over, not so great as in the early months. In sections of the placenta these buds appear as large plasmodial giant cells when cut transversely. They are frequently very numerous around infarcts, suggesting a compensatory regeneration. Rarely the placenta at full term is increased in size, and shows active proliferation of the villi as in the early months. With this hyperplasia there may be associated a myxomatous or hydropic degeneration of the stroma of the villi, giving rise to the condition known as vesicular or hydatid mole. A similar process takes place after abortion or delivery in case the placenta is retained, but is much more frequent and extensive the earlier the death of the fœtus occurs. The retained placenta, after abortion, may live with not only a lessened, but frequently an increased vitality many months after the death of the fœtus, and this may lead to a hyperplasia of the villi resembling in character that of a new growth. These hyperplastic growths of the chorion may be found in all stages from the slightly increased activity in the formation of syncytial buds to the development of atypical buds infiltrating the uterus wall and producing metastases in other organs. Various names have been given to these growths, and their terminology is at present confused. It is difficult to draw any close distinction between the simple hyperplasias on the one hand and the new growths on the other. For reasons of convenience they will be described under the head of tumors of the chorion.

Placentitis (*Chorionitis*).—Though the term placentitis occurs frequently in the literature, the actual anatomical observations of inflammatory conditions of the fœtal placenta are so rare and the interpretation of these so varied and conflicting that the condition cannot be said to have a definite status in pathology. Throughout the older literature there are references to placental inflammation as an occurrence possible but rare. It is first definitely mentioned by Guillemeau in his "Oeuvres de Chirurgie," 1648. Mauriceau, Portal, and Morgagni gave clinical descriptions of "placentitis" unsupported by anatomical observations. Cruveilhier, Murat, Brachet, Stein, d'Outrepont, Wilde, Simpson, and Rokitansky made many observations by which they declared the existence of placentitis to be conclusively proved, that it was both acute and chronic in its course, and in common with other inflammations possessed three stages. The conclusions of these observers were based wholly upon the gross appearance of the organ and upon a theoretical application of the changes seen in croupous pneumonia. The modern reader can have no doubt that they saw and described the placental infarct. In 1849 Scanzoni gave a more exact description of infarction, showing that the yellowish areas consisted of fibrin. He, however, interpreted the condition as inflammatory in nature and designated it as "phthisis placentæ." He was supported in these views by Mattei and Geoffroy.

But doubt as to the correctness of these views soon arose. Verdier and Bustamente explained the phenomena as due to retrograde metamorphoses of blood clots. Robin especially opposed Scanzoni's views, and gave the process a wholly different interpretation. According to his observations there did not exist a true inflammation of the placenta, at all events no one had yet seen it. What had been taken for placentitis he affirmed was nothing more than a "fibrous" degeneration of the chorionic villi arising from the obliteration of the chorionic vessels. Doubt as to the existence of placentitis then took a more definite form. Millet, in 1861, said: "Nothing is less precise than the symptomatology of this affection, nothing is less exact than its pathologic anatomy, in a word, nothing is less proved than this inflammation itself." In 1862 Maier and Hegar studied very carefully the phenomenon formerly described as placentitis. After a minute description of the well-known appearances and structure of the white infarct, they decided that the partial necrosis of the villi near the fibrin masses was the expression of an interstitial placentitis similar in its nature to hepatic cirrhosis. Maier noted also the periarteritis of the chorionic vessels, and considered the two conditions to be closely related and to lead to the same result, atrophy and degeneration of the villi.

In 1869 Charpentier doubted very much the existence of a true inflammatory condition of the placenta. The pathological anatomy of the organ became more and more unsettled, and the disposition to reject placentitis altogether rapidly increased. Many opinions were given asserting the impossibility of a placental inflammation in the Cohnheim sense of the process, since there "are no capillaries in the maternal placenta through which a migration might take place and no nerves to regulate the contractility of the vessels throughout the organ." The opinion of Robin came to be most generally accepted: "What has been taken for inflammation of the placenta is nothing else than a condition of transformation of blood clots at different stages. What has been regarded as pus is only fibrin in the course of disorganization, and in those cases where true pus has been found the pus did not come from the placenta but from an inflammation of the tissue of the uterine walls and an accidental deposition in the tissue of the placenta."

In 1884 Ackermann declared all previously entertained views to be incorrect, and explained all of the observed phenomena on the theory of an anæmic infarct of the placenta. His views were accepted by the majority of pathologists in so far as the question of the existence of placentitis is concerned. An immense quantity of literature concerning placental infarction now sprang into existence, and from 1884 to the present time the question of placental inflammation has been practically relegated to a far background. In summing up the gleanings from the literature we find that the older writers believed in the existence of a placentitis which they constructed from clinical phenomena, inaccurate gross observations, and still more inaccurate microscopical examinations and weakly grounded reasonings from analogy. As the knowledge of pathological histology developed the status of placentitis became more and more unsettled, and the changes which the older observers looked upon as inflammatory were shown to be of the nature of infarction, and finally, in our own day, to be considered as only the expression of senile decay of the organ. The literary history of placentitis is the history of the placental infarct, and inflammation of the placenta as a definite pathological entity has almost disappeared from the later literature, and its existence is affirmed only by scattered and unsatisfactory observations.

Small foci of leucocytes are not infrequently seen in the ripe placenta, especially in the neighborhood of infarcted areas, but their significance is unknown. A true purulent inflammation of the maternal portion of the placenta is relatively common. It may be due to gonorrhœal infection, or to an extension of a neighboring purulent process to the endometrium and decidua. In the earlier months it may be due to infection resulting from attempted abortion. The cells of the decidua undergo a liquefaction necrosis, and there is a large leucocyte infiltration which may assume the proportions of abscess formation. The leucocyte infiltration extends along the decidua and into the decidual septa, but only in rare cases involves the chorion, the uppermost layer of the decidua apparently acting as a barrier to the extension of the process. The chorion is most likely to be involved when the decidual inflammation occurs in the early months. In such cases the syncytium becomes swollen and hyaline, gradually losing its nuclei, and taking a very deep stain with eosin, but does not give the fibrin stain with Weigert's method. The stroma of the affected villi is partly or wholly liquefied and replaced by leucocytes. Each villus may come to have the appearance of a small abscess surrounded by the syncytium, which remains intact long after the stroma of the villus is entirely destroyed. The formation of syncytial buds may take place even at this stage. Fibrin is formed in the intervillous spaces, but is not so homogeneous and hyaline in character as in the placental infarct. Leucocytes may or may not collect upon the surfaces of the affected villi, and in the early stages of chorionitis there

may be no coagulation in the intervillous spaces, even when the changes in the syncytium and stroma of the

FIG. 1300.—Purulent Chorionitis. Placenta and fœtus removed one week after attempted abortion in the third month. Stroma of the villi almost wholly liquefied and replaced by pus. Syncytium swollen and hyaline, gradually losing its nuclei. A few well-preserved buds lying in the intervillous spaces which contain fibrin and an increased number of leucocytes. Camera lucida drawing. Leitz objective No. 3; eyepiece No. 2; reduced one-third.

villi are far advanced. The place of extension of the purulent process from the decidual septa to the chorion may usually be discovered. Ultimately the complete destruction of the affected portion gives rise to a well-defined abscess of the placenta. Death of the fœtus, general sepsis on the part of the mother, may result, or in rare cases the pus may burrow downward through the decidua, escaping from the uterus, and the gestation go to full term. Purulent inflammation of the decidua and chorion is of frequent occurrence in the retained placenta after abortion and delivery, and in the placenta of ectopic gestation.

Interstitial chorionitis, both acute and chronic, has been described. The acute forms occur only in the early months of gestation, and are practically always due to syphilis or tuberculosis, and will be described under those heads. The condition described as chronic interstitial placentitis is in reality only the fibroid change which takes place in the chorionic stroma as a result of the normal or premature obliteration of the chorionic vessels, and can hardly be considered as inflammatory in nature.

Periarteritis, Endarteritis.—The changes in the chorionic vessels were at a very early period regarded as inflammatory in nature. As before mentioned, the present view is to consider them in the mature placenta as being of the nature of senile changes. They acquire a pathological significance only when occurring prematurely or to an excessive degree. The process begins as a rule in the terminal and medium-sized arterioles, later in the larger arteries, while the veins and capillaries are not affected until the blood supply is almost shut off by the arterial changes. The first indication of the process is shown in a thickening of the adventitia. Very soon the intima is involved, the subendothelial cells proliferating irregularly, forming localized growths into the lumen, or a more regular narrowing of the lumen as a whole. When the lumen is almost entirely obliterated, there is usually a loss of the endothelium and the obturation of the vessel is completed by thrombosis. In the tissue about the affected vessels there may or may not be any increase in the number of wandering cells. These changes begin as early as the seventh month and progress until delivery. They are the direct cause of infarction.

Syphilis.—The changes in the chorion ascribed to syphilis are numerous and varied. Infarction, fatty change, calcification, sclerosis of vessels, fibroid degeneration, interstitial placentitis, etc., have all been regarded by various authors as being of syphilitic origin. As seen above, many of these conditions are physiological within certain limits, and in the present state of our knowledge it is impossible to say definitely as to when they acquire a pathological significance, and their relation to syphilis is still more obscure. Fränkel has observed in cases of early fœtal syphilis transmitted from the father a form of acute interstitial chorionitis in which there is a marked proliferation of the cells of the villous stroma. This loses its myxomatous character, its blood-vessels are obliterated, the villi become greatly thickened, and as a result the intervillous spaces are decreased in size. The newly formed stroma is of a fibroblastic character, rich in large spindle-shaped cells, having more or less vesicular nuclei. The change may affect the entire chorion, or scattered areas of the condition may be found throughout the organ. The former is most likely to occur when the infection occurs at conception, the latter in syphilis acquired in the first months. In the former case death of the fœtus and abortion invariably result, in the latter gestation may go on to full term with the birth of a child showing marked syphilitic manifestations. The fibroblastic character of the affected villi may be maintained until delivery or the stroma may gradually assume a hyaline character. Early and marked scle-

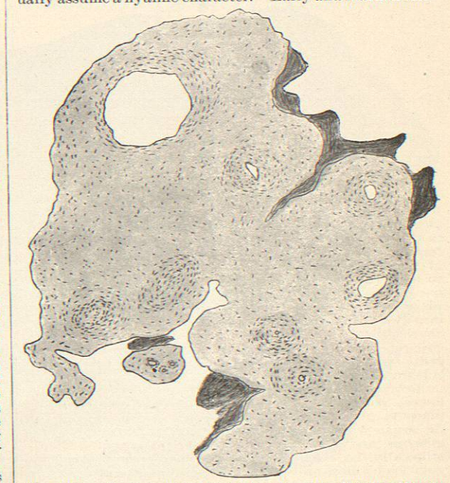

FIG. 1301.—Chorionic Stem from Ripe Placenta, Showing Normal Obliteration of Chorionic Arteries. Fibrin adhering to edge; Camera lucida drawing. Leitz No. 3 objective; eyepiece No. 2; reduced one-third.

rosis of the chorionic vessels accompanies the process, and, as a result of this, infarction occurs to an excessive degree. This form of interstitial placentitis does not, however, occur in all cases of early congenital syphilis, and fœtal syphilis may exist without any apparent changes in the placenta. Syphilis of the mother, acquired in the later months of gestation, may or may not affect the chorion. The general effect of maternal syphilis upon the chorion is the production of a premature senility as shown by early and extensive sclerosis and infarction. None of the chorionic conditions caused by syphilis has anything specific in its character. A similar interstitial chorionitis (localized and not diffuse) may be caused by the tubercle bacillus. Gummatous growths are not found in the chorion, though they have been described as occurring in the maternal portion of the placenta.

Tuberculosis.—Tuberculosis of the fœtal placenta has been repeatedly observed during the last decade (Schmorl, Kockel, Warthin, etc.). Typical caseating tubercles, with giant cells, and containing tubercle bacilli, have been found in the chorionic villi, and the writer has seen one case in which there were localized areas of interstitial

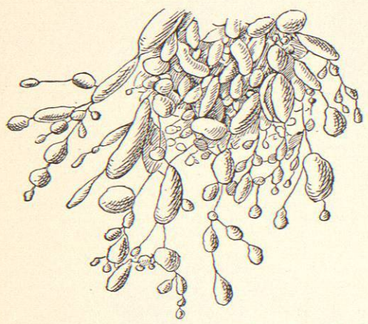

FIG. 1302.—Hydatid Mole. Myxomatous degeneration of chorion. (After Ziegler.)

chorionitis, with giant cells but without caseation, containing tubercle bacilli. The fœtus may be infected secondarily to the tuberculous condition in the chorion, while in other cases, as the one reported by Schmorl and Birch-Hirschfeld, tubercle bacilli may be found in the fœtus without the occurrence of changes in the chorion characteristic of tuberculosis. It is doubtful if the bacilli in the blood of the maternal sinuses pass through a perfectly normal syncytium, the tuberculous proliferation of the cells of the villous stroma being either secondary to a necrosis of the syncytium, upon which the bacilli have lodged, or the diseased villus had previously lost its plasmodial covering, whereby the bacilli were brought into direct contact with the stroma. Tuberculosis of the chorionic villi may also occur in ectopic gestation, as in a case reported by the writer in which the cyst wall, placenta, and fœtus, as well as both Fallopian tubes, were tuberculous.

Atelectasis of Chorion.—Küstner has observed in the lowest portion of normal placentas, and especially in placenta prævia marginalis, certain areas in the edge of the organ which were thinner, firmer, dryer, more homogeneous, and of a browner color than normal. Microscopically, no other pathological changes are found beyond such a close crowding of the villi that the intervillous spaces are obliterated. He has explained these changes as being the result of a continued pressure from overlying parts of the fœtus, and comparing the condition to atelectatic changes in the lungs he has designated the condition as chorionic atelectasis.

Tumors.—The most frequent and important of the new growths of the chorion are those resulting from increased activity in the syncytium. These form a large class of closely allied conditions, ranging from a simple hyperplasia of the villi to atypical growths of a malignant nature. Since all writers are not yet agreed upon the genesis of the syncytium, the terminology of these growths is unsettled and confused. They may, however, be divided into two large classes: the moles or benign syncytiomata, and the syncytioma malignum.

Moles.—A simple hyperplasia of the chorionic villi may occur at any time during the progress of gestation, or in the retained chorion after abortion or delivery. This hyperplasia is essentially due to a proliferation of the syncytium, the growth of the stroma being secondary. When the growth is toward the uterine cavity, it may be termed a benign placental polyp (fleshy mole, placentoma, etc.). If the newly formed villi consist of fibrous connective tissue a fibroid placental polyp is produced which has been called fibroma chorii. The hyperplasia is, however, most frequently associated with a myxomatous degeneration of the stroma of the new villi. This may involve a limited number of villi or the entire chorion (hydatid mole, vesicular mole, grape mole, bladder mole, myxoma chorii, etc.). The myxomatous change begins in the centre of the villus, while at the same time there is a peripheral growth of the syncytium and subsyncytial layer. As a result of this coincident proliferation and degeneration the villi come to appear as small cysts or bladders filled with a mucin-containing fluid, which are strung together by delicate pedicles in a manner suggesting bunches of grapes or resembling the variety of seaweed known as bladder wrack. The individual cysts have a diameter of 0.1 mm. or greater, and are fastened to slender pedicles which arise from other cysts or directly from a chorionic stem, which as a rule shows much less change. The cyst walls are very thin and delicate. Microscopically the cysts are found to be much-changed villi, the central portion consisting of a cavity filled with a serous or mucin-containing fluid, through which a few strands of stroma remain preserved. Toward the periphery there is a transition into a more dense myxomatous tissue, while just beneath the syncytium there is a thin layer of stroma of normal type. If the degeneration of the stroma occurs early in the development of the villus, the stroma may become entirely fluid; but if later, fibrous threads remain, giving an appearance more nearly resembling œdematous tissue than myxomatous. According to Marchand, no mucin is found in the cysts, the process being a hydropic rather than a myxomatous degeneration. The ends of the degenerating villi are either free or fastened to the uterine wall. If these infiltrate the wall, the hydatid mole may take on a malignant character and should then be classed as a malignant syncytioma. The causes of the hyperplasia and degeneration are unknown. It may be assumed that after the death of the fœtus the excess of nutrition supplied to the chorion leads to increased formative activity on the part of the syncytium, and the degeneration of the stroma may be due to an inability of the latter to keep pace with the former in proliferation. Hemorrhages from the decidua frequently accompany the proliferation of the villi, and large masses of fibrin may be formed around the new villi (fibrin mole). Though these chorionic hyperplasias most frequently follow abortion and delivery, they may begin during the course of an apparently normal gestation and lead to abortion. After the death of the fœtus the growth may continue indefinitely, and may at any time assume malignant characteristics. They may also arise in the vagina or in the cyst walls of ectopic gestations, wherever chorionic villi may be retained after death of the fœtus. Since by some writers the syncytium is regarded as being ectodermal in origin, the various forms of benign moles have been styled benign chorio-epitheliomata, since in all cases it is the syncytium which governs the development of the stroma. These

growths must then be placed among epithelial tumors, as the process is essentially a syncytial overgrowth; and not among connective-tissue tumors with which they were formerly classed.

Syncytioma Malignum (*Chorio-Epithelioma*).—Since the chief factor in the formation of the hydatid mole is the

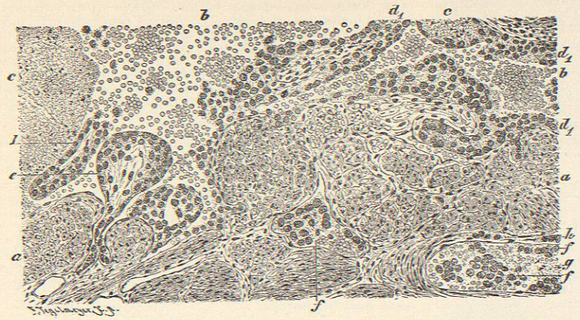

FIG. 1303.—Syncytioma Malignum. Infiltration of muscle of uterine wall by atypical plasmodial masses arising from the chorionic syncytium. (After Ziegler.)

proliferation of the syncytium, this growth bears a close analogy to an adenoma, and when the syncytial buds and cells infiltrate the maternal tissues, giving rise to metastases, it becomes carcinomatous in nature. The histological structure of the mole does not in itself give evidence of its benign or malignant character. Apparently benign moles may give rise to metastases, while, on the other hand, transported villi may fail to give rise to new growths. The behavior of the transported villi is governed solely by the covering epithelium. Marchand distinguishes between two forms of chorio-epithelioma, typical and atypical. In the former the chorionic epithelium appears as in the early months of gestation, consisting of irregular, branching plasmodial masses. In the atypical form the cells are irregular and compact, with very large and deeply stained nuclei, and while multinuclear cells are present there are no continuous plasmodial masses. The atypical form, when infiltrating, may resemble either sarcoma or carcinoma, but in the secondaries no stroma is formed as in carcinoma. Intravascular growths may occur, and either lymphogenous or hæmatogenous metastasis. Secondaries are most frequently found in the vaginal walls and lungs, but occur also in the liver, spleen, etc. The combination of branching plasmodial masses, the presence of cells corresponding to those in Langhans' layer, the absence of blood-vessels and connective-tissue cells in the intravascular growths are characteristics seen only in growths arising from chorionic epithelium.

The weight of authority at the present time accords to the syncytium a fœtal and ectodermal origin. The ectodermal nature of Langhans' layer seems to be definitely established, and between it and the syncytium there exist transitional forms which point to a common origin for the two layers. The term chorio-epithelioma would then seem to be justified. Both the hydatid mole and the chorio-epithelioma arise as a result of the increased formative activity of the chorionic epithelium caused by the increase of nutrition occurring after the death of the fœtus. The normal inhibition being thus removed, the syncytium may continue to grow indefinitely and in an atypical manner. Its cells become loosened and through chemotaxis enter the maternal vessels giving rise to metastases. The necessity of an early removal of all retained placentas is therefore clearly indicated, as malignant changes may occur at any time.

Connective-Tissue Tumors.—The number of reported cases of circumscribed connective-tissue growths of the chorion is very small, only 36 having been observed up to the present time. Of these 14 were diagnosed as *myxoma fibrosum*, 10 as *fibroma*, 9 as *angioma*, 2 as *sarcoma*, and 1 as a *hyperplasia* of the villi. Of these the two diagnosed as sarcoma showed no evidences of malignancy, and the

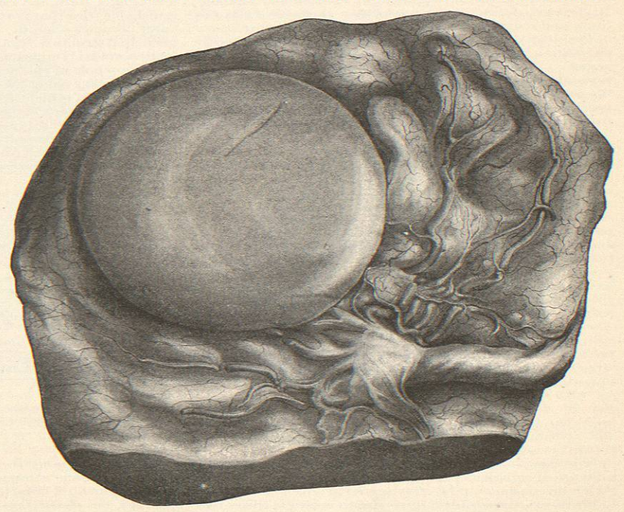

FIG. 1304.—Cyst of Chorion. (After Fenomenow, *Arch. f. Gyn.*, Bd. XV.)

one designated as hyperplasia was described as a circumscribed tumor. It is probable that these three belong to the same class as the others, and the similarity of all these growths in structure would make the diagnosis of *angioma fibro-myxoma* applicable to all. In the case of all these tumors it has been shown that the chorion is the seat of growth, and that neither decidua nor amnion is concerned in their origin; further, that the tumor arises from one cotyledon, even when multiple, having but one artery and vein. No degenerative changes were found in the remaining portion of the chorion that could be in any way associated with the origin of the growth. According to Albert these circumscribed chorionic tumors arise not from the degeneration of an existing cotyledon, but represent an atypical proliferation of a portion of the allantois, which through some unknown error of development has failed of normal growth, and retaining its embryonic impulse to proliferation leads to the formation of an atypical mass which may consist chiefly of myxomatous tissue (myxoma), connective tissue (fibroma), branching vessels (angioma), or of combinations of these. If the resulting formation is very cellular, it may be taken for a sarcoma, but has not the malignant characteristics of this form of growth. No name has as yet been proposed for these growths which will indicate both their origin and nature.

Cysts.—Aside from the cases which are very evidently to be classed with the hydatid mole few cysts of the chorion have been reported. These have been large thin-walled cysts filled with a clear fluid, situated on the fœtal surface. In a few an epithelial-like lining has been observed. According to Jaquet, four varieties of placental cysts are found: Gelatinous, arising from the space between the chorion and amnion, perivascular cysts, blood cysts, and degeneration cysts of the villi. Hegar and Maier believe that chorionic cysts may arise from the encapsulation and absorption of old hemorrhages. They may also arise from the simple softening of infarcts or as a sequel of chorionitis. Further, cysts may arise from disturbances in the development of chorion and amnion. In the case of the large monolocular chorionic cysts the cause and manner of formation are wholly obscure.

Parasites.—The micro-organisms found in the maternal circulation will appear in the blood of the maternal sinuses. It is a question whether these are able to pass through a perfectly normal and intact syncytium, but the present opinion is that the chorionic epithelium in a normal state acts as a barrier to the passage of organisms from the maternal blood to that of the fœtus. Some lesion of the syncytium, such as necrosis or desquamation, seems to be necessary for the passage, and this injury may be caused by the organisms themselves or be due to other causes. In the human species the bacilli of tuberculosis, typhoid and cholera, the pus cocci, pneumococcus, recurrens spirillum have been known to have passed through the chorion and to have infected the fœtus. The virus of smallpox, scarlatina, and syphilis may pass through the placenta, either with or without apparent local changes. In the case of tuberculosis the bacilli may be found in the fœtus when none can be demonstrated in the chorion. The important problems associated with placental transmission of disease have as yet been barely touched upon, and remain a ripe field for future investigations.

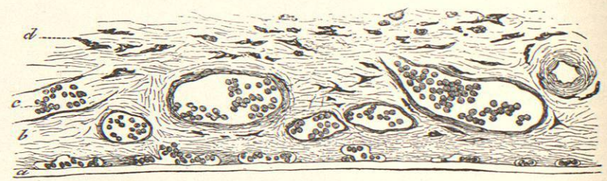

FIG. 1305.—Section of Choroid. (From Quain, after Cadiat.) *a*, Membrane of Bruch—the choriocapillaris is just above it; *b*, vascular layer; *c*, vein with blood corpuscles; *d*, lamina suprachoroidea.

Eclampsia.—Schmorl has observed in this disease the presence of emboli of giant cells in the pulmonary vessels, which he considers to be of syncytial origin. According to his theory these emboli give rise to multiple thrombosis of the pulmonary vessels, which are the direct cause of the eclampsic convulsions. This theory cannot at present be said to stand upon a very secure foundation. It is possible that in some of the cases of eclampsia in which pulmonary emboli of giant cells have been observed the giant cells were of bone-marrow origin and not placental, inasmuch as the differential diagnosis between placental giant cells and those of the bone marrow is impossible except in those cases in which large syncytial masses are present. Emboli of placental cells have been observed in cases in which there were no symptoms of eclampsia. Further, multiple thrombosis of the lungs produced by multiple emboli of liver cells does not give rise to eclampsic symptoms. The whole question of placental-cell embolism is at present somewhat unsettled. Infarction, hemorrhage, fatty degeneration, etc., of the chorion have also been believed to bear a causal relation to the phenomena of eclampsia.

Abortion.—The minute pathology of abortion is for the chief part an unknown field. Fatty degeneration, infarction, hemorrhage, calcification, myxomatous degeneration, etc., have in many cases been supposed to have been the exciting factor of the death and expulsion of the fœtus. Just what relation these changes actually bear to the production of abortion it is at present impossible to say. In the majority of cases we are unable to say whether the placental changes are primary or secondary. The association, however, of these changes in the chorion with a relatively large number of cases of abortion makes it possible that when present to an excessive degree they may lead to abortion.

Aldred Scott Warthin.

CHOROID, DISEASES OF.—ANATOMICAL CONSIDERATIONS.—The choroid is of mesoblastic origin: in connection with the ciliary body and iris it forms the vascular tunic of the eye. It originates as a differentiation of mesoblastic formative cells over the outer surface of the secondary eye vesicle; its first appearance being manifested by a plexus of capillaries lying next to the outer surface of the secondary eye vesicle. These capillaries eventually form the choriocapillaris. At first indistinguishable from that portion of the mesoblastic tissue which later becomes sclerotic, it soon becomes differentiated, its principal structure being separated from the sclera by the perichoroidal lymph space. The choroid extends throughout the whole of the posterior part of the globe, its anterior boundary being the ora serrata. The choroid consists of five layers, which, from without inward, are:—

First, *the suprachoroid layer*, consisting of a few non-vascular lamellæ of pigmented fibres which remain attached to the sclera when the choroid is torn from it. Numerous trabeculæ pass from the lamina suprachoroidæa to the layer of large vessels traversing the suprachoroidal lymph space.

Second, *the layer of large vessels*, which contains large arterial and venous trunks, numerous nerve fibres, and connective-tissue stroma throughout which are scattered many pigmented branching cells.

Third, *the elastic layer of Sattler*, a thin, rather dense layer composed largely of elastic connective-tissue fibres.

It lies between the layer of large vessels and the choriocapillaris.

Fourth, *the choriocapillaris*, a layer of capillaries which anastomose freely, forming a close network. The capillaries are most dense at the yellow spot, the meshes becoming larger as the periphery is approached. The choriocapillaris is continuous through the entire area of the choroid. The capillaries anastomose with those of the optic nerve, but never with those of the retina. The function of this layer is to supply nourishment to the posterior layers of the retina.

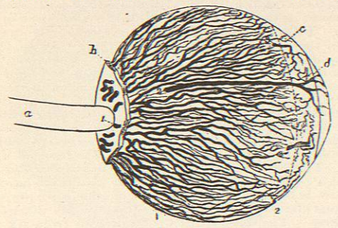

FIG. 1306.—Lateral View of the Arteries of the Choroid and Iris. (From Quain, after Arnold.) *a*, Optic nerve; *b*, part of the sclera left behind; *c*, region of ciliary muscle; *d*, iris; 1, posterior ciliary arteries piercing the sclera and passing along the choroid; 2, one of the long ciliary arteries; 3, anterior ciliary arteries.

Fifth, *the lamina vitrea*, a thin homogeneous membrane separating the pigment layer of the retina from the choriocapillaris.

Blood Supply.—The blood supply to the choroid is by the short posterior ciliary arteries, twelve to fifteen in number, which pierce the sclera diagonally about the optic nerve. On reaching the choroid they anastomose rather freely in the layer of large vessels, sending numerous small branches to the choriocapillaris into which the arterial blood flows. About the optic nerve the arteries form a vascular circle, known as the circle of Haller, from which some small branches pass to the retina. Anteriorly they anastomose with the anterior ciliary arteries. The blood is returned from the choriocapillaris by numerous small veins, which pass to larger venous branches in the layer of large vessels. The veins anastomose very freely and finally converge to form from four to six large veins, known as the venæ vorticosæ, which pierce the sclera at the equator of the globe, emptying into the large venous trunks of the orbit.

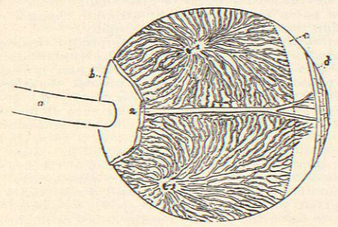

FIG. 1307.—Lateral View of the Veins of the Choroid. (From Quain, after Arnold.) 1, 1, Two trunks of the venæ vorticosæ at the place where they leave the choroid and pierce the sclerotic coat. The other lettering is the same as in Fig. 1306.

The amount of pigment present in the pigmented cells of the choroid, as in the pigment layer of the retina, differs largely in different individuals. In the blond it is relatively scanty, in the brunette and negro very dense. In the albino the pigment is wanting and, by means of the ophthalmoscope, the larger veins and arteries of the choroid can be distinctly seen.

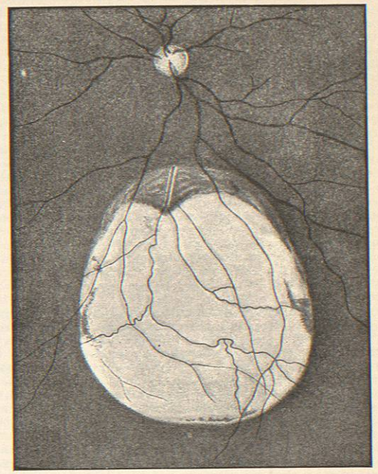

FIG. 1308.—Coloboma of the Choroid. (Haab.)

CONGENITAL ANOMALIES.—*Coloboma of the Choroid* is a defect which is usually situated in the lower part of that membrane. It is primarily due to imperfect closure of the retinal fissure in the development of the eye and the consequent imperfect formation of the choroid over the secondary eye vesicle. The retinal fissure, which begins to close at its posterior extremity, should be completely closed by the end of the second month of fœtal life; it may be arrested at any stage of development; hence the various differences in shape seen in coloboma of the choroid. The defect is usually separated from the optic disc by a bridge of normal choroid; it broadens out and ex-

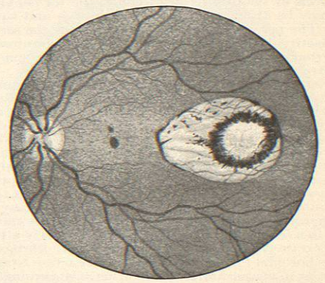

FIG. 1309. — Macular Coloboma of the Choroid. (De Wecker and Masselon.)