

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 fetal 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 monocular 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 fetus. 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 fetus. 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 fetus 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.

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 eclamptic 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 eclamptic 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 fetus. 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 lamellae of pigmented fibres which remain attached to the sclera when the choroid is torn from it. Numerous trabeculae pass from the lamina suprachoroidea to the layer of large vessels traversing the suprachoroidal lymph space.

Second, the *layer of large vessels*, which contains large

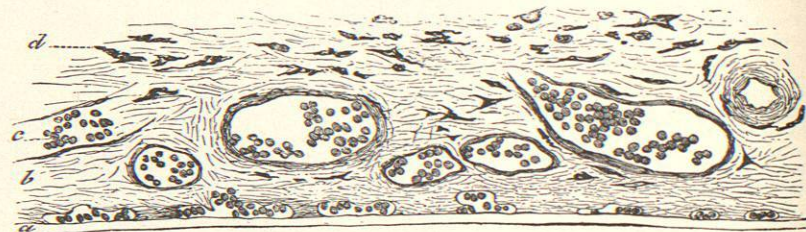


FIG. 1305.—Section of Choroid. (From Quain, after Cadiat.) a, Membrane of Bruch—the choriocapillaris is just above it; b, vascular layer; c, vein with blood corpuscles; d, lamina suprachoroidea.

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

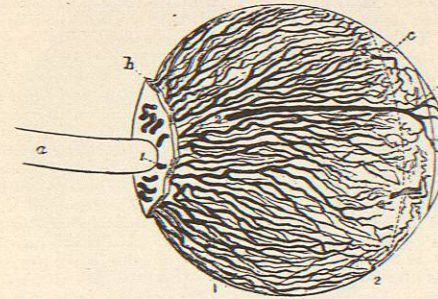


FIG. 1306.—Lateral View of the Arteries of the Choroid and Iris. (From Quain, after Arnold.) a, Optic nerve; b, part of the sclera left behind; c, region of ciliary muscle; d, iris; 1, posterior ciliary arteries piercing the sclera and passing along the choroid; 2, one of the long ciliary arteries; 3,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

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.

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

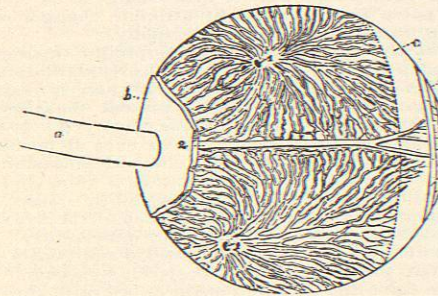


FIG. 1307.—Lateral View of the Veins of the Choroid. (From Quain, after Arnold.) 1, 1. Two trunks of the *venae vorticosae* at the place where they leave the choroid and pierce the sclerotic coat. The other lettering is the same as in Fig. 1306.

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 *venae vorticosae*, which pierce the sclera at the equator of the globe, emptying into the large venous trunks of the orbit.

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

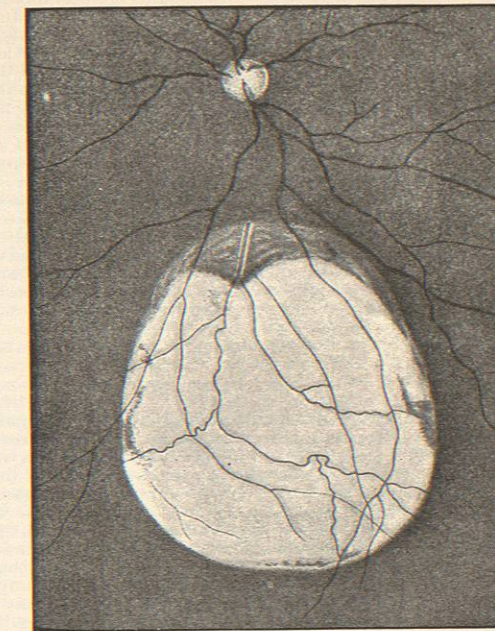


FIG. 1308.—Coloboma of the Choroid. (Haab.)

the ophthalmoscope, the larger veins and arteries of the choroid can be distinctly seen.

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

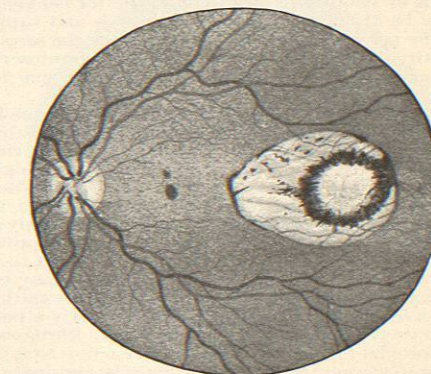


FIG. 1309.—Macular Coloboma of the Choroid. (De Wecker and Masselon.)

closed by the end of the second month of fetal 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-

tends toward the ciliary body. The border of the defect is irregularly pigmented. The anterior layers of the retina pass over the defect, but all perceptive elements are wanting. Viewed with the ophthalmoscope the defect presents a pearly-white appearance traversed by a few retinal vessels. Coloboma of the iris not infrequently accompanies coloboma of the choroid, and coloboma of the lens is sometimes observed. Bulging of the sclera at the site of the coloboma is sometimes present; this may assume the character of a cyst. The globe in these cases is frequently microphthalmic. Imperfect development of the central nervous system and of the cranium is sometimes observed in these cases.

Extra-Papillary Colobomata are sometimes seen. These are irregularly circular in shape and are generally located in the macular region. So often is this the case that they are termed macular colobomata. Various theories have been advanced to explain the origin of these defects, but none of them has been substantiated. Angio-cavernomata are sometimes observed as congenital defects in the choroid. They may occur in any part of the membrane.

Hyperemia of the Choroid.—This condition is undoubtedly present much more frequently than it is recognized. Hyperemia of the choroid is present under the following conditions: In congestion of the head; as the first evidence of a general or localized inflammation of this part of the eye; as an accompaniment of an exudative retinitis; and as a symptom of either leucocythæmia or pernicious anæmia. A deepening in the color of the fundus and slight reddening of the optic disc accompany this condition; however, unless one eye alone is affected, the change in the appearance of the fundus is so slight that it is impossible to diagnose it by means of the ophthalmoscope. It is held by some writers that hyperemia of the choroid is accompanied by certain subjective symptoms, such as meteoric flashes of light, wavy effects like those produced by the atmosphere when radiated from a heated surface, and at times photopsia. These phenomena are due to disturbance of the perceptive elements of the retina, and, as they often precede destructive processes, they should be given careful attention.

Choroiditis.—The term choroiditis is not sufficiently comprehensive to include all of the pathological processes which affect the choroid, since not all of the processes are inflammatory. It is therefore logical to divide the affections of the choroid into inflammatory and non-inflammatory; to the first the term choroiditis properly applies, to the second the term choroidal atrophy applies. All forms of choroiditis are exudative; for convenience they may be divided into non-suppurative and suppurative. The non-suppurative forms include the sero-plastic, plastic, disseminate, diffuse, areolar, circumscribed, hemorrhagic, sclero-choroiditis anterior, and some forms of central choroiditis.

Causes. The causes of choroiditis are the same in all except the embolic or metastatic, the purulent, tuberculous, traumatic, and atrophic forms, and may be considered before descriptions of the various forms are given. The most frequent cause is syphilis, either inherited or acquired. That due to inherited syphilis (which is by far the most frequent cause) is often discovered only after the clearing up of an interstitial keratitis. It is probable that in some cases the choroidal disease precedes the corneal, but no doubt the reverse is true also in many cases. The choroiditis of acquired syphilis may follow the initial lesion in a few months, but it is rare at so early a period; it may occur years later. In all but the sero-fibrinous and retino-choroidal forms the choroidal lesion due to syphilis is circular in shape, and in some forms of disseminate choroiditis the arrangement of the spots suggests the papillary syphilitic as observed on the skin. Rheumatism and gout may account for some of the cases of choroiditis for which syphilis, as a cause, cannot be traced. The history is apparently sufficient in some cases to warrant this supposition, and the treatment by the salicylates is sufficiently productive of results to sustain the opinion. Leprosy is, according to Knies, a cause of irido-choroiditis in

rare cases. Gonorrhœa in a very few cases is undoubtedly the cause. The form produced is the sero-fibrinous variety, with involvement of the whole uveal tract. Relapsing fever is sometimes accompanied by a diffuse, sero-plastic anterior choroiditis; typhoid fever is also sometimes a cause of this form of choroiditis.

Sero-Plastic Choroiditis.—This disease is characterized by a relatively sudden onset and the appearance of a sero-fibrinous exudation in the vitreous. The same exudation is present in the posterior and anterior chambers in many cases. There are more or less congestion of the deep vessels of the ocular conjunctiva and diminution of vision.

Symptoms. Unless the ciliary body or iris is involved no pain is experienced in the cases which do not result in increase of tension. Acute glaucoma may supervene. If this occurs the pain becomes intense. The vitreous becomes filled with minute particles of fibrin which obscure the view of the fundus, and the aqueous may be invaded by the particles which often become attached to the posterior surface of the cornea in the form of a pyramid. The vision is correspondingly obscure. Photopsia is not a prominent symptom. The anterior chamber is shallow because of an increase in the contents of the vitreous chamber. The pupil is moderately dilated.

Course. The disease runs its course in from six weeks to as many months. The opacities in the aqueous humor gradually disappear. Those in the vitreous may entirely disappear, but in many cases some large shreds of exudation remain and are movable in the vitreous, indicating a partial fluidity of that body. No evidence of changes in the choroid is discernible in some cases; in others some atrophic patches at the periphery may be seen after the acute stage has passed. In some cases vision returns to the degree of acuity present before the inflammation began, in others there is a permanent partial loss. If atropine is not instilled, adhesion between iris and anterior lens capsule may occur and the synechia become permanent. Recurrence is not uncommon.

Pathology. In this form of choroiditis pericorneal injection is sometimes found; the choroidal vessels are engorged with blood. The choroid is slightly œdematous. The exudation contains some fibrin. When the acute stage has passed there may be little change in the choroid, but in not a few cases slight atrophic changes occur, affecting the choriocapillaris principally.

Treatment. Quite vigorous antisyphilitic treatment is indicated in all cases which are not clearly attributable to some other disease of the system. If there is reason to believe that a rheumatic element is present, the salicylates, and the bicarbonate of soda may be employed. It is not wise to omit antisyphilitic treatment even if no history of syphilis can be elicited. The effect of potassium and mercury to prevent plastic formations is valuable in the non-syphilitic as well as in the syphilitic cases. Iron tonics are also of value. If the tension of the eye is not increased, it is necessary to apply atropine locally. The solution of this drug should be instilled sufficiently often to prevent the formation of posterior synechia. It is better to have the patient remain in bed until the acute stage of the disease has subsided. Leeches and cold or hot applications are of doubtful value.

Chorioretinitis Syphilitica.—Since in all forms of choroiditis the retina is more or less implicated, it would be more exact to include the retina in all of the terms used to indicate the different forms of choroiditis; but as the involvement of the retina is not a striking part of the picture portrayed by the disease in many of the forms of choroiditis, that membrane is not mentioned. Some of the forms involve the retina very seriously, and to one of these the term chorioretinitis syphilitica is given; to another, the term chorioretinitis pigmentosa is applied. Chorioretinitis syphilitica is a disease closely allied to sero-plastic choroiditis, although it produces more permanent damage to both membranes than does the latter.

The diffuse haziness of the vitreous is present usually to a moderate extent, shreds of exudation appear in the vitreous, and isolated large or small spots of exudation occur in the choroid. The retina appears hazy because of

œdema, and retinal hemorrhages may occur. The evidence of perivasculitis of the retinal and choroidal vessels is unmistakable. The vision is greatly diminished, and flashes of light, photopsia, micropsia, and macropsia may be experienced. Slight injection of the ocular conjunc-

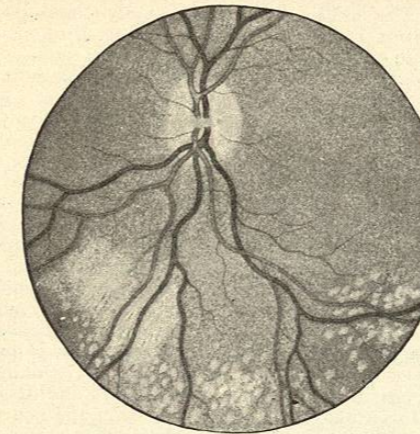


FIG. 1310.—Syphilitic Chorioretinitis. (De Wecker and Masselon.)

tiva may occur in the acute stage. Pain is not present. In many of these cases the diagnosis can be made at any stage of the disease, as the vitreous remains sufficiently transparent to permit of a more or less satisfactory examination of the fundus. The exudation into the choroid and retina is serous and plastic. The plastic exudation, which takes place in the form of circular patches, undergoes the same changes as those which characterize the exudation of disseminate choroiditis; atrophy being the terminal stage in both cases. As a result of plastic exudation in the retina, cicatricial bands may develop in that membrane, and they may also develop in the vitreous, in the plastic exudation that sometimes extends into that part of the eye. Detachment of the retina as a result of the traction of cicatricial bands in the vitreous is rare, owing to the fact that the choroid and retina are adherent in many places; but detachment of the choroid and retina together is sometimes seen following degenerative changes in the vitreous. A cataractous condition of the lens and atrophy of the globe are rare results.

The inflammatory stage usually responds quite readily to treatment, but the treatment must be vigorous and long continued.

Diffuse Choroiditis.—This form of choroiditis is characterized by the appearance, in the choroid, of large pale plaques of exudation which are found to be most extensive at the periphery. These patches at first are of quite a uniform orange or pale yellowish-pink color, not bordered by pigment. The retina over the areas of exudation is slightly œdematous. The patches coalesce, forming irregular areas with indentations of the normal fundus. The forms assumed have been likened to continents and islands and have been termed "map-like." Sometimes they assume the shape of leaves, which has caused them to be termed "leaf-like."

The disease is extremely slow in its development. The exudate slowly appears and as slowly disappears, leaving irregular scanty pigmentation over the affected areas and in some places a little pigmentation at their borders. The pigment layer of the retina and the superficial layer of the choroid, the choriocapillaris particularly, undergo atrophy and disappear. The deeper layers of the choroid remain nearly if not quite intact. There may also be found evidences of perivasculitis and occasionally of almost complete conversion of the larger vessels of the choroid into connective-tissue bands. By ophthalmoscopic examination the larger choroidal vessels and the deep choroidal stroma can be readily seen. The retinal vessels in severe cases become reduced in size, and the

optic disc presents hazy edges and becomes slightly pale, giving evidence of partial secondary atrophy of the optic nerve.

Individuals thus affected almost always present the stigmata of inherited syphilis.

Symptoms. The patient experiences no pain nor is there any injection of the external ocular tissues. Night blindness is sometimes experienced, and there is limitation of the visual fields in the parts corresponding to the affected areas of the choroid.

Prognosis. The prognosis for recovery of vision is not good, but much can be done to prevent the further advance of the disease. Treatment is similar to that of disseminate choroiditis.

Disseminate Choroiditis.—This form consists in the development of foci of exudation which are spread out over a large part, often the entire fundus, in the shape of circular patches which vary in size, but as a rule average in diameter less than that of the optic disc. After a time the exudation becomes absorbed or changed into fixed products, and atrophic areas occupy the site of the areas of exudation. The disease may for convenience be divided into two stages—the acute and the stage of atrophy.

The Acute Stage.—In the early part of the acute stage circular spots of exudation appear in certain parts of the fundus, usually beginning at the periphery; they are paler than the surrounding normal fundus. These spots have not very sharply defined borders. The retina immediately over the masses of exudation is not elevated, as may readily be determined by examination with the ophthalmoscope, but there is often an invasion of the retina by the exudate, as evidenced by the hazy appearance of the retina at these points. The spots multiply in number if not interfered with, resembling in many cases the groupings of papillary syphilitides as they appear on the skin. The areas of exudation are primarily discrete, but may eventually coalesce. The greater part of the fundus may become studded over with these spots of exudation in the course of two or three weeks, but in not a few cases some of the spots will be found to have passed to the second stage when others have but just appeared.

Ordinarily the cornea and media remain clear, but in some cases a parenchymatous keratitis may occur at the time of the onset of the choroiditis, and the presence of the affection of the choroid may not be discovered until the cornea again becomes clear.

The Stage of Atrophy.—The first stage passes slowly into the second stage. Gradually the exudation disappears,

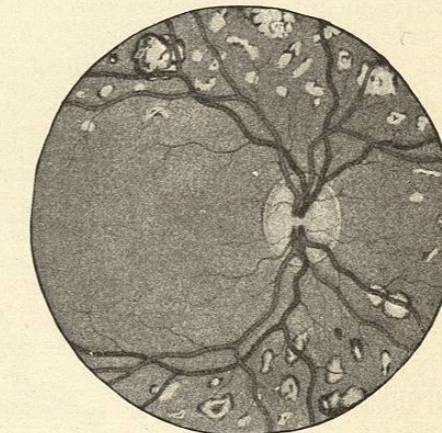


FIG. 1311.—Disseminate Choroiditis. (De Wecker and Masselon.)

the spot becomes paler, and the border of the affected area ordinarily becomes irregularly pigmented, the pigment being heaped up at certain points. Flecks of pigment are often observed over the area of the spot itself. Entire absence of pigment is not infrequently observed. The exudate either entirely disappears or a portion is converted into cicatricial tissue. All degrees of change