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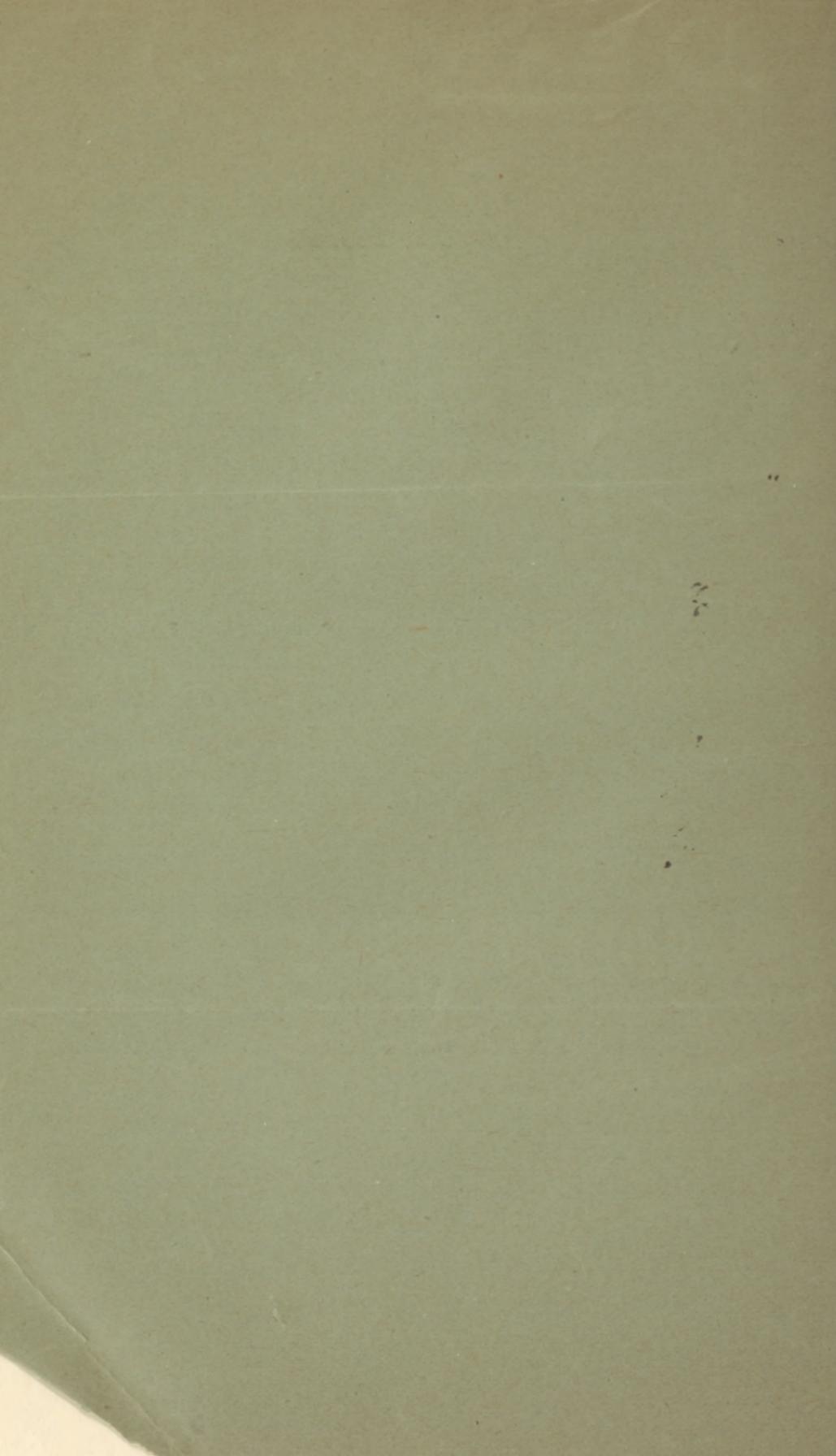
The Etiology and Early Management
of Glaucoma.

—BY—

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THE ETIOLOGY AND EARLY MANAGEMENT OF GLAUCOMA.*

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Although much attention has been given by authors to the subject of glaucoma, a great deal of the mysticism of the past has clung to it, causing most observers to class it with the inscrutable; while the few ventured to explain it by fine-spun theories.

Believing that the theories of the causation of this important disease of the eye which I learned from the text-books of a dozen years ago or more and that which I heard taught in Vienna in 1887, must, from their very nature, be nothing better than fragmentary, I have been making some observations independent of theory, either dignified by age or newly preconceived.

If the conclusions arrived at should—as I venture to hope—throw some light upon the etiology of the disease, give definite aim to the labors of the pathologist, and lead up to a more rational plan of prophylaxis or treatment than has prevailed in the past, I shall feel more than repaid for the effort expended in the researches.

My first observation was that the theory given in the excellent treatise of Sælberg Wells—mostly quoted from Græfe, and which we may call the anterior inflammation theory—however attenuated, would not apply to all cases; and was therefore untenable as a general theory.

* Read in the Section of Ophthalmology, Pan-American Congress, Washington, D. C., 1893.



My next observation was that the theory taught us in the clinic of the celebrated Prof. Stellwag, by his first assistant, and which we shall call the choroiditis theory, while approaching a little nearer the bottom facts, did not at all cover the ground, it being evident that while degenerative choroiditis might be present, in a good many cases it was as likely to be a *part* of the pathological process as to maintain the higher relationship of cause to effect.

Then I observed year after year, that—with few exceptions—the cases of decided glaucoma, of threatened glaucoma and of glaucomatous cupping without active symptoms, were in persons affected with astigmatism (frequently conjoined with hypermetropia); and it seemed to matter little how small the degree, 0.25 of a dioptré being a not infrequent showing.

Later I began to observe that the exceptional cases were myopes in whom a more or less similar condition of back-pressure existed; or what is relatively the same, posterior weakness of the eye-ball.

My next investigations led me into a study of the so-called “physiological cupping,” and I found it such an elusive subject that I at last concluded that it was to a very considerable extent a *delusive* one. Perhaps the fairest explanation of it in all respects that can be given in brief, is to compare the whole subject with the inguinal canal in its healthy state, and its possibility of acquiring inguinal hernia. The conditions are analogous in the main, except as to size and the possible difference in time required to produce a similar effect from a similar cause.

In both instances we have a point of weakness about certain vessels and subjected to more or less pressure from within. In each we may have this weakness exaggerated—it may be anywhere from a slight to an extensive degree—by a funnel-shaped depression or cupping outwards which may be either congenital

or produced. It is easy to understand that a tolerably constant undue pressure may gradually increase the depth of this cup or broaden it, and thereby gradually increase the element of weakness; or it may yield suddenly at the base and admit of a hernia *de facto*.

In the case of the optic disc, we have in perfect health, as it appears, an exceedingly slight depression about the *vena centralis retinae*, and also some less rigid tissue there, apparently intended by nature to afford greater safety to the vein. Now in the highest state of nature, this slight depression has an area of elevation around it which acts as an excellent bulwark of protection: but let it be subjected to a continued excess of pressure, slight though the accretion may be, as in the eye-strain of defective refraction, and we may observe that the interfibrillar substance of the nerve is absorbed, leaving a broad shallow cup (the atrophic cup of Fuchs); or, apparently independent of that, we may notice in other cases quite deep retrocession at the temporal side of the central vein without any very noticeable lowering of the general surface of the optic disc. In the former cases (atrophic) there is apt to be also atrophic change at the periphery of the disc, while in the latter cases this factor is at least not so noticeable.

I am disposed to regard both these conditions as intermediate stages between a healthy condition of the nerve on the one hand and glaucoma on the other; more especially as I have seen inflammatory glaucoma in an eye with one of the deep narrow cups and a decided tendency towards glaucoma simplex in another, and because it appears that atrophy of this kind favors the developing of glaucomatous cupping. The optic nerve is placed very much like the cork in a wine bottle, i. e., as if forced in, so that outwards and inwards it is larger than in the middle; hence an atrophy which reduces its diameter tends to make it *push out* more readily, thus displacing the lamina

cribrosa backwards and admitting of the well-marked glaucomatous cupping.

Having given a brief *resume* of the course of utterly unbiased reasoning which led up to the conclusion, though without introducing the cases, which would be interesting enough if time permitted, we shall unhesitatingly announce the prime underlying cause of the disease

The key-note of glaucoma is eye-strain; and that usually from some defect of refraction.

There is no need to make a great mystery out of glaucoma any longer. If it were possible to correct all the errors of refraction in a given community, correct them early and keep them continually corrected by suitable and absolutely accurate glasses, then in that community, glaucoma apart from a few secondary and traumatic cases, would disappear. We speak advisedly in saying a few, for it is highly probable that these cases have largely been predisposed to by eye-strain. We apprehend that the elucidation of this subject has been delayed somewhat through reverence for the opinions of the immortal Graefe. It is indeed a high tribute to his genius that his doctrine on the subject should have been accepted as the law governing research almost up to the present time. Graefe did wonderfully well—as well as it was possible to do in his day; but the age is progressive and the greatly improved refraction work, at least of some of the more careful men in this country, makes it possible to take a deeper and more comprehensive view into the etiology of this and certain other diseased conditions of the eye.

Our object should not be to combat the theory of this or that individual, but to lay a foundation under them. Some of them at least, and perhaps most of them, contain good material and make such a presentable showing that they deserve to have something better than mist to stand upon.

While we have already been laying considerable stress upon back pressure, and while we believe that in the average case the posterior portion of the eye earlier receives the impression which tends in the direction of glaucoma, we must never lose sight of the general truth that eye-strain, the cause, is bi-polar in its action; hence we must comprehend the effects produced both posteriorly and anteriorly, and must not be surprised nor disconcerted by the variations in type met with in studying the disease. It is but natural that when a force acts in more directions than one, the manifestations produced may vary in character. Of course in speaking of posterior strain, we are referring to that which takes place in and about the optic disc; and of anterior strain, referring to that which occurs in and about the ciliary region, including a tolerably large and important area.

We further must not ignore the fact that there is lateral pressure as well, and that the *venae vorticosae* are important elements in the circulation of the eye. And perhaps there has not enough stress been laid upon the ease with which the retro-choroidal lymph space may become affected.

Concerning the effects of anterior pressure, it is not necessary to say much here, for the reason that it is so well described in the text books. Most of them doubtless attribute undue importance to disturbances in Schlemm's Canal, as etiological factors; they are important enough to be sure, but it should be borne in mind that there are other and larger lymph channels in the eye. A close observer may notice that in a great many cases, interference with filtration in that direction is the final act of a rather long ocular tragedy.

A strong argument that the early pressure effects are not so much forwards as backwards, is that so little scleral congestion is observable in the inflammatory cases compared to what is seen in iritis, cyclitis, etc.

No wonder the scientist looked in vain for the cause *during* the attack, when the cause has been acting more or less quietly for years until the attack obscured it, and rendered critical study impossible.

It should be remembered that in thousands of non-glaucomatous cases the same cause (eye-strain) is acting, but finds enough resistance to prevent a glaucomatous effect from being produced.

The optic nerve often withstands the strain so long and with so little local deleterious effect that we can but marvel at its power of endurance. And even when some retrocession has occurred, oftentimes no startling symptoms arise until the lamina cribrosa has been forced back to such an extent that the pressure in part is applied in a lateral direction almost directly upon the nerve-sheath, upon the lymph channel surrounding it and, somewhat less directly, upon the posterior ciliary circulation.

Likewise forward pressure in the ciliary region (i. e., practically in the opposite pole) is endured fairly well until it causes an impingement upon the absorbent angle of the anterior chamber.

But it will be observed that these are only climaxes in a process long since begun.

It is highly probable also that in a considerable percentage of cases, strain at one pole is endured until too much strain is applied at the opposite pole; then the patient, suffering eye succumbs to overwhelming forces. In another of the diseases to which flesh is heir, viz., peritonitis, we have what may answer as an example in some respects of what is meant. So long as a peritonitis (non purulent) remains localized, it may seem comparatively insignificant, but let it assume a general form and all will admit its gravity.

Space will not here admit of going exhaustively into the subject of eye-strain, but a few points may be briefly touched

upon. We shall premise by saying that the term has too often been restricted in many minds, almost entirely to the apparatus of accommodation, rather ignoring the important part played by the recti muscles.

It is true that, apart from myopic eyes, a large percentage of the strain comes from near work, and hence glaucoma, as a fully developed disease, is largely confined to persons of more or less mature years, when the eyes find some added resistance to the efforts of accommodation. It accounts also for its relatively greater frequency in hypermetropes. We should not be surprised to find, if sufficient statistics were available, that the average onset was a little earlier in hypermetropes than in some other classes of cases, just as we notice in them the relatively earlier necessity of wearing glasses for reading.

A very possible reason why myopic eyes are less frequently glaucomatous arises from the fact that many of them do not find much difficulty of accommodation. Another factor is found in the *general* weakness of the posterior wall of the globe, making the excess of pressure relatively less felt at the disc than in hypermetropic cases.

Understanding that astigmatism is one of the greatest causes of eye-strain, we must consider that the ideal eye for the production of glaucoma, would be one with hypermetropic astigmatism.

It is known to be rather frequent in such eyes, and doubtless a great many glaucomatous eyes classed simply as hypermetropic, really had a slight astigmatism which was undiscovered.

Statistics show that very few emmetropic eyes have been affected with glaucoma, so few indeed that it is exceedingly probable that they were cases of mistaken diagnosis.

If space permitted, we would refer to general conditions of the circulation and the system at large; to tonic spasm of the ciliary muscle, intra-nasal pressure, etc., as possible etiological

factors. For the reason indicated, it is impossible here to give a detailed explanation of our views regarding the mechanism of eye-strain.

If the pathologist will take his cue from the clinical facts that eye-strain is the general underlying cause of glaucoma, and, further, that the earlier effects, so far as tending towards glaucoma are concerned, may be exerted backwards quite as frequently as forwards—perhaps far more frequently—he will soon find that the study of this disease does not lead him into such a mystical field as it has been pronounced by most of our authors.

If then he will turn his attention to the admirable schematic cut in Fuschs' "Text-book of Ophthalmology" (page 251) showing the lymph passages of the eye, and, observing how extensive they are, decide to make a *general* study of them in connection with glaucoma, he may find a rich field for investigation, and produce some decidedly definite results.

EARLY MANAGEMENT OF GLAUCOMA.

It has been thought best to refer to the plan of treatment provisionally adopted as the "early management of Glaucoma" for the reason that it applies primarily to those cases of Glaucoma and threatened Glaucoma, which early come under the observation of the oculist; and it would be manifestly unfair to apply it only to a few neglected and desperate cases found in metropolitan hospital practice, and then reject it as unworthy.

Of course, in this disease even more than most others, prevention is better than cure. The chief measure of prophylaxis is the early use of accurate glasses; but medication is also useful in that direction. The medication must, however, be followed up by glasses, and no pains must be spared to make them exact.

Of therapeutic measures perhaps the best we have as yet found are acetanalid internally, pilocarpine solution instilled into the eye, and the hot solution of boracic acid applied externally.

The acetanalid has mostly been given in eight grain doses, three or more times a day to the average adult, and four grain doses to specially susceptible females. Its action should be watched so that it shall not cause too great depression; and if the temperature is found sub-normal, the dose should be lessened.

My reasons for preferring pilocarpine to eserine are two-fold: first, that while it may not be so strong a myotic, it seems to have a better effect in reducing ciliary congestion; secondly, it allays pain instead of causing it, as eserine does.

We know of no reason why the strength of the solution should not be increased in critical cases, but 4 gr. ad $\bar{3}$ i is as strong as we have used. Whether the salicylate of sodium does good of itself, we cannot say positively, but at any rate it preserves the solution, and we have sometimes fancied that the pilocarpine solution was more efficacious when combined with it. So we invariably use it at the rate of ten grains to the fluid ounce.

Very possibly it may be found that hypodermic injections of pilocarpine ($\frac{1}{8}$ gr.) may be of service, but I have thus far never used them in this connection, preferring to rely upon acetanalid as internal treatment. Unless the patient were kept in a recumbent position and the circulation and temperature noted, it would not be safe to make use of both these depressing agents at once. Perhaps we may find cases wherein this, as in so many other diseases, the recumbent position is highly advisable; but oftentimes the pain is of such a character as to make the patient restless instead of causing him to seek repose.

For the hot external applications the patient was allowed to make an extemporaneous solution of boracic acid; but the mode of applying was as specific as possible. They were made with soft cloth dipped frequently (once a minute), kept at about the highest comfortable temperature, and continued twenty minutes each time. The frequency of repeating them varied with the case from once a day to every three hours or oftener.

We had hoped to obtain favorable results from the use of gelsemium, but, at least in the dose employed, it did not yield as favorable results as acetanalid.

The early employment of *temporary* glasses is a measure not to be neglected in certain cases; for in these irritable eyes a sensible man will not attempt a great amount of subjective testing for glasses; and we doubt if many cases retain the same refraction when the acute and sub-acute symptoms have passed, that existed during those stages.

Regarding iridectomy in this disease, we are confident that it is an operation which will fall more and more into disuse. It was an empirical operation at best, and though it served a very useful purpose in its day, it has comparatively little place in attending to the better cared-for eyes of the present. While it may still remain a desirable procedure for urgent or neglected cases, it does not always produce satisfactory results, and should by no means be lightly adopted as routine practice.

NOTE—(NOT READ AT WASHINGTON).

For valuable aid in the preliminary work of this paper, I am indebted to my friends Dr. C. E. Conner, of Scranton, and Dr. W. A. Shoemaker, of the New York Eye and Ear Hospital.

As intimated in the closing remarks, the clause in my paper referring to iridectomy was made more as a prophecy for the future than a statement of present practice.

The operation *is* at present more or less "an empirical one", but I trust that it will one day become a truly scientific one, confined within its proper limits.

Let him who would criticise the statements I have made first study his suspicious cases in the region of the optic disc with a Morton's ophthalmoscope; for I doubt if this paper would have been written had I not been the possessor of that admirable instrument.

