THE VITREOUS—SOME MODERN CONCEPTS

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The vitreous body consists of a transparent gel and a transparent framework made up of a reticular pattern of fine fibres.

It differs from the aqueous in that it contains collagen fibrils and larger quantities of protein and hyaluronic acid, less phosphate and sodium, but more potassium. It is more alkaline and is of a very high viscosity. Owing to the presence of cells, with nuclei, first discovered by Hannover in 1840, it cannot be quite as clear as the aqueous. These cells may act as phagocytes and help to produce hyaluronic acid, which is important in stabilizing the vitreous.

The fibrillary structure of the vitreous has been definitely confirmed by electron-microscopy and even by slit-lamp studies. With the electron-microscope, secondary systems, or membranules, or fibrils, can actually be seen entering the vitreous from the posterior vitreo-retinal limiting membrane. Such fibrils can also be seen extending from the plicata into the vitreous.

Anatomical Consideration

The hyaloid canal or Cloquet's canal, appears to be the most important structure of the vitreous, being its axis. It arises at the back of the lens, at Wieger's ligament, runs in a S-shaped manner through the centre of the vitreous to attach to the epipapillary material of the optic disc (Fig. 1). Bussaca has demonstrated that in some cases the funnel of the canal is prolonged as a filament to insert into the border of the physiological cup.

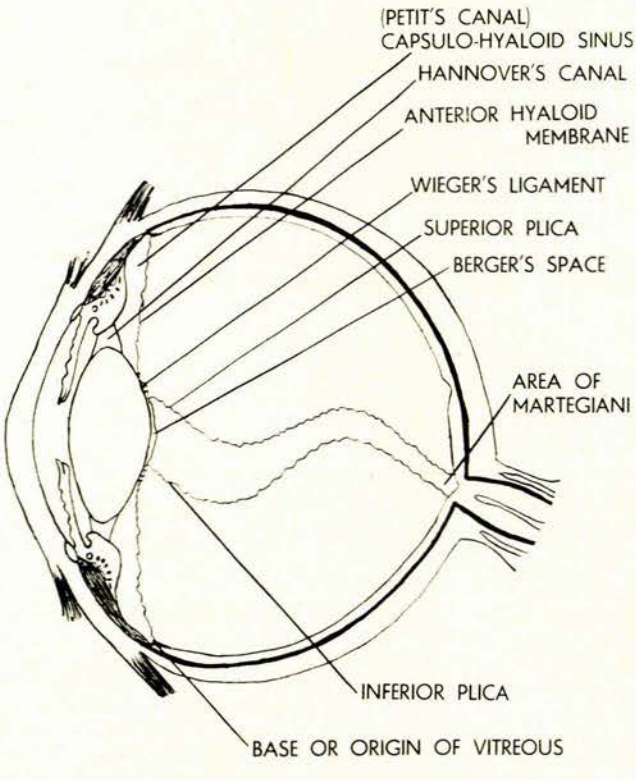

Fig. 1. Anatomical considerations.

As the individual ages, the transparency of this canal decreases, so that it may eventually appear very thin, yet its integrity is usually maintained. Haemorrhage, chalcosis and siderosis accentuate or outline Cloquet's canal, but this does not imply that it is otherwise invisible.

It is best studied in strongly hypermetropic people, such as aphakics, because of the lesser anteroposterior diameter of the eye. It is best seen and examined in the microzonuloscopic position. At an angle of 60°, move the microscope as close to the eye as possible; wait until the 'ascension phenomenon' comes to a complete rest; it is also necessary to have complete cooperation of the patient, who must maintain absolute rigid fixation, when this is required. In this microzonuloscopic position, we can see the canal of Cloquet even beyond the focal range of the microscope, occupying the middle portion of the field, so that it can be kept constantly under biomicroscopic examination. It can be seen bobbing to and fro like an air bubble in a leveller, never moving much beyond the field under observation. Its posterior third can be viewed with the Hruby lens and best in a red-free light.

The plicata represent the biomicroscopic outline of the canal of Cloquet. They are actually remnants of the walls of the embryonic hyaloid system. They are well seen when the anterior vitreous is well formed and the plicata thickened and luminous, but not when the vitreous is rarefied as in albinos, myopes and the aged.

All this applies also to the *ascension phenomenon* which is really the visible movements or gyrations of the anterior vitreous, the hyaloid canal and its walls or plicata, as observed with the slit-lamp in the microzonuloscopic position (**MZP**).

The anterior hyaloid membrane extends from the ora serrata to the posterior capsule of the lens, being attached circumferentially to a retrolental space 8 - 9 mm. in diameter, at the so-called Wieger's ligament. Here it fuses with the posterior group of zonular fibres, from which it is difficult to differentiate.

In front of the anterior hyaloid membrane and behind the cilary body and zonule is the capsulohyaloid sinus (or space), or Petit's canal.

The patellar fossa, or *retrolental space of Berger* is a potential space present only under pathological duress, between the posterior lens capsule and the underlying anterior hyaloid membrane. It is delimited by Wieger's ligament, and thus, that portion of the anterior hyaloid membrane which is contained within this circle, may be referred to as the patellar portion of the anterior hyaloid membrane. This separates the retrolental space from the canal of Cloquet.

Wieger's ligament. In 1883, Wieger described the occurrence of some degree of normal adhesion of the lens capsule to the hyaloid. He named it 'ligamentum hyaloideocapsulare'.

Reese and Wadsworth presented evidence that adherence between the posterior lens capsule and the anterior hyaloid occurs sufficiently in degree and frequency to be reckoned with, in performing an intracapsular lens extraction. It makes Kirby's sliding operation sometimes a hazardous procedure—'tumbling' is favoured in the USA because of this. The erysiphake seems to be of particular value in combating this adhesion syndrome. Possibly the forward movement of the posterior capsule when in the erysiphake, may help to free it from the vitreous adhesions.

Some have argued that extracapsular extraction is safest, but alphachymatrypsin (ACT) may be solving this problem.

The posterior hyaloid membrane (or vitreoretinal limiting membrane). Electron-microscopic studies by Gärtner and others, have shown the following: Between the cell membranes of Muller's marginal glial cells (internal limiting membrane of retina) and the basement membrane of the vitreous body (posterior hyaloid membrane) is a small potential space, which is occupied by the so-called 'cement substance of Pease'. By this,

the posterior vitreous adheres to the basement membranes of the retina. However, this substance does not exist in the papillary region and at the ora serrata—the origin or base of the vitreous. At these sites the posterior hyaloid membrane is firmly attached.

VITREOUS DETACHMENT

It is only in pathological states when the vitreous becomes detached from the retina, that the vitreous really acquires a posterior surface. This surface may then be termed a posterior hyaloid membrane. Clinically, identification of 2 surfaces, one at the posterior surface of the vitreous body and another at the inner surface of the retina, is indicative of some pathological process. Since, with age the vitreous tends to shrink and becomes detached posteriorly, it is difficult to assess the severity or significance of this pathological process.

The changes seen in the vitreous with advancing age are well described by Goldmann (Fig. 2). After 40, fibrous

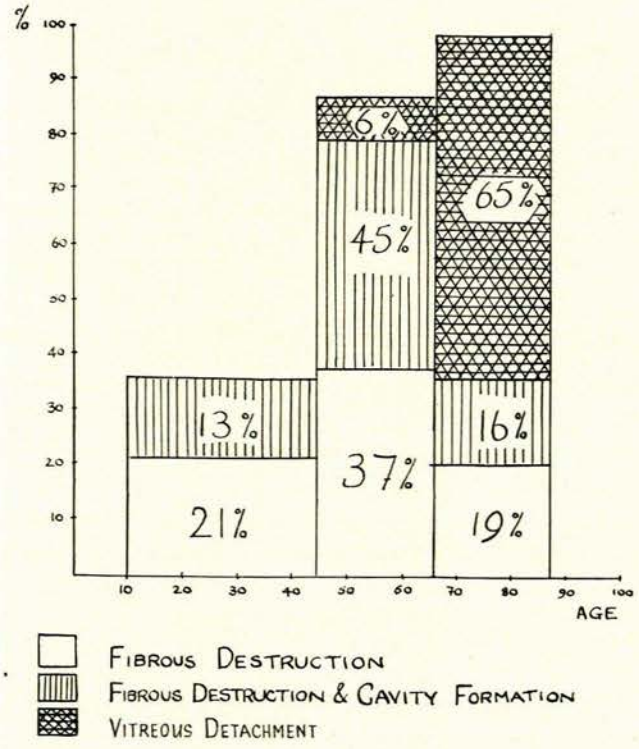

Fig. 2. Changes in the vitreous with advancing age.

elements become visible and the plicata less prominent. Later, between the newly-formed thicker fibres, which partly anastomose with each other, there appear optically-empty spaces, that is, spaces filled with fluid inside the vitreous gel. These spaces often become fairly large. With advancing age, more and more fibres and cavities appear, the walls of the hyaloid canal become less visible and finally, after the 65th year, there is usually a posterior vitreous detachment with collapse.

The patient has for some time seen threads when he looks at a white or bright surface. When he experiences lightning streaks or flashes and notices a large opacity in the temporal field, he then seeks medical advice.

Foster Moore, in 1935, reported many cases, mostly in middle-aged women, with the following symptoms—sudden onset of lightning streaks or flashes in the temporal field, with the simultaneous appearance of spots, 'flies', 'tadpoles', etc. before the eyes. The streaks are elicited only on movement of the eyes, and are vertical in direction and are only present towards the temporal side. Associated with the subjective symptoms is the simultaneous development of vitreous opacities.

In 1947, Moore reported the personal experiences of 3 ophthalmologists, Rutter Williamson, Professor Verhoeff and himself. He held the view that these lightning streaks have no sinister meaning, either at the time, or what is more important, ultimately.

F. H. Adler,[1] in 1959, wrote: 'According to Moore's description, they are seen only in the temporal field, and they are always vertical in direction. This, of course, eliminates the other type of similar entoptic phenomena, such as the flashes seen in the eye as the result of detachment of the vitreous.'

However, as we can easily see the detached membrane, we must accept (despite Adler) that Moore's lightning streaks are due to acute posterior detachment of the vitreous. These streaks are not noticed on the nasal side of the visual field, because the pars caeca retinae—the non-functioning blind periphery—is far larger on the temporal side of the retina.

These symptoms are due to the vitreous becoming detached. The lightning flashes are caused by the floating vitreous striking against the retina after rapid movement of the eye, causing stimuli that are interpreted as lightning streaks or flashes. Further shrinking of the vitreous will result in the disappearance of these annoying symptoms.

Usually one can see a circular opacity undulating on the posterior surface of the hyaloid; this is the separated attachment of the vitreous to the optic disc and means that the detachment is complete. Sometimes this ring is torn open to form a band from which fibrous opacities project.

Hruby's classification is generally accepted:

(*i*) Complete vitreous detachment
  (*a*) Simple vitreous detachment
  (*b*) Vitreous detachment with collapse in the normal vitreous framework.

(*ii*) Partial vitreous detachment is very rare and of little importance.

Simple complete posterior vitreous detachment is a separation of the posterior hyaloid membrane from the retina, together with a more or less regular retraction of the vitreous gel, which becomes a spherical segment of variable height. The vitreous framework is as a rule intact. The posterior hyaloid membrane shows discrete opacities, often ring-shaped, which is the cardinal ophthalmoscopic sign of posterior vitreous detachment. This simple type is generally encountered in younger persons and in non-myopic patients.

Complete posterior vitreous detachment with collapse of the vitreous structure occurs mostly in older persons and in myopes, with advanced cavity formation and fibrous destruction of the vitreous framework. The detached posterior hyaloid becomes visible behind the lens as a wavy membrane. Detachment of the vitreous is a common find-

ing after 50 years. Wadsworth found it in 75% of eyes of patients over 65 years. Hruby considers it usual after 65 years of age. Hauer and Barkay found posterior vitreous detachment in every one of 100 aphakic eyes, whereas in the same age-group, phakic eyes showed an incidence of 50 - 70%.

Is There a Relationship Between Vitreous and Retinal Detachment?

Goldmann estimates that there is only one retinal detachment to about 200 - 300 cases of vitreous detachment. He regards vitreous detachment as a change of senescence and almost 'physiological'.

However, posterior vitreous detachment is not a sign of health. It shows that the eye is less than perfect—that the intimate union between the vitreous and retina has been lost. In a previously healthy eye, detachment and shrinkage of the vitreous may have no damaging effect on the eye and adds but little to the prognostic significance. The passage of time has proved that these cases do *not* go on to retinal detachment.

As a result of inflammation, trauma, or haemorrhage vitreous strands and membranes may develop and adhere to the retina. Thus, in the presence of choroidoretinitis and intravitreal haemorrhage, it is possible for the vitreous to adhere to the pathological focus in the retina. Where there is such an isolated vitreoretinal adhesion to some part of the retina, the vitreous may be expected to exert its greatest traction upon the retina. This type of tugging is called 'vitreous shock' by Lindner. Movements of the eye cause jerk-like traction on the retina and may sometimes precipitate the occurrence of retinal tears, when the retina is atrophic. Here, I think, it is the underlying retinal disease and not the detached vitreous which is the real culprit. An interesting fact is that star-shaped folds of the retina are not sites of vitreoretinal adhesion, the vitreous is always detached at these sites.

Vitreous opacities. To understand and interpret these we must consider their nature, site and prognostic significance (Table I). Apart from embryonic remnants, invasion of the vitreous body by foreign bodies, parasites (*Taenia echinicoccus* and *Cysticerus cellulosae*) and connective tissue (retinitis proliferans), opacities may be related to inflammation, tumours, pigment deposits from cilary epithelium, systemic diseases (such as amyloidosis) and most commonly, to haemorrhage into the vitreous chamber. Physiochemical changes within the gel and vitreous framework produce opacities, quite apart from those that accompany posterior detachments. Consistency of position of an opacity suggests some association with the vitreous framework, whereas unlimited excursions of the opacity and the tendency to settle down at the bottom of the chamber, means that the vitreous gel has liquified.

Asteroid bodies are calcium-containing lipids, producing the well-known 'snowballs or stars on a clear night', appearance. Suspended in substantially normal vitreous, they do not gravitate to the bottom of the vitreous chamber, but return to their original position after limited excursions. This indicates an intimate association between vitreous fibrils and asteroid bodies. Rodman *et al.* suggest that these lipid bodies arise from some type of degeneration of the vitreous fibrils.

TABLE I. OPACITIES AND OTHER CHANGES IN THE VITREOUS

1. EMBRYONIC REMNANTS		Muscae volitantes Mittendorf's dot Bergmeister's papilla Clear cysts Persistent primary hyperplastic vitreous Vitreous veils
2. INVASION BY FOREIGN ELEMENTS	(*a*) *Exogenous*	Foreign bodies Parasites
	(*b*) *Endogenous*	Blood Exudates Pigment Connective tissue (retinitis proliferans) Cholesterol (synchysis scintillans) Calcium salts (asteroid bodies) Amyloid Glial tissue

3. ALTERATIONS IN VITREOUS STRUCTURE

(*a*) *Framework*	(*b*) *Gel*
(*i*) Fibrillary and membranous condensations (→ ascension phenomenon)	(*i*) Liquefaction or syneresis
(*ii*) Detachment of posterior hyaloid	(*ii*) Shrinkage (fluid separates from gel)
(*iii*) Senile degenerative changes (→ opacities)	(*iii*) Cavity formation
(*iv*) Lipin degeneration (→ asteroid bodies)	(*iv*) Other physicochemical changes (→ opacities)

Synchysis scintillans are really cholesterol crystals found in a fluid vitreous. These iridescent crystals move as a shower of glittering particles on a quick movement of the globe, but settle down to the floor of the vitreous chamber after movement has stopped. They occur in eyes that have suffered from long-standing uveitis or a vitreous haemorrhage. Most of the cases I have seen, have been in diabetics.

Retrolental haemorrhage is undoubtedly the most common and important cause of vitreous opacities (Fig. 3). It is necessary to diagnose the site of the haemorrhage (or opacity) and 4 clear sites can be identified.

SITES OF RETROLENTAL HAEMORRHAGES

(*i*) In front of the anterior hyaloid membrane, in the capsulo-hyaloid space, or Petit's canal. This means that the blood, pigment or exudates have come from the cilary body and have not invaded the true vitreous. This is also the route along which blood sometimes enters the lens, through some rupture in the posterior lens capsule. Blood in this space, tends to stay fluid and red for some time. Sometimes where inflammation is severe, exudates may organize to form a retrolental cyclitic membrane.

(*ii*) When the blood or opacity is behind the anterior hyaloid membrane but below the inferior plica, it is in the vitreous structure and this is always a serious matter. Blood stays fluid and cannot easily be absorbed. The haemoglobin of the blood causes the vitreous to liquefy. Haemolysis and phagocytosis of disintegrated erythrocytes take place, but some blood-pigment and cell debris remain. Sometimes a retinitis proliferans with newly-formed

vessels and connective tissue, enters the vitreous to dispose of the blood.

Such intravitreal haemorrhage arises from the retina, anywhere between the ora serrata and the edge of the optic disc. If the haemorrhage is small, fundal examination may show the source.

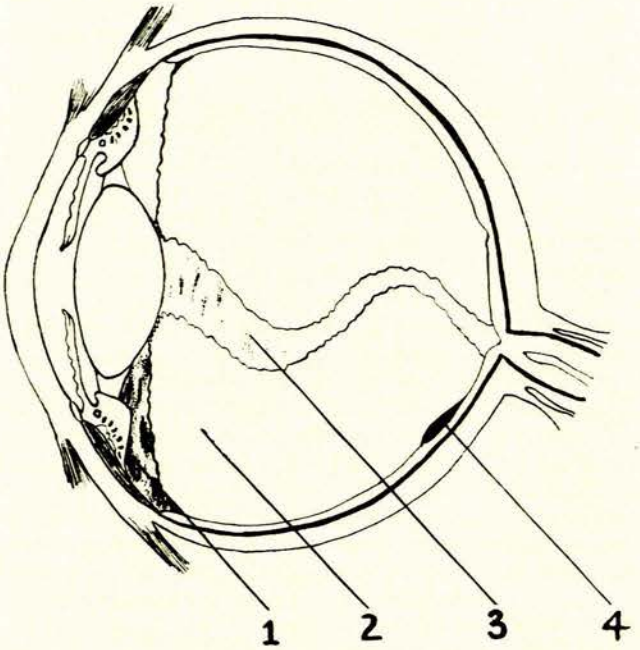

Fig. 3. Sites of retrolental haemorrhages:
1. In capsulo-hyaloid sinus—arising from ciliary body.
2. In vitreous framework—intravitreous—arising from retina anywhere between ora serrata and edge of optic disc.
3. In canal of Cloquet.
4. Subhyaloid—preretinal.

Diabetic or hypertensive retinopathy, traumatic retinitis, angiomatosis retinae, retinal vascular disease due to Eales' or Coats' disease, or retinal tears may be found. Although quite often a retinal hole is found, it must be admitted that in the vast majority of cases, retinal detachment does not occur. The bleeding is from a branch of the central retinal artery and since these lie in the nerve fibre and inner reticular layers of the ten-layered retina, it is not really surprising that retinal detachment does not often occur. Nonetheless, every patient past middle age, who has blood in the vitreous and particularly if he complains of flashes of light, must be meticulously examined for a retinal tear and be kept under surveillance until impending retinal detachment has been excluded or appropriate surgical treatment has been initiated.

According to Haessler, the haemorrhage that arises in a retinal tear, may be so great that it obscures the fundus, but it never obliterates the red, fundus reflex. When a haemorrhage is encountered that is so large that it obscures the fundus reflex, one need not fear retinal detachment. Such massive vitreous haemorrhage, usually due to trauma, may be recognized as such, only because no red reflex can be obtained in an eye with a clear lens.

After a few weeks, the vitreous opacities resulting from haemorrhage are difficult to differentiate from other vitreous opacities. They are black and tend to settle to the bottom of the vitreous chamber. After ocular movements, most do not return to their former form or position, as do the opacities accompanying vitreous detachment.

(*iii*) Cases have been reported of blood or exudates, within the hyaloid canal. This is behind the anterior hyaloid membrane and above the inferior plica.

This haemorrhage arises from any source, communicating with the physiological cup, along with any structure which may impinge upon and push through the canal walls. Cases have occurred of haemorrhage from a persistent hyaloid artery, also of exudation arising from the optic disc and occupying the area of Martegiani and travelling forwards up the canal.

(*iv*) Preretinal or subhyaloid in position. Large haemorrhages can clear up in a few days if the hyaloid is intact, but once the blood ruptures through the hyaloid into the vitreous, a serious position is produced. Blood and vitreous are incompatible and lead to lysis of both components.

Inflammation causes an increase in the albuminous content of the vitreous, giving a flare and the well-known haze seen in uveitis. In posterior uveitis, e.g. acute choroidoretinitis, fine, dust-like opacities are constantly seen, but if they appear early, then the inflammation is severe and will take months to subside. In anterior uveitis, the presence of dust-like opacities in the vitreous adumbrates a prolonged course and a less favourable prognosis. Opacities that occur with intraocular neoplasms have no special significance. However, Haessler[4] states that if dust-like opacities are so numerous, in the eye of a very young child, that they obscure the fundus, it may be a sign of retinoblastoma, because choroidoretinitis is rare in young children.

SUMMARY

A plea is made to recognize vitreous detachment as an entity and of no grave prognostic significance.

An attempt has been made to elucidate the problem of vitreous opacities.

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