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CHRONIC INTERSTITIAL OPHTHALMITIS.
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By S. O. Richey, M. D.,
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THE reader of the literature of glaucoma must be impressed with the regional consideration given to the subject by all writers, since the announcement of Von Graefe's high-tension theory. The whole process, and its explanation, have been confined to the ocular structures alone. In 1892,[1] the writer attempted to show *why* glaucoma should be regarded as a local expression of a diathesis. One of the ablest advocates of the retention theory (Knies), of increased intra-ocular tension, Priestley Smith, thinks "*that the high tension depends more upon an excess of blood in the eye than upon an excess of intra-ocular fluid.*" In favor of this is the *rapid* advancement of the lens and iris in acute glaucoma; the engorgement of the venal system; and the pulsation of the retinal arteries, intensified by slight pressure upon the globe; thus showing increased general arterial tension, accentuated by the added resistence to the entrance of blood into the eye.

High tension of the bulb is not glaucoma, but with the other symptoms of glaucoma, is a localized expression of a general affection, which is aggravated by violent emotion, by shock, by excess of food and indigestion, by hunger, by loss of sleep, by the climacteric, by constipation, and by palpitation of the heart; just such disturbances as aggravate this disease

[1] *Trans. American Ophthal. Society*, 1892.

expressed elsewhere in the system. Such passing conditions do not *cause* glaucoma, but they precipitate the attacks when a predisposition already exists, and the *modifications* of tension can be more readily understood, attributed to this factor, than when referred to either of the most prominent theories of high tension, that of hypersecretion, and that of retention, of the intra-ocular fluids, to each of which fatal objections exist. For, the mechanical resistance of the eyeball excludes the possibility of hypersecretion beyond the point at which the two would balance each other; and this objection stands equally against the theory of retention, as the secretion of fluids can not be continued beyond a certain fullness of the globe, which is reached more quickly if the secretions are retained. Also, the channels of excretion are often found open after the highest degree of tension, which takes place in the acute form of glaucoma, when the heart's action is usually excitedly strong, in its effort to overcome spasmodic contraction of the remote arterioles; a condition shown by the pallor of the face, followed by dilatation of the vessels and congestion of the face and head, with slight protrusion of the eyeball, and some edema of the lids, the condition in which we usually first see the cases.

The bilateral character of glaucoma must be remembered, for in nearly all cases, sooner or later, both eyes are affected in this way. Attention has already been called to its close resemblance to another local expression of the same constitutional malady in another extremity of the body, of which high tension is as constant a peculiarity, and which is accepted as due to turgescence of the blood vessels. Von Graefe believed all forms of glaucoma to have one cause, which he judged to be local, increased intra-ocular tension; by which the attempt has been made to account for all the other phases of the disease, local and general, symptomatic and anatomic; hence, much of the confusion.

Corneal opacity, which has by no means been ignored, as the reports of the vast majority of cases mention it, has not had due prominence, nor the importance attached to it, which its constant presence deserves. It is, perhaps, more constant than increased intra-ocular tension, which it often precedes;

and, in such instances can no more be due to increased tension than can disc excavation in a case in which tension has never been above normal.

To take a lesson from the affection, and begin at the beginning, is a course which is simple and logical. Donders indicated the beginning when he declared chronic simple glaucoma to be *the type* of the disease. The other forms of glaucoma have the same origin, the cause acting with a varying degree of force or virulence. Chronic simple glaucoma, the primary and uncomplicated form, is the initial localization of the process, from which arise all other forms and complications. It may be so slight that its known symptoms are not recognizable, for the disease has often developed beyond the first stage, lacking most of the diagnostic symptoms, until an added impulse has declared them; and yet, in nearly every case, there is an antecedent history of headaches, called neuralgic, which seem to start from the occiput, like the headache due to breathing coal gas. To quote Foster:[2]

"The dominating center of the vaso-motor nerves lies in the upper floor of the medulla oblongata. Stimulation of this central area causes contraction of all the arteries and great increase of arterial blood-pressure, with swelling of the veins and heart. Paralysis causes dilatation of the arteries and fall of blood pressure. This center may be excited directly and reflexly. * * * Psychical excitement influences this center, causing constriction of the small arteries. * * * A pale, cold, collapsed side of the face, contraction of the temporal artery like a whip-cord, *dilatation of the pupil*, and secretion of thick saliva, are sure signs of intense stimulation of the cervical sympathetic nerve, which may be brought about by some poisons, and by emotion. Lactic acid (1-10,000 saline solution) passed through the blood vessels of a frog always enlarges their caliber (Gaskell)."

Simple glaucoma has fairly constant features. Acute glaucoma is of many varieties, whose features in common are increased intra-ocular tension, advancement of the lens and iris, dilatation of the pupil, and opacity of the cornea; the variation depending upon the quantity of impulse, the special tissues involved, or upon the stage which has been reached by the chronic process, on which the acute form is engrafted.

[2] Foster's Text-book on Physiology, second edition, pp. 695-701.

That the acute form is propagated upon a local process already existing,* and more or less entangling the tissues of the whole organ, would seem to be supported by the irregularity and uncertainty of its development and response to measures adopted for its relief. The anatomic and microscopic changes noted, in the different series of glaucomatous eyes examined, may disagree; at times they directly contradict each other. This is not due to incapable or imperfect observation, though it causes great confusion in the mind of the student; but to the fact that the underlying process has attacked a different set of tissues, or the same tissues in varying order; has not pursued an identical course in its encroachments, or has done so with diversity of power or rapidity. Observations have included all the tissues of the eyeball. In one case, at a given period of development, the disc will be deeply cupped, while in another the excavation may be shallow, if it exists; the anterior chamber may be shallow in one case, and deep in another; tension may be the same in all; or with any, or all, of these changes it may not be increased. The sinus of the anterior chamber may be closed with normal or subnormal tension[3]; in another, aniridia[4], with the filtration angle closed by the *rudimentary* iris, and with increased tension. Any combination of symptoms and conditions existing together point to two processes at work: one, chronic and constantly progressive, the other spending itself in recurring attacks of more or less violence; both due to the same influence, which manifests itself in the two ways, in different degrees, in individual cases. A remote nervous influence manifests itself when mental perturbation excites an exacerbation, or when rest and quiet are followed by an improvement in the physical symptoms.

Whatever other changes are observed, those of the cornea are nearly always present. That glaucoma may begin in the cornea there is hardly room for doubt, for repeated slight injuries to the cornea are frequently followed by acute glaucoma, as is shown by the interesting cases given below in

* May not always be apparent.

[3] Schnabel, *Archiv. Ophthal.*, Vol. VII., pp. 37, 38, 302.

[4] Collins reports several cases, *Ophthal. Review*, Vol. X., p. 101.

abstract; even two of Mr. Collins' three cases of aniridia with glaucoma were marked with initial corneal changes; one with central leucoma, consecutive to a perforating ulcer of the cornea, and a staphyloma in the ciliary region; another, with a corneal cicatrix and a staphyloma; the third had a glaucomatous cataract at 34 years of age.

Von Graefe[5] reports a woman 50 years of age, with an old eczema for many years, with an opaque and swollen spot with a yellow center, in the cornea opposite the pupil. Tension, which was not tested at the time of his first interview, was normal in the fourth and sixth week. While the corneal disease remained stationary, a subacute glaucoma developed. Iridectomy relieved the glaucoma and cured the corneal affection.

Saemisch[6] relates a case, in which a *striped* opacity of the cornea existed previous to the development of a vesicle. When the corneal trouble was at its worst, acute glaucoma supervened. Iridectomy; cure.

Pooley[7] observed a case of keratitis vesiculosa, ending in absolute glaucoma. A Jewess, 40 years of age, left eye. The anterior chamber, fundus and field normal, V. = $\frac{20}{50}$, in August. In October, pupil wide and immovable, anterior chamber shallow, T (r) +, no perception of light, great pain. Iridectomy reduced tension and relieved the pain, but did not restore vision.

Saemisch[8] gives another case, blind from glaucoma, in which corneal vesicles developed. The eye had to be enucleated.

Bowman reports a case of keratitis bullosa in an ill-fed woman; the eye was destroyed by glaucoma.

Landesberg[9] details seven cases of keratitis bullosa, illustrative in this connection:

Case I. A child 8 years of age, in whom severe ciliary neuralgia, and increased intra-ocular tension, accompanied the development of each vesicle, which occurred at intervals of from eight to fifteen days. Reparation began in the deeper layers of the cornea.

Case II. A man 38 years of age; the corneal affection pursued the same course for four months, ending in acute glaucoma, cured

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[5] *Archiv. f. Ophth.*, Vol. XV., p. 108.
[6] *Berliner Klinische Wochenschrift*, No. 37, p. 449.
[7] *Archiv. Ophthal.*, Vol. IV., p. 46.
[8] *Handbuch der gesammten Augenheilk.*
[9] *Archiv. Ophthal.*, Vol. VI. p. 135.

by iridectomy. His family physician judged this man's trouble to be of *rheumatic** origin.

Landesberg thinks there is such an interdependence between keratitis bullosa and glaucoma that every eye suffering from this form of corneal trouble is liable to be seized with glaucoma.

Case III. In a man 22 years of age, ulceration of the cornea, consecutive to a foreign body (bit of metal) in the cornea, in October. Vesicles formed six times before the end of December, always announced by the presence of several small ulcerations in the deeper layers of the cornea, from which vertical parallel *stripes* proceeded. In four of the six attacks increase of tension was evident.

Case IV. A child, four years of age, with *eczema* of the head, nose and face, had frequent attacks of keratitis bullosa; irritation, pinhead infiltrations in the deeper layers of the cornea, from which proceeded vertical *stripes;* ciliary neuralgia and increased intra-ocular tension. The eye recovered with a circumscribed leucoma.

Case V. A man, 36 years of age, had frequent attacks of keratitis bullosa (seven to fifteen days interval), with the already described appearance of the cornea, and increased intra-ocular tension. He recovered with a firm cicatrix at the site of the affection.

Case VI. A man, 71 years of age, with a like appearance and condition of the cornea, anterior chamber shallow, pupil moderately dilated, increased (?) tension. Nine days later, a vesicle; acute glaucoma; iridectomy. Two weeks later, another vesicle, tension increased. Cured, with a circumscribed central leucoma.

Case VII. A girl, 20 years of age, without previous trouble. The appearance of the cornea as in the cases described; tension normal. Three weeks later, vesicles formed with increased tension. A number of attacks followed in the next four months, when the patient discontinued her visits. At the last visit, the lower part of the cornea was dim, and tension was normal.

Atropin was used in all these cases, but atropin will not *cause* increase of tension in a normal eye. A disposition to high tension must pre-exist, and its occurrence is only precipitated by the mydriatic; possibly by the retraction of the iris,† and the consequent diminished resistance to the influx

* The identity of the cause of gout and rheumatism can not be discussed here.

† Eserin acts by resisting this influx; when the *vis a tergo* is too great or too persistent, it fails.

of blood into the eye in the presence of general high arterial pressure, as shown by the tendency to intra-ocular hemorrhage if too rapid escape of aqueous is permitted during glaucoma operations. Landesberg found the best treatment of these cases to be by *scarification* and *compression*. He refers to the very rapid onset and course of the corneal phenomena, save the opacity; to the increased intra-ocular tension, which is constantly present and in proportion to the corneal irritation; to the immunity of the iris and choroid, and to the secondary (?) glaucoma.

Bullous keratitis, according to Graefe, *is one of the symptoms of a deep morbid process.* In its nature it is a herpes, and not of true inflammatory character; it is an expression of irritation at the origin, or in the course, of a nerve, an evidence of which we have in the neuralgic pain inseparable from the development of the vesicles. The periodicity of its recurrence (seven to fifteen days) suggests an irritation of chronic character, and in this is supported by its apparent kinship to herpes preputialis, which has the same history, expresses itself in the same manner, runs the same course, and of whose origin there is no doubt; lithemia. The vesicles may precede the development of glaucoma, and be of some prognostic import; they may develop only in the stage of glaucoma degeneration. Appearing thus at widely different stages of this disease, the two must be due to the same cause, and form part of the same process.

Landesberg[10] further relates two cases of *ribbon-shaped* keratitis (?) followed by glaucoma, which are not without interest.

Case I. "A man, 60 years of age. When first seen, the right cornea was the site of a wide ribbon-shaped and continuous opacity, corresponding to the palpebral fissure, and extending transversely over the whole cornea. The opacity was a *reddish-brown*, and had an equal degree of color intensity from the margins to the center of the cornea. The center itself appeared whitish-gray, as if a metallic salt had been precipitated there. The ribbon-shaped opacity had a sharply defined boundary above and below." Photophobia, tension normal, pupil reacted sluggishly to atropia; ophthalmoscopic exam-

[10] *Archiv. Ophthal.*, Vol. III., p. 65.

ination impossible. The left eye showed the first stage of the affection; near the inner and outer margins of the cornea, a narrow opaque stripe of slightly brown shade. The opacity had the appearance of a thin stripe of brown color, laid quite superficially on either side of the cornea with the most delicate touch of a brush; the center of the cornea clear. He rejected an iridectomy on the right. Three months later he returned. The right eye was *hard*, with a shallow anterior chamber, posterior synechia, perception of light, subconjunctival injection; corneal opacity unchanged. Iridectomy with good result. Three years later, there was no irritability, tension normal, and the corneal opacity had a tendinous appearance, having gained so much as to leave a very narrow rim of clear cornea.

Case II. A man, 55 years of age. When he presented himself, the right eye was the seat of subacute glaucoma. All the phenomena of glaucoma were present, with numerous hemorrhagic spots in the retina. The cornea was affected with ribbon-shaped opacities, brown in color; the corneal epithelium was smooth and shining. Iridectomy reduced the tension and cleared up the hemorrhagic spots, and the eye remained in a satisfactory condition, with no gain in the ribbon-shaped opacity, a year later.

Hirschberg[11] records a case of acute glaucoma, following the use of atropin, in "an apparently healthy lady," 64 years of age. He found "fine maculæ cornearum centrales *of old date* and punctate opacities of the front surface of the cornea." Atropin was used after iridectomy without mischief, and H. raises the question if glaucoma malignum *belongs to the eye or the individual.*

In but few of the cases cited above was the cause suggested for the corneal changes observed. A notable exception was that in which a bit of metal excited the changes which followed; and yet a constitutional predisposition existed, or the formation of vesicles would occur more frequently from foreign bodies in the cornea. This predisposition was probably "rheumatic," as suggested by the physician in another of the cases.

The cornea may be measurably tolerant of injury, but just what persistent mechanical irritation will accomplish (if that alone did it) is shown by the cases reported by Dr. Hock,[12] of Vienna, almost in the nature of an experiment.

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[11] *Archiv. Ophthal.*, Vol. IV., p. 203.

[12] *Archiv. Ophthal.*, Vol. V., p. 382.

"A right eye had been lost six months previously from Egyptian ophthalmia, leaving a dense cicatrix in place of the cornea. Fingers could be counted at 2 feet, tension normal. The cornea was tatooed, and during the evening there was severe pain on the right side of the head, extending from the eye. Next day the bulb was of stony hardness. A former corneal fistula was reopened, and the globe immediately became quite soft. Tension was increased four times during the treatment; each time the lens was advanced, with bulging of the middle part of the scars.

"The curative effect of artificial interference after each relapse (opening the fistula), and especially the recovery and persistence of normal tension after the last sitting (when the fistula was again reopened) proves to me that in this eye there were none of the conditions (!) predisposing to glaucoma, but that the effect was due entirely to the multitude of little wounds."

"Irritation of the cornea is sufficient, without mediation, to give rise to glaucomatous increase of tension" (Von Gräefe). Hock adds the case of a stout woman, 54 years of age, with palpitation of the heart, whose right eye had been lost a year before by glaucoma. "Just below the middle of the cornea was found a circular opacity as large as a hemp seed. Evidently there had been circumscribed corneal infiltration preceeding the glaucoma which had now passed by. Considering all the circumstances developed by the history of the right eye, we can hardly avoid the conclusion that here was a succession of corneal infiltrations, with consequent glaucoma."

Mauthner[13]: A man, 71 years of age, injured by powder explosion a long time previously. In youth, he had some inflammation, which left several facets as large as a pin's head, stretching across the area of the pupil. Acute glaucoma of the right eye, with remarkable *striped* haziness of the corneal parenchyma; left eye hazy from an *old* parenchymatous grayish-white and striped cloudiness. Sclerotomy, and the stripes became squares, so that forty-eight hours later the cornea looked like a chess board, the stripes giving way to squares, which disappeared in a week. Fourteen days after operation the cornea was perfectly transparent except for the old central facets; cured.

Mauthner (*Loc.cit.*): A man, 64 years of age. Glaucoma simplex. Both eyes had been struck by the bowstring of a mouse trap a year before. Both corneæ faceted; pterygium covers the pupil of the right eye; circular abrasion of the left cornea, upward and outward.

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[13] *Loc. Cit.*, Vol. VII., p. 224.

Tension moderately increased. Iridectomy of the right eye; sclerotomy of the left. Tension notably lessened.

Mauthner (*Loc. cit.*, p. 235): Man, 74 years of age, with both corneæ cloudy in distinct parenchymatous spots, which look like the remains of previous inflammation. Increased tension. R. V. = $\frac{1}{2}$; L. V. = $\frac{1}{8}$; F. F. free. Sclerotomy. Tension normal; spots in the cornea unaltered; vision unchanged.

Idem: Boy, 6 years of age; blow on the head; traumatic cataract of the left eye and glaucoma of the right eye. T. + 3, globe very large. Cornea cloudy in *stripes*, diameter of the cornea = 18 *mm.*, disc deeply cupped; no pain; no perception of light. Sclerotomy. T —; fingers at 10 feet. Left cornea clear, diameter = 15 *mm.* (3 *mm. less* than that of the glaucomatous eye).

Schöler[14] produced glaucoma experimentally by burning the cornea. This was probably due to the irritation of the cornea, and the consequent disturbance of the normal supply of corneal nutritive elements or to reflex nerve influence upon the vaso-motor "dominating center" in the floor of the medulla.

Dr. Elizabeth Sargent[15] made an anatomical dissection of six eyes enucleated for absolute glaucoma. She found *pannus of the cornea in all;* superficial ulceration in one; masses of round cells about the scleral and episcleral vessels, or in the cornea, in all; sinus of the anterior chamber patent in three; closed in two; partly open and partly obstructed in one; iris atrophic, and adherent to the cornea, in all; Descemet's membrane pierced by inflammatory tissue (showing *how* this membrane may not *always* be intact), in one; retina atrophic, and detached, in three; with sclerosis of the arterial walls in three; optic nerve excavated, with connective tissue hypertrophy, or celular infiltration of the nerve, and atrophy of the nerve, in all.

It is with difficulty that an abstract is made of such a detailed examination and reference is made to the paper. The examination was very pains-taking and offers evidence of the constancy with which corneal structure changes, cellular infiltration into the corneo-scleral region, and into the optic nerve and its sheath, and connective tissue hypertrophy, are met. These are the ultimate features of the structure changes,

[14] *Berliner Phys. Gesellschaft*, June 20, 1879.

[15] *Centralb. für Augenheilk.*, December, 1884, p. 353.

for, in these cases, the malady had already run its course. To specify all the changes that had taken place at any particular period of its progress is manifestly impossible, as they can not be just the same in any two cases, and thus give rise to a diversity in the severity of the symptoms, and of the result of a given operation, in different cases. This examination seems to have been made with no purpose to support a preconceived theory, and is reliable.

The examination of seven glaucomatous eyes by Birnbacher and Czermak[16] is of much the same character as the preceding, only it shows how persistently an accepted view possesses the mind. The four, first in order, had chronic inflammatory glaucoma; the next was one of hemorrhagic glaucoma; the last two, of glaucoma degeneration.

Case I. Cornea rough and exfoliated; T. + 2; slight edema of the corneal structure; slight rupture of Bowman s membrane, and two or three-fold layers of spindle cells between it and the pavement cells; spindle-form, and large groups of round cells, with single epitheloid cell forms about the episcleral veins. Cell infiltration into Schlemm's canal, membrane of Descemet, iris, ciliary body, choroid, in the sclerotico-choroid about the papilla; and masses of them rest in the perivascular spaces, and take on the form of young granulation tissue. The choroid is thinned about the papilla. An exuberant growth of endo-epithelium was found everywhere in the intervaginal space.

Case II. Retina and choroid adherent in places. Pavement cells of the cornea destroyed. Bowman's membrane broken through, and epithelium thickened. In the upper layer of the corneal structure, and in Bowman's membrane, was found a thick, fibrous, richly nucleated tissue, having vessels, and penetrating the surface of the membrane, always at least to the extent of the area of the membrane. This tissue was found between Bowman's membrane and the epithelium, and in the epithelium, causing small *elevations* of its surface. Blood vessels extended to the center of the cornea, and along them thick rows of round and spindle cell nuclei. The canal of Schlemm was effaced. Fibrous connective tissue was found on the surface of the iris; the retina, posterior to the ora serrata *was greatly thickened, and changed into an irregular meshwork of course and fine fibers;* the posterior choroid thin and adherent to the

[16] *Graefe's Archives*, Vol. XXXII., 2, pp. 1 to 75.

sclera; nuclei of the optic nerve neuroglia increased in number; about the central vein a mass of granulation cells.

Case III. Relation and position of the cells of the pavement epithelium of the cornea changed. No edema. Between the corneal epithelium and Bowman's membrane was found a tissue of coarse fiber bundles, and numerous long spindle-form nuclei, supplied sparsely with vessels, and extending over the whole cornea. Bowman's membrane was broken through in many places by lines of spindle cells, which sink into the corneal structure. Scleral and epi-scleral vessels extended forward through all the layers. The corneo-scleral sinus was almost obliterated. In the anterior section of the corpus vitrei were numerous wander-cells.

Case IV. A hemp-seed sized sclerectasia; in the corneal epithelium were circumscribed small prominences; between Bowman's membrane and the epithelium the space was filled with a fine mass of *striped* network. Slight edema.

Case V. In the corneal substance, especially near the periphery, and in the deeper layers was found a coarse *network* whose contents were invisible: membrane of Descemet intact.

Case VI. Edema of the cornea, and colored nuclei. Bowman's membrane broken through by spindle-shaped cells.

Case VII. A woman, 75 years of age, with high arterio-sclerosis and irregular heart action; urine normal. Pericorneal injection, keratitis punctata, cornea uneven, anterior chamber abolished. In the cornea fine granulation masses were found in the epithelial spaces. Glaucoma pannus; spindle-celled formation in the corneal substance.

It will be seen that the new-formed connective tissue was found in all these cases and in more than one structure, but especially marked and general in the cornea.

I have herein referred to no cases of my own, for, in the language of Mooren, "I prefer to call attention to the statements of other observers rather than to mention cases of my own practice, in order to leave no room for the insinuation that my observations are biased in favor of a certain pathological view, thus bringing into connection with each other things which may be only the result of accident."

I would say further, that, for obvious reasons, I have avoided reference to the "steaminess" of the cornea, which is always manifest in acute glaucoma, and which may, or may not, be due to edema of the cornea from increased intra-

ocular tension, as claimed by Fuchs[17]; nor to the arcus senilis (the early result of the affection of the corneal nutrient vessels, as the ulceration, or necrosis, of glaucoma degeneration is the late development) so often present in glaucoma; nor to the lenticular changes, though Rheindorf describes glaucoma with *acute* opacity of the lens, the cataract being incipient when the glaucoma developed. I would suggest that a closer relation exists than is realized, between the *cause* of glaucoma and the *cause* of senile cataract.

Angelucci[18] thinks that glaucoma is due to sclerosis of all the membranes of the eye, and especially of the walls of the blood vessels, as he found the arteries sclerosed, their caliber diminished, and the veins swollen and chronically inflamed.

Steamy opacity of the cornea may precede increased intraocular tension, and two weeks may be required for its disappearance after tension is reduced. It is an open question, if fluid from the anterior chamber can penetrate to the corneal lamina while the membrane of Descemet remains uninjured. The cases referred to show true corneal tissue changes, often of some duration when the glaucomatous attack developed: stripes, brown ribbon shapes with smooth and shining corneal epithelium, chess-board squares, vesicles, facets and leucoma; which differ greatly from the possible appearance of an intralaminar edema, with rough and desquamating corneal epithelium. Fuchs and others have seen intralaminar edema, and it, therefore, sometimes occurs, but only when the membrane is deficient at some point, as in Sargent's case, in which Descemet's membrane was perforated by inflammatory tissue. A few cases, however, do not show that the corneal opacity is due to edema from high tension. It *may* happen so, and another form of defficiency in the membrane of Descemet by which edema of the cornea from the interior of the eye may occur is shown by Tartuferi,[19] who, in a microscopic examination of the corneæ of a collection of glaucomatous eyes, found in most of them tissue changes of important character: shortening of the corneal diameter by connective tissue between

[17] *Graefe's Archives*, Vol. XXVII., 3, p. 66.

[18] Trans. Internat. Med. Cong., London, 1883.

[19] *Giorü. di R. Acad. di Med. di Torino*, Nos. 5 and 6, 1882.

the epithelium and the membrane in the periphery of the cornea; the presence of wander-cells which are easily changed into connective tissue; the epithelium often separated from Descemet's membrane by connective tissue, etc.

Here again we find corneal changes which are of the same character as those noted in the other structures of the glaucomatous eye, for by anatomical and microscopical examinations connective tissue has been found in the ciliary nerves[20], muscle,[21] and processes,[22] the cornea,[23] the iris,[24] the choroid[24], the retina,[25] and the optic nerve.[26] Fuchs,[27] himself, from the dissection of the eyes of a woman, dead at 64 years of age, which had been iridectomized for acute glaucoma eight years before, in which the lamina cribrosa was only slightly convex backward, with atrophy of one-fourth of the optic nerve fibers, concludes that simple hyperplasia accounts for the changes in the ciliary body, as they seem to have existed *before* the glaucoma, and to have excited the attack. Connective tissue hypertrophy was most marked in the region of the macula.

Schnabel[28] thinks opacities of the cornea do not depend upon increased tension, and that they are only a symptom; that they are not of inflammatory character in the sense of a keratitis, differing so much in appearance, some being dotted, some ribbon-shaped, and others diffuse, occupying the whole corneal tissue. Yet, some of them have as much of the character of inflammation as the so-called inflammatory glaucoma; we do not see *pus* associated with either. It is true that ulcers and abscesses of the cornea in absolute glaucoma, are sometimes accompanied by hypopion, but only as

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[20] Hocquard, *Archiv. d'Ophth.*, Vol. III., No. 3.

[21] Stolting, *Graefe's Archiv. f. Ophth.*, Vol. XXXIV.

[22] Brailey, *Royal Lond. Ophth. Hosp. Reports*, Vol. X., p. 86.

[23] Birnbacher and Czermak, *Graefe's Archiv.*, Vol. XXXII, 2.

[24] Knies, Ueber das Glaucom., *Archiv. f. Ophth.*, Vol. XXII., p. 163.

[25] Schnabel, *Archiv. Ophthal.*, Vol. VII., p. 307.

[26] Alt, Lectures on the Human Eye, p. 156.

[27] *Graefe's Archiv.*, Vol. XXX., p. 123.

[28] *Wien. Med. Presse*, 22-26.

a result of necrosis, for the glaucomatous character of the affection has about ceased. It would, therefore, seem proper to attach to the corneal changes, observed in glaucoma, their true importance as *part of the process* instead of trying to account for them as a result of increased tension, a superficial view which the evidence does not justify. If, however, they *must* be regarded as a symptom, let it be as a symptom manifested in the cornea, exposed to view, of a tissue change progressing elsewhere in the structure of the eye, out of view; in the sclerotic. The epi-scleritis, which often foreshadows a glaucoma, favors this view.

Dr. J. Kostenitch[29] reports the microscopic examination of a case of scleritis:

"A woman, 24 years of age, left eye blind, *larger* than the right and sensitive to pressure; Tn. Entire cornea a white scar. Optic disc excavated; anterior chamber shallow; iris atrophic, and adherent to the cornea at its periphery. Sclera, cornea, conjunctiva, iris, ciliary body and vitreous affected by cellular infiltration, and occupied by numbers of wander-cells (leucocytes); detachment of the membrane of Descemet; disappearance of Bowman's membrane; new-formed tissue in the anterior chamber, and the corpus vitrei, the fibrillæ being well marked; thickening of the walls of the arteries in the ciliary body and choroid; atrophic degeneration of the retina."

This is a typical picture of glaucoma, lacking the feature of high intra-ocular tension. It could not be examined ophthalmoscopically because of the corneal opacity, and was designated *scleritis* from the most prominent clinical symptom of active localized disturbance.

Thus, instead of being a consequence of increased tension, the process which it shows to be in action is the probable cause of high tension, and all the other symptoms for which high tension has been held responsible. By reflex action through the vaso-motor center in the medulla oblongata, either from the encroachment of new connective tissue upon the ciliary and other nerves of the bulb, or by the excitement of these same nerves by an irritant contained in the blood; or by the direct influence of this irritant itself upon the vaso-motor

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[29] *Archiv. Ophthal.*, Vol. XXIII., No. 4, p. 416.

center, causing contraction of the arterioles and increased heart's action (palpitation) to overcome the added resistance to the onward motion of the blood-stream, with consequent high arterial pressure and venous stasis, followed by dilatation of the arterioles in reaction, and the symptoms of inflammation, is the picture presented by irritative and inflammatory glaucoma, with increased intra-ocular tension, often relieving itself without interference, though not perhaps until visual power is abolished. A continued high tension is due to persisting venous stasis, as when tension *slowly* falls to the normal, after operation. If high tension depended upon the occlusion of the channels of filtration, nothing but mechanical interference would relieve it, when once "the vicious circle" had been formed; but we know that *passing attacks* of high tension are frequent. Eserin is effective in emptying the veins by the *spasmodic* pressure exerted by the iris muscle in its efforts to contract the pupil, and hence the greater efficiency of the weaker solutions, as a strong solution of eserin causes *rigid* contraction* of the iris.

High intra-ocular tension, due to the increased amount of blood in the vessels of the bulb, explains those cases in which the sinus of the anterior chamber is open, the variations of tension so common in *irritable* glaucoma, the advancement of the lens and iris, and the shallow anterior chamber; it accounts for a *hardness* of the globe which may at least balance the *vis a tergo*, the general blood pressure.

"When connective tissue is being supplied, the part becomes inflamed and swollen, owing to the exudation of plasma. The blood vessels become dilated and congested, and, notwithstanding the slower circulation, the *amount* of blood (in the part) is greater. The blood vessels are increased owing to the formation of new ones. Colorless blood corpuscles pass out of the vessels and reproduce themselves, and many of them undergo fatty degeneration, while others take up nutriment and become converted into large uninucleated protoplasma cells, from which giant cells are produced."[30]

Vascular disturbance is essential, but however this may be, connective tissue germination is wont to be accompanied by

* Perhaps well-devised manipulation of the eyeball would relieve tension. I have not tried it.

[30] *Foster's Text-book on Physiology*, 2d Ed., p. 405.

edema, and would be a more natural explanation of the edema of the cornea described by Fuchs.

Atrophy of the choroid, appearing earliest about the optic nerve in which an extra growth of connective tissue is so generally found, gives some color to Mauthner's[31] view:

"That glaucoma is a serous choroiditis by which vision is destroyed, and not by high intra-ocular tension; as the worst cases are those in which tension never rises above the normal."

The observation of Straub,[32] that when a meridional section of a glaucomatous eye is made, the choroid is found to have lost its elasticity, and does not withdraw from the sclera as in the healthy eye, is offered in evidence of its sclerosis, and its atrophy is due, at least in part, to this condition. Ulrich[33] regards sclerosis of the iris and infiltration as the two great factors in the pathogenesis of glaucoma, and Heyne[34] found in chronic glaucoma hyaline degeneration of the vessels. Valude[35] reported observations in four cases of hemorrhagic glaucoma, *all* of which gave evidence of arterio-sclerosis. The retinal vessels showed hyaline degeneration and peri-vasculitis. Optic nerve cupping, and closure of the iris angle were not always found, but dilatation of the iris and ciliary bodies was constant. He thinks this form of glaucoma due to an alteration of the retinal blood vessels, secondary to a disease of the general vascular system. Garnier[36] judges the compensatory endarteritis of glaucoma to begin in degenerated hyaline, or connective tissue masses between the elastic membranes. Schnabel[37] did an iridectomy on two eyes. There was nothing remarkable in the left; but from the right eye, with and behind the iris, a grayish mass prolapsed, which proved to be "a thin whitish membrane, pervaded by delicate straight and somewhat tortuous connnective tissue fibrillæ, and containing a few elastic fibers, numerous larger and smaller blood vessels filled with red blood discs; around these a larger quan-

[31] *Wien. Med. Bl.*, 10, p. 300.
[32] Report of Seventh International Ophth. Congress, Heidelberg, 1888.
[33] *Trans. Ophthal. Society*, Heidelberg, 1884.
[34] Inaugural Dissert., Königsburg, 1884.
[35] *Trans. Ophthal. Society*, Heidelberg, 1892.
[36] *Archiv. f. Augenheilk.*, Vol. XXV.
[37] *Archiv. Ophthal.*, Vol. VII., p. 277.

tity of fibrillar connective tissue, in which numerous cells were imbedded. * * * The exsected iris was thinner than normal." Prof. Jaeger had a similar experience in three iridectomies, and Dr. Kerzendorfer, in one. Schnabel[38] also reports sixteen, or more, cases of glaucoma, in all of which he found structure changes of the cornea (a cicatrix, an ulceration, or a staphyloma), or sclera, and closure or obstruction of Schlemm's canal, though some of them did not show increased tension. In some instances an increase of connective tissue was found in the ciliary muscle; and atrophy of the ciliary body first observed by Brailey,[39] exists in the great majority of cases of simple glaucoma, sometimes even before the glaucoma is manifest.

While only a portion of the recorded evidence is here offered, it would seem sufficient to establish the claims of this and a preceding paper (The Disease Process, Glaucoma, *American Jour. Med. Sci.*, June, 1893) that the local affection is a fibrosis, or connective tissue hyperplasia which, by the growth of the degraded tissue chokes to death the special functional tissues. The testimony of the eminent observers favors the further contention of this paper that the corneal appearance is *not symptomatic* of high tension, but is an organic change, shared in common with the other parts of the eye, especially with the sclerotic, whose density and opacity deny to us the observation of such slight manifestations as may be detected in the normally transparent cornea.

Repeating that it is the *type* of the disease, primary simple glaucoma, the basis of the varieties, which is under consideration, and recognizing that the evidence may not be conclusive to some minds, perhaps, because it has been lamely presented; or, because, from a long acceptance of the idea that high intraocular tension and glaucoma are almost synonymous, mental adaptability to any other view is impossible, the paper proceeds to the question:

What are the factors which most probably promote connective tissue hypertrophy?

Some of them have been named in the preceding pages.

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[38] *Archiv. Ophthal.*, Vol. VII., pp. 24 to 33 and 249 to 257.

[39] *Royal Lond. Ophth. Hosp. Rep.*, Vol. IX, 2, p. 199.

The chief examples of overgrowth of connective tissue in organs of high functional character are interstitial hepatitis, interstitial nephritis and sclerosis of the spinal cord.

Interstitial hepatitis may begin in a primary degeneration process, or in an irritative congestion. In either case cell degeneration seems to be the process, *and is often preceded by pain and enlargement for three or four years before the establishment of the sclerosis.* It may be secondary to malarial(?) hyperemia; due to abuse of alcohol; to diffusion of degenerative processes (as others coexist) which cause hypertrophy first, and are not inflammatory (Handfield Jones). It has been seen as early as 10 years of age (Frerichs). The morbid process involves extravasations of blood, complete destruction of the secreting structures, and disintegration and *partial absorption* of the component tissues of the organ.

Interstitial nephritis was shown in my paper, "The Disease Process, Glaucoma," to bear a close resemblance to glaucoma in its clinical history; in anatomical features it is of the same type. The *capsule* is less *transparent* than normal, with small vessels ramifying on its surface. In laying open the organ, the cut surfaces become convex, showing compression of its elements (tension). If the process is recent, the tissues are friable; if connective tissue overgrowth has taken place, the tissue is tough. There may be an accumulation of small round cells, and multitudes of new cells lying without their capsules.

Semmola[40] claims that true interstitial nephritis always consists in a general nutritive disorder, to which nephritis is secondary; beginning in diminished cutaneous respiration, followed by the imperfect digestion and transformation of albuminous foods. Palpitation of the heart, without organic change, conjoined with atonic or irritative dyspepsia is often found; observed in children as a heredity. A. Weber[41] found heart disease in all young persons having glaucoma, and that iridectomy served little purpose in such cases.

Sclerosis of the cord is a process of degeneration, though it may develop with some rapidity with symptoms of second-

[40] *Gazette Medicale de Paris*, 1875.

[41] *Graefe's Archiv.*, Vol. XXIII., No. 1.

ary degeneration; it is a primary irritation, with consecutive connective tissue proliferation (increase of neuroglia), and absorption of nerve fiber. *Causes:* Inherited tendency, constitutional syphilis, possibly sexual excesses, chronic alcoholism, repeated over-exertion and exposure to cold, and lead poisoning.

Thus, according to the most trustworthy views, connective tissue overgrowth is due to *dirty* blood; made so by imperfect digestion[42] (to which the habitual use of alcohol contributes), so-called struma, syphilis,[43] lithemia and lead poisoning; by the retention of effete matter and partially metamorphosed ingesta, through the inefficient action of the excretory organs. And glaucoma is found conjoined with albuminuria, eczema, palpitation of the heart, general high arterial tension, and constitutional syphilis; in fact with all the conditions which are supposed to foster the propagation of retrogressive tissue in organs of high functional value. Observation shows that excretion by the skin is very imperfect; that most of those who suffer from glaucoma have been accustomed to take cold still baths, thus increasing the contraction of the superficial arteries, and without the exercise which restores the circulation; and they eat too much for the waste they effect, and are habitually constipated.

It is claimed that high arterial tension is never absent in glaucoma, is always found in vaso-renal changes; is due to a chronic irritant of low intensity, in action through a considerable period of time; that it is the expression of Nature's effort to rid the circulation of the irritating element, and that it is always accompanied, or followed, by connective tissue hyperplasia.

The term *glaucoma* has had no real significance since the invention of the ophthalmoscope, and conveys to our minds no idea of the disease; hence, the substitution of the term *interstitial ophthalmitis*, descriptive of the pathologic changes common to all the structures of the globe.

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[42] "There is hardly any condition which is more certain to produce intense uric-acid-edemia than gastric catarrh." (Haig.)

[43] Within the past three years, I have seen two cases of simple glaucoma, clearly due to constitutional syphilis.