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Etiology of glaucoma

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ETIOLOGY OF GLAUCOMA

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PART I.

Introduction and History.

The term "glaucoma" is not the title of any one single disease but is a clinical name for a large group of pathological conditions with a complex of symptoms. The distinctive feature common to all is a rise in the intra-ocular pressure.

There seem to be many varied causes of this condition, and the pathological findings differ greatly in each case. Therefore, it is a difficult problem to unite these dissimilar manifestations into one common factor which may cause this rise in the intra-pressure.

The hardness of the glaucomatous eye, or its rise in tension, merely refers to the outward manifestations of an increase of the fluid pressure within the globe. All the causes of glaucoma lead up to this increase in pressure.

This subject covers a multitude of theories as to the origin of the fluid within the eye, and also many reasons as to the cause of this increase in pressure. This paper will be mainly a discussion of the etiological factors involved in the production of this condition. In order that I may adequately accomplish this end, it will be necessary to describe the anatomical details of the structures concerned; something of the physiology of the eye;

and, the pathological anatomy in reference to its aid in causing the condition.

Before beginning the discussion at hand, it may be of some interest to the reader to review some of the landmarks in the history of glaucoma.

Over four centuries before the Christian era, Hippocrates mentioned and described glaukos as among the known affections of the eye. The Greek word "glaukos" was used to describe the disease, because he saw a gray green reflex from the pupil. The word means "gleaming" or "bluish gray."

The Greeks and Latins, following Hippocrates, spoke of those causes which gave rise to blindness and were unaided or unrelieved by operation as glaucomata.

The discovery of the ophthalmoscope in 1851 by Helmholtz aided in the recognition of glaucoma. Adolph Weber soon followed with a clinical discovery of the cupping of the disc. Heinrich Muller confirmed this by his studies of the pathological anatomy of the eyeball. Much earlier than this (1830), MacKenzie noticed the hardness of glaucomatous eyes, and he maintained that this hardness was due to overfilling of the globe. About this time, Von Graefe discovered the venous and arterial pulse in the retinal vessels, and observed this was dependent on the increase of the intra-ocular pressure.

Bowman, recognizing the value of these discoveries, examined a number of hard, normal and soft eyes, and

introduced the finger-tip method of taking the ocular tension.

Leber, by his classic physiologic research, established the secretion of the intra-ocular fluid by the ciliary body. Following this lead, and working independently, Max Knies and Adolph Weber, simultaneously discovered the important fact that the angle of the anterior chamber was closed by adhesions of the iris base to the cornea, in cases of glaucoma. Priestley Smith published these valuable discoveries and improved them by a lifetime of very careful research.

Since this time, many of the preceding theories have been approved and disproved until the present day. The concepts and progress of modern ophthalmologists shall be discussed further in the body of this paper.

PART II.

Anatomy and Physiology.

Before the processes of disease in an organ can be studied, one must know something of the anatomical details of the structures concerned.

ANATOMY. It is known that the sensory nerves of the body are provided with end organs, by means of which they receive specific physical stimuli and transform them into nerve impulses. The eye is the highly differentiated and complex organ of the sense of sight.

The wall of the globe is composed of a dense, elastic supporting membrane. The anterior portion, which is transparent, is called the cornea; the remainder is opaque, and is called the sclera. The anterior portion of the sclera is covered by mucous membrane or conjunctiva, which is reflected from its surface on to the lids.

The cornea is composed of three layers: the epithelium, the substantia propria and Descemet's membrane. The epithelium is stratified, and is regarded as the continuation over the cornea proper. It lies upon a homogeneous lamina of the substantia propria, or Bowman's membrane. The substantia propria is regarded as the continuation forward of the sclera. Descemet's membrane is a thin elastic membrane, covered on its posterior surface by endothelium; it is regarded as the continuation forward of the uveal tract.

The cornea is set into the sclera somewhat like a watch glass, i. e., the sclera overlaps the cornea around the periphery. The cornea is devoid of blood supply with the exception of a few minute festoons at the periphery, so it must be dependent for its nourishment upon diffusion of lymph, which is supplied by the conjunctival vessels.

An outer membrane lining the sclera is chiefly concerned in the nutrition of the eye and it comprises the majority of the uveal tract; an inner membrane, of nervous origin, is concerned in the reception and transformation of light stimuli, and is called the retina.

The uveal tract consists of three parts. The two posterior, consisting of the choroid and ciliary body, line the sclera, while the anterior portion forms a free circular diaphragm, the iris. The aperture of the diaphragm is the pupil. Behind the iris is the crystalline lens.

The anterior chamber is a space filled with lymph, the aqueous humour; bounded anteriorly by the cornea, posteriorly by the iris and a portion of the lens. The sclera forms part of the boundaries of the anterior chamber at the part known as the angle of the chamber. A network of venous spaces lies in the inner layers of this portion of the sclera and is known as the canal of Schlemm. At the periphery, just anterior to the canal of Schlemm, Descemet's membrane splits up into fibrillae, which are

continuous with a meshwork of fibers stretching between the sclera and the iris; this is known as the cribriform (pectinate) ligament. The tissue separating the cribriform ligament from the canal of Schlemm is quite dense. There is no free communication between the anterior chamber and this venous plexus, as a thin membrane is interposed which is covered on each surface by endothelium.

The angle of the chamber is quite a narrow space in the normal eye. Since the outflow of the aqueous fluid is said to take place here, it is most necessary that the angle remain open as widely as possible. Its potency may be infringed on in several ways:

(a). Extreme dilatation of the pupil will crowd the membrane out to its periphery, thus filling the angle with an impediment of fluid passage.

(b). The ciliary body may become swollen by venous congestion, which will push the apices forward. The apices will press upon the base of the iris, and push it against the cornea, closing the angle. Also, if the apices of the ciliary body move forward, the attachments of the suspensory ligament of the lens do likewise, the lens advances and presses on the base of the iris.

(c). It is stated in the literature that as life advances, the lens enlarges while the tunic of the eye remains stationary in size. The enlarged lens will tend to press the iris forward, and thus occlude the angle.

(d). When the iris and cornea are approximated, as just described, their adjacent surfaces may become adhered together by the exudate thrown out, thus forming synechia and a permanent closure of the angle.

The uveal tract, previously mentioned, consists of the ciliary body, the iris and the choroid. From anterior to posterior, they lie (1) iris, (2) ciliary body, and (3) choroid.

The iris is composed of a stroma, consisting of branched connective tissue cells, usually pigmented, but not pigmented in cases where the iris is blue. On the anterior surface of this membrane, pit-like depressions are found, called the crypts of the iris. These crypts are most numerous at the ciliary border, and lead to the depth of the iris stroma, thus placing its tissue spaces in direct communication with the anterior chamber. In this stroma the fluid comes into intimate contact with the thin-walled iris veins. The direct communication with the anterior chamber is probably a device for insuring rapid transference of lymph from the iris to the anterior chamber and vice versa, so as to facilitate quick movements of the pupil in response to variations to light intensity. The crypts are of importance when the angle of the chamber is in an early stage of closure, as the fluid from the chamber can enter through them and find its way along the iris stroma to the neighborhood of the cribriform

ligament, across which it then passes to enter the canal of Schlemm.

The ciliary body and its adjoining parts are considered as the "cock-pit" of glaucoma, because it is said that here the fluid of the anterior chamber originates. There is some dispute as to whether the cells actually secrete or whether it is a transudate from the capillaries in the neighborhood. The generally accepted view is that the lining cells of the ciliary body have the power of taking up fluid transuded around the cells from the capillary vessels, and passing it across to the free surface by a definite act of secretion.

It is thought that the fluid thus poured out passes out by two streams, one backward into the vitreous, the other forward into the posterior chamber, then it moves forward through the pupil into the anterior chamber, continuing outward through the anterior chamber to reach the angle.

The vessels and nerves which supply the ciliary body and iris pass forward between the choroid and the sclera. Here they are exposed to the force of the intra-ocular pressure, as they lie against the hard unyielding scleral coat. Perforating vessels which pierce the sclera just behind the cornea form a free communication between the vascular system in the interior of the eye and that on the exterior.

The choroidal and retinal circulation lies between

the fluid contents of the eye and the unyielding sclera, therefore it suffers when the pressure within the eye increases. The arterial and venous circulations seem to react differently. The pressure diminishes the amount of blood entering the eye through the arteries and also diminishes the amount leaving the eye through the veins. The result would be to establish a venous congestion. If the pressure rises slowly, the circulation will be able to adapt itself to the change of conditions and the glaucoma will remain simple. If, on the other hand, there is a rapid increase in pressure, the adaptation is impossible and there will result an attack of congestive glaucoma. It seems quite probable that the anatomical conditions, governing the escape of the venous blood from the eye and those of the reservoirs into which this blood is passed immediately after its exit, exercise a definite influence on the incidence of the vascular factor when high tension supervenes.

According to Textbooks of Ophthalmology, there are two quite separate vascular systems within the eye. One supplies the retina and the retrobulbar segment of the optic nerve, the other is responsible for the uveal tract, the sclera and some of the episcleral tissue.

The uveal tract is supplied by the ciliary arteries, namely, the short posterior, the long posterior and the anterior. The short posterior are the most important

channels for the uveal tract and consist of about twenty vessels. The other two branches are merely contributaries to the short posterior.

The ciliary veins also form three groups, the short posterior ciliary, the venae vorticosae, and the anterior ciliary. The venae vorticosae, being the most important, are made up by the fusion of a number of capillaries which meet in a vortex, and in turn form the large vortices. The ciliary processes are almost entirely made up of branches of veins. Even though there seemingly is a large number of anastomoses between the anterior and posterior venous systems, the suppression of either leads to a marked venous stasis of the eye (35).

The blood, lymph and aqueous humour make up the fluids of the eye. The aqueous differs from the other two in that it contains very little albumin and a high percentage of sodium chloride, while the others contain much albumin and less salt. The origin of the aqueous has been a subject of great discussion for many years, and at present is still debatable. In the discussion on the physiology of the eye, I shall endeavor to explain a few of the modern concepts of this question.

The scleral coat is perforated for the passage of the second cranial nerve into the globe. The hole is not cleanly punched but is partially blocked by a fibrous membrane, the lamina cribrosa, which is a thinned out portion of the scleral tissue. Therefore, it is evident that this is the

weakest part of the wall of the eye, and consequently, it is the first part to yield under a rise of intra-ocular pressure. Further description of the anatomy of the "entrance of the optic nerve" need not be considered in reference to the subject of "Etiology of Glaucoma."

PHYSIOLOGY. If the eye must satisfactorily perform its function as an organ of vision, it is quite essential that a clear-cut image of objects on the outside shall be formed upon the retina. This is accomplished by means of curved surfaces; the curvature of these and their relative positions to each other must be kept constant. To attain this end, the walls of the globe must be held tense.

Parsons found that by inserting a canula, connected to a mercury manometer, into the anterior chamber, that the mercury would rise about 25 mm. This shows that the contents of the eyeball, mostly fluid, exerts a pressure on the inner walls of the globe which is about 25 mm. of mercury greater than the atmospheric pressure on the outside of the globe, thus the walls are kept stretched.

This pressure is called the intra-ocular pressure, or tension, of the eye. In order that the tension be maintained, the organism must do work, and the evident source of energy is the blood pressure. By experimenting with animals, it has been proven that the intra-ocular pressure follows passively the changes in the general blood pressure.

The aqueous fills the anterior and posterior chambers,

and permeates the vitreous (36) (26). Many writers compare it with the lymph in the other parts of the body, where its function is to carry food to the tissue cells and carry away the waste products of metabolism. Another function is the maintenance of normal tissue tension. Both functions play an important role in the eye. It is known that the whole of the lens and the vitreous have no blood supply, therefore they must be dependent upon the lymph for their nourishment; and the necessity for maintaining the normal tension of the eye has already been shown.

Due to the fact that the lymph is nutritional, it must be removed constantly in order that the waste products may be carried away and a fresh supply of nourishment brought to the cells. Therefore, a circulation of lymph through the eye is evident, if not, the tissue cells would die due to the deleterious effect of the excreted products of the cells.

It is an established fact that the lymph in other parts of the body is formed by filtration out of the capillaries, the amount being dependent upon the difference in pressure on the two sides of the capillary wall (a filtering membrane). In the eye the intracapillary pressure is higher than in other parts of the body, and the extracapillary pressure is also higher than usual, i. e., the intra-ocular pressure. The difference between the two pressures is, however, less in the eye than elsewhere in

the body. Because of this, the amount of lymph produced in a given time is small, and too, its chemical composition will differ from the other lymph of the body. It is probable that the osmotic conditions, under which lymph and aqueous are formed, play more of a part in this process than previously thought. By some the fluid is regarded as a dialysate. Duke-Elder (5) claims that the aqueous is not a secretion, but on the other hand, is a dialysate of the capillary blood, formed by the same processes as the other tissue fluids, the only difference being that the process is modified by the relative impermeability of the ocular capillaries in order to allow the physiological requirements of the eye to be met. In this article (5), he claims it is a physico-chemical process, an essence of which follows: A dialysate in equilibrium with its parent fluid must have a precise and definite chemical composition, it must have a very definite osmotic pressure, a definite reaction, a definite electrical potential, and there must be a definite relationship between the hydrostatic pressure at which it is maintained and that of the parent fluid. Duke-Elder gives considerable proof in his article for this statement, but even so, other ophthalmologists do not accept his views as yet. In spite of the fact that the conditions of the production of aqueous are not quite consistent with the view that it is a simple process of filtration, it is still generally spoken of as a

secretion. Even if the process is essentially a filtration, one would not think that membranes covered with living endothelial and epithelial cells would act as dead inert membranes. It has been shown by various authors that certain substances, such as, agglutinin and precipitin, pass freely, others do not pass into the aqueous. Therefore, Parsons (36) and others feel justified to use the term secretion.

Most ophthalmologists have found by ciliary body experiments and with their clinical and pathological observations that the ciliary processes are the chief, if not the only, site of the production of the aqueous in the human eye. They found that if they remove the iris and ciliary body, as can be done in the rabbit without immediate destruction of the eye, aqueous formation ceases and the eye becomes soft and shrinks. This effect was not due to the absence of the iris because of the fact the aqueous is normally secreted when the iris is almost completely absent as a congenital defect. Therefore, the conclusion is that aqueous is secreted by the ciliary processes.

The majority of the lymph thus produced is said to pass into the posterior chamber. As has already been stated, it then passes forward through the pupil into the anterior chamber, which communication is made possible by the continual movements of the iris. A small percent of the aqueous passes backwards into the vitreous, which it nourishes. A

small tubular lymph space is found in the vitreous, running forwards from the optic disc. The lymph of the vitreous reaching this space, which communicates with the perivascular lymph spaces which surround the central vessels of the retina. By this route the lymph of the vitreous passes out. Now that only a small part of the lymph secreted by the ciliary body passes out by this route, a more adequate drainage must be present to care for that in the anterior chamber. An old theory was that it transuded through the cornea and escaped into the conjunctival sac. This has been disproven and it is at present known that it filters away through the angle of the anterior chamber into the venous plexus, or canal of Schlemm. Having entered the canal of Schlemm, the worn-out lymph is carried away by the anterior ciliary veins.

Parsons(36) states, "Owing to the difference in temperature between the iris and the cornea, convection currents are set up in the aqueous, the fluid moving upwards in front of the iris and downwards behind the cornea. These currents can be seen with the slit-lamp, when, as is often the case, the aqueous contains particles in suspension."

The choroid is quite a vascular structure and some writers think it is concerned in the production of lymph. It is probable that it only produces enough lymph to nourish its own tissues and the outer layers of the retina.

Because the retinal capillaries do not extend deeply into the retina, the rods and cones and the pigment epithelium are dependent upon the choroid for their nourishment. The choroidal lymph passes out of the eye by way of perivascular spaces around the vorticosse veins, part of it passes into the subchoroidal space and into the lymph sheaths of the posterior ciliary vessels. The lymph of the retina passes away by perivascular lymph sheaths along with that of the vitreous. The choroid and retina must only produce sufficient lymph for their own requirements, and so do not contribute to the maintenance of intra-ocular tension.

One might think the vascular iris provides part of the aqueous of the eye, but as has been shown, the iris may be concerned with the process of absorption and it is unlikely that it would carry on such opposite functions.

Therefore, it will be considered that the aqueous is secreted by the ciliary body and the point of exit is at the angle of the chamber and then into the canal of Schlemm.

PART III.

Etiology of Glaucoma.

In order to give an adequate discussion on the etiology of glaucoma, it is necessary to include the pathology of the structures as involved in this disease.

In 1886, Birnbacher and Czermak found changes in the vortex veins in patients with glaucomatous eyes. The vessels and adjoining lymph spaces showed cellular infiltration, with a proliferation of the endothelium of the veins. Their contentions, according to Elliot (8), were that the changes lead to venous obstruction, followed by congestion of the surrounding areas, and thus increase the intra-ocular pressure. Koster (27) summarized all the works of those who preceded him and then cited his own observations, which follow: When all four veins were ligatured, the tension would increase immediately; the pupil dilated, the anterior chamber became shallow; the vessels of the iris were engorged with blood; and there was a marked swelling of the ciliary body. He noticed that after three weeks time, the eyes appeared normal with the exception that the iris remained adherent to the margin of the cornea. It is quite possible that the obstruction of these large veins might be a determining factor in the outset of a case of glaucoma which had been started by other changes. Modern literature hardly mentions this factor in

the production of glaucoma, therefore no more space shall be used for discussion of it as a definite cause.

Disease of the vessels of the eye has long been considered as a possible cause of glaucoma. Such suggestions were based on the fact that venous congestion is very often found in glaucoma. Many writers speak of it as "a vascular disease", although they do not give proof for such a statement. Almost three-fourths of a century ago, Von Graefe (49) wrote: "The great distention of the retinal capillaries is also, in my opinion, caused by the escape of the venous blood being impeded through the increased pressure." To this statement no proof was brought forth and just by mere supposition one could not definitely say that increased intra-ocular pressure is due to "vascular disease", and consequently it does not bear sufficient evidence to consider it further.

Schnabel (45), in 1892, brought forth the idea that the cupping of the glaucomatous disc was due, not to an increase of intra-ocular pressure, but to an atrophy of the nerve itself. He observed microscopic holes in that portion of the nerve which lies within the scleral canal. These holes, by coalescence and enlargement, led to the formation of a "cavernous degeneration" (as he described it). With progression of the condition, the spaces became larger until a single huge cavity remained. This cavity was the cupped disc. Not long after Schnabel put forth

his views, other ophthalmologists observed that cavernous degeneration of the optic nerve was found in eyes which had never been involved by an increase in intra-ocular pressure; too, no signs of it were found in many glaucomatous cases. Other evidence against this view is the fact that the glaucomatous cup lessens in depth and often disappears following a successful reduction of ocular tension by iridectomy. Holth (24) has shown that Von Graefe was the first to observe a transitory leveling of a glaucoma cup, after a successful iridectomy. Present day ophthalmologists have observed and confirmed the same phenomenon. Schnabel was considerably wrong when he advanced the view that the cupping of the disc was the result of changes within the nerve, without the aid of increased pressure. It is now positively known that the cupping of the disc in glaucoma is the result of a prolonged attack of increased intra-ocular pressure. Although his views were wrong, his work was still of value because attention was drawn to cases which had a continuance of nerve atrophy after the apparent cause had been removed.

Ischikawa (25) examined ten glaucomatous eyes and found Schnabel's "caverns" in five cases of recent origin, but none in the other five which were chronic. He also claimed that the caverns were characteristic of glaucoma and they formed a specific class of disease of the optic nerve. He stated that the condition began as an edema of the nerve fibers, which were later dissolved, leaving the caverns.

The glaucomatous cup was due to the coalescence and disappearance of the caverns.

The main fault with Schnabel's theory was that he just assumed that an increase of intra-ocular pressure had nothing to do with the cupping of the disc. Such assumption is of very little value in the collection of facts with proof as is needed to arrive at any conclusions on this subject.

T. Henderson (19) put forth the view that a structural change in the cribriform ligament obstructed the aqueous in reaching Schlemm's canal. The main points in his discussion were as follows:

(1) The cribriform ligament is a continuation of the inner lamellae of the cornea; which ligament is composed of regular and evenly disposed intermingling fibers and ends as the ligament of origin of the ciliary muscle.

(2) The boundaries of the angle of the anterior chamber are cribriform ligament anteriorly, iris root posteriorly, thus excluding the ciliary body from being in direct relation with the anterior chamber.

(3) The cribriform ligament, purely cellular at birth, undergoes sclerotic changes as life advances, and in old age, its fibers resemble those of the adjacent sclera.

(4) The ligament is merely a regular and open network of interlacing fibers, which continue directly with the longitudinal and circular bundles of the sclera around

the venous sinus of Schlemm's canal (8) (5).

(5) The spaces of the cribriform ligament are directly continuous with those between the corneal lamellae, and the ligament is considered as part of the corneo-scleral envelope.

(6) The results of sclerosis of this ligament are to reduce the contrast between the cribriform ligament and corneo-sclera to one of size of fibers only, and to reduce the interspaces and alveoli of the retiform tissue. This does not affect the thickness of the ligament which lies between the anterior chamber and Schlemm's canal.

(7) On purely an anatomical basis, a free communication is postulated between the suprachoroidal space and anterior chamber, along the posterior portion of the cribriform ligaments.

(8) Sclerosis of the ligament makes a mechanical obstruction, which predisposes to glaucoma by impeding the access of aqueous to Schlemm's canal.

(9) He assumes, also, on an anatomical basis, that the action of the ciliary muscle in accommodation facilitates the escape of the aqueous into the suprachoroidal space, thus producing a physiological deepening of the anterior chamber.

It is quite true that this sclerosis of the cribriform ligament may act as an obstacle to the escape of aqueous from the eye, if other conditions remain constant, and so

predispose the eye to a rise in tension. Henderson seemed to have the opinion that this was the chief of the predisposing anatomical causes of glaucoma. According to him, the fibrosis is universal; but on the contrary, it is known that glaucoma is not a very common disease, although the tendency increases with age. The literature shows that there is not always a rise of intra-ocular pressure as age advances, as would be expected if these fibrotic changes actually oppose an increasing resistance to the passage of aqueous from the eye. Most of the present evidence is that it is decidedly lower in the elderly than in young adults. PriestleySmith (43) pointed out that Henderson's theory does not account for the shallowing of the anterior chamber found in so many glaucoma cases, nor does he account for the characteristic closure of the filtration angle (angle of the anterior chamber). From Henderson's theory one may safely admit that the sclerosis of the cribriform ligament may well be one of the predisposing causes of glaucoma for it tends to upset the normal balance between the secretion and excretion of fluid from the eye. One can be positive that it is not the only cause.

Thomson (47) described the cribriform ligament as an open meshwork of fibro-elastic tissue, which he traced forward into direct continuity with the homogeneous elastic layer of Descemet. By tracing Descemet's membrane posteriorly, he found that it broke up into a number of fibrillae arranged in brush-like fashion (8). He also contended that

the spaces within the tissue were lined by extensions from the layer of endothelial cells, which covers the posterior aspect of Descemet's membrane. It is said that the majority of fibers of this ligament are attached to the anterior portion of the scleral process; the meridional division of the ciliary muscle inserts into the posterior surface of the scleral spur. Thomson, by his experiments and anatomical observation, evolved the following theory: "The escape of fluid from the anterior chamber into the canal of Schlemm, and from there into the neighboring vessels, is the result of a pumping action, the muscles of the ciliary body and iris draw back the scleral process and thus the canal of Schlemm and the trabecular spaces of the cribriform ligament are opened. The elasticity of the ligament and of the scleral spur tends to close the spaces and canal, and so drive the aqueous fluid into the surrounding veins." It is easily understood that an encroachment on the spaces of the cribriform ligament may be an important factor, but it is quite likely that a lack of elasticity in these thickened fibers may impede the usefulness of the parts as to the function just ascribed to them. That is, the pump action, whose force is furnished by the muscles of the ciliary body and iris, may be impaired in contraction and rebound by a change which would damage the elasticity and extensibility of this ligament. In this way, a predisposition to glaucoma may be established.

Fischer (9) has made an extensive study on the production of edema by the action of acids and alkalies on the colloidal tissues of the body. From his findings, he applied the principles as an explanation of the cause of glaucoma. He described the condition as an edema of the eyeball (10) in which the hydrophilic colloids of the eye hold an increased amount of water. He made no effort to distinguish which structures were involved but supposed the whole globe took part in the change. He thought the cause of the hydrophilism was an increase in acid content of the organ and that the existing causes of an attack of increased tension were those which might be liable to an abnormal production or accumulation of acid in the eye. According to this theory, all parts of the globe are edematous in glaucoma. Modern texts mention nothing of a swelling of the ocular tunic of the eye in these cases. From this I conclude that this has been disproven by later experimentors, or else it would be considered as a possible cause of glaucoma by authors of the present day.

Now the closure of the filtration angle must be considered. The function of this angle as a filtration area was first demonstrated by Leber in 1873; Knies and Weber (1876), each working independently on this principle, discovered simultaneously the frequency with which the angle of the anterior chamber was obstructed in glaucomatous eyes. Priestley Smith (43), after careful research and

experimentation, expressed the following ideas of the closure of the filtration angle:

(a) In the early stages, the closure of this angle is caused by the pressing of the base of the iris against the periphery of the cornea. At the onset, the closure is simply mechanical. The surfaces are in opposition, escape of aqueous is impeded although the fluid from the aqueous chamber can pass along the meshes of iris stroma into the loose structure of the cribriform ligament and then into Schlemm's canal. According to this writer, this is the condition which exists in the early stages of glaucoma, and up to a somewhat later stage in simple glaucoma.

(b) The opposed surfaces of iris base and corneal periphery become adherent to each other because of a plastic exudation thrown out between the two membranes (36). This is said to occur very early in acute cases, more slowly in the subacute and only after a considerable lapse of time in the simple glaucoma. It seems to be just a matter of time for the closure of the filtration angle, as it occurs sooner or later in all cases of unrelieved primary glaucomas. The adhesions first formed may be very peripheral and may not extend around the entire circumference of the eye. As time goes on, the belt of adhesion widens and becomes firmer and firmer, while the whole periphery of the anterior chamber eventually becomes involved, (8).

(c) It is believed that a further stage is reached when the iris tissue is not only in contact with and sealed to the periphery of the cornea, but is also firmly compressed against it in such a way as to occlude the spaces which normally allow the free passage of fluid through the iris stroma. Fuchs (11) pointed out, that so long as the iris tissue remains normal in structure, the fluid of the anterior chamber can find its way freely along the loose meshwork of its stroma, and thus pass from it into and through the cribriform ligament. In the cases studied by Fuchs, the determining factor of the onset of glaucoma was the state of the iris, which "looked normal or even a little distended in cases with normal tension, while it was compressed and atrophied in cases with excess of pressure" (11). He considered the atrophic change to be the result of the increased tension rather than the cause.

(d) Atrophy of the iris tissue - This stage is reached much earlier in the acute and subacute forms of glaucoma than in the simple chronic form. It is merely the result of interference with the normal circulation through the part.

(e) The final stage is a drawing back of the iris when the ciliary processes retract; this process may be so extreme as to cause the iris to become greatly stretched or torn.

According to Elliot (8), the closure of the filtration

angle varies with the form of glaucoma. It is produced in each form in the following manner:

(1) In congestive glaucoma, the ciliary processes are enlarged, due to the increased supply of blood to the parts, and their apices extend farther forward than normal. In this way, they come in contact with the iris anteriorly and sometimes with the lens margin internally, and so may acquire a wedge-like shape from compression between these two structures. The processes become thickened transversely, with the result that the spaces between them are narrowed or obliterated (1). This explains the pushing forward of the base of the iris which occurs early in congestive cases, and is accompanied by a corresponding shallowing of the anterior chamber. In more simple terms, the congestion of the ciliary processes leads to their distention or swelling; the result is a forward and inward displacement of their apices. This carries the iris directly forward and tends to join its base against the periphery of the cornea (36); and, at the same time, it causes an advancement of the lens which aids to shallow the anterior chamber.

(2) In simple glaucoma, the forward displacement of the iris is the result of the enlargement and advance of the lens, which takes place as life progresses. In the early stages, these changes do not cause any serious compression of the iris; fluid can still gain access to the cribriform ligament through the iris crypts. Later, as the tension

increases, the area of iris contact becomes larger, the channels in the iris gradually become compressed and closed, and the surfaces become adherent to each other (38) (36) (5).

Summarizing, one may say: That all forms of glaucoma are eventually brought about by a closure of the filtration angle; this closure in congestive cases is due to a swelling of the ciliary processes, and, in simple cases to an alteration in the size and position of the lens.

Shallowing of the anterior chamber of the eye is met with sooner or later in all cases of glaucoma. This condition is closely associated with the closure of the angle of the chamber. Priestley Smith, in a personal communication with Elliot (8), put forth the following views on the "Shallowing of the Anterior Chamber of the Eye":

"In primary glaucoma the anterior chamber is usually shallow. In the main this is a pre-existing and predisposing condition -- a cause than rather a consequence -- as provided by the fact that persons suffering as yet in one eye only, usually have a shallow chamber in both. It is a physiological condition proper to the life-period in which primary glaucoma commonly arises. The explanation lies, in part at least, in the continuous growth of the lens (36). That the chamber is shallower in some eyes than in others at the same time of life may be explained by the fact that lenses belonging to the same life-period differ considerably

not only in total bulk but in the ratio of their axial and transverse diameters; some are relatively thick but small transversely, others thin and wide.

"Another possible cause for the senile shallowing of the chamber, and for individual differences, is this: -- The lens is held in position chiefly by innumerable cords which pass at various angles from its peripheral zone to the ciliary processes, and, as experiments have shown, when the processes advance the lens advances with them. Now the processes alter in size and shape with the advance of life; they are more prominent and bulky in the old than in the young; moreover, individual eyes belonging to the same life-period present great differences in this respect.

"It is likely that these changes in the processes involve change in the position of the lens.

"But the physiological shallowing of the chamber is only half the question. In acute congestive glaucoma the chamber becomes shallower during the attack. The iris no doubt is partly responsible; it becomes thicker as the pupil dilates; it may thicken still more through venous hyperemia; its periphery is carried forward by the swollen processes. But this is not all. The chamber becomes shallower in the area of the pupil; the lens moves forward. Why is this? High pressure in the chamber does not explain it unless we assume an excess of vitreous over aqueous pressure.

"We know that a slight excess of this kind suffices to displace the lens and processes, to close the filtration angle, and therewith to arrest escape of fluid from the eye. A likely cause for such excess in acute glaucoma is found in the condition of the circumlental space. In elderly people the space between the lens and processes is narrowed by the increased volume of the lens (36); it is further narrowed, sometimes in a marked degree, by the increased bulk of the processes. The grooves between the processes become narrower for the same reason (Hess) (23); during an acute glaucoma the processes are turgid with blood (18) and have been found jammed against each other laterally and against the lens. Under these conditions it is likely that vitreous fluid is unable to enter the aqueous chamber - that it is imprisoned behind the lens and drives it forward.

"When the ciliary processes (in the rabbit) are rendered turgid by ligaturing the vortex veins, their apices advance and the lens advances with them. The same result has been obtained by cauterizing the limbus of the eye externally, and when only one-half of the circle was cauterized the swelling of the processes was limited to this half, and the lens was tilted forward at this side only. These observations suggest that when the processes become turgid and advance, the lens is carried forward by reason of its attachment to them.

"In any case they show that extreme swelling of the processes even in a healthy eye (of a rabbit) shallows the chamber, closes the filtration angle, compresses the base of the iris, and arrests the escape of fluid from the chambers, so as to produce an exaggerated picture of acute glaucoma."

The dimensions of the eye as seen in glaucomatous patients as seen by a number of ophthalmologists may be summarized as follows:

(1) The eyes of women, on the average, are a little smaller than those of men; this is in correspondence with the relative size of the bodies in the two sexes.

(2) The eyes of glaucomatous persons are, on the average, a little smaller than those of the non-glaucomatous.

(3) Smallness of the eye is not always a constant factor in primary glaucoma, but is a determining factor in a large number of cases.

(4) It is due to defective growth, and not to senile shrinkage of the eye.

(5) The percentage of very small corneas is much greater in the glaucomatous than in those with healthy eyes.

(6) The decisive factor in determining the onset of glaucoma is not the absolute size of the eye, but its size relative to that of the lens it contains.

Thomson (47) found that in the full term fetus, the

cornea amounts to about one-fifth of the circumference of the globe on section; its transverse diameter measured about 10.6 mm. in the specimens examined. According to this in comparison with Priestley Smith's measurements (cornea 11.17 mm.) of adult eyes, there is little increase in the size of the eye after birth. Priestley Smith (43) also states that the cornea increases little, if any, in its diameters after the age of five, and possibly diminishes after the age of forty. The sclera is probably fully grown somewhat before the rest of the body. Since these two structures determine the size of the eye, the growth of the globe must cease before that of the body generally. The lens, on the other hand, continues to increase in size up to an advanced period of life (8)(12) (28) (36).

Priestley Smith stated (43), "The salient fact is that while the lens grows larger as life advances, the globe does not, so that the older the eye becomes, the more likely it is to suffer from the disproportion in question; and the smaller it is, the earlier in life is the disproportion likely to arise." An enlargement of the lens tends to push the iris base into contact with the periphery of the cornea, a tendency which will obviously be exaggerated in eyes with a corneal periphery. Thus the consequences would be:

- (1) A mechanical closure of the angle of the chamber, leading to an obstruction to the free flow of fluid towards

the canal of Schlemm, and

(2) A compression of the iris tissue, resulting in a still more serious interference with the excretory currents of the eye.

In a very extensive article on the etiology of glaucoma, Duke-Elder (5) explained glaucoma as merely a pressure symptom. His conclusions were, that there was a derangement of the capillary circulation involving a capillary dilatation, which produces a rise in capillary pressure, or an increased permeability of their walls allowing an excess of colloids in the fluids of the eye; also, there were changes in the vitreous which are physico-chemical in nature. He admits that little is known about either but perhaps it may serve as a lead for further investigation to obtain an actual explanation of the condition. In reviewing the literature, I have been unable to find other writers with this view.

It is probable that the changes just discussed are active in the causation of all cases of glaucoma, but the changes in the size or position of the lens seems to be most evident in the chronic non-congestive forms. In the congestive attacks they are overshadowed by the evidence of the vascular upset, while in simple cases they are not so obscured.

The remainder of my paper shall deal with the accepted causes of glaucoma, but one must still consider that a

pathological excess of ocular tension may be determined either by (a) an interference with the balance normally held between secretion and excretion in the eye, or (b) by an obstruction to the escape of blood from the eye, or (c) by a combination of (a) and (b).

AGE. As stated by Priestley Smith (43), "The frequency of glaucoma increases slowly at first, more rapidly later in each decade, until about the sixtieth year. Between sixty and seventy, it is about as frequent as between fifty and sixty. After seventy its frequency diminishes." His conclusions were based on approximately a thousand cases, which were scattered through the practice of a large number of surgeons. Haag, analyzing a series of 1032 cases of primary glaucoma, found that 4 occurred in the first decade of life, 16 in the second, 26 in the third, 74 in the fourth, 176 in the fifth, 288 in the sixth, 329 in the seventh, 116 in the eighth, and 3 later. Thus it is evident that advance of life bears a strong causal relationship to glaucoma, but we must consider just how the advance of life causes glaucoma. There are many views but according to the late literature, the following ideas are presented:

(a) Advancing age may act by causing alterations in the anatomical condition of the parts, such as, enlargement of the lens, slackening of the zonule of the lens, allowing an advance of its anterior surface, or of its whole

bulk, and thickening of the fibers which constitute the cribriform ligament. The manner in which these factors operate has already been discussed so no further mention of them shall be made.

(b) Next in order is the influence of degenerative changes. This includes an increase in the volume of fluid secreted by the eye, as a result of (a) nervous irritation of secretory fibers (35), (b) degeneration of glandular elements, or (c) alterations in the vaso-motor conditions present; also an alteration in the nature of the secretion poured out by the ciliary body. (5).

The view that an increase in the amount of fluid poured into the eye might bring about a pathological rise in tension, was first revealed by Von Graefe and Donders. Von Graefe (49) attributed the excess of fluid so produced to a serous choroiditis, while Donders (3) thought that an irritation of the secretory nerves was to blame. Priestley Smith (43) made the following statement: "Over-production of the intra-ocular fluid is at least a possible cause of chronic glaucoma, as suggested long ago by Donders." About this time, Bjerrum (2) advanced the view that glaucoma arises from an inflammatory process, which affects the ciliary body and leads to hyper-secretion from that structure. Kuemmell (29) supported these views two years later. Elliot (8) was impressed by a particular case in his own practice. A very good trephining operation was complicated

by an injury to the patient's eye from the hat of a lady visitor. The chamber filled with blood, which soon cleared, but, in spite of the quite good filtration, the tension remained high. He stated that there were probably other features in the case, but he states that it was difficult to exclude the possibility that an increased secretion had resulted from the injury.

After reviewing the literature and considering the facts both pro and con as to the secretory function of the ciliary body, the conclusion arrived at has been that the production of the intra-ocular fluid is probably to be regarded as a combination of pressure filtration (5) and active secretion (8).

In support of the suggestion that degenerative changes in the ciliary body may lead to excessive exudation in the chambers of the eye, Priestley Smith compared it to the kidney in chronic nephritis. He had maintained that even though fresh channels of excretion may be opened as a result of the increased force of secretion, the result will inevitably be a rise in intra-ocular pressure, due to the fact that the fluid is secreted under pressure higher than normal.

As to the influence of alterations in vaso-motor control within the eye, it has been shown experimentally that irritation of the fifth nerve or of the sympathetic may be productive of a marked increase in intra-ocular

pressure (15). This has been attributed to the influence of local vaso-motor dilatation. Nervous conditions seem to have a powerful influence on the progress of an attack of glaucoma. Sleeplessness, anxiety, fatigue, hunger and other facts may be determining events in the onset of an attack of glaucoma, or may convert a simple into a congestive form of the disease. Slight injuries to the head also may cause an attack of glaucoma. Considering that a tendency to a loss of vaso-motor control is a constant accompaniment of the advance of life, this phenomena is easily explained. It is merely a deduction, but the physiological basis would seem to be well founded and reliable.

An alteration in the nature of the secretion poured into the eye has been submitted as one of the possible causes of primary glaucoma. It is generally assumed that such a change in the chemical constitution of the aqueous is a powerful factor in the production of increased tension, secondary to chronic and subacute forms of iridocyclitis (8) (5) (36).

Priestley Smith (43) gave the following hypothesis: "Chemically the aqueous and vitreous fluids are nearly, but not quite, identical, and they both proceed from the ciliary body; but, they are probably produced by different zones, the aqueous by the processes, the more sluggish vitreous by the non-plicate zone. It is likely that senile degeneration may attack these zones separately or unequally, and

that vitiation or overproduction of the vitreous fluid may occur with normal production of the aqueous. In the final stage of chronic glaucoma there is certainly sometimes undue retention of fluid in the vitreous chamber, for the aqueous chamber becomes inordinately shallow, and the vitreous, if examined in the bisected eye, shows signs of serous infiltration (8), but here again one must beware of mistaking consequences of high pressure for initial causes." He supports this view by citing some experiments made by Wessely, who, by injections into the vitreous, obtained an atrophy of the retina and choroid, with little involvement of the ciliary processes. The vitreous body was totally abolished while the production of the aqueous was apparently unaffected.

These ideas seem interesting but they appear to be purely speculative. Therefore, it is impossible to make any definite assertions as to which view is to be accepted in that they seem to be ever-changing.

(c) It has been suggested that glaucoma is caused by a pathological tendency to hydrophilism on the part of the part of the tissues of the eye. This tendency is supposed to be brought about by an overloading of the tissues with acid products of metabolism (9). It is assumed that the tissues swell with the increased absorption of water, thus we might suppose that the swelling of the tissues might impede the exit of the intra-ocular fluid by narrowing

the channels of excretion, and also, it might obstruct the circulation by constricting the blood vessels that pass in or out of the eye; and in that case, the veins would be compressed first and more so than the arteries, thus a venous congestion would be set up. It is also known that the elimination of waste products from the tissues becomes more difficult as life progresses; and so, as Fischer (9) has claimed, the advance of years would predominate this factor.

(d) During the past few years, much attention has been directed toward the influence of the secretions of the ductless glands (29) on the problems of health and disease, and likewise there have been some suggestions that glaucoma is caused or influenced by changes in the quantity or quality of these secretions. With all the possibilities ascribed to these secretions, we are unable to discredit any supposition made in that respect because of inadequate knowledge at present. Hertel (22) believed that the osmotic ebb and flow of the body fluids, inclusive of those of the aqueous and vitreous chambers, is influenced by the thyroid gland; he contends that certain phases of the secretion of this gland may predispose to the occurrence of hypertension in the eye. Both Magitot (35) and Goldenburg (14) state that the knowledge of the influence of the endocrine glands in glaucoma is very rudimentary, not only because of the interdependence of the glands themselves, but also on

account of their relations with the nervous system.

Such suggestions are purely speculative at present, as no proof is offered; while in future years, these secretions may be proven to be the cause or a factor in the cause of glaucoma.

(e) As life advances, many extensive changes occur in the vascular system. These, undoubtedly, have considerable bearing on the causation of glaucoma. From physiological possibilities, it suggests that a pathological rise in the intra-ocular pressure may be due to one or more of the following factors: (1) An increase of the vascular pressure in the body generally; (2) A similar increase in the eye; (3) A lowering of the vaso-motor control in the body generally; and (4) A similar change in the eye especially (15).

It is true that arteriosclerosis, like glaucoma, is a disease whose occurrence is much more frequent as life advances. A number of years ago, it was thought that arteriosclerosis was a factor in the causation of glaucoma, but in the past few years, a number of investigators have shown that high arterial tension (present in arteriosclerosis) is not necessary and apparently not even a leading factor in the etiology of glaucoma.

As to the influence of an increase of vascular pressure localized in the vessels of the eye, although it may have its influence, present methods of examination do not afford

adequate proof of its regular occurrence (6).

Now we must consider the loss of vaso-motor control in the body generally and in the eye especially. It is generally accepted that interference with the normal circulation through the eye is a leading feature of congestive glaucoma. It has also been previously stated in this paper that vascular disturbances play an important part in simple cases as well. Most of the exciting causes of glaucoma act by interfering in some way with the vaso-motor control. Causes of disturbances of the vaso-motor control are: cold, hunger, starvation, sleeplessness, nervous or bodily exhaustion, anxiety, sorrow, constipation, bronchitis, cardiac or hepatic obstruction, etc. Therefore, one or several of these factors, acting upon the vaso-motor control, may precipitate a potential case of glaucoma.

SEX. More than thirty years ago, Priestley Smith stated that females suffer in rather larger numbers than males; his ratio was 56.9 per cent. against 43.1 per cent. Kagoshima from Japan, found the percentages in that country to be 61.9 for women and 39.9 for men. It is generally considered that the chronic, non-congestive cases are slightly higher in the males as compared with females, while the congestive forms are nearly twice as common in females as in males (32) (36) (8). The unstable condition of the female nervous system at the time of the menopause is probably sufficient to account for this great difference. Some of this difference may be

accounted for by the fact that perhaps a large number of potential cases of glaucoma in the male never pass the boundary line so that they are manifested by subjective signs and symptoms. Too, some cases may remain simple in nature and are thus overlooked. Therefore, the greater liability of females to congestive glaucoma appears to be due to a wider and more frequent prevalence of the exciting factors in them as compared with the predisposing factors of the disease. This leads to the conclusion that eye affections of a painful or congestive type in elderly females should always be carefully examined and watched, while failing vision, without an obvious cause to account for it, in either sex, should invariably be watched with suspicion, until a definite cause can be assigned for the trouble.

HEREDITY. The influence of heredity on the incidence of glaucoma is found both in the race and in the family. It is usually stated that Jews (36), Egyptians and certain races of negroes are especially prone to the disease. Laquerer (30) pointed out that it was not accidental, that of three ophthalmologists who had suffered from glaucoma, two of them, himself and Javal, were Jews. The various possible modes of action of hereditary influence are: (1) A small size of the eye; (2) A want of normal development, especially of the parts around the angle of the chamber; (3) Any condition which would predispose to nervous or vascular storms; and (4) conditions of environment (8) (19) (44) (36).

ERRORS IN REFRACTION. It has long been recognized that hyperopia is the most common refractive error met with in eyes affected with primary glaucoma. Fuchs (11) made the following statement: "A disposition toward inflammatory glaucoma appears to belong principally to hypermetropic eyes, whereas strongly myopic eyes are to be regarded as having almost complete immunity against the disease." Priestley Smith (43) challenged the correctness of this statement on the grounds that, according to Donders (3), emmetropic eyes become hyperopic after the fiftieth year; small eyes are not necessarily hyperopic; hyperopic eyes are not necessarily small; and hyperopia is more widely prevalent than myopia or even than emmetropia. These conclusions were drawn by Priestley Smith following extensive research of statistics. Elliot (8) found, as the result of clinical observation, that glaucomatous eyes are frequently abnormally small, and that the type of eye one commonly meets with in hyperopia is likewise small. Although he does not have a statistical foundation for his views, he contends that they are hard to reject because they are so closely woven into his clinical experience.

Gilbert (13), from the statistics of the Munich clinic, found that of 71 cases of simple glaucoma, 62 per cent. were myopic or emmetropic, and 38 per cent. hyperopic; whereas of 115 cases of congestive glaucoma, 77 per cent. were hyperopic, the remainder being equally divided between

myopes and emmetropes. He thought the clinical form of the disease to be largely influenced by the state of refraction of the eye. Many variations in these percentages have been found by various ophthalmologists throughout the world, because of this, a definite conclusion is wanting. It is obvious that what we really need is a mass of reliable statistics of the state of refraction in earlier life of those who subsequently develop glaucoma. It would be difficult to obtain such figures from the out-patient records of hospitals, or from the private practice of any one man. If such figures were obtainable, there would possibly be some doubt, since it would be extremely difficult to calculate with any degree of accuracy the percentage ratios of emmetropes, myopes and hyperopes in the class of patients from whom the returns come. Regardless of what has been said, statistics show this one thing, that the myope is not safe from hypertension, even though his myopia may be comparatively high.

MYDRIASIS. Anything that causes a dilatation of the pupil tends to block up the angle of the chamber, and thus causing an obstruction to the outflow of intra-ocular fluid, to raise the tension of the eye. The causes of dangerous mydriasis may be divided into a number of headings: (1) The abuse of drugs; (2) The exclusion of light from the eye; (3) The influence of violent emotions; and (4) the effects of depressing diseases or of allied conditions.

The principal one of these four to be considered here is "the abuse of drugs." There is NO drug which produces mydriasis, which can safely be instilled into an eye pre-disposed to glaucoma, unless the surgeon takes the precaution to neutralize the effect by the subsequent use of a miotic. Cases are on record in which an excess of hypertension has followed the use of each of the following drugs, holocaine, cocaine, euphthalmin and homatropin.

There are two important points in connection with the use of mydriatics which should never be forgotten, viz: that a mydriatic should never be used without first ascertaining the tension of the eye and the condition of the optic disc; and that no doubtful patient should be allowed to leave the surgeon's care until miosis has been established. Even then, the patient should be provided with a prescription for a mild miotic to be used at intervals until all danger is past. The reason for the last statement is, that often miosis is evident shortly after an instillation, but may gradually pass away, giving place to mydriasis, which may last for several hours or even for several days. Therefore, it is necessary to warn the patient to watch his pupils and to instill the miotic, if necessary.

NERVE SHOCK AND STRAIN. It is generally known by ophthalmologists that any condition which leads to powerful mental excitement, or to exhaustion of the mind or body, is an exciting cause in persons whose eye is anatomically

predisposed to the disease (33) (8) (36). Many of these patients give a history of family bereavement, of haunting fear, of business anxieties, of long fatiguing and anxious night watches, of prolonged mental overwork, of sleeplessness, or of some similar condition or set of conditions, all of which are manifestations of nerve exhaustion. Many cases have been reported where the second eye becomes glaucomatous while the patient is in the hospital for operation of the other eye. This may be due to some one or more tendencies to ocular congestion and the influence of mydriasis. The former has previously been discussed. The latter may be caused either by strong, mental impressions, by nervous exhaustion, or possibly by the shutting off of light from the eyes by means of the bandage (5) (36) (8).

FEBRILE DISEASES. If nerve shock and strain are accepted as possible etiologic factors of glaucoma, it is quite obvious that disease should from time to time be encountered after various febrile diseases. The weakening of the heart's action and the impairment of the vasomotor tone which is common in such conditions, explain how this is possible. In many infective diseases, the influence of the invading organisms is often found far distant from their recognized areas of activity, and therefore, is it not quite possible that the eye should sometimes be attacked by these expeditionary forces of the disease? Bacterial or toxic conditions introduced into the system by this canal

may seriously affect either the vascular arrangements of the eye, or the integrity and efficiency of the excretory passages. The secretory activity of the ciliary body may be pathologically augmented in certain cases, or the chemical composition of the excreted fluid may be altered so as to form a barrier to its free excretion (5). Also these morbid conditions may favor the presumed state of hydrophiliam upon which Fischer (9) and his followers laid so much stress. The foregoing statements are merely deductions which are quite possible, but as yet, have not been definitely proven.

INJURIES. It is a well known fact that an injury of the eye or of the head may be the determining cause of an attack of glaucoma. This does not have reference to cases of secondary glaucoma in which some coarse lesion of one or other of the ocular structures can be demonstrated, as these cases are readily explainable. It is quite a difficult matter to explain why a blow on the head, not necessarily a severe one, should excite an attack of high tension, or why glaucoma should result from a slight abrasion, burn or other injury of the cornea or of the limbal region. Very little has been worked out regarding the explanation of these phenomena. It has been suggested by Elliot and others that we have to deal with an interference, probably vaso-motor in nature, with the normal circulation of the eye. So little is known about the

nervous mechanism of the intra-ocular circulation that the above suggestion is merely a guess.

PART IV.

Conclusion.

In the foregoing discussion, it has been attempted to put forth the most important factors concerned in the etiology of primary glaucoma, with only an occasional mention of the secondary causes. Of course, the various theories are quite confusing because of the unceasing change of ideas as to the physiology of the eye and chemistry of its fluids. At present, the aqueous has been accepted as a secretion of the ciliary body. According to Duke-Elder, evidence seems to be accumulating that the aqueous is not a secretion but a dialysate of the blood. If so, the vascular disturbances would be of considerable importance in the formation of the fluid and in the production of glaucoma. The nervous influence on ocular-tension, surely present, has its effect on the vessels of the eye, especially the capillaries. Evidence is accumulating that blood chemistry and the chemistry of the aqueous have considerable effect upon the production of glaucoma. At present it is quite an open question. Swelling of the vitreous may be found to be an important factor. Low grade inflammation is probably a factor in many cases, even of apparently simple glaucoma, producing venous stasis or by increasing capillary permeability.

The endocrines are being investigated for their

influence in glaucoma. There seems to be no end in the number of theories as to the cause of glaucoma, and in the final analysis, it will probably be found that each case has many factors in its origin. Inflammation, secretion, excretion, mechanics and general nervous elements all may have their part in the cause.

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