in the birth of the second child is investigated that its extra-uterine existence is noted.

TREATMENT.—*Before Rupture.*—When an extra-uterine pregnancy is diagnosed before rupture, it must be regarded as a malignant growth and removed by laparotomy as soon as possible. This procedure cannot be too urgently advised, for until it is removed the patient is in imminent danger of a fatal rupture. All methods which have as their end the destruction of the fœtus, such as electricity or the injection of medicinal substances, are to be strongly condemned; for, as we know, even the death of the child does not preclude the possibility of rupture of the tube. However, from what has already been said, it is apparent that such a line of treatment will rarely be thought of owing to the uncertainty of diagnosis at this time.

After Rupture.—Although laparotomy was advised some years ago for the removal of a ruptured tube, operators were not willing to accept this plan of treatment until Tait demonstrated the ease with which the diseased tube could be removed and hemorrhage checked. This method is now almost universally employed, and all of us can recall numerous instances in which life has been saved by it.

The diagnosis of ruptured tube and intraperitoneal hemorrhage once having been made by the very characteristic symptoms already discussed, the abdomen must immediately be opened (unless the degree of collapse is so great that the patient is moribund when first seen). Then immediately afterward long clamps are to be passed down into the pelvis and applied on both sides of the tubal mass. This will at once check all hemorrhage, and the injured tube can be excised without unnecessary haste. If the peritoneal cavity is filled with clotted blood which obscures the field of operation, the clamps must be applied by the sense of touch alone.

An excellent method of counteracting the effect of the loss of blood is by the infusion of normal salt solution. This can be done during the operation and the infusion allowed to run into the submammary subcutaneous tissue or injected directly into the circulation.

In tubal abortion some operators have made the attempt to save the affected tube. The advisability of such an attempt appears to be of doubtful nature, as it has not been proven that the tube once affected ever regains its functional activity.

Operation through the vagina has lately been advised and even now has many adherents. As it has many obvious disadvantages, it is mentioned here only to be condemned. Among the disadvantages may be mentioned the small field of operation in the vaginal method as compared to the large one in the abdominal, and the frequent necessity of having to open the abdomen to arrest hemorrhage which has been started in the vaginal operation.

Hæmatocele.—It has been shown that the majority of cases of pelvic hæmatocele undergo spontaneous cure and rarely require operation. Thorn reports 157 cases, in only 6 of which was operation thought necessary, the rest having been treated by careful watching and rest in bed. The total mortality here was only six-tenths of one per cent. Fehling has reported 91 cases, treated in a similar manner, without a single death. A disadvantage of the method, however, is that it is extremely slow and the patients have to remain many weeks in bed.

On the other hand, there are certain conditions which render operation imperative in cases of hæmatocele: such indications as rapid increase in the size of the tumor, signs of suppuration, or pressure on important organs. When such conditions are present the hæmatocele is best attacked by way of the vagina. An incision is made through the posterior vaginal wall, the clot is evacuated, and the cavity is at once packed with sterile gauze. This usually gives most excellent results, but all operators agree that it is frequently necessary to open the abdomen in order to check hemorrhage that cannot be controlled by the pack; therefore one should always have the abdomen prepared for a laparotomy before making the vaginal incision.

Treatment during the Latter Months of Pregnancy.—Occasionally in the latter half of pregnancy we shall find the fœtus enclosed in a large tubal or ovarian sac; more commonly, however, the fœtus has escaped through the rent in the tube wall and we have to deal with a secondary abdominal pregnancy in which the placenta is attached to the pelvic floor while the fœtus lies free. In such cases, as there is always danger of a secondary rupture, prompt laparotomy is indicated. If the child is within a few weeks of viability some have advised a waiting policy, thus giving the child a chance that it would not have were immediate operation done. If such a course be decided upon the dangers to the mother should always be explained to the family.

As to the best method of operating in such cases, unquestionably the ideal procedure is to open the abdomen and remove the entire gestation sac; but frequently, on account of adhesions to neighboring organs, such a course is inadvisable, and here the sac should be incised, care being taken to avoid wounding the placenta if possible. Then the child should be removed and the edges of the sac should be stitched to the margins of the abdominal wound, the cavity being packed with gauze and the placenta allowed to come away piecemeal. This entails a longer convalescence, but on the whole is better than endangering the life of the patient by attempting to remove an adherent placenta. In certain cases this plan cannot be carried out, and then the entire sac must be removed no matter how serious the procedure may seem.

When the fœtus dies the danger to the mother materially diminishes, for, when the fœtal circulation ceases, the hemorrhage from the placental site is not so much to be feared. If a diagnosis of the death of the child be made, operation should be deferred for a short time in order to favor closure of the maternal blood spaces and separation of the placenta; it being understood, however, that immediate operation may be necessary at any time.

George W. Dobbin.

EXUDATION, PATHOLOGICAL.—One of the chief phenomena of inflammation is the passage of a portion of the fluid and cellular constituents of the blood from the blood-vessels into the tissue spaces or out upon one of the free surfaces of the body. This process is known as exudation, and the fluid and cells which have left the vessels as an exudate. *Exudation* is distinguished from *transudation* in that it is always a part of an inflammatory process, while the latter term is commonly restricted in its application to an increase in the formation of lymph not due to inflammation, but dependent upon changes in blood pressure, alterations in the blood, or in the vessel wall which favor an increased lymph production (œdema). *Exudates* differ from *transudates* in their higher albumen content, higher specific gravity (1.016–1.025), and their greater richness in cellular elements. Transudates have a low specific gravity (1.006–1.008, rarely reaching 1.012), contain but few cells, and differ but little in their composition from that of normal lymph, their albumen content being very slight, sometimes lower and sometimes slightly higher than that of lymph. Exudates are usually turbid from the large number of leucocytes contained in them, while transudates are usually clear or but slightly cloudy.

The source of both the fluid and the cellular constituents of exudates is the blood, and the escape of these from the vessels is due to some alteration in the vascular walls which forms an essential part of the inflammatory process. Evidences of this alteration are seen in the dilatation of the vessels, the increased resistance offered to the blood current with consequent slowing of the circulation, the increased adhesion of the cellular elements of the blood to the vessel wall, and, most important of all, the increased permeability of the walls. The composition of the exudate is therefore dependent upon the degree of pathological alteration as well as upon the peculiar property of the affected vessel. The greater the injury the greater the exudate and the amount of albumen and cellular elements contained in it. Moreover, since the

properties of the blood-vessel walls differ in different regions of the body, the character of inflammatory exudates in different parts will also vary; thus the albumen content of pericardial and pleural exudates is higher as a rule than that of peritoneal. Exudates differ further in the proportionate amounts of the various blood elements contained in them. In one case the exudate may be almost entirely fluid with a very small number of cells (serous exudate); in another, the leucocytes may be so numerous as to give the exudate a more or less thick, creamy appearance (purulent exudate); or the escaped fluid may contain large amounts of fibrinogen and fibrin ferment (fibrinous exudate). Further, many red blood cells may escape by rhexis or diapedesis (hemorrhagic exudate), or large numbers of blood plates may be present in the exudate. Combined characteristics exist, so some exudates may be properly styled sero-fibrinous, sero-purulent, fibrino-purulent, etc. Besides the blood constituents inflammatory exudates also contain tissue débris, fluids arising from cellular liquefaction and degeneration, etc. The high proportion of albumen in many exudates is due partly to proteids derived from cellular destruction. In exudates there may be contained also poisons arising from tissue destruction or, in infective inflammations, from the growth of micro-organisms; the bacteria themselves, and cells arising from the proliferation of fixed cells of the tissue, may likewise be present.

The various problems regarding the manner of escape of the exudate from the blood-vessels have offered abundant scope for investigation and speculation, and a survey of the various theories evolved to solve these problems forms one of the most interesting historical chapters in the development of pathology. The existence of pathological exudates was very early recognized. Rokitansky believed that they arose from an increased permeability of the wall of the vessels due to thinning from overdistention. Vogel, Paget, and others explained their formation as being due to an increased attraction between the tissues and the elements of the blood. Virchow distinguished two kinds of exudates: one the result of mechanical pressure in the vessels, pressed-out blood serum; the second as nutritive, the result of an increased attraction between the tissues and blood constituents. The cellular portion of the exudate he believed to originate from a proliferation of the tissue cells in the inflamed area.

Dutrochet in 1842 and Waller in 1846 first observed the migration of leucocytes from the vessels, but these observations were forgotten until Cohnheim in 1867 rediscovered the phenomenon. This observer affirmed that the passage of leucocytes from the vessels formed the essential feature of inflammatory exudation; that the cells in inflamed areas did not arise from the tissue cells; and he explained the origin of the fluid portion of the exudate as due to a molecular change in the vessel wall. This view was opposed by many, notably Stricker, who held that the greater part of the cells in inflammatory exudates were not leucocytes but new cells arising out of the inflamed tissue, pus cells therefore taking their origin from connective-tissue cells.

Heitzmann believed also that the cells of the exudate were not leucocytes, but were embryonic cells arising from the softened or dissolved intercellular substance. More recently Grawitz has taken a similar position, in that he declares the cells of the exudate to arise from so-called "slumbering cells" in the intercellular substance of connective tissue which under normal conditions are not visible. The views of Stricker, Heitzmann, and Grawitz find little acceptance at the present time, the almost universally accepted opinion being that the chief part of the cells of inflammatory exudates at least arises from the blood.

Heidenhain explains the formation of lymph as a process of secretion on the part of the cells of the vessel walls and not as a pure filtration process. Pathological stimulation of this process or alterations in the vascular walls will change the amount and character of this secretion. A portion of the fluid of exudates at least must be regarded as being of the nature of a pathological cell secretion. According to Arnold, Thoma, and Engelmann the formation of the exudate is due chiefly to changes in the lines of cement substance lying between the endothelial cells of the vessel wall. Through distention of the vessel the cement substance may be so thinned as to form small openings through which both fluid and cellular elements escape. Other writers hold that the escape of the leucocytes is not due to alterations in the vascular walls but to chemotactic influences acting from outside of the vessels and causing the cells to pass through the wall.

At the present time the exact nature of the process by which exudates are formed cannot be said to be definitely fixed. The most commonly accepted view is that the chief part of the fluid and cellular elements comes from the blood, and that both pathological secretion and alterations in the wall, as well as chemotaxis, play parts in the formation of the exudate. The escape of the leucocytes is considered an active process brought about by means of their amœboid power in response to chemotactic stimuli. In the very earliest stages of the inflammatory process the cells of the exudate are wholly leucocytes; later, tissue proliferation begins and a part of the cells of the exudate may thus be derived from tissue cells.

Recently much attention has been given to the closer study of the leucocytes found in exudates. Lubarsch divides the cells of inflammatory exudates into hæmotogenous wandering cells and histogenic wandering cells, in the former case descendants of immigrated cells, in the latter arising from fixed connective-tissue cells. The relative proportion of these cells varies in different forms of inflammation, and in different stages of the same form; but the immigration of leucocytes forms the most prominent feature of the majority of inflammations. Until recently it was affirmed that the migrating leucocytes were chiefly polynuclear forms and that these only possessed the power of active movement. Welch, Councilman, and others have shown that lymphoid cells may also migrate, a fact which was formerly denied. The so-called plasma cell also appears to play an important rôle in many exudates, particularly those of chronic inflammation. This cell is now believed by the majority of writers to be of hæmatogenous origin, most probably a form of lymphocyte. It is characterized by a protoplasm staining blue with methylene blue, an eccentrically placed nucleus, a perinuclear clear space, and a nucleus possessing a number of coarse particles of chromatin arranged on the inner surface of the nuclear membrane. Plasma cells occur in large numbers in the cellular exudate of chronic inflammations, particularly, as Councilman has shown, in the interstitial nephritis of scarlatina. This observer believes that they are lymphocytes which migrate as such or as plasma cells from the blood, and multiply outside of the vessels in response to chemotactic stimuli. It would appear, then, that there are selective chemotactic stimuli; part influencing the migration of lymphoid and plasma cells, part the polynuclears or other varieties of leucocytes. Some exudates contain large numbers of eosinophiles and mast cells, but the significance of such occurrence is not yet known.

Wieniarski confirms the assertion of Korczynski and Wernicki that lymphocytes predominate in exudates not due to malignant disease or showing a tendency to become purulent. When neutrophile polynuclears predominate the exudate is caused either by malignant disease or by pus-forming agents. According to these observers the presence of eosinophile cells and red blood cells in exudates is without significance. In pleural and peritoneal transudates the cells are almost exclusively lymphocytes; in pleural exudates of serous nature they are also lymphocytes; the predominance of polynuclears always indicating a purulent process in case no malignant disease is present. In one case in which but few leucocytes were present in the exudate, but these all polynuclears, a purulent exudate developed later; in another case in which 6,230 lymphocytes were present in a cubic millimetre of the exudate the inflammation did not

become purulent in character. The same facts apply also to peritoneal exudates. In serous pleural exudates the number of lymphocytes varies from 270 to 9,270 per cubic millimetre, in transudates from 60 to 300. In peritoneal transudates even smaller numbers of lymphocytes are usually present. During the absorption of exudates the number of leucocytes increases; during fibrinous exudation they are found in smaller numbers. Much work yet remains to be done along this line, as many important problems regarding the fate of the migrated cells have yet to be settled.

The fluid and cellular exudates from the vessels collect first around these, but soon spread into the lymph spaces of the neighboring tissues forming an infiltration. If the exudate is very abundant the neighboring healthy tissue may be infiltrated to such an extent that it may be damaged and the inflammatory process spread. The tissue elements may absorb a portion of the exudate, and become swollen or vacuolated; or all of the tissues may become dissolved in the exudate. If fibrinogen and fibrin ferment are present, coagulation may take place either before or after liquefaction of the affected tissue takes place. Open spaces in the tissues, such as the lung alveoli, may become filled with the exudate; if the inflammation involves a body surface the exudate may either collect in one of the body cavities or be discharged from a free surface.

The first pathological changes which find expression in the formation of a pathological exudate are vascular phenomena which are due either to irritation or paralysis of the vaso-motor system, or to a direct action upon the walls of the vessels themselves, the result in either case being a dilatation of the vessel. This change may, however, take place without the production of an exudate, but it always precedes exudate formation when the latter occurs. Following the dilatation of the vessel there is a slowing of the blood stream and with this a marginal collection of the leucocytes along the vessel wall; and finally diapedesis of the cellular elements takes place simultaneously with the passing out of the fluid portion of the exudate. The passage of the leucocytes is an active process accomplished by means of amœboid movement, and is only an increased degree of a process which takes place to a limited extent normally (wandering cells). The cause of the increased diapedesis of leucocytes in inflammation is probably partly chemotaxis and partly changes in the vessel wall. According to Arnold and Thoma the leucocytes pass through the endothelial cement substance which is stretched and thinned by the distention of the vessel. The leucocyte first sends a process through the thinned portion of the wall, and through the opening thus made the remainder of the cell body slips after. Through the opening formed in this manner the red blood cells and fluid elements of the blood may also pass.

The passage of a serous exudate into a body cavity, as the pleural or peritoneal, is spoken of as a serous effusion. Serous exudates on mucous membranes are styled catarrhs; localized collections of fluid beneath the horny layer of the skin are blisters, vesicles, bullæ, or blebs. The serous infiltration of tissue spaces is an inflammatory œdema. On mucous surfaces serous exudates may contain a large percentage of mucin derived from mucous degeneration of the epithelium (mucous catarrh); or a loosening of the epithelium into the exudate may occur (desquamative catarrh). If flakes of fibrin are formed in the exudate it may be styled sero-fibrinous; if the number of leucocytes is so great as to make the fluid slightly turbid, sero-purulent.

Fibrinous exudates occur either upon mucous or serous surfaces, into the lung alveoli, or into tissue spaces. On mucous membranes they form whitish or grayish croupous or diphtheritic membranes which are either loosely or firmly adherent to the tissues beneath. These membranes are made up chiefly of granular or fibro-granular fibrin, occasionally in coarse bands or hyaline masses. In fibrinous effusions large masses of stringy fibrin may float in the exudate, while the serous surface may be covered with a thick layer of stringy fibrin, or in mild cases only small strings or flakes may be scattered over the surface. In the lung the alveolar spaces may be completely filled with a network of fine fibrin fibrillæ enclosing leucocytes, red blood cells, and desquamated epithelial cells in its meshes. In the kidneys fibrillar or hyaline fibrin may be found in the tubules. In the follicles of the spleen and lymph glands the spaces between the cells may be filled with a fibrinous exudate. The intercellular spaces in tubercles are also commonly filled with an exudate of fibrin. According to Neumann the fibrinous material found in certain inflammations of serous membranes is not an exudate, but the result of a "fibrinoid degeneration" of the superficial connective tissue of the membrane. This theory is not supported by the actual findings, and has not been generally accepted.

If the inflammatory exudate contains a large number of leucocytes, but not in a sufficient number to cover up the structure of the tissue, it is spoken of as a small-celled infiltration. When the leucocytes in the exudate are so numerous as to give it a white or creamy appearance it is called pus; such an exudate on mucous membranes is a purulent catarrh; in the body cavities a purulent effusion or empyema. Purulent vesicles are styled pustules; deep-seated purulent exudates, abscesses. Sero-purulent exudates may infiltrate the tissues (purulent œdema). Fibrin is a very frequent accompaniment of purulent exudates (fibrino-purulent exudate); and the inflammations of serous membranes usually produce exudates of this kind. Usually with an increase of pus formation the amount of fibrin decreases and the fibrin masses present in the exudate begin to dissolve. Fibrin coagula infiltrated with leucocytes become white and friable. The presence of large numbers of red blood cells (hemorrhagic exudate) and blood plates is found usually in association with fibrinous exudation, but they may also be present in serous inflammations.

The exciting causes of pathological exudation are those of inflammations in general. Serous, sero-fibrinous, and fibrinous exudates may be caused by thermal and chemical means; but are most frequently due to infection with the Diplococcus pneumoniæ, Bacillus diphtheriæ, and the Streptococcus pyogenes. Purulent exudates may also be caused by chemical action, but are most frequently the result of infection by the Streptococcus pyogenes, the staphylococci, gonococcus, etc. After the causes which have produced them cease to operate exudates are discharged from the body, absorbed, organized, or under certain conditions encapsulated and calcified. In the processes of repair and organization it is possible that the exudate may serve as a source of nutriment for the new-formed tissue cells.

The significance of pathological exudations is practically that of inflammation in general. The old view, and the one still commonly accepted by clinicians, is that they are essentially harmful. From the pathological side, however, inflammation must be regarded as a reaction on the part of the body to local injuries, having for its purpose protection and repair. Though both protective and reparative measures may be imperfectly carried out and even many times in themselves cause further damage, it must be granted that inflammation is essentially an attempt at self-preservation. In this attempt the constituents of the exudate, both cellular and fluid, play the chief part. The leucocytes of the exudate act as phagocytes and as sources of bactericidal and antitoxic substances, or may even furnish nutriment to formative tissue. The fluids of the exudate likewise possess important germicidal and antitoxic powers; they serve further as diluting and irrigating agents; and may also carry nutriment to the newly forming cells. Even fibrinous exudates may serve a beneficial purpose in limiting the action of bacteria. Masses of leucocytes may also form mechanical barriers to the spread of germ growth.

In the exudate we therefore find the means of counteracting or destroying harmful agents, neutralizing their poisons, protecting the organism, and aiding in healing. But in the attempt to fulfil these functions exudates may in themselves create new conditions that may damage

the body. The pressure upon the brain of meningeal exudates, the disturbance of respiration and heart's action by pleural and pericardial effusions, the closure of the glottis from inflammatory infiltration, etc., are examples of the imperfect adaptation of the process, which though in the main intended to be beneficial may often overreach its aim.

(See also *Inflammation.*) *Aldred Scott Warthin.*

EYE.—General Arrangement.—The eyeball (see Fig. 1952) is a spheroidal expansion attached to the end of the optic nerve, optically equivalent to a camera obscura and filled with fluid or semifluid contents. It has three concentric envelopes or coats: an outer one, protective in character, formed of fibrous tissue, called the *sclera* behind, where it is white and opaque, and the *cornea* in front, where it is clear and transparent; a middle one, nutritive in character, composed of vascular tissue intermingled with pigment, called the *choroid* behind, in front assisting to form the *iris* or diaphragm which regulates the admission of light through a central aperture, the *pupil*; an inner one, percipient in character, formed of nervous elements and called the *retina*.

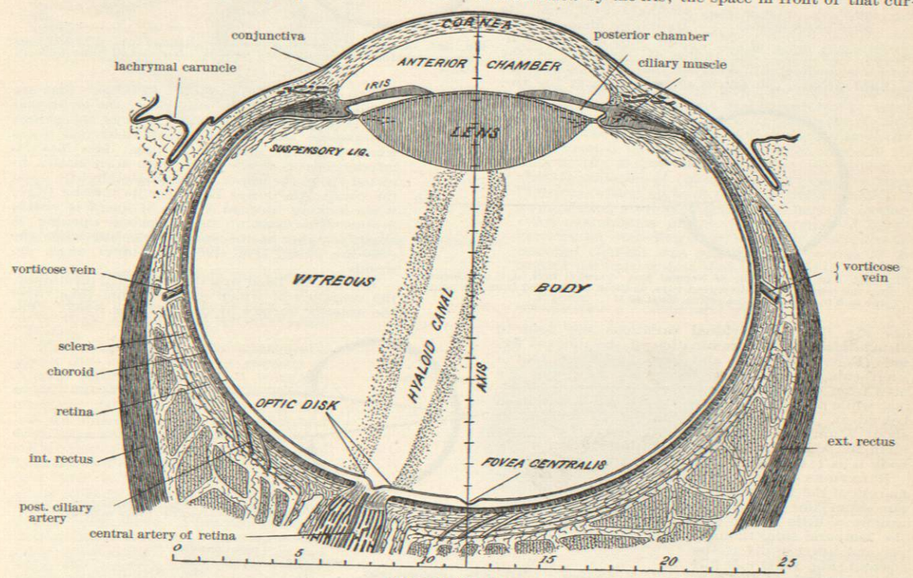

Fig. 1952.—Diagram of Horizontal Section of Eyeball Drawn to an Accurate Scale. (Flemming.)

The rays of light that pass through the pupil are focussed dioptrically by the refractive action of a clear, lenticular body, the *crystalline lens*, which is set behind the iris and divides the cavity of the eyeball into two compartments or chambers: the *aqueous chamber*, situated in front and containing a watery fluid called the *aqueous humor*, and the *vitreous chamber* behind, containing the semifluid *vitreous humor* or *body*. The aqueous chamber is subdivided by the iris; the space in front of that curtain being known as the *anterior chamber*, that between it and the lens as the *posterior chamber*.

In describing the eye terms applicable to a spheroid are used (see Figs. 1953 and 1954). The antero-posterior diameter is termed the *axis*, the ends of that diameter the *poles*. The *equator* is a line on the surface equidistant from these poles; circles and directions parallel to it are

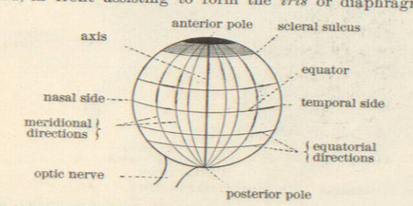

Fig. 1953.—Diagram of Right Eye, as Seen from Above, to Illustrate Terms of Orientation.

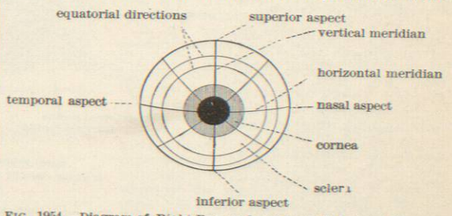

Fig. 1954.—Diagram of Right Eye, as Seen from Before, to Illustrate Terms of Orientation.

termed *equatorial*. Circles drawn from pole to pole are termed *meridians*, directions along these are *meridional*. Any half of the ocular globe is called a *hemisphere*, any fourth a *quadrant*. The lateral aspects of the globe are distinguished as *temporal* and *nasal*.

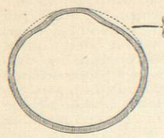

FIG. 1955.—The Eyeball Compared with a Sphere.

The eyeball is not a perfect sphere. Its antero-posterior diameter averages 24.2 mm., its transverse diameter 23.6 mm., and its vertical diameter 23.2 mm. The radius of curvature of the cornea is considerably less than that of the sclera and a slight groove, the *scleral sulcus*, separates the two

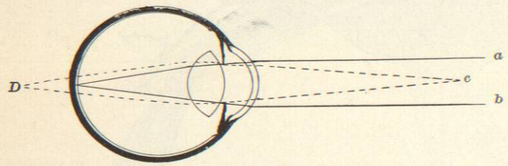

FIG. 1956.—An Emmetropic or Normal Eye. Parallel rays, *a b*, are focussed upon the retina; while divergent rays, as those proceeding from the point *c*, come to a focus *behind* the retina, as at *D*.

(see Fig. 1955). Individual variations may lead to visual defects. An examination of the annexed figures (Figs. 1956, 1957, and 1958) will show that a lengthening of the axis of the ball may cause the rays from distant objects to focus in front of the retina, while a shortening of that axis may focus them behind it. The average weight of the eyeball is about 7 gm., its specific gravity about 1.077. At birth its weight as compared with the rest of the body is as 1:419; at maturity it is as 1:4,832.

RELATIONS.—The eye is placed just within the margin of the pyramidal cavity of the orbit, 16 to 18 mm. from the optic foramen (Figs. 1959 and 1960), usually a little nearer to the upper margin and to the temporal side, though equidistant from the upper and lower walls at the equator (Fig. 1961). It fills about one-fifth of the cavity, the remainder being occupied by the ocular vessels, nerves and muscles, the lachrymal gland and the bulbar fascia, loose orbital fat lying in the interstices. The edge of the orbit retreats somewhat on the temporal side so that the eyeball is there somewhat more accessible for operation.

In front the eye is protected by two movable folds of integument, the eyelids, or *palpebræ*, which, when open (Fig. 1962) form an aperture known as the *rictus palpebrarum* or palpebral opening that exposes nearly the whole of the cornea and a considerable portion of the sclera. About 30 mm. in length and 12 to 15 mm. high, it is curved above and below, uniting internally and externally at the *angles* or *canthi* of the eye. The external angle is sharp, the internal rounded, forming a slight recess

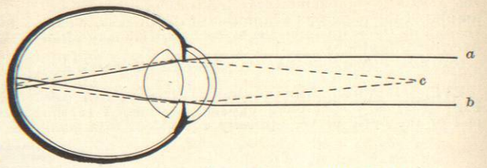

FIG. 1957.—A Myopic or Short-sighted Eye. Parallel rays, *a b*, converge before reaching the retina. Rays must be *divergent* in order to focus there.

known as the *lachrymal bay*. It is here that are found passages for the removal of the lachrymal fluid (Fig. 1963). Two small openings, the *puncta lachrymalia*, lead into the short lachrymal ducts which discharge through the nasal duct into the inferior meatus of the nose. With horizontally directed vision the upper lid impinges slightly upon the cornea, the lower falls near its inferior edge; when looking upward a strip of sclera is visible below. The axis of the palpebral opening is slightly higher at the outer angle, but when the eyes are closed (Fig. 1964) the outer angle descends.

The lids are lined by a thin epithelial membrane, the *conjunctiva*, which extends from them over the anterior surface of the ball (Fig. 1965). The

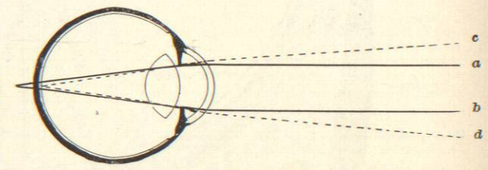

FIG. 1958.—A Hypermetropic or Far-sighted Eye. Parallel rays converge *behind* the retina. Rays must be *convergent* in order to focus there.

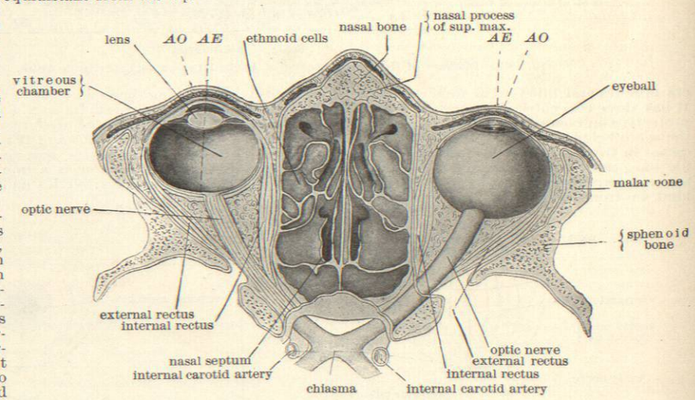

FIG. 1959.—The Eyes and Optic Nerves Seen from Above after Removal of the Roof of the Orbit. *AO*, Axis of Orbit; *AE*, axis of eye.

place of reflection is known as the *fornix* of the conjunctiva and is situated 13 mm. from the upper lid, 9 mm.

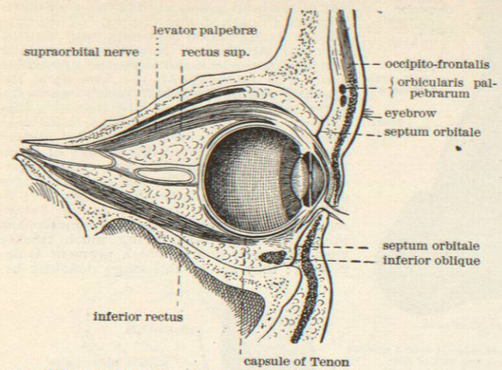

FIG. 1960.—Sagittal Section of the Visual Apparatus Through the Summit of the Cornea and the Optic Foramen. (Merkel.)

from the lower one when the eyes are open. Its distance from the cornea is 10 mm. above, 8 mm. below, 14 mm. at the outer angle, and 7 mm. at the inner angle (Testut).

Within the orbit a thin aponeurotic sheet, the *bulbar fascia* or capsule of Tenon, separates the eyeball from the orbital fat (Fig. 1966). This invests the posterior part of the ball with two layers, one closely adherent to the sclera and sometimes called the *episclera*, the other reflected over the orbital contents. The two layers are united by fine trabeculæ, and there is between them a loose lacunar interval, the *perischeral space*, that communicates with the perineural space of the optic nerve. Processes from the bulbar fascia invest the structures that pass to the eyeball. Those that extend upon the ocular muscles are of considerable surgical importance. Extensions also pass to the fornix of the conjunctiva, to the eyelids, and to the edges of the orbit. As the latter assists to retain the contents of that cavity, it is known as the *orbital septum*.

DEVELOPMENT.—The percipient portion of the eye is, like the essential part of other organs of sense, derived, either directly or indirectly, from the ectoderm. In its simplest form, as seen in invertebrates, it consists merely of tegumentary cells, forming eye spots or retinas, surrounded by pigment and communicating by filamentous processes with nervous ganglia. Often, by a thickening of the epidermis, one or more lenses are developed in connection with these.

In vertebrates the ectodermal integument becomes infolded to form the central nervous system, and the development of the eye is more complicated. In Amphioxus there appears, toward the close of the embryonic period, on the swollen anterior end of the simple tube that forms its central nervous system, a pigmented spot that is believed to represent an eye. To this spot rays of light pass through the pellucid jelly that covers the animal externally, and, arrested by the pigment, excite directly the cells of the neural tube and apparently impart an obscure sensation of vision. In this primitive form, therefore, vision appears to be a direct function of a specialized portion of the central nervous system.

An examination of the visual organs of higher vertebrates, including man, shows that here too the essential element was primitively a portion of the central nervous system which, extending peripherally, became separated and associated with other elements to form a complicated apparatus. The retina may be regarded as a specialized portion of the cortex of the brain, the optic nerve as a fibre tract, the lens as an epithelial invagination, the cornea, sclera, choroid, and contents of the eyeball as connective-tissue derivatives.

In the human embryo of three weeks (corresponding to the chick of two or three days) the central nervous system is also tubular, being formed by the approximation and gradual concrescence of the edges of a groove. The area that is to form the retina lies at first along the outer edges of the groove, in the region known as the neural crest, from which also originate the cells for the sensory nerves. The retinal cells differ from the latter in that, as the edges of the groove fold over to form the tube, they are gradually drawn in until they lie along the sides of the cavity (Fig. 1967, A, B, C, D).

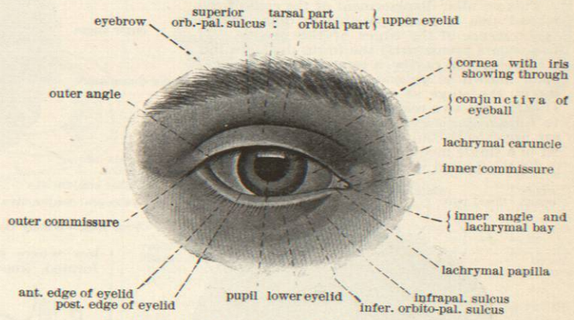

FIG. 1962.—The Eye as Seen with Lids Open. (Toldt.)

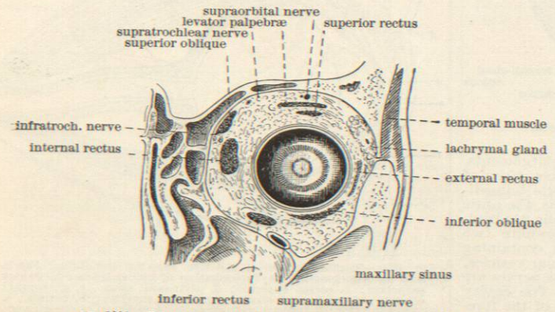

FIG. 1961.—Frontal Section of the Eyeball in the Orbit. (Merkel.)

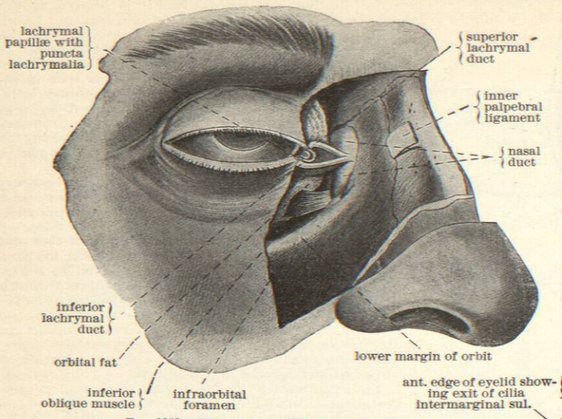

FIG. 1963.—The Lachrymal Passages.

This peculiar displacement of the cells of the retinal area is made possible by delay in the concrescence of the edges of the groove. If this occurs prematurely the fœtus develops but a single orbital cavity, a teratological phenomenon known as *cyclopia*, which differs according to the stage at which union takes place. If early, the two retinas may be fused together to form a single eye; somewhat later, the two are imperfectly united; still later, two eyes exist in a single median orbit.

At the period we are considering the neural

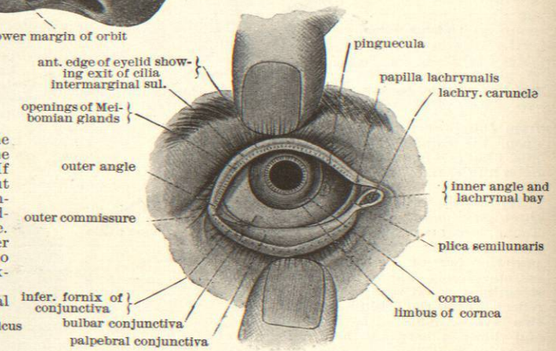

FIG. 1965.—Widely Opened Eye of an Elderly Person; Lower Lid Everted.

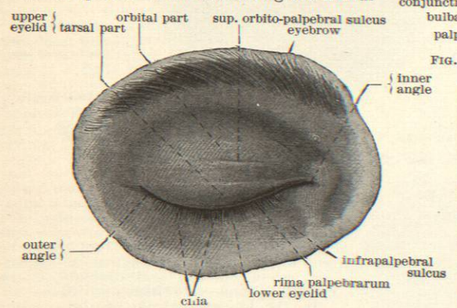

FIG. 1964.—Eyelids of Right Eye when Closed.

tube shows at its cephalic end three bulbar enlargements, the primary cerebral vesicles. It is from the anterior of these, the forebrain, that are formed the parts immediately concerned with vision: the retinal areas, the optic nerves, the corpora geniculata, and the optic thalami. The forebrain now widens out laterally, forming two hollow protrusions, whose bulbar extremities, containing the retinal areas, are called the primary ocular vesicles, the constricted pedicles, from which the optic nerves are formed, being known as the optic stalks. The cavity of each still communicates freely with that of the forebrain (Figs. 1968, 1969).

The ocular vesicles in their outward growth soon reach the epithelium or ectoderm of the side of the head which, at the place of contact, becomes thickened and cupped. This cupping or invagination continues until another vesicular structure is formed, the lenticular vesicle, from which develops the crystalline lens. The intrusion of the lens upon the growing end of the ocular vesicle causes the latter to assume a shape like that of a thin hollow rubber ball with a marble somewhat smaller in size pressed against its surface (Fig. 1970). Because of this change of form it is henceforth called the optic cup (secondary ocular vesicle). The successive changes are shown diagrammatically in Figs. 1971 to 1975. The edge of the cup is the future margin of the pupil; its walls are double, the inner or retinal layer being much thicker than the outer one in which pigment is deposited; they are at this stage deficient below where growth is less rapid and a fissure is thus formed, known as the choroidal cleft, through which

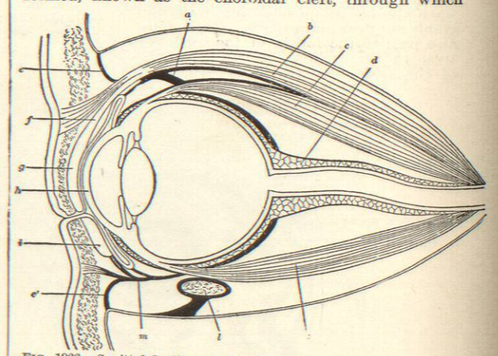

FIG. 1966.—Sagittal Section of the Eye, showing the Bulbar Fascia and its Attachments. *a*, Prolongation of bulbar fascia; *b*, levator palpebræ; *c*, rectus superior; *d*, perineural space; *e*, *e'*, septum orbitale; *f*, tendon of levator; *g*, tarsus superior; *h*, cornea; *i*, tarsus inferior; *k*, inferior rectus muscle; *l*, inferior oblique muscle; *m*, prolongation of bulbar fascia.

wandering mesenchymal cells penetrate between the retina and the lens to form the vitreous body. The interval between the layers is soon obliterated. The central artery of the retina enters the cleft back of the cup, upon the optic stalk, thus becoming finally enclosed

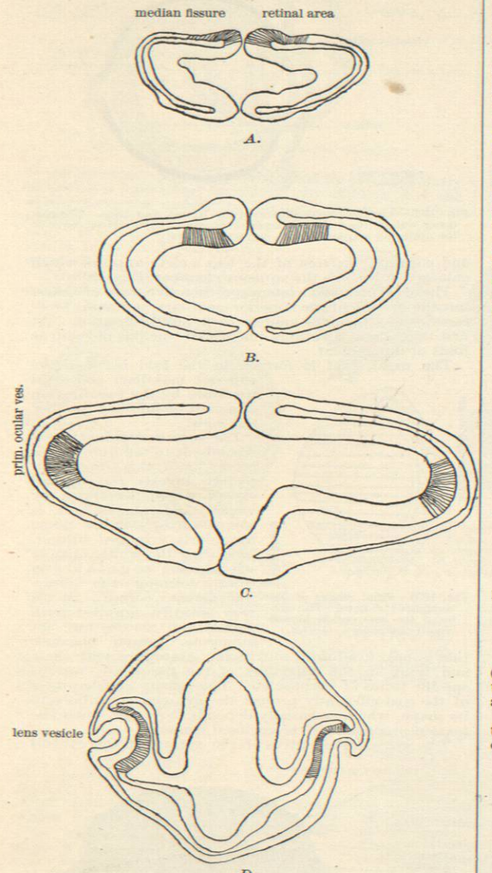

FIG. 1967.—Diagrams showing the Displacement of the Retinal Cells and their Relation to the First Cerebral Vesicle. (Dejerine.) *A* and *B* show the early development of the first primitive cerebral vesicle; *C*, the primitive ocular vesicle; *D*, the secondary ocular vesicle and the lens vesicle.

within the optic nerve. The cleft closes early but a trace of it may often be found in the adult. If the closure is incomplete the ocular defect known as coloboma occurs, causing distortion of the pupil or a defect of the visual field.

The retina develops like a part of the cerebro-spinal axis. Spongioblasts and neuroblasts arise in it, the former developing into the supporting tissue of the retina or fibres of Müller, of which the expanded bases form the internal limiting membrane, the distal arborescent extremities the external limiting membrane in which spaces are left which eventually enclose the rods and cones. The neuroblasts appear first in the area centralis, afterward extending peripherally as far as the ora serrata. Beyond this, in the retinal layer of the ciliary region and the iris, spongioblasts alone occur. The neuroblasts develop axis-cylinder processes that grow inward along the optic stalk, gradually obliterating its cavity and constituting the optic nerve. The percipient elements

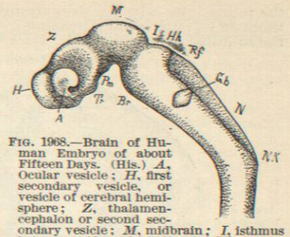

FIG. 1968.—Brain of Human Embryo of about Fifteen Days. (His.) *A*, Ocular vesicle; *H*, first secondary vesicle, or vesicle of cerebral hemisphere; *Z*, thalamencephalon or second secondary vesicle; *M*, midbrain; *I*, isthmus between mid- and hindbrain; *Hb*, fourth secondary vesicle; *N*, fifth secondary vesicle; *Gb*, otic vesicle; *Rf*, fourth ventricle; *Nk*, neck curvature; *Br*, pons curvature; *Pn*, mammillary process; *Tr*, infundibulum.

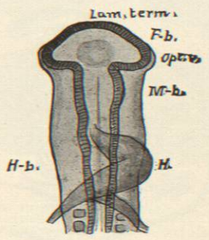

FIG. 1969.—Dorsal View of Head of Chick of Fifty-eight Hours. (Mihalkovics.) *Lam. term.*, Lamina terminalis; *F.b.*, forebrain; *Opt. v.*, ocular vesicle; *M.b.*, midbrain; *H*, heart.

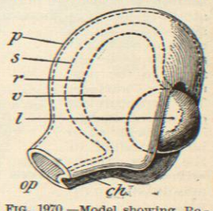

FIG. 1970.—Model showing Reconstruction of the Optic Cup. (Hortwig.) *p*, Outer wall of cup, which becomes the pigmented layer of the retina; *r*, its inner wall, which becomes the retina; *s*, temporary space between the two walls; *v*, vitreous chamber; *l*, lens; *ch*, choroid fissure; *op*, hollow stalk of cup which becomes the optic nerve.

(rods and cones) are the last to develop, not appearing until the fifth month in man and after birth in kittens and rabbits.

In its early stages the lenticular vesicle is proportionately larger than at birth, filling the cavity of the optic cup so that there is but little space left between it and

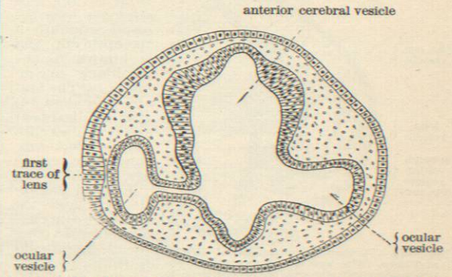

FIG. 1971.—Section of Head of Chick, Second Day of Incubation. (Duval.)