close relations to the embryo lead, however, to pathological processes peculiar to itself.

Hæmatoma.—An effusion of blood between the chorion and the amnion may occur as the result of accidental or voluntary trauma, or of diseased conditions of the chorionic villi. Rupture of the umbilical vessels may lead to the formation of a very large blood clot between the two membranes. The small extravasations from the capillaries of the chorionic villi are relatively frequent and have little significance, but large ones may strip the amnion from the chorion over a large area, producing abnormal pressure upon the embryo and alterations in the amniotic secretion. Death of the embryo and abortion may result from these causes, or the pressure upon the amnion may produce adhesions between it and the fœtus, leading to disturbances of development. The small clots are absorbed and replaced by new chorionic villi or fibrous connective tissue, or they may become calcified.

Retrograde Changes.—The placenta and fœtal membranes at term must be regarded as senile structures, and certain retrograde changes must be recognized as a part of their physiological decay. These signs of age in the amnion begin as early as the fourth month, and manifest themselves chiefly by degenerative changes in the mesodermal nuclei, as shown by diffusion of chromatin, hydropic and fatty degeneration. Marked alterations in the shape of these cells occur in the later months, but these changes are probably dependent upon the tension of the membrane.

Fatty Degeneration.—Minute fat droplets are very frequently found in the mesodermal cells of the mature amnion, and are to be regarded as physiological. In retention of the membranes after abortion this change may reach a pathological degree.

Hydropic Degeneration.—This may occur to a limited extent in the mature amnion. In the rare cases of œdema of the chorion the cells of the amnion become hydropic; and frequently, after death of the fœtus, both the cells and intercellular substance of the mesenchyma undergo liquefaction.

Myxomatous Degeneration.—A myxomatous degeneration of the amnion may be associated with a similar change in the chorion. The mesodermal cells become branched, the intercellular substance more gelatinous in character, and small mucin-containing cysts may be formed in the mesenchyma. A hyperplasia of the mesodermal cells may precede this change, and the amnion may be greatly thickened throughout, or villous-like projections into the amniotic cavity may be formed. In very rare cases these may acquire such size that they may be classed as myxomata. These changes are of most frequent occurrence in the earlier months of pregnancy and usually follow the death of the fœtus.

Colloid-like Change.—The mesoderm of the amnion not infrequently becomes homogeneous and hyaline, loses its cells and slight fibrillation, and stains as colloid. The exact nature of this change is not known. It occurs rather frequently after the death of the fœtus.

Hyaline Change.—Portions of the amnion may undergo a proliferation of the connective-tissue cells, leading to the formation of a more mature connective tissue in which fibres are formed. The intercellular substance acquires a true hyaline character and stains rose red with Van Gieson's stain. This sclerosis in a limited degree may be considered as a senile change, but in the normal amnion it is never extensive, and the membrane for the greater part preserves its embryonic character. Any extensive hyaline change must be regarded as pathological. The causes and conditions of occurrence of this change are not known, but it may occur after the death of the fœtus or in connection with syphilitic changes in the chorionic villi.

Calcification.—Small plaques of calcification are not infrequently found at full term upon the inner side of the amnion, most frequently of the placental amnion. These most probably are calcified masses of fibrinous exudate. After the death of the fœtus lime salts may be deposited in the amniotic mesoderm. This deposit may be preceded or accompanied by fatty, myxomatous, or hyaline change. The pathological significance of calcification, either of the chorion or of the amnion, is probably very slight, and the deposits of lime salts found in these structures at delivery, unless very extensive, are to be regarded as senile phenomena.

Hyperplasia.—After the death of the fœtus the amnion may become much thickened from a hyperplasia of the mesodermal cells. The new tissue formed usually undergoes hyaline or myxomatous change or calcification. Localized hyperplasias may appear as new growths. The processes underlying these changes are practically unknown, but hyperplasia of the amnion is associated, at least in some cases, with syphilitic hyperplasia of the chorionic villi.

Amniotitis.—Since the amnion contains no blood-vessels, a primary inflammation in the ordinary acceptance of the term cannot occur in the membrane. But the tissues of the amnion may become involved in changes which are so analogous in character and sequelæ to inflammatory processes that the use of the term Amniotitis may be accepted for practical reasons. Œdema and liquefaction of the intercellular substance of the mesenchyma may occur; fibrinous exudates may take place, leading to the formation of extensive deposits of fibrin on the epithelial surface of the membrane, and to the presence of strings and bands of fibrin in the lymph spaces of the mesenchyma. The gelatinous tissue connecting the amnion and chorion may wholly or partially liquefy, its number of wandering cells may be increased, and in very rare cases pus may be formed between the two structures. In these cases there is also present a small-celled infiltration of the chorionic villi. The umbilical cord may show a similar infiltration. Purulent placentitis is apparently very rare, and no well-studied cases have been reported. I have seen two cases of placental abscess resulting from infection of the placenta through attempted abortion. In these the space between the chorion and amnion contained masses of fibrin and collections of leucocytes; the amnion was swollen and colloid-like, containing fibrin strings and large numbers of leucocytes.

In both of these cases the amnion was involved by extension from the chorion; and it is probable that the fibrinous exudates, which are rather frequently found in and upon the amnion, are the result of primary pathological changes in the chorion or decidua. The existence of a primary amniotitis is yet to be proved. It has been stated that in cases in which the amniotic fluid is absent or greatly reduced in quantity, the friction of the fœtus upon the membrane leads to the formation of plastic exudates and adhesions. It yet remains to be proved that such exudates are the direct result of changes in the amniotic cells.

That fibrinous exudates do occur has been confirmed by numerous observers, but we are as yet at a loss to explain either their etiology or the manner of their occurrence. Through the organization of fibrinous adhesions between the fœtus and the amnion, fibrous bands may be formed which may lead to the production of marked abnormalities in the fœtus. These adhesions may also be formed between the coils of the umbilical cord or between it and the body of the fœtus. Amniotitis is also regarded as one of the causes of hydramnion, the overproduction of the amniotic fluid being explained as of the nature of an acute serous inflammation. The proof of this remains to be established, but the fact that hydramnion and the formation of adhesions between fœtus and amnion have followed traumatic injuries to the mother may be taken as support of this theory.

In the later months of pregnancy the epithelium of the fœtal surface of the amnion may be torn away in strips. According to Ahlfeld, this is the result of fœtal movements, the epithelium being scratched by the finger and toe nails of the fœtus. The amnion may burst in the last months of pregnancy, the ovum being preserved by the chorion. Through the movements of the fœtus, the torn

membrane may be rolled up into bands, which may become entangled with the umbilical cord and constrict it even to the extent of shutting off the fœtal blood supply. Inflammatory changes have not been shown to follow these conditions.

Amniotic Bands and Adhesions.—During the early stages of development of the membrane there may occur total or partial union of the amnion with the developing skin of the embryo. This union may be the result of an imperfect development of the membrane, in that it does not become differentiated from the ectoderm, or fits too closely about the embryo, so that the amount of secretion is not sufficient to separate the amnion from the surface of the embryo. At the points of contact, union through direct fusion or intergrowth may take place; or a plastic exudate may be thrown out which unites the surfaces and later becomes organized after the manner of plastic exudates on any serous surface. It is still an unsettled question as to how far these adhesions between the amnion and the fœtus are to be referred to a primary failure of separation and fusion, or to inflammatory processes; but it is probable that in the majority of cases they are primary defects of development.

These adhesions play a great part in the formation of monsters and malformations, and their teratological importance can hardly be overestimated. Bands and strings of union not infrequently persist at full term, and their connection with the misshapen portion of the child leaves no doubt that they bear a direct causal relation to the malformation. The structure of these bands usually resembles that of the amnion, containing no blood-vessels; and they may be covered with epithelium. In other cases they are to be regarded as prolongations and outgrowths of the fœtal dermis, and contain blood-vessels which arise from those in the fœtal skin. Very frequently the only remnants of these bands at birth are short tags in the skin of the child. These have a structure similar to that of normal skin. Stretching of the adhesions through increase of the amniotic fluid may lead to their atrophy or to the formation of fibrous bands, which contain few cells and no blood-vessels and possess no epithelial covering.

A total adhesion of the membrane to the embryo causes marked disturbances of development of the head and extremities. Partial adhesions occur most frequently at the extremities of the embryo. An abnormal tightness of the cephalic cap may lead to marked malformations of the cranium, brain, or face (acrania, anencephalia, exencephalia, cephalocele, cyclopia, arrhinencephalia, etc.); while abnormal tightness of the caudal cap produces a deficient development of the lower extremities (amelia, phocomelia, etc.). Clefts of the thoracic and abdominal walls, failures of closure of the dorsal and genital furrows, etc., are also associated with deficient growth of the amnion. It is impossible to say to what extent this association is one of cause and effect or merely a coincidence.

If the amniotic fluid increases greatly in amount at an early period, portions of the adhesions may be separated and torn loose, floating in the fluid; or remaining attached at the ends, they may become stretched into fine threads and bands. These may entangle the extremities of the fœtus and affect their development through pressure and disturbance of blood supply, or even cause intra-uterine amputations. The variety of malformations produced in this way is very great. Larger bands of adhesions may divide the amniotic cavity into several chambers, and an over-accumulation of fluid in one or several of these cavities may result in the production of pressure malformations (club-foot, flat-foot, etc.).

Hydramnion (see *Hydramnion*).—The pathology of an abnormal increase of the amniotic fluid remains unsettled. It is evident that a number of factors may underlie this condition. It may be acute or chronic. The latter may be due to pathological changes in the mother (œdema and dropsy from nephritis, cardiac disease, etc.), hypertrophy of placenta and decidua, placental tumors, persistence of chorionic vessels which normally undergo obliteration, abnormalities of the umbilical vessels; or to pathological changes in the fœtus (increased blood pressure, cardiac hypertrophy, obstruction of the ductus Botalli, syphilitic cirrhosis, fœtal tumors, oversecretion of urine, as in the case of unioval twins, lymphangiomatous conditions of the fœtal skin, etc.). Deficient absorption of the fluid may also lead to an overproduction of the fluid. In some instances, as in syphilis, disease of both the mother and child may contribute to an excessive formation of the fluid. Acute cases following trauma to the mother have been ascribed to the occurrence of an acute serous amniotitis. Other cases of acute hydramnion arise without apparent cause. These cases are most common during the fourth and sixth months of pregnancy.

Oligohydramnion.—A deficient formation of the amniotic fluid may occur, but the pathology of the condition is as obscure as that of hydramnion. It is commonly found in cases in which extensive adhesions exist between the fœtus and the amnion, and in the case of twins in which one sac may present a deficiency of the fluid, the other an excess.

Abnormalities.—A large number of varieties of abnormal development of the amnion have been described. The most important of these, the bands and adhesions, have been mentioned above. Defects of the membrane, total or partial reduplication, formation of multiple cavities, etc., may occur. The etiology and the manner of production of these are unknown.

Tuberculosis.—Primary tuberculosis of the amnion has not yet been reported. In one of two cases of placental tuberculosis which I have seen, miliary tubercles were found in the chorion just beneath the amnion, which was thickened and adherent, showing small-celled infiltration and signs of connective-tissue proliferation.

Syphilis.—In syphilis of the fœtus and fœtal placenta a hyperplasia of the amnion similar to that of the chorion may take place. This may lead to a general or localized thickening of the membrane, and is associated with various degenerative processes (fatty, colloid-like, hyaline).

New Growths.—Cysts of the amnion have been described. These were small and without clinical significance. They were most probably due to a myxomatous degeneration of the mesenchyma. Small myxomatous projections into the amniotic cavity occur rarely. They are either localized hyperplasias or remains of adhesions which have undergone a myxomatous change. The existence of true amniotic neoplasms is as yet doubtful.

Extra-Uterine Pregnancy.—In extra-uterine pregnancies, either before or after the death of the embryo, the tissue of the amnion may undergo extensive hyperplasia, and become greatly thickened. It may contain new blood-vessels, which penetrate it from the external cyst wall. After the death of the fœtus the entire amnion may become calcified, forming a calcareous cyst wall, from which the mummified fœtus may be easily shelled out (lithokelyphos); or if adhesions exist between the fœtus and the membrane, these may also become calcified, while the remaining portion of the fœtus undergoes mummification (lithokelyphopædion).

Aldred Scott Warthin.

AMŒBÆ PATHOGENIC FOR MAN.—The amœba belongs to the class of the rhizopoda, of the system of the protozoa. The first recorded observation of an amœba pathogenic for man is that of Lambl, who, in 1859, described amœbæ and other organisms in the mucus from the intestine of a child with enteritis.

The amœbæ described as pathogenic for man may be classed as follows: (1) Amœba coli; (2) amœba oris hominis; (3) amœba urogenitalis.

1. The amœba coli, first described by Loesch in the stools of an individual with dysentery in St. Petersburg in 1875, has, within the last fifteen years, been found in the stools, intestinal contents and lesions, peritoneum, liver and lung abscesses, pleura and pericardium, and sputum of man by numerous observers in various parts of the world. It has been found in some or all these lesions in Egypt by Kartulis, Koch, Kruse and Pasquale;

in Greece by Kartulis; in Bohemia by Hlavna and by Epstein; in Austria by Manner, Kovác (patient from Sumatra), and Cohen; in Germany by Nasse (patient from Florida), Quincke and Roos, Boas, Borchart, Roemer, and others; in Italy by Calandruccia, Fenalglia, Vivaldi, Maggiora, and others; in Russia by Massiutin; in France by Peyrot and Roger; in Roumania by Babes and Zigura; in Siberia by Lobas; in England (in patients from India) by Mason and Galloway, Harold, Curnow, Marshall, Windsor, and others; and in Brazil by Lutz; in the Philippines by Flexner and Barker. Since Osler's first case in 1890, cases have been reported in the United States from Baltimore by Lafleur, Simon, Councilman and Lafleur, Howard, Thayer, Preston and Rührah, Lewis and others; from Texas by Dock; from Florida by Day and others; from Atlanta by Harris; from Charleston by Wasdin; from Birmingham by Wilson; from Virginia by Slaughter, Field, and Johnson; from Cincinnati by Eichberg; from Cleveland by Howard and Buxton, and others; from New York by Lockwood and others; from Buffalo by Stockston; from Boston by Councilman, Burrell, Strong, Fitz and Gerry, and others; from Columbus, Ohio, by Wilson.

Morphology.—The amœbæ described by Loesch varied in diameter from 8 to 37 μ, and had one or two blunt pseudopodia. The ectosarc was hyaline, but apparent on the putting out of pseudopodia. The finely granular endosarc contained a nucleus 5 to 7 μ in diameter, small non-contractile vesicles, red blood cells, and epithelial cells. Accurate descriptions of the organism have been given by Kartulis, Osler, Councilman and Lafleur, Kruse and Pasquale, Harris, and others. Most recent writers describe amœbæ larger than those of Loesch, from 10 to 50 μ in diameter; Kartulis, indeed, described giant amœbæ with a diameter of from 150 to 222 μ. When at rest the organisms are round or slightly oblong in shape, and show no distinction between ectosarc and endosarc, having the appearance of a somewhat refractive body enclosing clear, pale vacuoles of varying size. Some have fine, and others coarse granules, structures which, according to Councilman, represent fine vacuoles. Harris points out that the organism has a faint green or bluish-green color. A nucleus surrounded by a pale rim, and capable of changing its form, can sometimes be made out in fresh amœbæ. It can usually be demonstrated by appropriate staining methods.

Two kinds of motility are recognized: a progressive movement, and one characterized by the putting out and retracting of pseudopodia. When the latter are protruded, the ectosarc appears as a pale, hyaline, homogeneous substance, less refractive than the endosarc. Kruse and Pasquale distinguish four varieties of amœbæ in stools: (*a*) a form with poorly differentiated, highly refractive protoplasm—found in normal fæces; (*b*) amœbæ containing irregular and usually small granules—not uncommon in dysenteric stools; (*c*) amœbæ the endoplasm of which consists of larger and smaller vacuoles—most common form in dysenteric stools; (*d*) amœbæ containing many foreign bodies.

Quincke and Roos make three classes of amœbæ parasitic for man: (*a*) amœbæ intestini vulgaris, 40 μ in diameter, with large granulations, pathogenic for neither man nor cats; (*b*) amœbæ coli mitis, about the same size and appearance as the above, pathogenic for man but not for cats; (*c*) A. coli Loesch, or A. coli felis, about 25 μ in diameter, with finely granular endosarc. producing dysentery in both man and cats.

It should be noted that amœbæ have been found in the stools of individuals in health or with typhoid fever, and with cholera, by Cunningham, Lewis, Shuberg, Quincke and Roos, Kruse and Pasquale, and others. Shuberg and Quincke and Roos have found amœbæ in the stools of individuals after purging with Carlsbad salts. Here the serum poured into the intestine probably changed the reaction of the contents of the organ from acid to alkaline, and thus permitted the growth and survival of amœbæ reaching the alimentary tract by food or drink.

There are possibly several varieties of amœba coli, some of which are harmless. The form found in association with intestinal lesions usually has fine granulations.

Multiplication.—The amœba coli, like other amœbæ, multiplies by direct division; indirect division has not been observed. The organism is said to pass into an encysted stage, from which amœbæ may again be developed.

Cultivation.—The statement of Kartulis in one of his earlier articles, that he cultivated the amœba coli in straw infusion, has not met with general acceptance, and it has been suggested that he was dealing with the ordinary amœbæ of straw infusion. Celli and Fiocca have cultivated amœbæ from the intestinal contents of various animals and of man in both health and disease, as well as from the female genitalia, and from various waters. As a culture medium, they used a strongly alkalinized solution of fuscus crispus, a sea alga, in water or bouillon. On this solid medium they obtained cultures of amœbæ only slightly mixed with bacteria, and were also able to isolate different varieties of amœbæ. They describe the life history of amœba coli as follows: Amœboid state: the organism is lobular in form with numerous lobular hyaline pseudopodia, and measures from 10 to 30 μ in diameter. The ectoplasm is hyaline, the endoplasm uniform, and contains a vesicular nucleus, which may contain a vacuole. In the resting state the organism is from 1.5 to 2 μ in diameter, of single contour, and uniform, finely granular protoplasm. In the encysted stage, there is a double contour, an inner thick, and outer thin contour, while the cyst contents are finely granular. The cycle of development is given as follows: in from twelve to fifteen hours the organism passes from the encysted to the amœba stage, which usually lasts about forty-eight hours, after which the resting stage begins. After from sixty to sixty-five hours they again become encysted or degenerate.

Beyerinck cultivated on malt gelatin an amœba which he believes identical with amœba coli. The organism grew rapidly, both in separate colonies and as a spreading veil-like growth with marked liquefaction of the media. The latter, he thought, was due to trypsin. Spore and cyst formation were not observed. Multiplication took place by direct division. Schordinger claims to have cultivated amœba coli on hay-infusion agar from dysenteric stools. There is evidence that amœbæ may multiply in the intestinal contents.

Staining Reactions.—Living amœbæ are singularly resistant to ordinary dyes, which they take up very imperfectly. Harris has pointed out a marked affinity of living amœbæ coli for toluidin blue, which in weak aqueous solution stains the endosarc deeply at once, and the ectosarc somewhat slowly, giving a sharp differentiation. The stain also fixes the organisms and preserves their natural forms. Preparations after washing in water and mounting in Farrant's solution are said to keep for months. Both the coarser and finer methods of fixing and hardening amœbæ in fæces and other discharges, as well as in the tissues, have been tried with varying success. Councilman and Lafleur obtained the best results with Flemming's solution and safranin, but also got good pictures with methylene blue after alcohol hardening.

Mallory's differential stain gives excellent results with both pus and tissues, but not with fæces.

Mallory's Method: harden in alcohol; stain sections in saturated solution of thionin; differentiate with two-per-cent. aqueous solution of oxalic acid; wash in water, dehydrate in alcohol; clear in oleum origani cretici; wash off with xylol; mount in xylol balsam. "The nuclei of the amœbæ and the granulations of the Mastzellen are stained brownish red; the nuclei of the Mastzellen and all the other cells are stained blue" (Mallory).

After reviewing the various hardening and staining methods hitherto in vogue, Harris concludes that the internal structure of amœbæ is best shown by staining with Heidenhain's iron alum hæmatoxylin after corrosive sublimate hardening. He proposes a new method which gives excellent results.

Harris' Method: Harden in alcohol or corrosive sublimate, stain first in eosin or benzopurpurin, followed by a weak solution of toluidin blue for twenty or thirty minutes; wash in alcohol for three or four minutes; clear in cedar oil or xylol. Slight washing with alcohol shows the amœbæ stained dark blue with very dark nuclei; by further treatment with alcohol the bodies of the amœbæ take a reddish tinge, the vacuoles are distinct and only the periphery of the ectosarcic stained.

Reaction of Amœba Coli to Variations of Physical Condition and to Chemical Agents.—Extremes of heat have a decidedly harmful effect upon amœbæ. Celli and Fiocca have shown that in the amœbic stage they are killed by exposure to 45° C. for five hours, or to 50° C. for one hour, while in the encysted stage they resist 60° C. for one hour and 55° for four days. They also resist drying in either diffuse light or darkness, and in sunlight for two hundred and seventy hours at 12° to 15° C. While the amœba coli usually loses its motility when chilled, Harris has shown that it is not killed by exposure to a temperature of 0° C. Celli and Fiocca claim that both the amœbic and encysted stages resist 0° C. temperature.

The organism will not grow anaërobically, although it may be found 2 metres below the surface of the ground. In both the amœbic and encysted stages it is killed by the various chemical disinfectants used against bacteria. Solutions of quinine, permanganate of potash, hydrogen dioxide, and other substances used for irrigation of the intestine destroy the life of amœba coli. According to Harris toluidin blue rapidly kills the organism. It is very susceptible to the action of acids, in which it soon dies. It is killed by urine (Harris).

Physiological Characters of Amœba Coli.—This organism is phagocytic to an eminent degree, and commonly contains bacteria (both dead and alive), leucocytes, epithelial cells, red blood corpuscles (either unchanged or in various stages of degeneration). The cultural experiments of Beyerinck and the character of the tissue lesions (as first pointed out by Councilman and Lafleur) produced by this organism suggest that it, like certain other amœbæ, produces and sets free a digestive ferment, which is probably trypsin. The amœba coli exerts a direct dissolving action upon tissues, and *has very little if any positive chemotactic action on leucocytes.*

Lesions Caused by Amœba Coli.—(*a*) *Intestine.*—The intestinal lesions caused by this organism are peculiar and wellnigh characteristic. To Kartulis, and especially to Councilman and Lafleur, belong the credit of establishing amœbic dysentery as anatomically and etiologically a distinct disease. The lesions are most numerous in the sigmoid and descending colon, but commonly involve the rectum, transverse colon, and cœcum. The ileum and the appendix (Harris) may also be invaded. The process is characterized by the formation of ulcers, varying in size from a pin's head to that of the hand, which are usually deep, sometimes round, sometimes irregularly round or oval, or with irregular overhanging or undermined edges. The ulcers are often crater-like, and the tissue may have a peculiar honeycombed appearance (Councilman and Lafleur). The muscular coats, especially in the larger ulcers, are commonly laid bare and often dissected up, forming larger and smaller flaps attached at one side. The serosa opposite the ulcers is often thickened and may be covered with fibrin. In many places—and this is quite characteristic—larger and smaller abscesses are found in the submucosa, under an intact mucosa. These abscesses may communicate with the lumen or with ulcers by means of small openings, through which puriform material may be squeezed. The abscesses may burrow for a considerable distance in the submucosa. Councilman and Lafleur and others have established the fact that the process begins in the submucosa, and involves the mucosa and muscularis secondarily. In severe cases large sloughs may be cast off, and perforation occasionally occurs. Microscopically the walls of the ulcers and abscesses are covered with a granular detritus containing a few round cells, and sometimes a few epithelial cells and polymorpho-nuclear neutrophilic leucocytes. The absence of cellular infiltration, except where evidently due to bacterial mixed infection, is a marked feature of the process. The essential lesions seem to be swelling and disintegration of the tissue, which softens and breaks down. The line of necrosis is usually irregular. The blood-vessels may or may not be dilated and congested. The veins often show aggregations of round cells in their lumina and walls, and the latter may disintegrate and thrombosis may occur. Amœbæ are often seen in the veins, and may be seen penetrating their walls. Obliterative endarteritis of the vessels near the ulcers may occur. The lymph sinuses and lymph vessels often contain amœbæ and may show accumulations of cells. As the process advances from the submucosa toward the lumen, the muscularis mucosæ gives way, and, together with the mucosa, breaks down, giving rise to an ulcer. At the surface the mucosa may be reflected back over itself or may overhang the ulcer. Occasionally ulcers are partly lined with epithelium (Councilman and Lafleur). There is hypersecretion of mucus by the glands near the affected portions of the intestine, and in some glands fatty degeneration of the epithelial cells occurs. The intertubular stroma often shows an increase of cells. Amœbæ may be found in the glands and in the intertubular stroma, whence they make their way to the submucosa, as was first shown by Councilman and Lafleur. They probably pass through the muscularis mucosæ by means of the lymphatics. Amœbæ are found in varying numbers in the ulcers and abscesses, along their walls and often penetrating the neighboring tissue for some distance. When the muscularis is extensively invaded, there is dilatation of the vessels of the serosa, which shows cellular infiltration, fibrous tissue thickening, and even fibrin formation. There is reason for believing that amœbæ may reach the peritoneal cavity without marked lesions of the serosa and without perforation of ulcers. In addition to the typical ulcers above described, Harris found in one case superficial ulcers beginning primarily in the mucosa and involving the underlying tissues secondarily. These ulcers showed no amœbæ. Ulcers may be found in various stages of repair. When they heal, the new-formed scar tissue may contract and cause stricture of the gut.

(*b*) *Liver.*—There are two forms of the so-called tropical abscess of the liver, one with and the other without accompanying dysentery or a history of this disease. Abscesses belonging to the latter class are commonly called "idiopathic." In their gross appearances, distribution, and clinical history the two varieties are identical. Recorded accounts of the histological lesions of the so-called "idiopathic" liver abscess are wanting. The only published case of amœbic abscess of the liver in which dysentery has been excluded at autopsy is Buxton's. A woman, forty-one years old, entered the Philadelphia Hospital, September 14, 1898, and died four days later. She complained of weakness and of pains in the abdomen. There was no previous history of dysentery, but her bowels were loose for four weeks before admission. At autopsy the liver was found to contain four abscesses in the right lobe and one in the left, all filled with yellow pus containing amœbæ. The large and small intestines were apparently normal, and no healed ulcers could be found. The weak points in the case are the lack of microscopical examination of the stools for amœbæ, and of sections of the intestines for possible amœbic lesions too obscure to be seen with the naked eye.

The proportion of cases of amœbic dysentery complicated with liver abscess varies within rather wide limits in the experience of individual observers. According to the statistics of Councilman and Lafleur in 1,429 autopsies on individuals dying of dysentery in India liver abscess occurred in 306, or about 21 per cent. In Algiers, of 1,000 autopsies on dysentery cases, 180, or nearly 17 per cent., had liver abscess. The above figures, however, refer only to the cases of dysentery coming to autopsy,