(post-anal gut, Balfour[1]) of older embryos, which soon becomes divided, at least in birds, into a dilated terminal portion and a narrower neck communicating with the intestine proper. The posterior section then subdivides, and its narrow end segment lengthens out and unites with the spinal cord. This canal we may designate as Braun's canal. It is not improbable that it is homologous with the amnio-allantoic canal of Gasser,[7] which Rauber[12] has nicknamed the Cochin China canal, after the breed of hens in which it seems most constant. In the one case we may suppose the canal to open after, in the other before, the closure of the posterior end of the medullary groove. If the homology is correct it may be further said that the canal is identical with Kupffer's myelo-allantoidean canal; it cannot be brought into relation with the development of the allantois, as believed by Kupffer,[10] as the allantois and Enddarm are both formed before the canal appears.

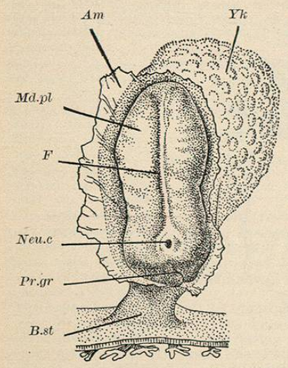

FIG. 5161.—Reconstruction of a Human Embryo 1.54 mm. Long. The amnion has been opened to show the dorsal surface of the embryo. *Yk*, Yolk sac; *Am*, amnion; *Md.pl*, medullary plate; *F*, dorsal furrow; *Neu.c*, neurenteric canal; *Pr.gr*, primitive groove; *B.st*, body stalk. (After Count Spee.) (From Minot's "Embryology," Blakiston, Phila., 1903.)

The significance of the three canals is obscure. The middle one seems unquestionably the temporarily open secondary blastopore. The first, or Gasserian canal, is distinct from the blastoporic, as the two coexist, and it seems desirable that this canal alone should be called *neurenteric*. As to its origin or significance nothing definite can be said, but it may be suggested that it was the excretory opening of the central canal of the spinal cord and that the cilia of the canal served to expel the fluid (compare Balfour[1,2]). This may have been the condition in the earliest vertebrates, and the neurenteric canal now persists as a rudimentary organ. As to the morphological or physiological interpretation of the third or Braun's canal no satisfactory suggestion has been made.

It seems not impossible that a persistent neurenteric canal may occur as an excessively rare anomaly in the adult.

Charles Sedgwick Minot.

LITERATURE.

The literature of the neurenteric canals is all comparatively recent, and is, for the most part, included in essays dealing with other embryological subjects. Great confusion has arisen from the failure to distinguish the several canals. The reader will find his way most readily by consulting first the text-books of Balfour,[2,3]; second, the article of Braun,[3]; and third, Rauber's note,[12] although Rauber's terminology is eccentric and perplexing.

[1] Balfour, F. M.: On the Early Development of the Lacertilia, etc. Quart. Journ. Microsc. Sci., xix. (1879); also in his Works, i., p. 644, with Plate 29.

[2] Balfour: Comparative Embryology, vol. ii., pp. 267–269.

[3] Braun, M.: Die Entwickelung des Wellenpapageis: ii. Theil, Semper's Arbeiten, v., 205–341. Taf. x.–xiv. (a valuable but excessively diffuse article. The most important passages on the neurenteric canals are on pp. 296, 301, and 308).

[4] Durham, Herbert E.: Note on the Presence of a Neurenteric Canal in Rana. Quart. Journ. Microsc. Science, xxvi., 509–510, Plate xxvii.

[5] Foster and Balfour: Elements of Embryology, second edition, 1883.

[6] Gasser: Der Primitivstreifen bei Vogel-Embryonen, Cassel, 1879.

[7] Gasser: Beiträge zur Kenntniss der Vogelkeimscheibe. His und Braune's Archiv, 1882, 359–398.

[7a] Götte: Entwickelungsgeschichte der Unke.

[8] Heape, Walter: The Early Development of the Mole (Talpa Europæa). The formation of the germinal layers, and early development of the medullary groove and notochord. Quart. Journ. Microsc. Sci., xxvi., 1883, 412–452, Plate xxviii.–xxxi.

[9] Hoffmann, C. K.: Die Bildung des Mesoderms, die Anlage der Chorda dorsalis, u. die Entwickelung des Canalis neurentericus bei Vogel-Embryonen, p. 109, 5 Tafeln. Amsterdam, 1883. (Abstr. in Hoffmann u. Schwalbe's Jahresber., 1883, 442–444.)

[10] Kupffer, C.: Die Gastrulation an den meroblastischen Eiern der Wirbelthiere und die Bedeutung des Primitivstreifs. Arch. Anat. u. Physiol., Anat. Abth., 1882, 1–30, Taf. i.–iv. Fortsetzung, 139–156. Taf. viii.–ix.; 2te Fortsetzung, 1883, 1–40, Taf. i.–ii.

[11] Lieberkühn, N.: Ueber die Chorda bei Säugethieren. His und Braune's Archiv, 1882, 399–438, Taf. xx.–xxi. Fortsetzung, 1883, 435–452, Taf. xix.

[12] Rauber, A.: Noch ein Blastoporus. Zool. Anzeiger, vi., 1883, 143–147, and 163–167 (*cf.* his earlier papers on the Blastoporic Canal, Zool. Anzeiger, ii., 1897, p. 499, and iii., 1880, p. 180).

[13] Strahl: Ueber Canalis neurentericus und Allantois bei Lacerta viridis. His u. Braune's Arch., 1883, 323–340, Taf. xiv.

PERITONITIS.—(Peritonæitis, Inflammatio peritonei.) The peritoneum is a thin membrane of connective tissue extremely rich in blood-vessels and lymphatics, and is covered by a single layer of flattened endothelial (mesothelial) cells (often called epithelial). These cells are chiefly polygonal, but the cell outlines are often very irregular, serrate, or wavy. By treatment with silver nitrate or by *intra vitam* staining with methylene blue, the cement lines between the cells can be made out; and by especial methods of technique the so-called intercellular protoplasmic bridges connecting the cells may be demonstrated. The connective-tissue layer consists of an interlacing network of fibrous connective-tissue bundles, numerous elastic fibres, and connective-tissue cells. Through this there runs a very abundant capillary and lymphatic network having a free anastomosis. The majority of writers hold that the lymphatics communicate directly with the peritoneal cavity by means of small openings between the mesothelial cells, known as stomata. Some of these writers regard the endothelial lining of the lymph spaces of the peritoneal basement membrane as continuous with the lining of the peritoneal cavity. This is not the case, however, and the mesothelial cells form a probably unbroken layer over both the blood and the lymphatic vessels, the processes of absorption and secretion being carried on through the two layers of cells, endothelial and mesothelial. The absorption capacity of the peritoneal surface is very great, being fully equal, in the case of experiments with certain poisons, to that of direct intravenous injections. Gases, fluids, and even morphological elements may be quickly removed from the peritoneal cavity. On the other hand, the secretory activity of the immense vascular surface of the peritoneum is very great, and in disturbances of the vascular secretion an immense amount of exudate may pass through the peritoneum into the peritoneal cavity. The endothelial and connective-tissue cells of the basement membrane respond very quickly to "irritation" of any kind, and fibroblastic activity is set up more quickly in the peritoneum than anywhere else in the body. The course of peritoneal inflammations is considerably modified by these factors.

The peritoneum lines the entire abdominal cavity, and is reflected over the organs contained within it. Over the organs it forms the serous coat or capsule (*tunica serosa*). The membrane is attached to the underlying parts by a subserous coat of adipose tissue, connective-tissue bands and elastic fibres. Over the organs the subserosa is but slightly developed. The anatomical relations of the peritoneum to the abdominal wall, its investiture of the abdominal organs, the intimate relations of the peritoneal cavity to the female genital tract, etc., in connection with its very vascular structure, peculiarly predispose this membrane to the occurrence of inflammatory processes. Such a predisposition is shown by the fact that peritonitis is one of the most common and important clinical conditions. It is also one of the most serious. The high mortality of acute general peritonitis makes it one of the most dreaded affections. Even in these days of aseptic surgery when so many operative procedures have been divested of their chief dangers, peritonitis still remains to the abdominal surgeon and gynecologist one of the complications most feared and most carefully to be avoided.

The occurrence of peritonitis is not dependent upon climatic, seasonal, or meteorological influences. Females are more frequently affected than males, this fact being dependent upon the important rôle which diseases of the female genital tract play in the causation of peritonitis. It is therefore seen more often during the years of sexual activity, but may occur at any age. It is of not infrequent occurrence in young children. The relation of peritonitis as a complication to many of the acute infections makes its occurrence to a certain degree associated with epidemics of these diseases. Alcohol is regarded as a predisposing factor.

ETIOLOGY.—Peritonitis may be *primary* or *secondary*.

Primary peritonitis is, if we except the surgical form, of much more rare occurrence than the secondary. It is usually *traumatic*, more rarely, *idiopathic*, *spontaneous*, or *rheumatic*.

Traumatic peritonitis is most commonly due to perforating wounds of the abdomen. Abdominal injuries in which the wall is not penetrated rarely, if ever, directly cause peritonitis. Nevertheless, blows, kicks, falls, etc., are often adduced as causes of peritonitis. The more severe the injury, the more likely is the occurrence of peritonitis. Hemorrhage into the peritoneal cavity is usually followed by inflammation of the peritoneum. Likewise, trauma may indirectly cause peritonitis through the perforation, laceration, tearing, or bruising of the stomach or intestines, rupture of the gall bladder, pancreatic duct, urinary bladder, etc., these lesions permitting the entrance of micro-organisms into the peritoneal cavity. The perforation of the œsophagus or stomach by the careless passage of a sound or stomach tube may also be a causal factor in the production of peritonitis.

Operative or *surgical* peritonitis may be mentioned in this connection. Before the days of antisepsis and asepsis a large proportion of laparotomies were fatal from the peritonitis following the operation. Even at the present day the chief anxiety attending operations involving the peritoneal cavity is the possibility of peritonitis. The vast improvement in surgical technique has, however, greatly reduced the number of cases of post-operative peritonitis, and in certain operations in which there is, with proper methods, no opportunity for infection the peritoneal cavity is opened with a certain degree of impunity. The occurrence of surgical peritonitis is dependent upon several factors: the character of the operation and the possibility of infection during the operative procedures, the amount of damage done to the peritoneal tissues, the state of the general resistance of the membrane to infection, etc. Surgical peritonitis is due to the introduction of bacteria into the peritoneal cavity. But not all bacteria which gain an entrance into the peritoneal cavity set up an inflammation there. It has been shown experimentally that rather large quantities of bacteria, even of the pus cocci, may be introduced into the peritoneal cavity of animals without causing peritonitis, the bacteria entirely disappearing after a short time. The presence of a tissue lesion, even though very slight, or the simultaneous introduction into the cavity of irritating substances, furnishes the conditions requisite for the growth of the bacteria and for the production of an inflammation. The peritoneum therefore when uninjured must possess a certain protective power against bacteria. This resistance is found to be lowered in the case of hemorrhage into the cavity, after the removal of ascitic fluid, and in cases of pseudomyxoma peritonei. The general condition of the body, conditions of intoxication, cachexia, etc., also play a rôle of importance. Fatal peritonitis may follow the tapping of the abdomen in ascites, the small number of bacteria introduced by means of a septic trocar being sufficient to overcome the lessened resistance. When modern methods of asepsis are properly carried out the dangers of surgical peritonitis are relatively slight in all cases in which the contamination of the peritoneum with septic material can be avoided. Even when this contamination does occur, the modern procedures of thorough irrigation have greatly reduced the chances of infection.

The existence of a *peritonitis rheumatica* (*refrigeratoria*) has been denied by many writers. While the effects of cold as a factor in the etiology of peritonitis were formerly greatly exaggerated, there can be no doubt, from the large number of observations made by reputable clinicians, that attacks of peritonitis may follow the sudden chilling of the overheated body, prolonged exposure to cold or wet, lying upon the cold damp ground, etc. The effects of such chilling of the body are especially noticeable when the abdominal vessels are overfilled with blood (*physiological congestion*). In women, particularly in girls at the time of puberty, a wetting or chilling of the body at the menstrual period is not infrequently followed by peritonitis. In all of these cases the effect of the refrigeration is not directly to excite the peritonitis, but to render the membrane less resistant to infection. The chilling plays the same rôle here as in the case of the mucous membranes. Diplococci have been demonstrated in the exudate of cases of so-called rheumatic peritonitis; and it is very probable that all such cases are infections following and dependent upon a lessened resistance of the body.

Cases of *idiopathic* or *spontaneous* peritonitis have also been reported clinically.

The autopsy findings in such cases show the presence of an infection proceeding from the female genital tract, the appendix, or some other of the abdominal organs. Such cases occur most frequently in females and in very young persons. In the former the infection is nearly always secondary to some unsuspected condition of the internal genitals. The gonococcus plays an important rôle in such cases, particularly in very young girls. It has been said that the peritonitis apparently arising spontaneously in young girls is usually gonococcal, a gonorrhœal infection of the genital passages existing at the same time. In young male children cases of apparent spontaneous peritonitis are usually found to be due to appendicitis. Further, spontaneous cases of peritonitis have been found at autopsy to be ambulatory typhoid, acute tuberculosis of the peritoneum, etc. On the whole it may be said that the existence of a true idiopathic peritonitis, while probable, has not yet been definitely demonstrated. A *cryptogenic hæmatogenous* infection of the peritoneum is of course possible and certainly occurs in tuberculous peritonitis, but has not yet been shown positively to occur in the case of the pyogenic organisms.

Likewise the existence of a toxic peritonitis, though assumed by some writers, has not been demonstrated. The occurrence of so-called *nephritic* or *uræmic* peritonitis seen in the late stages of Bright's disease is to be regarded as of the nature of a terminal infection, and similar to the pericarditis and pleuritis occurring in the same disease. The occurrence of peritonitis in association with or following various forms of intoxication is to be explained in the same way as the rheumatic—the lessened resistance of the body tissues and the pathological alterations of the body juices predispose to infection.

Since primary inflammations of the pericardium and pleura are of relatively more frequent occurrence, the greater rarity of primary peritonitis may be explained by the assumption that the peritoneum possesses a greater resistance and protective power than the other serous membranes. This relative greater immunity of the peritoneal cavity has been demonstrated experimentally.

SECONDARY PERITONITIS.—It should be noted at this point that various writers upon the subject of peritonitis are not agreed in their conceptions of primary and secondary peritonitis. In a very broad sense the term primary may be applied to those forms of peritonitis which are not secondary to, or do not occur in association with, any other acute or chronic disease, hence not metastatic; and, further, which do not arise by contiguity from any of the organs or structures covered by the peritoneum. Terminal infections should then be classed as secondary rather than as primary. A number of recent

writers, notably Flexner, in retaining the old classification of primary and secondary peritonitis, make a much narrower application of the term *secondary* as applying to peritonitis. According to Flexner, by *secondary* peritonitis we should understand those conditions which follow operations upon the peritoneum or the contiguous viscera, and those in which the abdominal cavity becomes inflamed through the mediation of disease-contained viscera. By *primary* peritonitis should be understood an inflammation, usually diffuse, of the serous cavity, occurring without the mediation of any of the contained organs and independently of any surgical operation upon these parts. Such a primary peritonitis may arise as an independent affection or develop in the course of infectious diseases in distant parts of the body. The micro-organisms causing the inflammation may be brought by the blood or lymph current or may pass through the intact intestinal wall. Flexner would therefore class as primary not only the terminal infections of the peritoneum occurring in the course of chronic diseases, but also those forms of metastatic peritonitis in which the micro-organisms come through the blood or lymph without the mediation of some diseased organ or part. The reason given for this more arbitrary and narrower use of the term secondary and the wider application of the term primary is that pathogenic micro-organisms may reach the healthy peritoneum without setting up an inflammation, but that "only an already inflamed peritoneum can be excited to peritonitis through the agency of pathogenic micro-organisms." Used in this way the terms primary and secondary indicate the character of the affection rather than the source of the cause or the manner in which the peritonitis arises. Since such a usage may be confusing, the writer has preferred to adhere to the more commonly accepted use of the terms as given above, viz., *primary peritonitis*, an inflammation of the peritoneum arising primarily and independently of other processes, as the result of chemical or mechanical irritation or as a direct or cryptogenic infection, or as the result of a lessened resistance of the peritoneum due to trauma, toxic conditions, or altered conditions of the peritoneum caused by foreign bodies, presence of fluid, etc., analogous to primary infections in other parts of the body; *secondary peritonitis*, an inflammation of the peritoneum, secondary to some other process, arising by contiguity or metastasis, or occurring as a terminal infection. Even with this broader standard of classification no hard-and-fast lines can be drawn. Inasmuch as cases of primary peritonitis are rare, and as the peritoneum possesses a normal resistance against pathogenic micro-organisms, practically all cases of peritonitis are secondary.

In the great majority of cases secondary peritonitis arises through an *extension* of an inflammatory process from some contiguous viscus or part. There is no organ or tissue in direct contact with the peritoneum from which such an extension may not occur. The possibilities are so numerous that it is necessary to refer here only to the most common or important. The extension to the peritoneum may or may not be accompanied by perforation into the peritoneal cavity. In the latter case the resulting inflammation is known as a perforative peritonitis.

Perforative Peritonitis.—This is the most common form of secondary peritonitis. It may result from the perforation into the peritoneal cavity of an infected wound of the abdominal wall or of a viscus (traumatic or operative), or from the perforation, as the result of inflammatory processes, of the wall of the stomach, intestine, gall bladder, pancreatic duct, urinary bladder, uterus, tubes, blood-vessels, lymph vessels, etc. Abscesses of the abdominal viscera, echinococcus cysts, tumors, burrowing suppurative processes in the retroperitoneal tissues, etc., may also give rise to perforative peritonitis. It is hardly expedient to enumerate here all the possibilities whereby a perforative peritonitis may arise, and only those of greatest clinical importance will be mentioned.

In the case of the *stomach* perforative peritonitis may be caused by carcinoma, gastric ulcer, abscess, phlegmonous gastritis, action of corrosives, foreign bodies, etc. In the *intestines* ulcers (typhoid, dysenteric, catarrhal, tuberculous, syphilitic, carcinomatous, etc.), new growths, foreign bodies, parasites, gall stones, fæcal concretions, fæcal obstruction, intussusception, ileus, hernial incarceration, etc., may lead to perforative peritonitis. Perforation of a suppurating *appendix* is one of the most common causes of peritonitis.

In the case of the *spleen* perforative peritonitis may be caused by splenic infarcts, abscesses, echinococcus cysts acute splenic tumor leading to rupture of the organ, rupture of a dilated splenic vein, metastatic tumors, etc. In the *liver* the same condition may result from abscesses, echinococcus cysts, rupture of bile passages, biliary concretions, new growths, etc. Rupture of the *gall bladder* or *common duct*, ulcerative processes in the walls of the gall bladder or ducts, presence in these of concretions, etc., may likewise lead to perforative peritonitis. This may be caused also by pancreatic concretions, abscesses and tumors of the *pancreas*, hemorrhagic and purulent pancreatitis, etc. Fat necrosis may or may not be associated with the peritonitis dependent upon pancreatic conditions.

Perforative peritonitis may also arise in connection with abscesses and tumors of the *kidneys* or *adrenals*, kidney tubercles, echinococcus cysts, renal calculi, purulent pyelitis, peri- and paranephritic abscesses, etc. Ulcerative and phlegmonous processes of the *urinary bladder*, new growths of the bladder, cancer of the *uterus*, purulent and gangrenous conditions of the uterine wall, purulent salpingitis, tubal pregnancy, tubo-ovarian abscess, ovarian tumors, rupture of Graafian follicle and ovarian cysts, suppurative processes of the retroperitoneal and mesenteric lymph glands, thrombophlebitis, and thrombo-arteritis of the abdominal vessels, rupture of abdominal aneurisms, rupture of the thoracic duct or dilated lymphatics, subperitoneal tumors and abscesses, psoas abscess, carious and purulent processes in the spinal column and pelvic bones, etc., may all lead to perforative peritonitis. In rare cases purulent processes proceeding from the mediastinum, pleura, pericardium, lungs, diaphragm, thoracic vertebræ, etc., may rupture into the peritoneal cavity and there excite an inflammation.

In the case of perforative peritonitis there will be found in the peritoneal cavity, in addition to the inflammatory exudate, various substances of very different nature according to the location of the perforation and the character of the contents of the cavity communicating with the peritoneal sac. Stomach contents, fæces, fæcal concretions, bile, biliary concretions, pancreatic juice, urine, urinary calculi, foreign bodies, parasites, blood, chyle, mucin, pseudomucin, colloid, etc., may be mixed with the exudate. According to the character and amount of the substances thus entering the cavity, and according to the character and amount of the peritoneal exudate the appearances seen at operation or autopsy will differ greatly.

Some writers distinguish a *secondary perforative peritonitis*, grouping under this head those conditions in which the exudate of a purulent peritonitis, originally not perforative, gains entrance to some neighboring organ, and through it finds an exit, as, for example, through the stomach, intestine, pleura, abdominal wall, etc. Such cases should more properly be styled *perforating peritonitis*.

Peritonitis Arising by Contiguity.—Peritonitis may arise secondarily by the direct extension of inflammation caused primarily by wounds (operative or traumatic) of the abdominal or pelvic wall, gastric ulcer, gastric cancer, phlegmonous gastritis, toxic gastritis, duodenal ulcer, intestinal ulceration (typhoid, dysenteric, catarrhal, tuberculous, syphilitic, carcinomatous), new growths of the intestine, coprostasis, intussusception, ileus, hernial incarceration, presence of intestinal concretions, parasites and foreign bodies, typhlitis, appendicitis, proctitis, liver abscess, new growths of liver, hepatitis, angiocholitis, inflammatory processes due to echinococcus, biliary

concretions, etc., pancreatitis, pancreatic abscesses and presence of pancreatic concretions, splenic abscess and infarction, hydatid disease of spleen, kidney abscess, peri- and paranephritic abscesses, inflammatory processes caused by renal calculi, pyelitis, ureteritis, cystitis, inflammations of ovary, tubes, uterus, vagina, prostate, retroperitoneal or mesenteric glands, psoas abscess, congestive abscesses of spinal column or pelvic bones, inflammations of abdominal or pelvic vessels, suppurating buboes in the inguinal region, inflammatory processes of the mediastinum, œsophagus, pleura, pericardium and diaphragm, etc. In the case of inflammatory processes in the pleural cavity (tuberculous, empyema, etc.) the peritoneum is not infrequently involved, inasmuch as the lymph vessels of the diaphragm form a direct connection between the pleural and peritoneal cavities.

Next to the gastro-intestinal tract the *female genital tract* is the most frequent source of peritoneal infection. In women genital peritonitis is by far the most common form of the affection. During the puerperium or as the result of attempts at abortion, operative procedures, intra-uterine injections, etc., the genital tract may be directly infected; less frequently the infection occurs at other times, as for instance at the menstrual period. The resulting endometritis, metritis, parametritis or salpingitis may extend to the peritoneum and excite peritonitis. Purulent processes of the endometrium may extend directly up the Fallopian tubes. In other cases the extension of purulent processes from the vagina or uterus may take place through the lymphatics or blood-vessels. Large abscesses of the uterine wall or tube may break directly into the peritoneal cavity. Further, cases of apparently primary peritonitis have been reported as occurring in pregnancy without any association with rupture of the uterus or other complications of pregnancy. The discovery at autopsy of the existence of extensive renal changes makes it probable that such cases of peritonitis are secondary to the nephritis.

Gonorrhœa of the female genital tract is also an important factor in the production of peritonitis (see below). It must be noted further that in many cases of septic puerperal peritonitis the uterus and tubes serve as avenues of entrance to the agents of infection while remaining themselves in a normal condition.

An especial factor in the production of peritonitis of the female genital tract is that offered by the twisting of the pedicles of ovarian cystomata or of subserous uterine myofibromata. As a result of the shutting off of the circulation from the tumor, the tissues of the latter undergo necrosis, and the necrotic material offers a most favorable field for infection, particularly from the intestine. Such infections are very likely to result in a gangrenous peritonitis. In case infection of the dead tumor tissue does not occur a non-infectious peritonitis may result, the dead tumor acting as an irritating foreign body. A very virulent peritonitis may be thus excited, the exudate as well as the necrotic tumor containing no micro-organisms. Dermoid cysts appear to be especially susceptible to infection after twisting of their pedicles.

The rupture of an uninfected ovarian cystoma and the discharge of a thin serous fluid into the peritoneal cavity rarely excite a peritonitis, the fluid being quickly absorbed. The absorption power of the peritoneum under such circumstances is very great. Only those cystomata containing a firm jelly-like substance (mucin, pseudomucin, colloid) are dangerous, since the cyst contents cannot be easily absorbed and act upon the peritoneum as foreign bodies, leading to an organization and vascularization of the material (aseptic peritonitis, pseudomyxoma peritonei). It should be remembered, however, that in such cases secondary peritonitis is very likely to occur owing to the diminished resistance of the peritoneum; consequently a combination of pseudomyxoma and an acute or subacute peritonitis is not infrequent. Operation with the removal of the jelly-like cyst contents from the peritoneal surface after organization has begun is especially likely to be followed by infection of the raw surface thus exposed. In the case of the rupture of cysts containing chemically irritating material (*pancreatic cysts, chyle cysts*), a non-infectious, acute, and very virulent peritonitis may be set up.

Secondary peritonitis is also not infrequently associated with metastatic carcinoma (*peritonitis carcinomatosa*) and tuberculosis of the peritoneum (*peritonitis tuberculosa*). These conditions will be discussed below.

In the *acute infections* peritonitis is occasionally met with as a complication. In *septicæmia* and *pyæmia* peritonitis is a very frequent complication. It occurs less often in *pneumonia, smallpox, varicella, measles, scarlet fever, diptheria, erysipelas, malaria, relapsing fever*, etc. From the increasing number of cases reported *pneumonia* seems to be somewhat particularly associated with peritonitis, and the pneumococcus has recently been found in the peritoneal exudate of a number of cases. The peritonitis may be secondary to the pulmonary symptoms or may appear to be the primary condition, the peritonitis manifesting itself before the pulmonary affection. It is probable that such cases represent a general infection by the pneumococcus, the localization varying in different cases. They may therefore be classed as *peritonitic pneumonia*, in analogy to the types known as cerebral and gastric. Pneumococcic peritonitis may also be secondary to pneumococcic pericarditis, empyema, and arthritis. The occurrence in various parts of the body of multiple metastatic abscesses containing peumococci is evidence of the pyæmic nature of the process.

According to Bednar peritonitis may develop after *vaccination*.

Metastatic peritonitis occurs in *malignant endocarditis* and in *pyæmia*. The primary focus may occur in any part of the body, and may be of very slight extent. Peritonitis has followed the infection of a circumcision wound, and very slight lesions in other parts of the body have led to the same fatal termination. It is not uncommon in such cases to find all the serous membranes involved—an acute panserositis; meningitis, pleuritis, pericarditis, and arthritis as well as peritonitis. Peritonitis may also develop during the course of *acute articular rheumatism*. The exact relation of the two conditions is unknown; the peritoneal inflammation is either metastatic or dependent upon a lessened resistance of the membrane to cryptogenic infections. Likewise acute peritonitis is sometimes associated with *acute nephritis*. It is possible that in these cases the intoxication plays some part in the development of the process, most probably by lowering the resistance of the membrane. The exudate in these cases is usually serous, and bacteria cannot always be obtained from it by culture methods; hence some writers regard these forms of peritonitis as of chemical origin and primary.

Terminal Peritonitis.—Peritonitis also occurs not infrequently as a terminal infection in chronic valvular disease, arteriosclerosis, chronic nephritis, cirrhosis of the liver, amyloid disease, tumor cachexia, scorbutus, etc. The peritoneum alone of the serous membranes may be affected, or the pericardium and the pleura may also become inflamed. Micro-organisms are usually present in the exudate. In those cases in which they are not found it is possible that they were present earlier in the disease, and later disappeared. It is of course possible that the peritonitis in some of these cases may be toxic or chemical, but it is more likely that the general intoxication has lowered the resistance of the membrane and cryptogenic infection has occurred.

There is also a close relationship between *syphilis* and peritonitis. Acute peritonitis not infrequently develops in the early stages of acquired syphilis, but is usually a secondary infection. It is not known whether an actual syphilitic peritonitis exists, though it is very probable that it does, inasmuch as a so-called idiopathic peritonitis is of common occurrence in syphilitic new-born. In the tertiary stage of acquired syphilis evidences of local peritonitis are common enough at autopsy. Chronic perihepatitis and chronic perisplenitis are more common in syphilitics than in non-syphilitics.

Foreign bodies (concretions, parasites, forceps, needles,

hairs, etc.) when aseptic produce a local encapsulating peritonitis When septic they may excite a diffuse purulent or fibrinous process. Free bodies, such as fat tissue, blood clots, pieces of fibroid tumors, etc., may become calcified or encapsulated, setting up a local adhesive peritonitis. In *extra-uterine pregnancy* hemorrhage into the peritoneal cavity following the rupture of the sac may excite an intense peritoneal irritation even in the absence of infection. In such cases the blood itself must exert a chemical irritation upon the peritoneum, or irritating toxins are contained or developed within it.

Finally, during *intra-uterine injections* irritating substances may be forced through the tubes into the peritoneal cavity and excite a toxic inflammation. Such cases may occasionally be properly classed with the primary forms of peritonitis, but inasmuch as some inflammatory condition of the uterus or tubes is usually present it becomes difficult to say to what extent the injection is alone responsible for the peritonitis.

Parasites.—A number of observers have noted at autopsy the presence, in the peritoneal cavity, of intestinal worms in association with a perforative peritonitis. In some cases the perforation has been regarded as caused by the parasite (usually the common round worm), the peritonitis following the perforation. Some of the reported cases are very doubtful. Katsurado several years ago reported two cases of purulent peritonitis which he regarded as caused by round worms. A very careful examination, both at the time of operation and at the autopsy, failed to reveal any other cause for the peritonitis. In both cases the affection ran a very severe course. Osler and other recent writers accept the view that *Ascaris lumbricoides* may penetrate the intestinal wall and excite peritonitis. The *Amœba coli* has been found in the exudate of peritonitis.

Under rare conditions peritonitis may follow other conditions not mentioned above. As stated before it is hardly feasible to enumerate all the possibilities, but the most common and important sources of secondary peritonitis have been given; and if the extensive anatomical relations of the membrane are borne in mind it will usually not be difficult to trace to its source any secondary peritonitis that may be encountered.

Bacteriology.—Inasmuch as the great majority of cases of peritonitis are caused by bacteria the bacteriology of peritonitis becomes a matter of very great importance. In recent years a number of important investigations have been carried out along this line, but much work yet remains to be done before we shall possess a fully satisfactory knowledge of the subject. It has been demonstrated that very different forms of bacteria may be found in the exudate of peritonitis. In the majority of cases there is a mixed infection, several varieties of pathogenic bacteria being present, or a number of varieties of pathogenic and non-pathogenic bacteria may be found together. According to Tavel and Lanz the hæmatogenous cases of peritonitis are mono-infections, while those arising by contiguity from a neighboring diseased organ are usually poly-infections. It is probable that in some at least of the cases of mono-infection the single variety found may have crowded out other varieties which were originally present. This is particularly likely to have been the case in those instances in which the only micro-organism present is a harmless saprophyte. In those cases in which the exudate is sterile there is also the possibility that the original pathogenic bacteria causing the inflammation have died out.

The pathogenic bacteria most frequently found in peritonitis are the *Streptococcus pyogenes, Staphylococcus aureus, Staphylococcus albus, Bacterium coli commune, Diplococcus pneumoniæ, Bacillus pyocyaneus,* and the *Gonococcus.* (Tuberculosis of the peritoneum will be considered separately.) Less frequently found are the *Bacillus typhosus, Bacillus aërogenes capsulatus, Bacillus proteus, Bacillus lactis aërogenes, Micrococcus tetragenus,* and a number of unidentified pathogenic and non-pathogenic bacteria.

In perforative peritonitis the *Bacillus coli communis* usually predominates; in puerperal peritonitis the chief rôle is played by the *Streptococcus pyogenes;* while in those cases of peritonitis which apparently arise spontaneously the *Pneumococcus* or the *Gonococcus* is the chief exciting factor. In all those cases of peritonitis in which the infection comes from without the flora resembles that of ordinary surgical infections, except for the prominent part played by the colon bacillus. In those cases which arise by contiguity from some one of the neighboring organs the staphylococcus plays a chief rôle, except in the case of intestinal infection in which the colon bacillus alone or combined with the former is usually found.

Much discussion has taken place as to the pathogenic rôle of the colon bacillus in peritonitis. The pyogenic properties of this organism have been quite generally accepted, and there can be no doubt of the existence of a primary colon-bacillus peritonitis. In the majority of cases in which the colon bacillus is found pyogenic cocci are also present, and it is probable that in some of the instances in which this organism alone was found in the exudate it had overgrown the cocci. Barbacci in experimental work upon dogs found that the colon bacillus often outgrew the pyogenic cocci. Infections of the peritoneum with the colon bacillus occur through the bladder and female genital tract as well as directly from the intestine. There are still several questions to be settled before the exact pathogenic value of the colon bacillus can be fixed. Its relations to the pyogenic cocci need to be more definitely determined in regard to the part played by each as primary excitants of inflammation. Further, many of the cases regarded as colon-bacillus infections were diagnosed as such from the results of the bacteriological examination made at autopsy some hours after death. As the colon bacillus may enter the peritoneal cavity after death and increase there, overgrowing other bacteria, it is necessary, in order definitely to determine the relation of this organism to a peritonitis, to make bacteriological examinations of the peritoneal exudate during life.

An effort has been made by a number of writers to make a bacteriological classification of peritonitis. Freund divided all cases of peritonitis into three classes: one due to pyogenic organisms, one to fæcal bacteria, and the third to toxic substances. Bumm distinguished a septic, aseptic, and a specific peritonitis. The septic he divided into a streptococcus peritonitis and a putrid peritonitis, the latter not caused by any one form of bacteria but by a multiple infection. The aseptic peritonitis he regarded as due to mechanical, thermal, or chemical influences alone without bacterial agency. In the class of specific peritonitis he placed tuberculous and gonorrhœal peritonitis. Tavel and Lanz have offered a more complex classification based upon the anatomical origin of the infection, special kinds of peritoneal infection being derived from the stomach and intestine, the gall bladder and liver, the female genital tract, the kidneys, urinary bladder, etc., other forms still being derived from hæmatogenous infections and operations.

Flexner has reported a series of observations upon one hundred and six cases of peritonitis studied from the bacteriological point of view. The material was obtained at autopsy, cover-glass and aërobic cultures being carried out. Few anaërobic cultures were made and the pathogenic properties of the bacteria isolated were tested but rarely. In spite of this incompleteness of the investigation the results are of interest. Twelve of the cases presented the character of a primary peritonitis according to Flexner's definition. In all these cases there was a previous chronic disease. In two cases no micro-organisms were found; in nine cases there was a single infection, and in one case a multiple infection. The *Streptococcus pyogenes* was found five times, four times alone, and once in association with the colon bacillus; the *Staphylococcus aureus* and *albus* twice, the *Micrococcus lanceolatus, Bacillus proteus, Bacillus pyocyaneus,* and an unidentified bacillus occurred twice each.

The secondary cases Flexner divides into two classes: *exogenous* peritonitis and *endogenous* peritonitis. In the former the infection entered from without (wound infection); in the latter the bacteria came in part or wholly from the intestinal tract. Thirty-four cases of the exogenous form were examined. Of these, 25 were mono-infections, nine were multiple. The following tables are taken from Flexner's article (Philadelphia *Medical Journal,* 1898):

Micro-organisms Found in Cases of Exogenous Peritonitis.

	Total number of cases.	Alone.	Combined.
Staphyloccocus aureus	15	12	3
Staphylococcus albus	3	2	1
Streptococcus pyogenes	10	5	5
Bacillus coli communis	7	2	5
Micrococcus lanceolatus	3	1	2
Bacillus proteus	1	0	1
Bacillus pyocyaneus	2	0	2
Unidentified organisms	3	0	0(?)

Combinations.	Number of cases.
Staphylococcus aureus and Streptococcus	1
Staphylococcus albus and Streptococcus	1
Staphylococcus albus and Bacillus proteus	1
Staphylococcus albus and Bacillus coli communis	1
Streptococcus and Bacillus coli communis	1
Streptococcus, Bacillus pyocyaneus and Coli-communis	1
Streptococcus and Bacillus pyocyaneus	1
Micrococcus lanceolatus and Bacillus coli communis	1
Micrococcus lanceolatus and liquefying bacillus	1

Of 60 cases of endogenous peritonitis 58 gave positive bacteriological results. Single infections occurred in 21 cases of these, and multiple infections in 37.

Bacteria Found in Cases of Endogenous Peritonitis.

	Number of cases.	Alone.	Combined.
Bacillus coli communis	47	9	38
Streptococcus pyogenes	39	7	32
Staphylococcus albus	4	2	2
Staphylococcus aureus	3	1	2
Micrococcus lanceolatus	4	1	3
Bacillus proteus	4	2	2
Bacillus aërogenes capsulatus	8	2	6
Bacillus pyocyaneus	3	0	3
Bacillus typhosus	3	0	3
Unidentified	3	0	3

Combinations.	Number of times.
Streptococcus and Bacillus coli communis	16
Streptococcus, Bacillus aërogenes, and Bacillus coli	2
Streptococcus and Bacillus aërogenes capsulatus	1
Streptococcus and Staphylococcus aureus	1
Streptococcus and Bacillus typhosus	2
Streptococcus and Staphylococcus aureus, Bacillus typhi, proteus and coli	1
Streptococcus and Bacillus proteus	1
Streptococcus and unidentified organism	1
Bacillus coli and Micrococcus lanceolatus	3
Bacillus coli and Bacillus pyocyaneus	3
Bacillus coli and Bacillus aërogenes capsulatus	2
Bacillus coli, Staphylococcus aureus, and Bacillus aërogenes capsulatus	1
Staphylococcus albus and orange sarcina	1
Staphylococcus albus and unidentified organism	1

The comparison of the table in the exogenous and endogenous forms is of interest. It will be seen that multiple infections are relatively more frequent with the endogenous form, and that the variety of bacteria is much greater. The streptococcus takes the place which the staphylococcus holds in the exogenous cases and the colon bacillus plays a much more important rôle. The small part which the pathogenic staphylococci seem to play in endogenous peritonitis is of clinical importance, inasmuch as the streptococcic infections may be associated with metastasis. The infections in the exogenous cases resemble those of ordinary surgical or traumatic infections. Basing his conclusions upon the observations, Flexner would distinguish three forms of peritonitis: a primary or idiopathic form restricted to a small number of terminal infections; a second variety analogous to surgical infections; and a third variety dependent upon disease of an intraperitoneal organ, whereby micro-organisms and other extraneous substances gain entrance to the peritoneal cavity, break down its resistance, and lead to infection.

Pneumococcic Peritonitis.—Cases of peritonitis due to the *pneumococcus* have been described by Frommel, Fraenkel, Charrier, Veillon, Beco, Bryant, Comby, Brun, Burckhard, and others. Brun reports fourteen cases of pneumococcus peritonitis in children. The majority of the cases of peritoneal infection with the pneumococcus occur in young girls. The avenue of infection is through the genital tract. The peritonitis may or may not be associated with inflammations of other serous membranes. The infection is frequently localized in the pelvis. The prognosis is favorable in those cases in which the exudate becomes encysted by adhesions, but metastases and localizations of the micro-organism in other parts of the body may occur. The diffuse form is usually fatal. In the case of operative procedures in pneumococcus pyosalpinx the greatest care must be taken to prevent infection of the peritoneum since the peritonitis thus excited is usually very virulent. Pneumococcus peritonitis may also result from extension or perforation in the case of a pneumococcus appendicitis. The relation to pneumonia and arthritis has been mentioned above. It is of importance in the diagnosis of this condition to bear in mind the fact that after death the pneumococci may entirely disappear from the peritoneal exudate, the colon bacillus or some other organism being left. Cases have been reported in which the examination of the pus from the peritoneal cavity during life, or soon after death, showed the presence of pneumococci in great numbers, while examinations made twenty-six to forty-eight hours later showed only the presence of intestinal bacteria.

Gonorrhœal Peritonitis.—The occurrence of an acute general peritonitis caused by the *Gonococcus* is now firmly established, and the condition is probably not infrequent in women affected with gonorrhœa of the internal genitals. Wertheim in opposition to Bumm was the first to maintain that gonococci may multiply within the peritoneal cavity and excite there an inflammation which may spread over the entire peritoneum. An increasing number of such cases is being reported. The view of Bumm and others, that the *Gonococcus* finds no favorable soil upon serous surfaces, and that its growth upon the peritoneum is slight and localized has been completely overthrown by recently reported cases. The infection of the peritoneum with the *Gonococcus* occurs chiefly in women, although cases of gonorrhœal peritonitis in men have been reported by Jadassohn, Horowitz, von Zeissl, and others. In the female the infection usually proceeds from the tubes. In the case of an infection of the woman with very virulent gonococci there may develop suddenly a most severe general peritonitis. The passage into the peritoneal cavity, from the mouth of the tube, of pus rich in gonococci always excites a general peritonitis; if the flow is small, and if but few gonococci are present in the pus, or if these are reduced in virulence a local peritonitis is usually set up. Gonococcic peritonitis is relatively more frequently a complication of gonorrhœal vaginitis and vulvitis in young girls. Such cases are unfortunately not rare in some of our large cities. The belief held by certain of the lower classes that gonorrhœa in the adult may be cured by rubbing the male organ over the external genitals of young girls is probably responsible for some of these cases. The peritoneal affection may appear as a mild or severe acute general peritonitis, or it may run a latent course as a chronic inflammation. The prognosis in these cases is always serious. Recovery may be followed by sterility.

Pathology.—The pathological changes found in the peritoneum in inflammation of this membrane are essentially the same as those occurring in inflammatory processes of the pleura and pericardium. According to the duration of the inflammation there may be distinguished an *acute, subacute,* or *chronic* peritonitis. According to the extent of the membrane involved a peritonitis may be