but the abdomen remains distended, and later the presence of an exudate becomes manifest. The prognosis in the cases of general infection is not good, but when the inflammation is localized in the pelvis it is more favorable.

Chronic Peritonitis.—In the majority of cases chronic diffuse peritonitis is the sequela of an acute inflammation, although such an occurrence is relatively rare. Usually no sharp line can be drawn between the two; the symptoms of an intense acute process gradually abate and are replaced by those of a chronic type. In some cases the chronic inflammation develops as the sequela of a number of acute attacks; in others the onset is slow and insidious. The pain, abdominal tenderness, and distention are never so marked as in the acute cases. The distention is usually moderate and often asymmetrical, certain coils of intestine being especially prominent. Sometimes the abdomen is flat or even scaphoid, the walls are hard and board-like. On palpation the thickening of the omentum and mesentery, as well as the fibrous adhesions between the coils, may be made out as tumor-like masses or as uneven prominences. When an effusion is present palpation may be negative. A friction rub may occasionally be felt. In some cases the abdomen may be so tense that nothing can be felt through it. Cases of chronic peritonitis, not due to tuberculosis or complicated with other conditions, do not usually show much exudation. The course is very protracted. Recovery may follow the absorption and organization of the exudate, or its escape by perforation from the cavity. Death usually results from general marasmus. The healing of the inflammation may be followed later by stenosis and intestinal obstruction, which may result fatally. Occlusions of the common duct by the contraction of adhesions may cause a chronic obstructive jaundice.

A peculiar form of chronic serous peritonitis occurs in children, appearing under the form of a chronic ascites. It occurs most frequently between the ages of two and ten, and in girls at the age of puberty. The abdomen is distended by the exudate, which is usually quite abundant, serous in character, and freely movable. The affected children are anæmic, more or less weak, but do not lose much in weight and do not suffer pain. The lower extremities may become œdematous. The exudate may apparently entirely disappear and then return. The course may last many months and may terminate in complete recovery. The exact nature of the affection is unknown. Trauma, inflammation of the gastro-intestinal tract, lymph glands, and genital tract, etc., have been adduced as etiological factors. It is possible that the condition represents an infection of the peritoneum with tubercle bacilli of low virulence. In the fatal cases death results from an increasing marasmus.

Tuberculosis of the Peritoneum.—Tuberculous disease of the peritoneum is of especial interest because of its peculiar clinical course and the fact that the condition may undergo spontaneous healing after laparotomy. A distinction is drawn by some writers between pure tuberculosis of the peritoneum and a tuberculous peritonitis; but such a distinction, while based upon pathological grounds, has but little practical value, the first condition rarely presenting clinical signs.

Simple tuberculosis of the peritoneum without accompanying inflammation or exudation is usually secondary to an acute miliary or chronic pulmonary tuberculosis. It may be secondary also to tuberculosis of the bronchial or subperitoneal lymph glands, genito-urinary tuberculosis, tuberculosis of the pleura, adrenal bodies, etc. In the great majority of cases the infection of the peritoneum proceeds from tuberculous ulcers of the intestine or caseating mesenteric glands. In rare cases it may be primary. Small grayish tubercles are found scattered over the peritoneum. On microscopical examination they consist of epithelioid cells and numerous giant cells containing tubercle bacilli. Caseating centres may be seen in the larger ones; but inflammatory changes in the neighboring peritoneum are usually wanting. The tubercles are almost always more numerous in the omentum. When very small the tubercles may not be recognized at operation or autopsy; the larger ones are recognized by their grayish color and yellowish centres. Though often very numerous the tubercles are usually circumscribed. There are no symptoms, the condition usually being discovered at autopsy, or in the event of a laparotomy.

Tuberculous peritonitis—tuberculosis with inflammation and exudation—exists in a great variety of forms which have been variously classified by different authors. The most common forms are: *acute miliary tuberculosis* with sero-fibrinous or sero-hemorrhagic exudation; *chronic tuberculous ascites* with small tubercles which show little caseation; *chronic fibro-caseous tuberculous peritonitis* with purulent or fibrino-purulent exudate; *chronic hyperplastic tuberculous peritonitis*; *chronic fibroid tuberculosis* with little or no exudation, the serous surfaces being fastened together by adhesions.

The appearances at autopsy or operation may vary