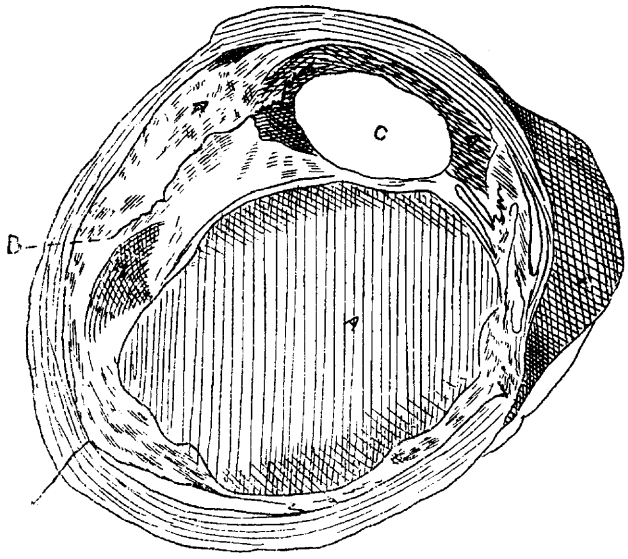
treated for acute mania and was restored to his normal condition in a couple of weeks. After his attack in Seattle he drank no spirituous liquors for a year, and had on previous occasions abstained four or five years at a time, for he had found by experience that he could not use liquor at all without using too much. During the past year he had been using Warner's Safe Cure, thinking that his kidneys were out of order. For the past three months his health had been poor; everything that he did required an effort and ten days before the accident in order that he might keep up with his work he took some wine. In a few hours he was drunk from the amount of port wine and whisky that he had imbibed, and for seven days remained intoxicated, eating nothing; at the end of that time he drank four bottles of ginger. He was cold to his knees, his jaws were set and he could not move his arms. After the first drink he seemed to be irresponsible and had no idea of how time passed. After the seven days' spree he stopped drinking absolutely, but was troubled with hallucinations and illusions, against which he struggled for twenty-four hours. Sunday morning he was feeling so weak and



A, hemorrhage: B, choroid and retina; C, lens. Retina was also found in hemorrhage.

miserable, with no passage of the bowels in five or six days, he commenced on a bottle of Fellows' hypophosphites and in twenty-four hours had used 12 ounces of it, which represented 1 3-5 grains of strychnin. Sunday night he was still feeling so distressed that he called in Dr. Clark, who gave him sufficient codein (I believe) to assure him a good night's rest. Monday he was very excited, and when they brought soup to him at 1 o'clock he would not eat because he thought it was poison. While lying on the lounge he had a spasm of the stomach and with the spasm a blackness came over his eyes; he fell from the lounge, but saw the carpet as he fell. He thought that he had gone to hell and that his brain was full of little devils. With the great pain of the spasm he lost control of himself and he tried to get his fingers into his brain through his eyes, but that caused so much pain that he tried to reach his brain through his ears, then he recollected nothing more till an hour and a half later.

After the eyeballs were removed they were hardened in Wickersham's solution and later in formalin. They were carefully cut, stained and examined microscopically by Dr. Alex. Bruce of Edinburgh. Both the eyes were practically the same, and the accompanying diagram is a drawing from one of the sections. This shows the parts very much displaced, the aqueous and vitreous spaces filled with blood, subconjunctival hemorrhage and blood separating choroid and retina from the sclera, with parts of the retina in the hemorrhage. But this gives us no definite information as to the cause of the accident. The pathologist after hearing the imperfect history writes that "the condition was a result of a sudden thrombosis of the sinuses, and of this condition I understand there are some cases on record."

Personally, I am unable to explain the cause of the conditions satisfactorily, although I incline to the theory of his having gouged them out with his thumbs or fingers, but if he did I do not understand why the intra-ocular tension should have been so great. If due to double aneurysm, why was there no aneurysmal bruit and no pulsation, and if due to double thrombosis of the sinuses why should there not be more general disturbance?

REMARKS ON THE CAUSES OF GLAUCOMA.

Read in the Section on Ophthalmology, at the Forty-seventh Annual Meeting of the American Medical Association, at Atlanta, Ga., May 5-8, 1896.

BY LEARTUS CONNOR, A.M., M.D.
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Judged by its literature, the causation of glaucoma is unsettled. No effort to harmonize undoubted facts has met with general support. Whether glaucoma is a deformity or a disease, remains an open question. Thus Priestly Smith and his followers claim that glaucoma is a deformity of the eyeball, that an engorgement of the blood vessels in the posterior chamber pushes forward the lens and crowds the ciliary body and iris into the anterior angle, effectually blocking the outlet from the eye for the intraocular secretion. From the resultant intraocular tension he deduces the phenomena of primary glaucoma. The failure of this view to account for glaucomatous attacks in the young, in persons having no iris, in cases of intraocular tumor or dislocated lens, etc., has prevented its universal acceptance.

Of those holding that glaucoma is a disease, part affirm that it is purely local; and part that it is the local expression of a constitutional affection.

Von Graefe called simple glaucoma "amaurosis with excavation," regarding it as quite distinct in origin and course from glaucoma. Iridectomy failed to exert any appreciable influence upon its progress, in contrast with its wonderful power in checking the destructive force of acute inflammatory glaucoma.

Previous to his time glaucoma was regarded as a local inflammation, and treated with antiphlogistics, or as an arthritic disease; the result was the same in either case, viz.: total loss of vision. Not content with the empirical fact that iridectomy would cure a glaucomatous outburst, ophthalmologists have sought out its mechanism and the processes leading to it. The questions raised by this study are most intricate, far-reaching and difficult of solution. Thousands of workers have each contributed something of a positive or negative nature, but much still remains undone. The object of this paper is to briefly discuss a few points bearing upon the causation of glaucoma.

1. An obstruction to the outward flow of the fluids

in the posterior chamber is the most uniform characteristic of an acute glaucoma. Hence the importance of a clear idea of the normal flow of these fluids, and the changes made by the glaucomatous disease. The course of the blood is readily determined, in health, by the course of its vessels. Elaborate experiments have seemed to show that the oblique course of the efferent veins, through the sclerotic, combined with such disease of the veins as partially occluded their caliber, obstructed the outflow and so increased the intraocular tension, but this view has not obtained large recognition. That a variation in the caliber of the blood vessels does occur in glaucoma is evident, but all the facts seem to place these secondary to obstruction to the outflow of the secretions.

Knies, Webber and others have experimentally shown that the aqueous is secreted in the posterior segment of the eye and passes anteriorly through the connective tissue of the circumlental space, Fontana's spaces, Schlemm's canal, and the sclerotic into the capsule of Tenon. In this course the outflow is through connective tissue, and tissue matrix, not reaching spaces lined with epithelium until it enters the capsule of Tenon and the efferent lymph spaces of the subconjunctival tissue. Knies has shown that dissolved substances may in addition pass through the lens capsule and Descemet's membrane, but not formed or solid matters. Posteriorly, he has shown that the secretion of the posterior chamber escapes through the connective tissue spaces of the optic disc. The obstruction to the outflow of this fluid by a change in its nature has been the subject of elaborate experiments by Knies as detailed in *Arch. Oph.*, Vol. xxiv. Aseptic irritants were injected into the posterior chamber, that formed coagula in the secretion which obstructed the outflow spaces, producing an increase of ocular tension, dilatation of the pupil, and cloudiness of the center of the cornea. On removing the eyes he found both anterior and posterior outlets obstructed by the changed secretion. Hence he thinks that glaucoma is an irido-cyclitis, that secretes a noxious fluid, which clogs the meshes of the outflow connective tissue spaces. Experimentally this change of the secretion in the vitreous was temporary, but in the irido-cyclitis of glaucoma, he thinks that the secretion of noxious fluid is of longer duration and so the symptoms of glaucoma longer continued.

The study of glaucomatous eyes which have been removed, before total destruction, has shown an actual change in the connective tissue spaces of the outflow channels. The fibers have been found thicker and shorter, encroaching upon the spaces, and often entirely obliterating them. It is not possible to remove an eye in the early stage of glaucoma, or to find one in a person dying of acute disease, but in those studied nearest such a time, the changes in the outflow spaces have been found. Other changes have accompanied these, but we pass them for the present as we desire to state the fundamental idea in its simplest form. When we examine carefully the persons having glaucomatous attacks, we find that in many there is evidence of gout, either acquired or inherited; of rheumatic gout, rheumatism, syphilis, of those whose entire bodies have been surcharged during many years with badly assimilated products. In many of these we find evidences of interstitial diseases in other organs than in the eye. It is quite fair to infer that the impure blood produced by these constitutional diseases, may affect the connective tissue outlets. Given then an obstruction of the connective tissue outlets of the posterior chamber by an ophthalmitis affecting the connective tissue, due to impure blood, and any one of the so-called exciting causes of glaucoma may so change the secretion of the posterior chamber as to clog the outflow spaces, and so induce a glaucomatous attack. Our knowledge of general, so-called, blood diseases, added to Knies' experiments, and well-known studies of glaucomatous eyes, makes a rational chain of events leading up to an attack of glaucoma.

2. The following considerations place this causation of glaucoma in a clearer light. Thus outbursts of glaucoma are most frequent during and following middle life. This fact admits of two interpretations. *First*, Priestly Smith argues, that the lens becomes hardened and enlarged by age, so that it more entirely fills the circumlental space. Farther, he thinks that glaucoma occurs mostly in the hyperopic, in eyes in which this space is already abnormally small. In such an eye a relatively slight disturbance of the circulation of the posterior chamber suffices to push the lens forward, to crowd the iris against the anterior angle of the eye and to prevent the outflow through Fontana's spaces. The so-called causes of glaucoma, according to this view, produce an engorgement of the blood vessels of the posterior chamber, and so push the lens forward, when owing to a deformity of the parts the anterior angle is closed, and a glaucomatous attack induced. *Second*, a broader view of the relations of glaucoma to age, adds to the changes in the lens, changes found in other portions of the body. One has but to make a very superficial study of individuals during and after middle life to find that all the conditions are present for the promotion of connective tissue degenerations, as well as vascular, secretive and nervous. Individuals continue to eat as when they needed food for growth and development, as well as for the actual work of each day and the maintenance of existence. This excess of food fails to be perfectly digested, and so never becomes a part of the living tissues, but is driven about the blood, lymph and secretory spaces, in its course disturbing the wholesome activity of each part. Secretion and excretion are more or less disturbed, so that effete materials are retained within the body, thus increasing the disturbing and dangerous elements of the circulating fluids. When the connective tissues have been subjected for years to this constant irritation, and auto-infection it is not surprising that there results a group of interstitial or glandular diseases. Certainly we ought not to be surprised, if a few among the whole mass suffered from a degeneration of the outflow connective tissue spaces of the posterior chamber, and so became liable to an outburst of glaucoma, when some especial cause rendered the secretion of the posterior chamber, so noxious as to occlude the contracted spaces.

Another factor of age, intensifying that already mentioned, is the diminished muscular activity of advancing years, by which the utilizing of excess of food is diminished, secretions and excretions made less active, and so the accumulation of impure blood accelerated. Many other elements might be added, all proving that impure blood is the vice of middle life, a vice dependent upon numerous physical, mental and moral factors, but all laying the foundation for some organic disease. Why in one, interstitial nephritis occurs, in another interstitial hepatitis, in another

spinal sclerosis, and in another that disease of the connective tissue outflow spaces of the posterior chamber, is unknown. Possibly the same individual might have the entire list if he could live long enough.

Since diminished spaces in the connective tissues of the outflow channels is the factor which makes operative the so-called causes of glaucoma, then a child may inherit from gouty or rheumatic parents such diminished spaces, and so be attacked with glaucoma.

3. But aside from age, certain diseases are certainly closely allied to the causation of glaucoma. In several recent papers Dr. Richey has admirably discussed this point, showing the relation between the manifestations of gout, gouty rheumatism, etc., and glaucoma. He regards simple glaucoma as the basis of all forms of primary glaucoma. In each an interstitial ophthalmitis exists, originating as other interstitial diseases. The varied forms of the disease he ascribes to the part of the eye attacked, the rapidity of its progress and the order in which different portions are disabled by the disease.

In simple glaucoma the connective tissue in and about the optic disc is first affected by the noxious elements; contraction of the tissues follows, leaving more or less extensive excavation of the optic disc, and destroying in varying degrees the optic nerve fibers. The central retinal vessels drop to the sides of the excavation, and by their peculiar appearance, when seen by the ophthalmoscope easily prove the presence of the excavation. If in such a case the anterior connective tissue spaces become constricted, a slight disturbance of the secretion of the posterior chamber may suffice to clog the outflow, and produce a case of acute, sub-acute or chronic inflammatory glaucoma, according as the obstruction be sudden and complete or slow and incomplete. The fluid pent up in the posterior chamber pushes forward the lens against the posterior wall of the cornea, the ciliary body against the anterior angle; dilates the iris, renders it immovable and crowds it into the anterior angle; increases the tension of the eye ball; renders the cornea steamy and insensitive; dilates the anterior ciliary vessels, and induces a group of subjective symptoms, varying with the stage and degree of the blockage. In hemorrhagic glaucoma the events already noted are complicated by the rupture of weakened blood vessels within the posterior chamber.

In many cases of acute glaucoma there is no cupping of the optic disc because its connective tissues have not been attacked, the ophthalmitis being limited to the anterior outflow spaces. It would seem that the relationship of chronic humeral diseases to glaucoma is very important.

Dr. Jonathan Hutchinson (*Oph. Rep.*, Vol. ii) says that there are many different forms of inflammation of the eye, or parts of it, which are in connection with gout and gouty rheumatism. He divides them into two groups: 1. Those which go with acquired humoral or renal gout. 2. Those which depend upon inheritance of structures, damaged or specialized by gout in predecessors. The differences between these two classes is very marked. In one the attacks are of a transitory nature, acute and attended with much pain. In the second group there is great tendency to chronicity and persistence, though there is a tendency to temporary recovery and recurrence. The invasion is often insidious, but the disease is usually in the end destructive. These two groups admirably describe the characteristics of acute and chronic glaucoma.

If we seek the mechanism by which these diseases induce glaucoma we have but to remember that in them the connective tissue outlets of the eye are constantly bathed in the secretion formed from impure blood, just as we found in the case of the over-fed, badly fed, over-worked, under-exercised person of middle life or beyond. In both the connective tissues of the outflow spaces undergo organic changes that admit of their readily clogging, as the secreting fluid of the posterior chamber is altered by any one of the many existing causes of the glaucomatous outburst. Dr. David Webster (Trans. Amer. Oph. Society, 1885) reports a case in which a glaucomatous outburst followed a very hearty meal, and another from the eating of a peach. Dr. S. D. Risley reports a case in which certain acid fruits would surely excite such an attack, the special fruits were the peach and grape.

The one condition present in all cases of primary glaucoma is a contraction of the outflow channels; the one cause of this contraction is a hyperplasia due to the long continued irritation of the secretions from impure blood; the disorders inducing such blood are many, to some of which we have called attention, as the pollution incident to over-eating in middle life, gout, gouty rheumatism, syphilis, etc.

The one factor which is essential to the precipitation of an outburst of glaucoma is such a change in the secretion of the posterior chamber that it will clog the meshes of the outflow spaces. This changed secretion may be due to many causes, as imprudence in diet, to mental worry, violent emotion or shock; to overstrain of the eye, as in uncorrected astigmatism or presbyopia; to circulatory disturbances, as cardiac weakness; chronic cough, vomiting, stooping or straining; to mydriatics, etc. No one of these events has been known to induce, in a sound eye, an outburst of glaucoma; otherwise they would be of common occurrence, because these events are incident to the course of nearly every person's daily experience.

CONCLUSIONS.

1. Glaucoma is a disease rather than a deformity, like hernia or astigmatism.

2. The disease glaucoma has two distinct factors: one inducing changes in the connective tissues of the body, including the outflow spaces of the posterior chamber of the eye; the other so altering the secretion of the posterior chamber that it clogs the crippled outflow connective tissue spaces.

3. The first factor results from the long continued action of impure blood upon the connective tissue elements of the outflow spaces, the second from a variety of conditions acting through the nervous vascular, digestive and muscular systems, or through local strain of the eye, as in presbyopia, uncorrected astigmatism, or intraocular tumors, dislocated lens, or from the use of mydriatics.

4. Impure blood may result from many distinct diseases, as gout, rheumatic gout, syphilis, or from chronic overloading of the body with food in excess of its assimilative powers.

5. In simple glaucoma the connective tissue at the optic papilla is so attacked as to induce an excavation of the optic disc. If the anterior outflow space remains patent, there may be no symptoms except diminishing field of vision and the excavation, but if the anterior outflow becomes obstructed, increase of

tension and other glaucomatous symptoms appear.

6. In acute inflammatory glaucoma the anterior outflow spaces are suddenly closed, inducing all the typical symptoms of a glaucomatous outburst.

7. In sub-acute glaucoma the obstruction to the anterior outflow is less sudden and complete, and hence the symptoms are less severe and startling.

8. In chronic inflammatory glaucoma the obstruction is more complete and permanent, and so the effects are more hopelessly destructive—glaucoma absolute marking the final stage when vision is totally lost.

9. In hemorrhagic glaucoma the outburst is complicated by rupture of weakened intraocular blood vessels, so that hope of relief is slight.

10. In secondary glaucoma the same obstruction to the outflow channels occur. This may be due to an intraocular tumor, a dislocated lens, a lens swollen after decission, or to occlusion of the pupil, etc.

103 Cass Street.

SOME QUESTIONS RELATING TO GLAUCOMA.

Read in the Section on Ophthalmology at the Forty-seventh Annual Meeting of the American Medical Association held at Atlanta, Ga., May 5-8, 1896.

BY ALEX. W. STIRLING, M.D., C.M. (EDIN.), D.P.H. (LOND.).

ATLANTA, GA.

I propose to bring forward for discussion certain parts of the subject of glaucoma, which though far from novel, yet appear to merit, from their importance and the still divergent views concerning them, a constantly recurring investigation. These questions are:

1. Is glaucoma always preceded by papillitis?

2. Are certain doubtful cases of chronic glaucoma with an apparently normal condition of the anterior segment of the eye, unaccompanied by any sign of spinal and cerebral disease, but showing what is known as the true glaucoma cup; are these really glaucoma, or are they optic atrophy?

3. Should all cases of primary glaucoma be operated upon?

4. What is the cause of the cataract which occasionally appears in eyes which have just undergone operation for glaucoma?

Question 1.—Those who hold that papillitis is a constant accompaniment of the early symptoms of glaucoma should be divided into two classes, viz., those who assert that it actually precedes the glaucoma, and those who refer to it merely as invariably present in the initial stages of the disease.

An abnormal condition of the blood vessels on the disc, with edema, is described by many well-known authors, and is no doubt familiar to most as a symptom of confirmed glaucoma, and may be dismissed as a possibly perfectly natural result of the glaucoma pressure.

But it is otherwise with the assertion that papillitis is a constant precedent of glaucoma. Klein supported Jaeger in this contention; Mauthner[1] thought the optic nerve was so softened by a morbid process secondary to choroiditis as to cup later under normal tension; Brailey and Edmunds[2] had reason to believe that in primary glaucoma "neuritis" precedes the increased tension, and that an inflammation is also present in the ciliary body and iris; Gruening[3] recognized a congested disc as visible along with the first premonitory symptoms in certain cases; Knies[4] has observed: "Among the earliest and most characteristic appearances . . . I found marked hyperemia and edema of the optic nerve. This appears to be the regular beginning which passes in weeks into cupping"; Bitzos[5] asserted that "one can accept as an absolutely certain fact" that glaucoma begins "by a papillitis constituting a lesion which is primary and at the same time fundamental," and that during this stage, before the advent of *plus* tension or of any outward sign, the cases being frequently discovered by accident, there is diminution of visual acuity and in the dimensions of the field.

If we admit that glaucoma is always necessarily preceded by optic neuritis (which has certainly not been universally observed), it practically follows either that the condition of the optic nerve blocks a path of exit for fluids from the eye in such a manner as to cause forward displacement of lens and iris, with secondary obliteration of the normal filtration area in the corneo-iritic angle, or that glaucoma is an inflammatory disease with iritis secondary to, or accompanying, the papillitis. Now, experiments have shown that only a very small proportion of the ocular fluid leaves the eye in the region of the optic nerve; and we also know that in ordinary undoubted optic neuritis glaucoma may be said never to follow either in the inflammatory or atrophic stage; and even if we were, for the sake of argument, to admit that, normally, a large amount of fluid has its exit in this region, it would yet be hard to see why its blockade should cause that adhesion between cornea and iris so nearly universal in glaucoma. We should rather expect merely a more rapid drainage at the angle.

Against the theory that glaucoma is an inflammatory disease and that the corneo-iritic adhesion is due to an iritis, a theory upheld especially by Knies, one might quote the opposite opinion of Bowman, expressed thirty years ago, and of De Wecker and many others since then, as well as that of the elder Critchett, Priestley Smith and Fuchs, that it much more nearly resembles a strangulated hernia. The following facts might also be arrayed against the inflammatory theory: Glaucoma with glaucomatous cupping, found in cases of mechanical obstruction to outflow through the corneo-iritic angle from affections limited to the anterior part of the eye, as from dislocated lens, in which case the tension may rapidly rise and fall according to the position of the lens, or, from hereditary ophthalmia, the result of malformation in or near the angle of the anterior chamber; the suddenness of the early attacks and their disappearance; the fact that an operation, and that on the anterior part of the eye, can quickly and permanently remove the symptoms; as well as the fact that a drop of some mydriatic may cause an acute attack in an eye previously absolutely free from inflammation.

Question 2.—The cases referred to are those designated "amaurosis with optic nerve cupping," by von Graefe; "glaucomatous optic nerve disease," by Edward Jaeger; and "excavation atrophy," by Stellwag, and which have been by some ophthalmologists, separated from other forms of glaucoma, but by others of equally high repute held to be merely one extremity

[1] Mauthner: Archiv. Ophth., N. Y., vii, 1878, p. 178.
[2] Brailey and Edmunds: London Ophth. Hosp. Reports, x, 1880, p. 86.
[3] Gruening: Trans. Am. Ophth. Soc., 1889.
[4] Knies. Centralblatt f. All. Path., April, 1890.
[5] Bitzos: Annal. d'Ocul., vol. 112., 1894, p. 92.