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Etiology and factors of retinal detachment

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The purpose of this paper is to give the reader an understanding of retinal detachment and its etiologic factors. For the sake of simplicity in writing and understanding the following outline fashioned somewhat from that in Duke-Elder's Ophthalmolgic text, vol. III will be used. In order to understand retinal detachment a review of the embryology, histology and anatomy is first presented.

Embryology:

The optic nerve and retina are formed from an outgrowth of the forebrain. The outgrowth is called the optic vesicle (see diagram I.) It is spherical with a connecting tube, the stalk, to the chamber of the forebrain. The optic vesicle is transformed into the optic cup by invagination of the distal layers. On the underside of the vesicle a notch occurs which affects both layers of the vesicle and continues along the optic stalk as a groove to the forebrain. This defect is called the choroid fissure. (See diagrams 1,2,3).

The choroid fissure closes so that a doubled walled cup is formed; thus, the stalk becomes a tube

within a tube. By the formation and closure of the choroid fissure, the inner layer of the optic cup continues directly backward within the optic stalk. This continuity creates a direct path along which optic nerve fibers, originating in the inner layer of the retina, pass to the brain. This same arrangement furnishes a tunnel which the hyaloid artery utilizes in reaching the interior of the eyeball without piercing its layers.

The two layers of the optic cup differentiates into the inner nervous layer, which receives and transmits images to the brain, and the outer pigment layer. These two layers make up the adult retina; there are no known connections between the two layers except at the ora serrata. Thus, these layers have a potential space between them. It is the separating of these layers that is called "Retinal Detachment," where in actuality it is intra-retinal separation.

Anatomy:

A quick review of the histology of the retina will follow with special emphasis put upon the areas and structures that are important in retinal detachment.

There are ten layers to the mature retina. The outer pigment layer gives rise to the pigment

epithelium. The other nine layers are derived from the inner nervous layer.

These layers, from without inward, are arranged as follows: 1. pigment epithelium; 2. layers of rods and cones; 3. external limiting membrane; 4. outer nuclear layer; 5. outer molecular layer; 6. inner nuclear layer; 7. inner molecular layer; 8. ganglion layer; 9. nerve fiber layer; 10. internal limiting membrane.

1. The pigment epithelium is a single layer of cells seen as a continuous sheet extending from the optic nerve to the ora serrata. This epithelium is firmly attached to the basal lamina of the choroid, but loosely to the overlying nervous layer of the retina except at the ora serrata and the optic disc. This pigment layer is composed of cells which are 12-18u long and up to 5u high. They are hexagonal in shape and fit together like flagstones. The cell is divided into three parts (a) the dome, next to the choroid; (b) the base, which contains pigment and from which (c) the pigment process project towards and between the rods and cones.

2. The rods and cones are the visual cells and constitute the light sensitive part of the retina.

The remainder of the retina is concerned with the transmission of impulses arising from stimulated rods and cones. The rods and cones are a neuro-epithelial layer that lies between the external limiting membrane and the pigment epithelium.

The rods are long, slender and cylindrical, 40-60u in length and 2u in diameter. Each rod shows a division into outer and inner segments. The outer segment is long and lies external to the external limiting membrane. The thinner segment approximates the external limiting membrane, from this end a thin fiber passes through the external limiting membrane and enlarges into a densely staining nucleus called the rod granule, then it terminates in a small end knob in the outer molecular layer. The rods contain rhodopsin or visual purple which functions for perception in dim light and movement at the peripheral visual field.

The cone consists of inner and outer segments. The cones change shape depending upon the area in which they lie (see diagram 4). The outer segment is shorter than the inner. The inner segment is continous with the cone fiber which passes through the external limiting membrane with a diameter barely

less than that of the inner segment. The cone fiber becomes the cone granule and then continues on as a short fiber and ends in the outer plexiform layer with numerous short branches called the cone foot. The cones contain no visual purple, but contain a pigment called iodopsin which functions to register acute vision in bright light.

3. The external limiting membrane is a thin chicken wire-like netting membrane formed by the chief supporting elements of the retina, the Müllers fibers.

4. The outer nuclear layer consists mainly of the rod and cone granules or nuclei.

5. The outer molecular layer is formed from the meeting of the axones of the cones and rods with the dendrites of the oipolar cells and the processes of the horizontal cells and the fibers of Muller. This layer is thickest at the macula and all but disappears at the fovea. This layer takes up fluid and becomes swollen on the slightest injury. It is responsible for macular detachments following trauma and in the post-mortem state.

6. The main body of cells in the inner nuclear layer are the sensory bipolar cells.

These are regular first order neurons and act only to pass the impulse received from the rods and cones. There are two types of bipolar cells. One, the midget bipolar cells synapse with only the cones. The other bipolar cells synapse with rods and cones.

Besides the bipolar cells this layer also contains the following cells. Horizontal cells which are flat and whose processes spread out horizontally to the surface of the retina and lie next to the outer molecular layer. The amacrine cells which have a single process which invades the inner molecular layer and some of them make connections with centrifugal fibers of the optic nerve. The nuclei of the fibers of Muller lie in this layer, also the terminal capillaries of the central retinal vessels are found here.

7. The arborisation of the axons of the bipolar cells and the dendrites of the ganglion are the principle constituents of the inner molecular layer. Also found in this layer are end processes of the above mentioned amacrine cells, the fibers of Muller, a few scattered nuclei, and branches of the retinal vessels. This layer is subdivided into substrata by the processes of the amacrine cells and the dendrites of the ganglion cells.

This layer is uniform in thickness throughout the retina except at the fovea contralis where it is absent.

8. The ganglion layer is almost self-explanatory. The ganglion cells are multipolar nerve cells that are neurones of the second order and are similar to those of the central nervous system. The axons of these cells terminate in the lateral geniculate body and the superior colliculus. Generally this is a single cell layer; however, on the temporal side of the disc there are two layer of nuclei. The closer to the macula(r) the greater the number of layers will be found, up to the depth of eight layers. More and larger retinal vessels are also present.

9. The nerve fiber layer consists of the axons of the ganglion cells; the retinal vessels, centrifugal cells, fibers of Muller, and neuroglial cells. The axones traverse this layer to the papilla where they exit via the optic nerve to the geniculate body and superior colliculus of the brain.

10. The internal limiting membrane separates the retina from the vitreous. This membrane may also be called the hyaloid membrane of the vitreous. This membrane is divided into two layers.

The outer, formed by the feet of the fibers of Muller and the inner which is the true hyaloid membrane. The outer layer is not present at the optic disc or wherever large blood vessels came close to the internal limiting membrane.

Other histologic structures mentioned whose functions appear to be supporting the structure of the retina are the following: (1) the Fibers of Muller, (2) the Golgi spider cells, (3) astrocytes, and (4) horizontal bands. Also found are microglia which are phagocytic wandering cells in the retina.

The retinal structure changes in different areas, some of which are important in retinal detachment.

The optic disc has only the nerve fiber layer and internal limiting membrane present. There are no visual cells here, resulting in the blind spot.

The macula lutea is a funnel shaped depression in the retina lying 3.5 mm. lateral to the edge of the disc and just below its middle. Here the cones gradually replace the rods so that at the center of the macula there are no rods, only cones. This is called the fovea centralis. Here the retina is thinner than any other portion the light perceptive

area of the retina. There are no supporting Fibers of Muller, no ganglion cells and no nerve fiber layer. The inner and outer molecular and inner nuclear layers are reduced to thin membranes. The cones of the fovea are connected to only one ganglion cell. The result of all this is a clearer purer image received by the brain from this region.

The ora serrata is the scalloped anterior border of the retina. Here the retina is attached to the choroid and the vitreous. The visual function of the retina ceases 0.5-1 mm. from the ora because of gradual thinning and finally loss of the rods and cones. The layers fuse together and the retina continues anteriorly as the pars ciliaris retinae which consists of two layers, an outer pigmented and an inner non-pigmented layer that lies on the inner aspect of the ciliary body.

The blood supply of the retina comes from two vascular networks. The main body of the retina receives its blood supply from the central artery. The outer portion of the retina, the rods and cones and outer nuclear layer, is a vascular and receives its nourishment from the chorio-capillaris.

The central artery is an end artery, it has no anastomoses; thus if it is obstructed, blindness

results. This artery enters the retina through the lamina cribrosa traveling with the optic nerve. In the nerve head the central artery lies very close to the vitreous, being separated from it by a single glial layer. The central artery climbs up the nasal side of the globe and divides into two branches, superior and inferior. The retinal vessels continue to divide dichotmously, as they proceed towards the ora serrata, where they end in capillaries which do not anastomose with any other system of vessels.

There are two layers of capillaries in the retina, superficial and deep. These two layers communicate freely with each other. The significance of these two layers is seen in that these different layers are affected by different systemic diseases, resulting in a different retinal picture. When the deep layer is affected and hemorrhage results, a circumscribed round area is seen. However, when the superficial layer hemorrhages it gives a flame appearance to the retina.

Two other small arteries supply the lamina cribrosa and a small area of the surrounding retina. These arteries are the circulus vasculosus nervi optici, which is formed by a circular anastomosis of

some short ciliary arteries, and the cilo-retina artery.

The venous drainage of the retina follows a similar course as the central artery, but not next to it.

The chorio-capillaris is a vascular layer of large bore capillaries. It lies next to the basal lamina, which is the structure that the pigment layer of the retina is so closely attached. The chorio-capillaris completely surrounds the retina from the ora serrata to the optic disc. Its sole function is to provide nourishment for the rods and cones. The arteries that feed these capillaries are the short posterior ciliary arteries. The veins that drain the chorio-capillaris are the venae corticosa. These vessels travel through the orbit. Consequently, any mechanism or infectious process that affects the orbit may also affect these vessels and the areas they serve.

There are four common theories as to the etiology of retinal detachment. These theories are distension, exudation, hypotonia, and traction.

The distension theory is that detachment which occurs through the stretching of the layers of the

eyeball. This theory was postulated by early observers of retinal detachment because they noticed a preponderence of myopes amoungst the affected individuals. These observers considered the stretching of the choroid and sclera as the actual cause for the detachment. In 1857 von Graefe estimated 50 to 60 per cent of all spontaneous retinal detachments occurred in myopes. Von Graefe explained that if the retina was not sufficiently extensible the increase in length of the axis of vision in myopia could produce a separation of the retina from the choroid. This preponderance of myopia in retinal detachments is well recognized today and it is found in about two-thirds of all the patients. However, the pausability of the theory was challanged by Leber in 1916. He demonstrated the retina to be as extensible as the choroid, or perhaps even more so, by demonstrating a contusion that frequently produced a choroidal tear only rarely produced a retinal detachment. Other findings which tend to repudiate this theory is that all people with severe myopia do not develop detachments and that most detachments are situated anterior to the equator, in myopia the greatest elongation occurs behind it.

These facts tend to disprove the distension theory per se'; however, it does point out that myopia is a predisposing factor to retinal detachment.

The exudative theory states that the retina is pushed up by an exudate from underneath. This theory was postulated by Arlt in 1853 and von Graefe in 1854 to explain spontaneous retinal detachments in the non-myopic eye. The exudate results from a change in the normal micro-anatomy of the choroid, e.g. choroiditis, capillary congestion, or a choroidal hemorrhage. This theory is not disputed. It is known a large number of detachments occur this way. However, with this type of detachment tears or holes are not usually present; consequently, spontaneous reattachment frequently occurs.

The hypotonic theory states that retinal detachment resulted from the lowering of the intraocular pressure in the vitreous cavity, thus removing the support that kept the retina in place. This theory was first proposed by Stellwag in 1861, since this time many variations of the same idea have been presented. However, today it is generally agreed by most, that low ocular tension is insufficient to produce retinal detachments unless it occurs suddenly.

Clinical observations, experimentations and accidental gross vitreous loss in cataract extractions prove this to be true.

The traction theory states that the detachment of the retina occurs from pull on the retina from adhesions in the vitreous body. Heinrich Müller was the first to present this theory in 1858. He perpetrated an analogy of the gross findings in cases of intra-ocular inflammation and hemorrhage to that of the histologis picture of the vitreous body. Müller postulated that the vitreous retracted anteriorly pulling the retina with it. In 1882, Leber noted tears and retinal detachment occuring together in eyes with retained foreign bodies. On experimentation he found that he could produce retinal tears that were followed with retinal detachment. Leber did this by aseptic1y introducing metal bodies into the vitreous. These tears had sharp edges which suggested to him they were due to traction rather than degeneration, and ophthalmoscopy failed to show any degeneration near the aperature. From these findings Leber proposed the presence of delicate structures in the vitreous which were adherent to the retina and consequently pulled upon it.

Leber stated the presence of these fibers could only be noted by their affect on the retina.

The traction theory is the accepted theory today to explain spontaneous retinal detachments. Wadsworth, in 1952, in presenting the etiologic and pathology for a symposium on retinal detachments, showed photomicrographs of retinovitreous adhesions and emphasized their importance along with subvitreal fluid in detachments.

Wadsworth in 1956 presented another paper on The Vitreous and Its Role in Detachment of the Retina. Wadsworth points out that vitreous changes noted by other authors were minimized or considered artifacts. He states that these vitreous changes are not artifacts, but they are important and fall into a general pattern depending on the age of the patient, history of eye trauma, or ocular diseases.

Shrinkage of the vitreous with posterior detachment occurs with age. (Pd Adsworth, 1952; Pische, 1953). Detachment of the vitreous is an extremely common finding in patients over fifty years of age. Wadsworth (1956) found shrinkage in seventy to seventy-five per cent of all the ages he examined above the age of sixty-five.

Vitreous detachment usually occurs at first in the superior portion of the vitreous then gradually spreads out from there. The space created by the vitreous detachment is filled with a fluid resembling that of aqueous. In the absence of retinovitreal adhesions vitreous shrinkage may cause no damaging affects to the eye. However, when vitreous detachment first occurs, the patient may be affected with a symptomatic manifestation of lightning flashes. The flashes are the result of floating vitreous striking against the retina with rapid movement of the eye. This symptom disappears with further shrinkage of the vitreous (Moore, 1935; Verhoef, 1941).

Age, ocular diseases or trauma can cause vitreal strands, retinovitreal adhesions, and vitreous shrinkage. The end result is a retinal detachment coming from the subvitreal fluid flowing through a tear in the sensory portion of the retina and dissecting the rods and cones from the pigment epithelium (Wadsworth, 1956; Teng, and Chi, 1957).

Schepens et. al., in 1960, stated that there is a definite union between normal vitreous and retina, but that it is destroyed by pathologic conditions

which produce liquifaction, separation, and contraction of the vitreous. For basis of this statement Schepens et al. use the observation that when a normal young eye is sectioned in half, retinal detachment invaribly occurs because the sensory portion of the retina stayes attached to the displaced portion of the vitreous. Also, they show specially stained photomicrographs of a fibrillary pattern which they claim demonstrates retinal vitreous union. At the present time these statements by Schepens et. al. are quite contraversial and are not generally accepted.

Retinal tears were first noted in the presence of retinal detachments in 1853 by Coccius. In 1863 von Graefe and Libreich individually noted their frequent occurance of this disease. This prompted von Graefe to postualte that they were the result of the detachment and part of the healing process. Von Graefe tried to heal detachments by causing more retinal tears so that the retina would settle out. In 1870 de Wecker emphasized the frequency of tears in spontaneous detachment and proposed they were the cause, not the result of the detachment. In 1882 Leber found breaks in the retina in 51 per

cent of 27 cases. At this time Leber postulated the theory of vitreous degeneration, liquidation, and retraction. In 1916 Leber found tears in 73 per cent of his cases of recent detachment; however, in those over two months duration he found tears in only 45 per cent. He contributed this difference to a tendency for the edges to heal; the atrophy of the retina makes the edges invisible as the time passes. With these findings Leber felt that "in every case of spontaneous and sudden detachment of the retina, a tear should be expected." Gonin found an incidence of 87 per cent in 1928 and 85 per cent in 1930 of retinal holes found with detachments. With the completion of his last survey Gonin stated that, "if sufficient time and trouble were spent looking for them, tears were almost invaribly found in spontaneous detachment." Arruga confirmed Gonin's statement by finding incidences of 90 per cent and 95 per cent in 1932 and 1935 respectively, in his recent cases of retinal detachment.

There are three types of breaks in the retina, round and oval holes, arrow-head and horse-shoe shaped rents, and dialyses.

Round holes are most frequently found.

Case reports and histologic examinations indicate that most of these holes are caused by "actual destruction of retinal tissue by peripheral retino-choroidal inflammation or degeneration." (Duke-Elder, 1940).

Arrow-head tears are thought to be the result of traction from an adherent vitreous on a weakened area of the retina. The majority of these tears occur in the upper half of the globe near the periphery where the vitreous has a stronger attachment to the retina and where movement of the vitreous has its greatest affect.

Dialyses is a tearing away of the retina at its attachments. Thus, it has anterior dialyses at the ora serrata and posterior dialyses at the optic disc. Anterior dialyses are the more common variety. Most dialyses occur at the infero-temporal quadrant in emmetropic young adults and are usually associated with some type of trauma to the head or eye.

The importance of retinal discontinuity was brought to light in 1916 when Gonin reported his first cures of retinal detachment by using chemocautery to seal the breaks in the retina.

This basic idea of repairing the hole or tear in the retina by coaugulation of the choroid is still the primary treatment for retinal detachments with breaks. Today the only improvements have been in technique and instruments; diathermy, light coagulation and Laser.

Even though the results of repairing retinal holes cannot be questioned, Duke-Elder in 1940 stated"..., but from many considerations, it would seem to be the case that the hole itself is not sufficient to cause the detachment: other factors, disease, degeneration, or severe trauma must operate as well." However, in 1952, Post stated, "It is our opinion that breaks in the continuity of the retina are the most essential factor in the production of retinal detachment and that the following may be the causes of such breaks: a localized atrophying process in the retina and a localized vitreous pull and trauma." Wadsworth (1952) also stated, "A break in the continuity of the retina is generally considered an all important factor in the development of the separation of the retina." Today, it is generally agreed by most authors that a retinal break is essential for all spontaneous retinal

detachments other than those produced by the exudative process.

According to Duke-Elder, Gonin (1934) reported a preponderance of retinal detachments in the Jewish race. He found a ratio of ten Jews to one of any other race. No other literature was found on the subject in this review to substantiate these findings.

There is a great deal of recent literature concerning the hereditary factors in retinal detachment. For many years heredity was a suspected factor in retinal detachment but not until the last thirty years has much work been done on the subject. Most of the important work has been done in the last fifteen years. The most recent work shows that heredity is responsible for many predisposing causes of retinal detachment, but not for the actual detachment per se'.

Sorby et.al. (1951, 1955) was the first to show that the predisposition for retinal detachment was hereditary and transmitted as a sex-linked recessive trait. In his pedigree, eight affected males appeared in three generations. In his report, Sorby stressed a wide range of clinical expressivity for predisposition

to retinal detachment. He included retinal cysts, vascular veils, pigmented high-water marks, pigmentary changes in the fundus sometimes taking the form of a pigmented central lesion, outlying white dots, and aborescent figures. Sorby relates any of these findings to an intermediate stage of detachment, and if they are followed the result will be extensive chorioretinal atrophy with exposure of sclerosed retinal vessels and retinal detachment.

Retinal detachment occuring from congenital retinal cysts has been suspected for many years. However, it wasn't until comparatively recent years that definite proof was reported. (Schapland, 1945; Duke-Elder, 1949).

Edmund (1961) reported a family where the males, who had severe myopia since birth, developed retinal degeneration. The degeneration started between the ages of fifteen and thirty-five. Once the degeneration had started it ran a rapid course and the patients had bilateral retinal detachments within six years. The degeneration may be due to myopia. However, other reports, as will follow, indicate that the degeneration is probably of hereditary origin.

Inherited retinal detachment in non-myopic males was reported by Levey in 1952. He described affected males in two different families. The first pedigree had seven affected males in five generations, presenting with the outstanding finding of vitreous veils and cystic retinal detachment. His second pedigree revealed three affected males in three generations whose essential findings were retinal detachment, hypermetropia, and macular degeneration. Levy points out that this predisposition is a sex-linked recessive disease with a predilection for the inferior temporal quadrant of the globe.

Retinoschisis, a splitting of the retinal layers derived from the inner layer of the optic, cup, which frequently results in retinal detachment is said to be a hereditary recessive disease affecting only males (Geiser, 1961; Delaney, 1963; Balian, 1960; Curtin, 1960; and Shea, 1960).

Other ocular conditions that are considered to be hereditary and are responsible for retinal detachments are vitreous degeneration (Condon, 1955) and peripheral retinal degeneration (Friedman, 1961; Delaney, 1963).

Age, sex, and myopia are three predisposing

factors that have been recognized for years. It is generally agreed that there is an increased incidence of detachment with an increase in age. Duke-Elder (1945) stated, "in the decade when senile changes usually begin to take place retinal detachments are most common." Schepens (1952) found in his large series a peak incidence in the fifty to sixty years age group.

The first reported study on incidence rate in the different sexes was done by Sattler (1905) in which he found males predominated by almost two to one over females. Most subsequent reports verify these findings. (Anderson, 1931; Duke-Elder, 1945).

The high incidence of myopia in retinal detachments has been noted since the beginning of recognition of retinal detachments. The prevalence of myopia was so great in early detachments that the distension theory was proposed to explain the cause of detachments. This theory was discussed earlier, and it was noted at that time that myopia was a prominent predisposing factor to retinal detachments. (von Graefe, 1857; Duke-Elder, 1945; Schepens, 1952).

Besides myopia the other earliest most common factor noted to be associated with retinal detachment

was trauma. Trauma of many varied types has been recorded to have precipitated retinal detachments. A direct blow to the eye, with and without puncturing it, has resulted in detachment. Trauma received to the body or to the head has been followed with detachment. Heavy lifting and straining has caused sudden detachments. A retinal detachment that occurred three years after an injury to the head was attributed to have caused the detachment. (Anderson, 1931; Mac Donald, 1935; Duke-Elder, 1945). In order for an indirect blow or non-piercing direct blow to cause retinal detachment, the retina must be already predisposed to detachment by the presence of vitreous shrinkage, vitreo-retinal adhesions, or a retinal break (Wadsworth, 1952; Teng and Chi, 1957).

Inflammatory lesions of the orbit and eyeball have been recorded as causing retinal detachment (Elwyn, 1953; Wadsworth, 1956.) Inflammation of the orbit or eyeball may affect the short cilary arteries or the venae corticosa resulting in inflammation and congestion of the chorio-capillaris with a resultant transudation and retinal detachment. Spontaneous reattachment usually occurs in this type of detachment as long as there is no retinal break.

Chorioretinitis, gross uveitis, scleritis, and retinitis have been reported to cause retinal detachments with retinal tears. These infections may cause retinal tears and detachment by producing vitreo-retinal adhesions and vitreous shrinkage. (Lewallen, 1957).

Reported bacterial infections are many and varied, ranging from the pyogenic bacteria to tuberculosis and syphilis, that have been responsible for retinal detachments. Also viral infections have been attributed to produce detachments, recent case reports include Herpes Zoster Ophthalmicus by Lincoff (1956) and Toxoplasmosis by Havener (1962).

Gross hemorrhages into the vitreous can cause retinal detachment. Detachments are caused most frequently when there are repeated hemorrhages. This results in organization of the blood and forming vitreo-retinal adhesions and finally resulting in a retinal break and detachment. Very rarely, hemorrhage breaking into the sub-retinal space may produce detachment. (Duke-Elder, 1945; Wadsworth, 1956).

Embryonic remains may cause retinal detachments. Remenants of the hyaloid system and failure of

complete formation of the iris and/or choroid leaving a coloboma have been reported to have caused detachment (Agarwal, 1954).

Certain systemic diseases are associated with retinal detachments. These include groups of infectious diseases, viral diseases, parasites, collagen diseases, vascular diseases, and hereditary diseases.

The infectious diseases have been already mentioned under the inflammatory lesions.

Some viral diseases have already been mentioned. Other diseases of suspected viral origin are Harada's disease, Vogt-Koyanagi Syndrome (Cowper, 1951) and Coat's disease (György, 1962). Duke-Elder suggested that Harada's disease was a mild form of the Vogt-Koynangi syndrome because of the similar clinical picture seen in these two diseases. The clinical picture associated with both of them is bilateral uveitis, alopecia, poliosis, vitiligo, and hearing defects usually in young people. The choroiditis seen in these becomes exudative and retinal detachment results. Coat's disease, which is also called external hemorrhagic retinitis or exudative retinopathy, is characterized with an external retinitis

hemorrhage which eventually hemorrhages into the vitreous and ending in retinal detachment.

Collagen diseases that have been reported to rarely cause or be associated with detachment include rheumatoid arthritis (Schreiber, 1920), lupus erythematosus (Maumenee, 1956) and periarteritis nodosa (Maumenee, 1956). The mechanism of detachment in his case uveitis and exudation. Whereas Maumenee attributed the detachment in the lupus erythematosis case due to straight exudation and in the polyartheritis nodosa case due to hemorrhage.

Retinal detachments resulting from renal retinopathy appear to be caused from the same mechanism seen in detachments from hypertension. Detachments in these diseases may occur from hemorrhage; however, they more often occur from exudation. Retinal detachment is not frequently seen in hypertension of renal diseases unless they are very severe. When they are seen it is frequently in the terminal stages of the disease. (Lewallen, 1957). The type and mechanism of detachment mentioned here is the same as that seen in diabetes mellitus. (Schlossman, 1956).

Toxemia of pregnancy is rarely complicated with

retinal detachment. Bosco (1961) reviewed 185,244 deliveries and found 10 cases of spontaneous non-traumatic retinal detachments. This is an incidence of 1/18,524, deliveries. However, in all the cases of detachment the patient had severe pre-eclampsia or eclampsia. In this series there was a ratio of 1 eclapmsia for every 911 normal pregnancy. Thus there were about 203 eclamptic patients, consequently in this series about five per cent of the toxemic patients developed retinal detachments. In all these patients the detachment was of the exudative type. And in all of these patients spontaneous reattachment occured within fourteen days following delivery.

The allergic phenomenon has been accused of causing bilateral exudative retinal detachments (Balyeat, 1937; Prewitt, 1937). It is conceded that detachment by this man may be possible, however, if it is so, one would expect to find more cases that could be attributed to this phenomenon.

One case of gout has been reported as a contributing factor to retinal detachment (Greef, 1929). However, no other similar cases have been reported, thus this one case may be the result of some other factor not

elicited in the report.

In summary, I have presented a review of the embryological formation and histology of the retina, with emphasis put on the potential space between the rods and cones and the pigment epithelium. A review of the theories of retinal detachment and their historical background was made. Emphasis being put on the traction theory and the present concept about it. The significance of retinal breaks were reviewed.

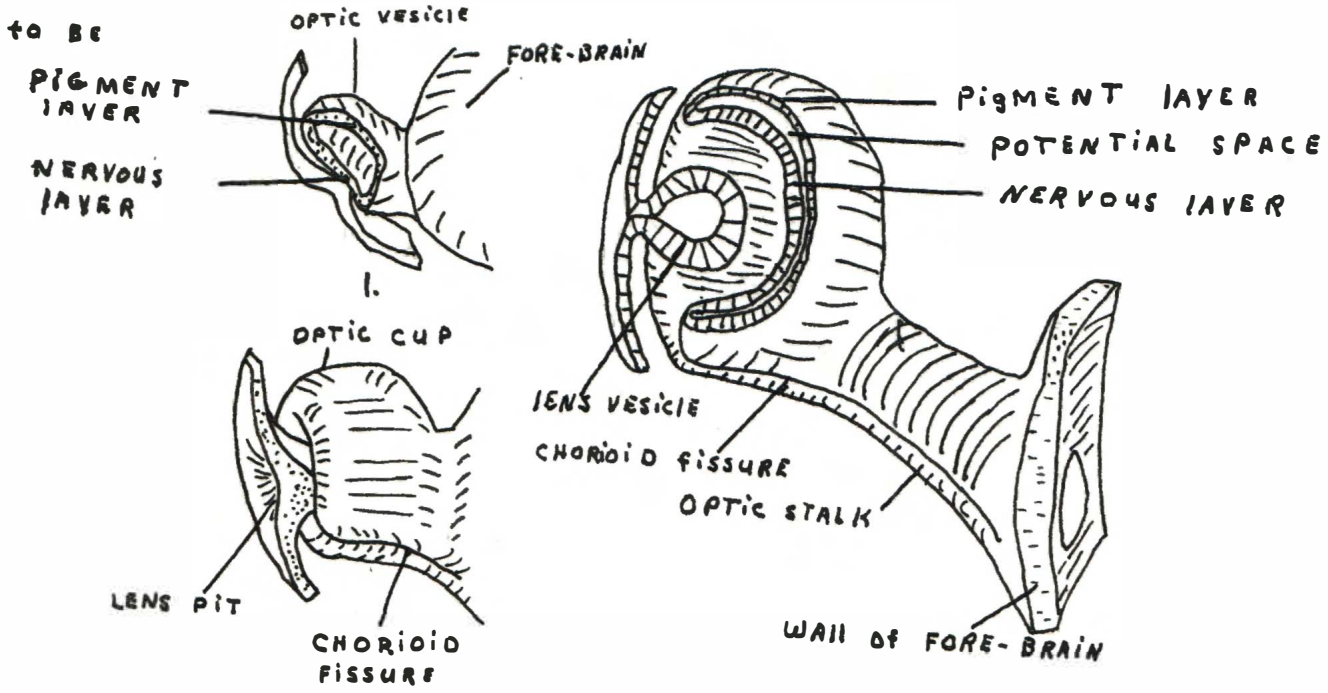
A review of recent literature was made on the subject of retinal detachment and its predisposing factors.

In conclusion of this paper, the following points are reiterated:

1. Retina detachment is actually intra-retinal separation.
2. Traction theory is the accepted theory.
3. Myopia, trauma, and hereditary retinal changes are the most significant predisposing causes.
4. Detachments are of two types, exudative without any retinal abnormality and retinal breaks.
5. Vitreous contraction and retino-vitreal adhesions are of primary importance to spontaneous detachment

by producing,

6. Retinal breaks which are always accompanied with some amount of retinal detachment.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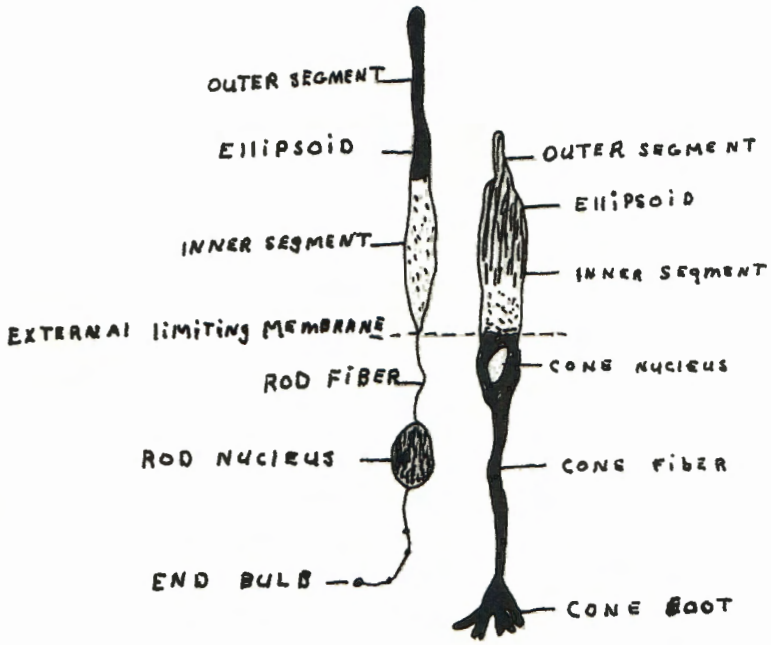
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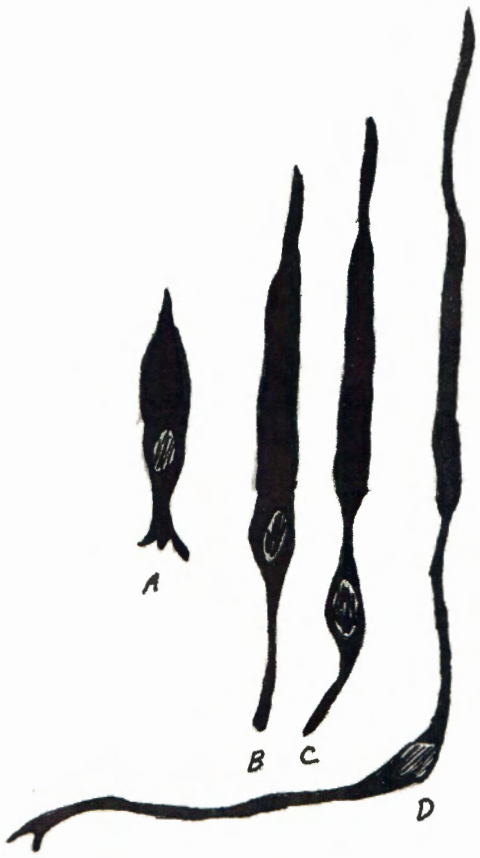
INNER SEGMENT
EXTERNAL LIMITING MEMBRANE
ROD FIBER
ROD NUCLEUS
END BULB
OUTER SEGMENT
ELLIPSOID
INNER SEGMENT
CONE NUCLEUS
CONE FIBER
CONE FOOT

ROD CONE

4.

Cones from different areas of the human retina. A, From near the ora serrata; B, from periphery of macula lutea; C, from the macula lutea; D, from the fovea centralis. (After Greeff.)



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