nature. The amnion of man is a non-vascular structure and contains no blood-vessels; the presence of an extensive system of lymph channels has not been definitely demonstrated, though such vessels may be present. The amnion of the chick is contractile, and is said to contain muscle cells.

(*b*) The outermost surface of the amnion, that directed toward the chorion, is lined, partially at least, by a single layer of thin, flat endothelioid cells (Figs. 107, 111). These are descendants and representatives of the mesothelial cells which line the cœlom and from which the endothelial cells of the pleura and peritoneum are also derived. These cells are naturally well marked in the early period of pregnancy, while the amnion is still unattached to the chorion and presents a free outer surface. A similar layer of cells probably lines the innermost surface of the chorion. After the amnion becomes united with the chorion, these cells would probably be suppressed at the points of union of the two membranes, though even at full term such cells have been seen at a plane corresponding to the deepest part of the amnion or innermost part of the chorion, perhaps lining spaces left between the membranes similar to lymph spaces in the body lined with endothelium.

In the specimen of afterbirth above referred to, in which the amnion remained permanently separate from the chorion and presented a free outer surface, this layer of cells was nicely demonstrated by the silver-nitrate method (Fig. 112). On surface view these cells were mostly of hexagonal shape (some pentagonal and heptagonal), quite uniform and regular in shape and size, with slightly rounded angles. They were united to one another by their edges, which were straight, not sinuous. Their size was small, measuring 0.0055 to 0.007 mm. in diameter. They did not form a complete lining over the entire outer surface of the amnion, or at least they appeared only in patches; perhaps many of them were lost from degeneration. No nuclei were visible in them—possibly another degenerative sign; if present, they did not take the nuclear stains employed. Patches of precisely similar cells were also observed on the inner surface of the chorion in this case.

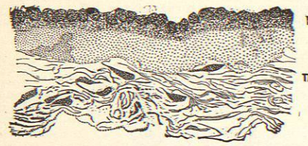

FIG. 110.—Section of Placental Amnion from Eight-Months' Embryo. *ect*, Epiblastic epithelial layer; *mes*, mesoblastic connective-tissue layer, showing non-cellular subepithelial stratum and deeper fibrous stratum. × 340. (Minot.)

The covering of the *umbilical cord*, which is continuous at the placental end with the amnion and at the fœtal end with the skin, differs in some marked characters from the amnion elsewhere. This covering consists of a superficial layer of epithelium, which rests directly upon the mucofibrous tissue composing the chief part of the cord. The integument of the cord is therefore intimately adherent to, or an integral part of, the cord, and cannot be stripped off as can the amnion elsewhere. The epithelial covering is composed at first of a single layer of cells, but later becomes stratified squamous in character, consisting of two to four layers of lenticular-shaped cells.

Union of Amnion and Chorion.—In its origin and early period the amnion is distinct from the chorion and separated from it by a space, which is the extra-embryonic part of the cœlom, and is homologous and at first continuous with the pleural and peritoneal cavities. After about the third month of pregnancy, in man, the amnion comes into contact with the chorion, and the two membranes grow loosely together. The precise character of the histological connection between the amnion and chorion has not been well made out.

ABNORMALITIES OF THE AMNION.—Very rarely is the amnion the seat of abnormal or pathological conditions.

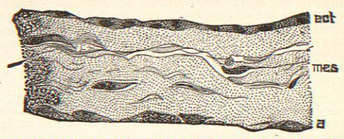

FIG. 111.—Section of Placental Amnion, at Term. *ect*, Epiblastic epithelial layer; *mes*, mesoblastic (mesenchymatous) connective-tissue layer; *a*, mesothelial endothelioid layer. × 340. (Minot.)

Such abnormalities may arise in two ways: from anomalies of development, or from pathological processes.

Among conceivable anomalies of development of the amnion might be: complete absence of the amnion; incomplete development of the amnion from failure of one of the amnion folds to grow; failure of the edges of the amnion folds to unite, leaving a hiatus in the amnion and chorion; persistence of a cord or connection of tissue between the amnion and chorion (the "amniotic cord"), such as normally occurs in ruminants; incomplete expansion of amnion after closure, compressing the fœtus. Some such anomalies of development have been occasionally observed in some animals, but in man they are exceedingly rare.

A couple of human cases are recorded (Hamard) in which there was a separate small amniotic pouch around the abdominal insertion of the umbilical cord. The reporter of one of these cases attributed the condition to a rupture of the amnion (the chorion remaining intact) with retraction of the amniotic membrane. Hamard, who reported the other case, considered the condition to be due in both cases to an early anomaly in the development of the amnion.

It happens, rarely, that the primitive separation of the amnion and chorion persists, in man, throughout pregnancy, so that the fœtus to the time of birth is enveloped in two separate sacs, the amnion internally and the chorion (united to the decidua) externally. This constitutes a rare anomaly of the human afterbirth, of which the writer has reported one case and cited seven other cases found recorded.

Small nodules or caruncles have been observed in the human amnion, scattered about in considerable numbers, some flat and sessile, some more or less pediculated, and ranging in size from that of a pinhead to that of a pea. Structurally, these are of two kinds, one composed of epithelium, the other of connective tissue. The epithelial nodules are commoner and have little or no pathological significance; they are small aggregations of epithelial cells. The connective-tissue nodules are composed of tissue like that of the mesoblastic portion of the amnion; they are very rare, and have been observed in connection with early fœtal death.

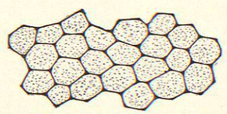

FIG. 112.—Endothelioid Cells of Outer Surface of Amnion (and Inner Surface of Chorion). Silver nitrate. × 1,000.

Adhesions of the amnion to various parts of the fœtus, with resulting deformities of the latter, have been observed. These adhesions have apparently been due to inflammatory action.

Deficiency or excess in the quantity of the amniotic fluid, with the resulting pathological consequences, are considered in other articles.

AMNIOTIC FLUID.—The amniotic sac is filled with a serous fluid, the amniotic fluid or liquor amnii, in which the fœtus is immersed.

In quantity the amniotic fluid at full term in the human female may vary greatly, but ordinarily ranges from about 500 to 1,000 c.c., averaging from 600 to 800 c.c. Abnormally there may be a deficiency (oligohydramnios) or an excess (polyhydramnios) of amniotic fluid, both conditions giving rise to certain pathological conditions and dangers. The differences in quantity at different periods of pregnancy are not well determined; it is quite possible that the fluid increases in amount during the earlier portion of pregnancy, and diminishes in the later portion.

The liquor amnii is a serous or watery fluid, containing in solution a small proportion of proteid, organic, and mineral substances. It is normally clear, limpid, and transparent, colorless, alkaline in reaction, and has a specific gravity of about 1.007 or 1.008. It contains from one to two per cent. of dry solids, besides a small amount of adventitious epithelial cells, hairs, vernix caseosa, and occasionally leucocytes. Proteids (albumin, globulin, mucin, etc.) are present in the early part of pregnancy in large amount (10.77 per cent. at four months, 7.67 per cent. at five months, 6.67 per cent. at six months), but undergo a great decrease toward the end of pregnancy, when there is only a small proportion present (0.82 per cent.). The inorganic salts present are those usually found in serous fluids, chiefly salts of sodium, potassium, ammonium, and calcium. Urea is present in slight proportion; the amount is less early in pregnancy and gradually increases, 0.03 or 0.045 per cent. being present at the ninth and tenth months.

Marked abnormalities in the physical and chemical characteristics of the amniotic fluid have been rarely encountered.

As to the source from which the amniotic fluid originates, there have been two opposing views: one that it is derived (in mammals at least) from the maternal tissues by transudation from the decidua through the chorion and amnion; the other that it is derived from the fœtus, being the excretory products of the urinary or sweat glands of the latter. The view that the liquor amnii is of fœtal origin has long been held; but in opposition thereto and in support of its maternal origin it has been urged by Minot that the fluid in its composition does not resemble urine, but is more of the nature of a serous fluid transuded from the blood-vessels; that the fluid appears before the urinary or other excretory glands of the embryo are developed and while the urethral outlet of the male is still imperforate; and that substances experimentally administered to the mother have afterward been found in the liquor amnii but not in the fœtal tissues. On the contrary, the fluid occurs in sauropsidan embryos which have lost their connection with the misternal tissues; and as to the finding of drugs administered to the mother in the liquor amnii but not in the fœtus, it is possible that the substances may have been entirely excreted and eliminated from the fœtus and discharged into the amniotic fluid. Possibly in mammals the fluid is derived from both the fœtus and the mother—from the mother at first and later from the urine of the fœtus.

The function of the amniotic fluid is largely to afford protection to the fœtus in utero, by equalizing the pressure on all parts of the fœtal body and preventing undue direct pressure of the uterine walls on particular parts of the fœtus. By maintaining a symmetrical shape of the uterus, and protecting the umbilical cord and uterine walls from excessive and unequal local pressure, it obviates interference with the umbilical, placental, and uterine circulation. The amniotic fluid also permits the movement of the fœtus in the uterus, and prevents adhesions of the fœtus to the amnion or of parts of the fœtus with one another from taking place. The symmetrical distention of the womb by it facilitates and assists in the dilatation of the os uteri during labor. It has been also asserted that the amniotic fluid serves as a source of water for the fœtus; as the fluid contains only a small proportion of solids, it could have little nutritive value except as supplying water. It is well settled that both mammalian and bird embryos swallow amniotic fluid; but whether this is done as a reflex act or for nutritive purposes, or whether the placental circulation is incapable of furnishing sufficient water to the fœtus, is not known.

J. B. Nichols.

AMNION, PATHOLOGY OF.—The amnion is the innermost of the membranes inclosing the fœtus. It is continuous with the fœtal epidermis at the umbilicus and forms a sheath about the umbilical cord. The exact manner of the development of the human amnion is as yet unknown, for in the earliest embryos examined it forms a complete sac about the embryo. Morphologically, it is a part of the body wall. It consists of two layers: an epithelial one continuous with the ectoderm, and a layer of embryonic connective tissue continuous with the somatic mesoderm. The epithelial layer is on the inside of the membrane, toward the fœtus; the connective-tissue layer on the outside, next to the chorion and uterus wall.

The membrane is thin and translucent, containing no blood-vessels, but is rich in large lymph spaces, forming lacunæ in which the mesodermic cells lie. These spaces are connected by a system of very fine lymphatics. In the earliest stage the tissue of the amnion consists of but two layers of cells (ectodermal and mesodermal), between which lies a distinct space. By the second month these layers have become united, and the mesodermal portion has increased greatly in thickness so that it is capable of being divided into two parts, a thin mesothelial layer covering the chorionic surface of the membrane, and the mesenchyma, which makes up the greater part of the fully developed amnion. The tissues of the amnion do not normally develop beyond an early embryonic stage; the ectoderm preserves its one-layered structure, and the mesodermal tissue remains embryonic in character. No blood-vessels or nerves have been found in the human amnion. In the later months of pregnancy, physiological degenerative changes occur in both mesodermal and ectodermal nuclei.

The amniotic fluid (liquor amnii) is most probably, for the greater part, a secretion of the amnion, but the manner of this secretion or the source of supply to the amnion is still unknown. In the later months of pregnancy some portion of the fluid is undoubtedly derived from the fœtus. It is probable that the fluid is secreted by the capillaries of the chorionic villi next to the amnion, and is passed on through the amnion by means of the activity of its cells. The fluid serves as a source of water supply to the fœtus; and, as a mechanical protection against blows, shocks, pressure, etc., it assists in maintaining a uniform temperature, allows room for fœtal movements, and aids in delivery.

During the first two months there is a definite space between the amnion and chorion, but in the third month the amnion is gradually pressed against the chorion, until an agglutination takes place between the two membranes through the formation of a homogeneous fluid or gelatinous matrix containing few cells. This union is always very slight, as the amnion in all normal cases can be readily stripped from the chorion. In the first three weeks the membrane is somewhat removed from the embryo; in the fourth week the rapid growth of the latter almost entirely fills the amniotic cavity. During the second month the membrane enlarges more rapidly, forming a larger space for the amniotic fluid, but after the fourth month it fits more closely about the fœtus, from which it is kept separated by the fluid.

The structure of the amnion is analogous to that of the serous membranes, and there is consequently a close analogy between the general pathology of the fœtal membrane and that of the latter. The tendency toward plastic exudations with the formation of more or less extensive adhesions, changes in the amount and character of the secretion, etc., occur here as upon other serous surfaces. The peculiar function of the amnion and its

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;