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THE DISEASE-PROCESS, GLAUCOMA.

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A fair hypothesis must be based upon all the known facts of a case, and must rationally explain them all.

"Scleritis[1] is more often met with in persons of a rheumatic, or gouty, habit than in others." Garrod[2] has seen two cases of sclerotitis with white deposits of urate of soda on the surface of the tissue; "likewise on the tarsal cartilage at the angles of the eyes." Increased tension is constantly a symptom in scleral metamorphoses. "During life the existence of scleritis is often overlooked, or is first recognized by its results, staphyloma or atrophy."[3] A chronic hypertrophic condition of the sclerotic *always* precedes total phthisis of an eyeball, and the process leading to it begins, without exception, in the posterior parts of the sclerotic around the entrance of the optic nerve, and progresses more and more toward the corneo-scleral margin.[4] Coccius[5] saw one case of fatty degeneration of the sclerotic in glaucoma, and Cusco and Coccius suggest that the starting-point of glaucoma is an inflammation of the sclerotic. *Lime* deposits in the sclerotic at this period of life might easily be mistaken for fatty degeneration—the presence of fat globules in the tissue would be essential to its proof. The effect of fatty degeneration of the sclerotic would be its friability and diminished resistance to intra-ocular pressure, with subsequent *lowered* tension. The occurrence of this condition in a very limited number of cases is possible, for any phase in this "combination of diseases" is conceivable. In nearly all cases of glaucoma, however, a state of increased rigidity of the sclera is a marked characteristic; high tension and increased rigidity of the sclerotic, to some extent, mask each other.

The sense of the discussion upon this malady at the Heidelberg Congress, in 1877, is that the apparent teachings of dissections are inconclusive. "Anatomical observations in explanation of this array of symptoms serve little purpose," and yet each one of "this array of symptoms" is an evidence of morbid change in some of the ocular structures.[6] The presence of *all* the symptoms, or of the most charac-

[1] Soelberg: Wells, Dis. of the Eye, Bull's ed., p. 273.
[2] Reynolds: System of Med., vol. i., p. 517.
[3] Stellwag: Dis. of the Eye. Am. ed., 1873, p. 336.
[4] Alt: Lectures on the Human Eye, 1880, p. 45.
[5] Archiv f. Augenheilk., B. ix., 1, p. 21.
[6] Alt, p. 155.

teristic of the observable structure changes, are not essential at any given stage, and all cases do not run the same unvaried course; but, taking the *whole* history of two cases to their conclusion, and usually the distinguishing symptoms *have* been present, and the result is much the same. If the varying symptoms, the different diseases which are included in the name, the natural explanation of the symptoms, and the theory of their causation can be made consistent with each other, and can be shown to be due to a common cause, the fog surrounding this question will be somewhat thinned.

The accumulated testimony of all observers, interpreted, points to morbid changes in the connective, or fibrous, tissues as the initial and permanently progressive lesion of the disease, indicating that these changes are interstitial, beginning in the sclera; that they consist in connective-tissue proliferation, with its usual debasing effect upon the higher functional forms of tissue enclosed by the sclera, which, by their protests against this inexorable degradation, give rise to the subjective symptoms present—the objective symptoms are phases of the progress of the disease.

By a histological consideration of the globe, one is impressed with the amount of connective tissue in its construction and its importance to the integrity of the eye, for this organ is *composed* of fibrous and connective tissue, an elaborate and pervading framework and support to vitally functional tissues, surrounding every structure. Because of its low vitality and consequent indisposition to resentment, it rarely (about 1 per cent. of all cases) acts viciously, and then we must look for a persistent cause of irritation, of low grade, with a tendency to reversion along the lines of evolution. In the inadequacy of the liver to convert uric acid into urea we have such a tendency; in the presence of uric acid in the blood and tissues we have such a cause. When a disease is bilateral it cannot be purely local in its origin; in the consideration of causes persistent irritation is an essential quality, and acute inflammatory action results when the irritation is increased. Uric acid is carried by the blood, and the envelope of an organ receives its first effect; hence, it has been observed that the sclerotic is denser and harder than normal before any other evidence is in existence of the presence of the dread disease, and as this occurs at a period of life when such condition, to some extent, is supposed to be natural, it is liable to attract little attention.

The morbid process, to my mind, *is a hyperplasia of connective tissue which diffuses itself through the whole organ*, possessing the property of pathological connective tissue to always *contract*. High arterial tension promotes the formation of new connective tissue, is present early in glaucoma, and is the conservative effort of Nature to relieve the blood of uric acid. This product acting mildly for a long time, will usually

cause connective-tissue proliferation; if the action be intense or if the urate of soda is formed in this region, the vascular apparatus chiefly is affected and violent inflammatory action results. In this we find the difference between chronic and acute glaucoma. The slow, insidious progress of simple glaucoma is a point in favor of this view, as all forms of glaucoma, including the hemorrhagic, are the outcome of connective-tissue growth, varying in degree of acuteness, or in the active functional tissue first invaded; it may be that the coats of the bloodvessels are first involved, or that there is a binding of nerve fibres by the encroachment of the connective tissue surrounding them, or that there is thickening of the stroma of the choroid, or retina, or even of the inner layer of the sclerotic, which last would, to some extent, shorten the antero-posterior diameter of the eyeball interiorly, producing increasing[1] hypermetropia.

In interstitial nephritis from the same cause, we have an analogue to the condition called glaucoma, with similar constitutional disturbances, and a like morbid process of the cortex and parenchyma. It is of low grade, insidious, and is often unmarked by positive symptoms until the beginning of the end; it always attacks both kidneys, though not usually at the same time, and therefore the disease is seldom at the same stage of progress in both; the progress of the malady has its interruptions and exacerbations, or it may be lighted into more acute action; after each exacerbation the disease is more advanced; step by step, the history of simple glaucoma.

Hyperplasia of the connective tissues offers an unforced explanation of all the sympathetic and anatomical phenomena of glaucoma: the so-called prodromata—impaired accommodation, premature presbyopia, increasing hypermetropia, halo, fogginess of vision, and heaviness of the brow; the local symptoms of an attack—increased intra-ocular tension, circumorbital pain, peri-corneal injection, protrusion of the globe, sluggish dilated iris, anæsthetic cornea, lachrymation, photophobia; the anatomical peculiarities—excavation of the disk, obstruction of the channels of filtration, the white ring of the papilla; the complications—

[1] Within the past few years, I have seen two cases of primary glaucoma in women aged fifty-three and seventy-one years), in which, without any observable change in the anterior section of the eyeball, *myopia* has developed and increased precisely as in the progressive myopia of early life. The individuals read more comfortably without convex lenses, and require concave lenses for distance. There is excavation and increased intra-ocular tension in both eyes in both cases, the two eyes of each person being different in depth of excavation and the degree of myopia. The vitreous appears to have receded instead of being advanced, and the eyeball is distended posteriorly, as in staphyloma posticum. Choroidal atrophy has not yet appeared: —3 D′ on the worse eye gives V. = 20/xx.

If any operation would serve here, it must be *posterior sclerotomy*, in which event the same operation would serve in staphyloma posticum.

These cases may be the exceptions which prove the rule.

coloboma iridis, total aniridia, nephritic retinitis, detached retina, high degrees of staphyloma posticum (with reduced tension). Wells[1] is my authority for the statement that Donders claimed inflammation to be not an integral part of glaucoma, but an unessential complication.

The constitutional disturbances are due to the constitutional cause of the interstitial process.

In fulminating glaucoma the irritation has been sudden and violent in its action (possibly urate of soda); in the hemorrhagic form the walls of the bloodvessels have first been affected and rendered brittle; in the chronic, irritable form the irritant has been mildly persistent, with periodical accessions of intensity. All are forms of one process, due to one cause, unless the disease is unilateral and the cause clearly some local injury; even then a predisposition exists.

Anæsthesia corneæ is not so great in degree or in the area involved in acute, as in chronic glaucoma; in acute glaucoma it may be limited to some one part of the cornea, while in the chronic form the whole cornea is about equally affected. If this symptom were due to high tension, it would be more pronounced in acute glaucoma; that this is not true, favors the idea of an invasion of the canal containing the nerve by new tissue in the chronic affection.

The *bluish-gray tint* of the media may be caused by the presence of an increased number of wandering cells or by exceeding fine fibrillæ of new-formed connective tissue, or by the presence of uric acid crystals.

Obstruction of the channels of filtration is not a cause of glaucoma, as seems to be understood by those who favor the "filtration theory," nor does this obstruction come from the interior of the globe by the advancement of the iris and its application to the posterior surface of the cornea. This may occur, but it is *secondary* to obstruction of the channels. If obstruction took place by application of the iris, the process would complete a "vicious circle," and two things would be expected which do not happen: (*a*) the exits being closed, the pressure of fluids would be toward the posterior channel of excretion (Leber) near the optic nerve, with *deepening* of the anterior chamber; (*b*) the channels being closed by the application of the iris to the posterior surface of the cornea, "thus establishing glaucoma," the obstruction and increased intra-ocular tension, in the nature of things, would act and react upon each other, and the condition would grow worse until relieved by (mechanical) surgical interference. Is this the case? "These slight attacks of glaucoma, often lasting only a few hours, clearly disprove the distention theory.[2] How is that theory to be reconciled with the fact that the attacks occur suddenly, and soon afterward disappear spontaneously?

[1] Wells, vid. sup., p. 583.

[2] Schweigger: Archiv f. Augenheilkunde, vol. xxiii.

How can the filtration at the angle of the chamber be so suddenly interrupted and so wonderfully soon re-established?"

Does not reason rather lean toward the following?

Scleral changes may be found in all cases of primary glaucoma, hypertrophy with increased rigidity, which begins always in the posterior portion of the sclerotic, about the entrance of the optic nerve (Alt), advances gradually to the corneo-scleral margin and encroaches upon the channels of excretion. Such changes, beginning posteriorly, reduce the size of the emissaries as they pass through the sclerotic and cause venous stasis: this, with the diminution of space in the vitreous chamber by hypertrophied sclera, causes advancement of its contents, and may lessen the depth of the anterior chamber without increasing the intra-ocular tension, as sometimes happens. The neoplastic connective tissue, during the interval, invades the optic nerve, and all is prepared for an increase of tension as soon as the morbid tissue so occupies the channels as to *slow* excretion. Now, some circumstance (an injury, mental emotion, exposure, imprudence in diet) violently disturbs the circulation (local or general), and precipitates an attack of glaucoma. Or, no such cause acting, simple glaucoma is developed with its whole train of symptoms.

In case of acute glaucoma, the vascular disturbance over, the engorgement passes off, leaving the channels much as they were immediately before the attack, which could not happen if the obstruction were due to the application of the iris to the cornea. Iridectomy probably acts by promptly relieving such engorgement; myosis, by creating a diverticulum. The narrowing of the channels by hyaline[1] and epithelial masses, or by cicatricial contraction, is of more permanent character, does not belong to the early stage, and cannot be cured by iridectomy, or any other operation anterior to the vitreous. Only the *result* of the operation can, at times, decide the character of the obstruction.

Dilatation of the pupil (which is not always present), which varies in degree and regularity with the same degree of tension, depends upon constriction of the motor filaments to the iris from the third nerve, as they pass through the thickened sclera.

The shallow anterior chamber is a result of the advancement of the corpus vitrei caused by venous stasis and the encroachment by the sclera upon the vitreous space (this being possibly a factor in the dilatation of the pupil); for, by scleral hypertrophy and the resulting atrophy of the uveal tract, the secretion of the aqueous humor is diminished and the resistance *ab fronte* is reduced; hence, the very temporary relief from paracentesis of the anterior chamber (*similia similibus*).

Excavation is rare in a first acute attack, and therefore *cannot* be entirely due to pressure, which is sometimes excessive in such attack. As the disease progresses the resistance at the papilla is lessened by

[1] De Wecker: Ocular Therapeutics, English ed., 1879, p. 260.

the same agent which so effectually degrades the other tissues and permits the effect of pressure to appear; or, the excavation is due to contraction of the new morbid tissue.

"A delicate connective tissue is frequently found in glaucomatous excavations, having been formed in the adjacent part of the vitreous body."[1] "I have not infrequently seen a well-marked glaucomatous excavation on one side, and a neuritis of mild but distinct form, with stretching (?) of the tissue and beginning excavation, in the other eye."[2] Is there any difference in appearance between glaucomatous excavation and that which follows gray atrophy?

In event that intra-ocular pressure alone in glaucoma could produce excavation in a papilla of normal resistance, it would be *funnel*-shaped. This is rarely true; the excavation is usually shaped like a *cup*, or *truncated pyramid*, the width being often greater than the depth, the structure disappearing laterally to a point under a ledge of the sclerotic, which protects it from *direct* pressure. This pathological peculiarity can be due, alone, to neither intra-ocular pressure, nor to modification of tissue in the papilla, but is the product of the action of both. The atrophic process in the optic nerve seems to be caused by the extension and proliferation of fine connective tissue, and its subsequent *contraction*. "There is a continuity of inner connective tissue of the optic nerve with that of the sclerotic and choroid."[3] To this same agent is probably due the grayish-yellow glaucomatous ring.

The acuteness and field of vision are controlled by the same influences, and *pari passu*, which modify the appearance and condition of the papilla.

Arterial pulsation[4] occurs when the normal balance between general arterial tension and intra-ocular tension is disturbed. *Reduction* of blood-pressure takes place when the lumen of the arteries is diminished by infiltrated new connective tissue, as is common in lithiasis, and thus we may have arterial pulsation without increase of intra-ocular tension, or excavation of the disk. *Impure blood excites spasm of the smaller arteries.*

Mydriasis reduces, and myosis increases the area of vascular distribution of the ciliary region. By mydriasis the arterial circulation is suddenly obstructed, resulting in temporary engorgement, which may be sufficient to excite an acute glaucoma if the channels of excretion are narrowed. Hypermetropia favors ciliary engorgement by constant excessive demands upon the ciliary muscle. Therefore, "chronic glaucoma[5] is a neurosis (vasomotor)—a progressive atrophy with the feature of inflammation with defective power; acute inflammatory glaucoma is a paroxysmal expression of the same affection."

[1] Alt, vid. sup., p. 156.

[2] E. G. Loring: Text-book of Ophthalmoscopy, vol. ii., p. 229.

[3] Löwig: Studien des Phys. Inst. Breslau, 1858, p. 125.

[4] Schweigger: Archiv f. Ophthalmologie, Oct. 1891, p. 491, 492.

[5] Richey: Amer. Journ. Med. Sciences, Nov. 1892; Trans. Amer. Ophthal. Soc., 1892.

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