Richey (S.O.)

The Prime Etiological Factor of Glaucoma is Constitutional.

BY

S. O. RICHEY, M.D.,

OF WASHINGTON, D. C.

FROM

THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES,

NOVEMBER, 1892.

Extracted from
The American Journal of the Medical Sciences for November, 1892.

THE PRIME ETIOLOGICAL FACTOR OF GLAUCOMA IS CONSTITUTIONAL.[1]

By S. O. Richey, M D.,
of Washington, D. C.

This paper will be occupied with the presentation of one idea, for a *résumé* of the literature of glaucoma would unprofitably occupy much space, as so much has been written offering diverse views of its different features, each with a show of reason.

Mr. Jonathan Hutchinson, in the Bowman Lecture, 1884, discusses the relation between certain diseases of the eye and gout. The tissues of an individual long subject to the causes of gout may become modified in such a way that they are liable to suffer in a peculiar manner when exposed to the ordinary causes of disease; the *nervous* and *vascular* systems are specially so disposed. Rheumatic gout may have such a relation to true gout, and he names "hot eyes," calcareous bands of the cornea, arthritic iritis, relapsing cyclitis, *glaucoma*, and retinitis hæmorrhagica as having such connection; and asks if it can attack any of the *structures of which the nervous system is composed.*

In the London *Lancet*, January, 1873, he describes an iritis occurring at an early age, differing from other forms of arthritic iritis, in being persistent and insidious, rather than paroxysmal. Without any attack of acute inflammation, adhesions quietly form between the iris and the capsule of the lens. . . . This affection usually begins in but one eye, and advances to almost entire loss of vision in it, before attacking the other. It is insidious, and for the most part painless, but is liable to exacerbations and periods of improvement. It is remarkably intractable, prone to attack both eyes, and to end in blindness.

Such is the position of an acute observer as to the influence of gout upon the eye.

The question as to the *cause* of increased tension is still open, whether due to *too rapid infiltration*, or to *impeded excretion*, with a leaning to the latter.

Opposed to Mr. Priestley Smith's theory, that "glaucoma of every form is essentially a disease of retarded excretion,"[2] are the conclusions of Schnabel,[3] supported by clinical and pathological studies, that "glau-

[1] Read at the meeting of the American Ophthalmological Society, July 20, 1892.
[2] Trans. Seventh International Medical Congress, vol. iii. p. 84.
[3] Archiv Ophthalmol., vol. vii. p. 14.

coma may be present without obliteration of the sinus of the anterior chamber; that the latter can exist without glaucoma; that glaucoma can be cured without obliteration of the sinus of the chamber being removed."

"It has been proved by Mr. Windsor,[1] of Manchester, that acute glaucoma may occur where there is congenital absence of the iris."

A doubt, which reaches almost a denial, is general as to whether excavation of the disc is due to pressure, or not. In Rydell's[2] case, blind from acute glaucoma of three weeks' standing, *without excavation*, pain was relieved and tension reduced, but vision was not improved. Mauthner[3] claims that "We find in the beginning of an excavation that pressure frequently is not increased. I have recently examined the left eye of a patient, in which there is the beginning of a pressure excavation, of which there was not the slightest sign a year ago, when I saw him for paresis of one of the muscles. The functional disturbance is extraordinary, and shows itself in transitory obscurations; central S. is less than in R. E, which has S = 6/VI, while with L. E. a few letters of 6 are not seen at 6 m. distance. Without glasses the patient, who is forty-five years old, reads with R. E., J. 2, with the left eye J. 3 : F. undisturbed. The well-known appearance of the vessels is very marked at the upper lateral edges of the papilla. T. is precisely the same in both eyes, and falls even below the physiological maximum. Would such a pressure produce such a picture?"

"Some morbid process has attacked the intra-ocular end of the optic nerve, causing a diminished resistance (softening) of the lamina cribrosa, so that it yields to even normal pressure in the eye, but at the same time there is going on in the optic nerve an alteration, which has the greatest resemblance to that in the lamina cribrosa, and leads to a softening, to a giving way of the supporting connective tissue."[4]

Reading these comments on glaucoma with a free mind, our previous ideas are subverted, because we must conclude that increased tension is not necessary to excavation; that excavation is not always present, even when increased tension has existed sufficiently long to produce it; that excavation may result from[5] "some morbid process" in the nerve,

[1] A Practical Treatise on Diseases of the Eye, by Haynes Walton, London edition, p. 1170. See Ophthalmic Review.

[2] Von Graefe's Archiv, 1872, vol. xviii. pp. 1–51.

[3] Archiv Ophthalmol., vol. viii. p. 38.

[4] Vide supra, p. 39.

[5] Garrod, on "Rheumatoid Arthritis," Reynolds' System of Medicine, p. 553: "In the early stage, when swelling is prominent, a considerable increase of synovial fluid is found, and the joint exhibits the same appearance as in case of ordinary inflammation. The lining membrane is often red from over-injection of the bloodvessels. If the bone be sawn through, it is often found spongy, and contains a large amount of *oily matter*, from the occurrence of a *species of fatty degeneration*." N.B. All italics are my own.

lessening its resistance; that increased tension is not dependent upon obstruction of the channels of excretion.

If the last proposition be true, that increased tension is *not* dependent upon obstruction of the channels of excretion—and Schnabel supports his conclusion by dissections of the organ which he had observed while affected with the malady—then increased tension *must* be caused by too rapid infiltration, or secretion. Schnabel argues further,[1] that glaucoma is a disease of the bloodvessels of the eye, which develops either gradually, or at once, in the region supplied by the long anterior and posterior ciliary arteries, the central bloodvessels, and those of the sclerotic circle; that the disturbances of nutrition and function are the direct result of these disturbances of circulation, etc.

Mr. Priestley Smith's theory was obviously derived from the study of glaucoma of local origin; and yet, as Mr. Brailey, of London,[2] says, "it fails to account for temporary glaucoma, for glaucoma without the characteristic application of the iris, for glaucoma in young persons, for one-sided glaucoma, for glaucoma in aphakic eyes, and especially for cases where a traumatic dislocation of the lens backward has been quickly followed by increased tension. It does not, also, explain the *invariable inflammation* and *atrophy* of the ciliary body and optic nerve."

To the theory of increased secretion, or more properly too rapid infiltration, a *vis a tergo*, some derangement of the general system, is a *sine qua non*. The uric acid diathesis, of which gout is a characteristic feature in many instances, offers the most satisfactory explanation: true gout, of acute inflammatory glaucoma; rheumatic gout, of chronic simple glaucoma.

In nearly all particulars acute gout of the toe and acute inflammatory glaucoma are alike. Observe the points of resemblance:

Acute Inflammatory Glaucoma.	Acute Gout of the Great Toe.
1. An inherited tendency.	1. An inherited tendency.
2. Most frequent after the period of presbyopia.	2. Most frequent after the beginning of senile changes.
3. First attack is usually in cold weather.	3. First attack, usually in winter, or spring.
4. Premonitory symptoms: Impaired A.; premature presbyopia, increased H.; halo, rising clouds or smoke, heaviness of brow, shooting pains in the eye, increased tension. These may be so slight as to cause no anxiety.	4. Premonitory symptoms may be so slight as to pass unnoticed, or may be very distressing.
5. Sudden seizure, usually at night.	5. Attack is sudden, usually between two and five o'clock in the morning. (Garrod.)

[1] Archiv f. Augenheilkunde, vol. xv. p. 311.

[2] Trans. Seventh International Medical Congress, vol. iii.

Acute Inflammatory Glaucoma.	Acute Gout of the Great Toe.
6. Constitutional disturbances; febrile excitement, with some nausea and vomiting.	6. Chilliness, heat of skin and perspiration, thirst, loss of appetite, a white tongue, constipation, and restlessness.
7. Circumorbital pain, peri-corneal and sub-conjunctival injection, slight protrusion of globe, sluggish, dilated iris; cornea dull and anæsthetic, humors greenish, ischæmia.	7. Toe is swollen, red, hot, and exquisitely tender. Veins proceeding from the toe are turgid with blood, and the joint is stiff. Great tension of the skin.
8. As the attack passes off there is great chemosis, lachrymation, and photophobia. The cornea becomes roughened.	8. As the attack passes off there is pitting of the skin (œdema), then desquamation.
9. The inflammatory attack passes off in a few days or weeks.	9. Duration, from four days to three weeks.
10. The disease is not arrested: there may be a recurrence of acute inflammatory attacks, chronic inflammatory exacerbations, or the disease may progress insidiously.	10. Gout recurs, and the frequency of the paroxysms increases.
11. No pus.	11. No pus.
12. Urine. ?	12. Urine scanty, high-colored, and deposits a colored sediment on cooling.
13. No analysis of aqueous humor, so far as I know.	13. Synovia contains urate of soda.
14. The disease may attack first one eye and then the other.	14. Gout not uncommonly seizes first one great toe, then the other.
15. Occurs most frequently in women.	15. Is rare in women.

Thus, each may be inherited and have the premonitory symptoms; the attack is sudden and at night; in each it is characterized by great pain, engorgement, and tension, followed by œdema and exfoliation; duration, from a few days to a few weeks; recurrence of the affection, possibly to attack the other side, or to become chronic. No pus.

Such is the clinical picture.

That acute inflammatory glaucoma is more frequent in females, and gout of the great toe more frequent in males, may be due to the greater emotional tendencies of women: for, according to Schweigger "mental emotion and loss of sleep favor acute glaucoma."

While women derive a certain immunity from podagra by reason of menstruation (Hippocrates), yet at the approach of the climacteric, a period of greater or less tendency to vascular cerebral disturbance, arising from the intermittence of the derivative action of this function, acute inflammatory glaucoma is most frequent, and chronic simple glaucoma develops.

"The great toe[1] contains a considerable amount of tissues peculiarly liable to become the seat of the deposition of urate of soda; as, for example, the cartilages and ligaments, tissues having either little vascularity, or nourished independently of bloodvessels; the great toe being

[1] Garrod, "Pathology of Gout," Reynolds' System of Medicine, vol. i p. 535.

very remote from the heart, the circulation is weaker there. . . . . The reasons for the great toe on one side of the body being affected apply equally to the other; and hence, the disease not uncommonly attacks first one toe and then the other, within the short space of a few hours or days."

Anatomically, the eye is an extremity of the body, not quite so far from the heart as the toe, and is exposed to variations of temperature and to injury; the sclerotic, the cornea, and the tendons of the extrinsic muscles are of dense fibrous tissue, with little vascularity; the stroma of the choroid and iris is of reticular connective tissue, supporting pigment cells, bloodvessels, etc., the zonule of Zinn is a *fibrous* perforated membrane, the lens capsule is a structureless membrane, the corpus vitrei depends upon bloodvessels not its own for nutrition, and contains mucin, and (Picard) 0.55 per cent. of urea, and about 0.75 per cent. of sodic chloride. The posterior surface of the iris and ciliary body secrete the aqueous humor (synovia?) which contains a small amount of albumin, sugar, and sodic chloride, equal to $\frac{1}{50}$ of its volume.

With increase of blood-pressure and intra-ocular pressure, there is increase of albumin and the production of fibrin in the anterior chamber. (Jessner and Grünhagen.)

Taken with the fact that a local derangement, as a dislocated lens, does not seem sufficient to cause the *whole* train of symptoms, general as well as local, called glaucoma (though it may precipitate an attack which would probably have taken place at a later date), the clinical history of a seizure and the anatomical peculiarities of the regions under consideration present a picture of such mimicry as we find nowhere else repeated. The crucial test, the presence of urate of soda, I have had no opportunity to apply since recognizing the resemblance.

To again read Garrod,[1] "The impure state of the blood, due to the presence of urate of soda, is probably the cause of the disturbance which often precedes the gouty paroxysm; that is, of the so-called premonitory symptoms. Urate of soda in abnormal quantity in the blood is essential to an attack of gout, ... but does not constitute gout; . . . . that the amount of deposited urate of soda is not in proportion to the intensity of the inflammation, and that in some the infiltration may ensue and give rise to scarcely any inflammatory action. . . . . The inflammation of the gouty paroxysm tends to the destruction of the urate of soda in the blood of the inflamed part, and probably of the salt also which is thrown out." Soelberg Wells[2] observes that "males who are attacked by glaucoma frequently suffer from gout, or disorders of the digestive organs:" of primary glaucoma, "when

[1] Reynolds' System of Medicine, vol. i. p. 533.

[2] A Treatise on Diseases of the Eye, 3d Amer. ed., 1880, p. 589.

once the one eye has become affected by glaucoma there is great tendency in the disease to invade the other also."

Mr. Hutchinson[1] asserts that "all forms of rheumatism, and all forms of gout, are included in the common term, arthritic. But we cannot limit the term to the joints, as its etymology might seem to require, but must allow it also to apply to certain affections of the muscles, fasciæ, tendons, and other fibrous structures which have been proved to be dependent upon the same peculiar state of health. . . . . Under the term rheumatism we include all arthritic maladies which are not proved to be gouty. . . . . I must protest, at once, against any attempt to limit the term gout to cases in which attacks of acute inflammation of the great toe occur. . . . . Rheumatism differs from gout in being of *nerve* origin, and due to reflex disturbance of nutrition; . . . . it is, according to my hypothesis, the basic diathesis to which a small minority of cases of gout is superadded."

The younger Garrod says that rheumatic gout lacks the distinguishing feature of gout, urate of soda.

Many of the manifestations of rheumatic gout are associated with chronic glaucoma, viz.: enlarged or distorted joints, a peculiar senile pallor, or muddiness of the skin; periods of mental depression, and other symptoms, attributable only to changes in the nervous system. I have found nowhere any reference to pathological alteration of nerve tissue in gout, although the existence, character, and specific cause of such changes, which are *presumed* to exist because of the nervous symptoms present in lithiasis, would have important bearing upon the subject in hand, in explaining the structural changes in the lamina cribrosa and the intra-ocular end of the optic nerve, the condition of diminished resistance associated with excavation without increase of tension, in cases of chronic glaucoma.

Dr. W. W. Johnston,[2] Washington, D. C., published some thoughts "On the Nature and Treatment of Forms of Disease characterized by Indigestion, the Presence of Bile, Urates, and Uric Acid in the Urine, and by Nervous Symptoms," which suggest a possible cause and explanation of the nerve changes in chronic glaucoma. In his own words, "The question of the continuous production of toxic substances in the intestinal canal in health, and the protection of the organism by physiological elimination, as well as the auto-intoxication of the organism by the absorption of poisons in alterations of the gastro intestinal tract, was developed in detail by Professors Albertoni and Silvia at the meeting of the Fourth Italian Congress of Internal Medicine, held in Rome. Professor Silvia enumerates the following substances as probable

[1] Trans. Seventh International Medical Congress, vol. ii. p. 92.

[2] The Medical News, March 12, 1892.

poisons: peptoxine, organic bases (ptomaïnes and leucomaïnes), indol, phenol, lactic acid, ammonia, sulphuretted hydrogen, acetone, etc. The direct proof of the fact that the nervous phenomena in such cases are due to the absorption of toxic matters from the intestines is not yet found, but the argument is a forcible one. The existence of indigestion is known by the symptoms: the presence of toxic matters in the intestine in health is proved. . . . . The relationship of acute indigestion and nervous disturbances, and the association of fermentative dyspepsia with nervous symptoms, and an excess of these products in the urine and feces, give sufficient grounds for adopting this theory as reasonable."

Dr. Johnston has given much attention to the subject of digestion, and if a reference to his able paper will induce those who have the care of cases of chronic glaucoma to read it, it will probably divert attention from glaucoma, except as a local manifestation of a general malady (although he does not refer to glaucoma), broaden the view of the subject, and enable us to comprehend the changes in nerve tissue going on elsewhere in the system in rheumatic gout, by that which takes place in the intra-ocular end of the optic nerve, exposed to observation, in chronic glaucoma.

Returning to the subject of intra-ocular tension, Mr. Priestley Smith[1] claims that "high tension depends more upon an excess of blood in the eye than upon an excess of intra-ocular fluid," while Dr. Spender[2] has observed, as early symptoms of arthritis, increase of pulse rate with high arterial tension.

Mr. Hutchinson[3] concludes that "it is probable that there are many different forms of inflammation of the eye, or of parts of it, which are in connection with gout. They may be divided into two groups: *a*, those which go with acquired, humoral, or renal gout; *b*, those which depend upon inheritance of structures damaged, or, at any rate specialized, by gout in predecessors. The difference between the two classes of affections is very marked. In the one, attacks of a transitory nature are the rule, and the attacks are often acute and attended by much pain. In the second group, although a tendency to temporary recovery and recurrence is often observed, yet, there is a great proneness to chronicity, and persistence. The invasion is often insidious, but the disease is usually in the end destructive."

If the difference between the forms of acute inflammatory and chronic simple glaucoma had been in the mind of Mr. Hutchinson the description could not have been more effective than in the specification of the two groups named above.

[1] Ophth. Rev., vol. vi. p. 196.

[2] Garrod: A Treatise on Rheumatism. Am. ed., 1890, p. 245.

[3] Ophth. Rev., vol. iii. p. 385.

His address will bear reading with this thought.

Ordinarily, when both eyes are attacked by the same disease process, we rationally conclude that the cause is constitutional, and do not treat an expression of the dyscrasia, but rather its cause.

In chronic glaucoma, a local manifestation is treated (for, sooner or later, both eyes are attacked), and then we wait to see what "turns up," with about the results presented by Dr. Bull,[1] of New York, to the American Ophthalmological Society, in 1889; the detailed history of ninety cases of chronic simple glaucoma, subjected to the operation of iridectomy, during a period of seventeen years. The paper is most interesting and instructive, especially the summing up: "One hundred and fifty-four operations were done on the one hundred and eighty eyes under consideration. Vision was temporarily improved by iridectomy in both eyes in two cases, and in one eye in six cases; but in all eight cases, after a few months, a steady loss of vision and narrowing of the field set in, and continued progressively as long as the patients were under observation.

"Vision remained unchanged, neither better nor worse, after the operation, for a period of one year or longer, in both eyes in eight cases, and in one eye in twenty cases.

"Vision grew slowly and steadily worse after the operation, in both eyes in forty cases, and in one eye in twenty-nine cases.

"Vision grew rapidly worse after the operation, in both eyes in two cases, and in one eye in eight cases."

He concludes that "the health and age of the patient exert a decided influence upon the operation, and any marked evidence of senility is distinctly unfavorable to the operation."

Dr. Gruening,[2] of New York: "In chronic glaucoma with degenerative changes, neither iridectomy nor anterior sclerotomy will give the patient the desired relief; posterior sclerotomy *may do it at times.*"

Mr. Power,[3] of London: "In cases of chronic glaucoma no operation is of much service." This terse statement, it seems to me, covers the whole ground.

The good results of operation in chronic glaucoma are in comparatively small ratio, and are therefore accidental, and not scientific; for it often precipitates disaster by additional irritation. So long as the two chief clinical characteristics of glaucoma, increase of tension and excavation of the disc, are not satisfactorily explained, the management of such cases must be empirical. The author of iridectomy for glaucoma acknowledged it to be empirical, and only experience has taught us in

[1] Trans. Amer. Ophth. Soc., vol. vi., part 2, pp. 246, 291.

[2] Ibid., 1889.

[3] Trans. Seventh Internat. Med. Congress, vol. iii. p. 106.

what cases it is of most value, those of acute inflammatory glaucoma; for here it saves the eye until another time; it does not cure the disease. Dr. Bull's statistics do not teach us to do iridectomy in chronic glaucoma, cases of which form of the disease are in excess of any other, unless upon the plea of *dernier ressort*—because we know of nothing better. They indicate that the majority of eyes are worse after an operation; in a few the *status quo ante* is maintained; in a still smaller percentage there is some improvement. With this diversity of result, who, save in the occasional case of *immediate* gain, or loss, to the eye, can say what influence is attributable to operation? Might the case not have done just as well without interference? Is the surgeon justified in a feeling of certainty that he has done a service? If all such cases followed *approximately* a given course he would have a guide; but they vary so much. If it progresses slowly after an operation, it might have done so without it. If it remains stationary for a time, can that be attributed to operation? If the patient goes rapidly blind, has he a right to reproach the surgeon? In operation is *possibility*, not *probability*. In simple glaucoma it has a questionable rationale, and experience teaches that, if done at all, it must be done with caution. It is double-edged, and may cut either way.

It is a prime necessity that a quiet, healthy, out-door life should be led, apart from occupations of much nervous excitement, causing loss of reserve force; that a condition of self-possession should be maintained; that the dietary should be regulated as to time, quantity, and quality; for over-feeding and bad feeding is a conspicuous vice of the age. In adult life the effort should be to preserve the balance between waste and repair, and to see that both processes are normal. This is a duty which the family physician may share.

As such cases pursue so chronic a course, it would seem wise to discover the constitutional cause, and to begin with that, instead of with the last expression of the disease, leaving the cause in action.

Rational management of the disease involves a study of the general condition and a correction of all the habits of the individual. This is difficult, but our function is advisory, and each sufferer must "work out his own salvation" with our guidance.

By controlling the quantity of food productive of uric acid, and by reducing the whole quantity to the possibility of easy digestion and assimilation, thus lessening the amount of toxic substances in the intestinal tract; by the regular entire excretion of what is excessive by way of the kidneys and bowels, harm in this way is obviated. Tonic aperients (not irritants and excitants), which encourage natural action of the intestines, serve a good purpose when used with judgment. Hunyadi water, taken at bedtime, lies in the tract all night, does not purge, but by its solvent power prevents accretions. Nothing should be

done to lessen the digestive power, and a quantity of food should be taken, small enough to *insure* its digestion and proper disposal. Anything (as coffee) which retards digestion must be rejected for obvious reasons.

Salicylate of phenol, it is claimed, has been found in the joints of gouty persons taking it; therefore its purpose is apparent.

Lithia waters secure the excretion of some uric acid; piperazine, a new synthetical compound, is commended as having twelve times the solvent power of lithia upon uric acid. Strychnine acts by stimulating the functional activity of all the organs of the physical economy.

Galvanism, if properly and steadily used, is profitable. After ten minutes' use of two milliampères direct current to the sympathetic, in an ordinary case, ocular tension is lessened, the pupils seem more active, and the patient becomes calm, often almost falls to sleep. By the experiments of Onimus and Legros[1] it has been shown that if the direct current (positive pole at the nerve centre) be employed, the circulation is augmented; within a few moments the arteries have increased in bulk, and the whole network of capillaries is seen in great commotion. Faradization contracted the bloodvessels, but after a time contraction ceased, and the arteries became larger than before the application. The continuous current, on the other hand, renders circulation more active, and reëstablishes it when it has been arrested. The induced current causes spasmodic contraction of the unstriped muscle, while the continuous current produces a *vermicular* contraction (Bartholow). The latter thus favors the natural movement of the vessel, and while *directly* increasing the amount of blood passing, by reaction the amount of blood in the part supplied by the vessel is reduced to the normal. The object to be gained, stimulation of the cervical ganglia, the trophic centres of the region of the trigeminus, is accomplished as well with the cathode held in the hand as in contact with the affected region; yet, when placed on the temple, or above the eye, it has some additional *mental* effect, which is not undesirable.

The writer has endeavored to cover the ground as concisely as possible: to offer the salient points of a view of the subject he has entertained for several years, especially in regard to *too much food*. He thinks that in the hypothesis discussed we find the true etiological factor of the most intractable of diseases, chronic glaucoma; that acute inflammatory glaucoma is a paroxysmal expression of the same affection; that local irritation, or trauma, excites an attack of glaucoma only in the presence of the dyscrasia; that operation saves the eye during a paroxysm; that operation serves little purpose in chronic glaucoma, even when it does not, by irritation, hasten the disease process or precipitate a paroxysm;

[1] Traité d'Electricité Médicale, Paris, 1872.

that chronic glaucoma is a neurosis—a progressive atrophy with the feature of inflammation with deficient power, varied by periods of *seeming* rest; that correcting and controlling individual habits, especially in the *amount* and character of food taken, will do more to preserve vision than operation; and that there may be a possibility of aborting chronic glaucoma, if the tendency to it be recognized at an early stage.