EYE, EAR, NOSE AND THROAT

UNRECOGNIZED GLAUCOMA AND THE GENERAL PRACTITIONER'S RESPONSIBILITY IN ITS DIAGNOSIS*

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SUMMARY

Attention is called in the paper to some details in the development of glaucoma which the physician should know. They are:

1. The significance of frequent changes in glasses in persons over 50, especially in the direction of seeming myopia. Dangers of the optometry examination.

2. Misleading influence of the term "incipient cataract" for "lens striations." The latter often do not advance. If they do, uncomplicated progress is painless, and pain or injection means something else. The cloudy appearance of the lens in glaucoma may be mistaken for cataract unless the ophthalmoscope is used.

3. The pain in progressive glaucoma is unlike that of eye strain. Often thought to be neuralgic or rheumatic. These troubles are now known to be usually due to an infection focus. Importance of search for it.

4. Gastric disorders frequent in progressing glaucoma. Importance of recognizing the direction of cause and effect.

5. Recognition of the ocular injection of glaucoma as distinguished from that of conjunctival. A physician should know the differential diagnosis of conjunctivitis, keratitis, iritis and glaucoma or else let every red eye alone.

The theme of this paper was assigned by our Chairman. When we met in New Orleans in April, he put his request somewhat as follows: "The general practitioner in the South regularly reads the special papers appearing in the JOURNAL of the Southern Medical Association. We ought to take advantage of this by writing on eye diagnosis. Won't you bring to Louisville a paper upon glaucoma based on observed errors of diagnosis? Such a paper should point out to the ophthalmologist the lines along which he should try to enlighten his professional associates and would, at the same time, be generally read by physicians."

In complying as well as I can with Dr. Stucky's request, I shall be compelled to

review certain things entirely familiar to all my hearers.

When ocular hypertension occurs because of a previous or existing eye disease, the intelligent practitioner will naturally seek advice. In primary glaucoma, early eye symptoms may not be noticed unless looked for. The problem is one of interpretation. Not always, even when things turn out badly, can the physician be justly blamed, for the early eye symptoms may be so trivial in the patient's opinion that no special stress is laid on them. Extra-ocular signs may be wanting. But when the latter are present the doctor should be prepared to properly interpret them. He should know when an eye examination is needed. The physician's responsibility to glaucoma is thus expressed by Fuchs:

"Primary glaucoma is a common disease, constituting about 1 per cent of all cases of eye disease. Its accurate recognition is of the greatest importance for the general practitioner, the more so because here prompt and proper therapeutic interference can save everything, but a false diagnosis and improper treatment may destroy everything. Unfortunately, we still constantly get under observation many cases of glaucoma which have been incorrectly diagnosed by the general practitioner, and which come to the ophthalmologist only when help is no longer possible. Cases of inflammatory glaucoma are often confounded with iritis or iridocyclitis and are accordingly treated with atropin, which has a particularly deleterious action in glaucoma. Cases of glaucoma simplex which present no external symptoms of inflammation are not infrequently regarded as commencing cataract, and the patients are put off in expectation of the cataract's becoming ripe, so that they delay until it is too late for iridectomy."

We may accept the following as established: In glaucoma harm comes to the eye from increased intra-ocular pressure. When the hypertension is intermittent and slight, the eye may remain free from congestion and visual symptoms be transient and of slow progress. This is chronic or simple glaucoma. Again, if the eye possesses a "glaucomatous tendency," as it is called, sudden hypertension may develop with injection, i. e., evidence of eye inflammation or inflammatory glaucoma. In a third class the onset of hypertension may be so sudden as to overwhelm the eye. This is fulminating glaucoma. The two

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latter forms demand prompt operative interference. Let us consider the symptoms of the first and second of these forms.

CHRONIC GLAUCOMA

It presents no signs of inflammation. Indeed the diagnosis often escapes the oculist unless he forces himself to follow a definite routine. There may be temporary obscurations of vision; apparently, frequent need for changing of glasses (often in the direction of myopia and diminishing presbyopia until visual acuity lessens and then a magnifying glass may be selected). Sometimes there are haloes about artificial lights; tension may be slightly increased to the fingers or the tonometer. However, with these visual symptoms one frequently will not discover elevation of tension. It is in studying field vision that one makes the diagnosis. Contraction, especially on the nasal side, or scotomata, with certain definite features plus the gradual glaucomatous cupping of the optic disc, give the diagnosis. It must be admitted that these things concern the oculist alone and that the general practitioner would be apt to inquire about them only if previous observation had shown him the association between a slowly developing and destructive eye disease and certain symptoms easily under-estimated. But these refraction changes present features which the physician should appreciate. At this time, when that pseudo-scientist called the optometrist is so prominent, the medical adviser should be put on his guard. *The fact that a patient of fifty years or more requires frequent changes of glasses is of itself a matter of grave suspicion provided refraction work is correctly done in the first place.*

The refractive changes in chronic glaucoma are thought to be due to one of two influences: (1) lowering of accommodation due to hypertension with consequent increase in presbyopia, and (2) swelling of the lens with just the opposite effect on refraction, i. e., increase in refraction with apparent myopia and lessening of presbyopia. This latter may be the precursor of peripheral lens striations which can easily escape notice. Every case of so-called incipient cataract should be carefully watched from this standpoint. The hypertension in such a case may be regarded as

secondary to the lens swelling, thus making the glaucoma technically secondary. However, this fact does not interfere with the point under consideration, namely, that apparently progressive myopia in an individual over 50 (so called second sight) may lead to glaucomatous changes due to lens swelling. The following case illustrates:

Some years ago a lady of 58 consulted me about an increasing myopia for which an optometrist had, every few months, ordered concave glasses of increasing strength. He strengthened the glasses as long as he could give satisfactory vision. When he was unable to do this, he told her she was developing "second sight," the forerunner of cataract. Minus 8 sph. gave her 20/70, her field was limited to 10°, tension +1 to the fingers (it was before the days of the tonometer), and there was typical glaucomatous pitting. Under treatment with miotics, the case was kept under control during the rest of her life, three or four years. Better vision probably could have been preserved by an earlier diagnosis.

Here was a patient who, under the advice of her physician, accepted the diagnosis of an optometrist, or optician as he was then called, because the aforesaid optician had properly corrected the doctor's own presbyopia. The characteristic greenish appearance of the glaucomatous pupil had finally led to the diagnosis of cataract. The patient was waiting for its maturity. This case illustrates not only the possible evils of optometry, but, with the following case, emphasizes a real danger hanging over persons with so-called incipient cataract:

The late Dr. Russell Murdoch, of Baltimore, who was one of the most careful diagnosticians I ever knew, found in a lady's eye peripheral lens opacities. Her central vision was normal. He told her of these lens cloudings and warned her to come for periodic examinations. She stopped after a time, and after Dr. Murdoch's death came into my hands. One day, six months after the last previous examination, she consulted me because of an acutely congested eye, dilated pupil and pain. She told me that these symptoms had developed two or three months previously; that she had not been to see me because she was sure it was only the development of the cataract and she wanted to know if it was ready for operation. She had sub-acute glaucoma and an iridectomy saved the vision that was left, about 20/50, with contracted field. The so-called incipient cataracts never ripened, but they misled. Often they do not mature.

This case, considered with the other, presents two very important truths. Even if peripheral lens opacities are found, the term "incipient cataract" is a misnomer.

It gives the patient mental torture and, worse still, makes him take a wrong view of his entire eye condition. In my judgment the term "incipient cataract" should be left out of our nomenclature. If, however, the lens opacities are mentioned at all (and undoubtedly they should be to some member of the family) there should be added and forced home the statement that "ripening" cataracts do not give pain and that when pain or congestion come, something else is indicated. Prior to this experience I had not made it a habit to give this warning to patients with lens cloudings. Now I invariably do. I tell the patient or some one in the family that there is no reason why an eye with lens cloudings should not develop something

whose fields are herewith presented illustrate the first class. One had and still has, by refraction correction, normal central vision. But there is a paracentral scotoma starting from the normal blind spot, passing above the fovea and over into the nasal field (Bjerrum's symptom). (Fig. 1). The other patient had no idea that his eyes were involved. I saw the case in consultation with my friend, Dr. L. F. Barker, who had been consulted for unexplained gastric disturbances with severe attacks of neuralgic head pains. A similarly situated scotoma was found. (Fig. 2). Both of these patients presented cupping of the discs. Had the physicians who saw these cases before they reached Baltimore realized the importance of these neuralgic pains, valua-

Fig. 1.—Glaucomatous scotoma discovered in routine examination. No previous suspicion of disease.

Fig. 2.—Glaucomatous scotoma discovered in routine examination. No previous suspicion of disease.

else; that regular inspections are necessary and that if pain comes, it may be very significant. This is another line along which our general practitioners should be instructed. The word "cataract" should not be used till it is known that the opacities are progressive.

Apart from visual changes, there are symptoms during the progress of glaucoma which might readily take the patient to his physician.

I want to discuss one—pain. The neuralgic pains of glaucoma may vary from a mysterious sense of discomfort and tension in reading to severe pain of a perfectly characteristic nature. The two patients

ble time might have been saved. But in each case the "neuralgia" had been confounded with the headache of ametropia. There is a vast difference between the constant band-like supraorbital pains of eye strain and the periodic boring eye or temporal pains of glaucoma. Either might be called "neuralgia" by the patient and would naturally take him to his physician. He should be able to distinguish between the two classes of pain, or ought, at least, to know that such symptoms demand eye study by a trained ophthalmologist.

Passing allusion must be made to another easy and wrong diagnosis. For many years, and to a certain extent even

now, rheumatism or a "rheumatic tendency" has been accepted as an explanation for various sensory disturbances. In most of our text books there is description of "rheumatic" eye diseases. Inflammatory glaucoma was once called "ophthalmia arthritica." Chronic glaucoma has often been thought rheumatic because of the occurrence of muscular pains, etc., during its course. Consider this for a moment in the light of what we now know about rheumatism and neuralgia. We may at the same time consider arterio-sclerosis, based on the feeling of radial resistance. Neuralgia, rheumatism and beginning arterio-sclerosis are sometimes necessary, but always insufficient diagnosis. All have a cause. Focal infection is now recognized as a frequent producer of just these things. When we speak of chronic primary glaucoma we mean an eye going blind with moderate hypertension, slow development of the glaucomatous cup and nerve atrophy, accompanied, more or less irregularly, by other symptoms, with fundamental cause unknown. May it not be possible that a focal infection can produce temporal neuralgia and intermittent ocular hypertension of low degree just as we know it sometimes produced choroiditis with secondary hypertension; the same process but of less severity and acting on the neuro-retinal rather than on the uveal circulation? In other words, I suspect that sometimes our inadequate and unsatisfactory explanation for chronic simple glaucoma may be displaced by better knowledge of the effects of focal infection. Be this as it may, the lesson for us is plain and it should be passed along to the internist. These sensory and vascular disturbances ought to be studied from the standpoint of focal infection. Whether the symptom be neuralgic, rheumatic or hypertonic, search for an infection-focus or peripheral irritant will necessarily involve the sinuses and eyes. Thus, study of remote disorders may reveal an unsuspected glaucoma. The case seen for Dr. Barker in a routine diagnosis study illustrates this.

Sometimes in chronic glaucoma, but more regularly in the congestive variety, there are gastric disturbances of more or less severe type. They may vary from nausea to what looks like acute cholera

morbus. After a few days the eyes look red and there is complaint of dim vision. For such things as these, the physician would naturally be consulted. Unless he knows that the relation of cause and effect may be from the eye to the stomach, he can easily reverse his process of reasoning. A single case will suffice to illustrate.

In 1901 a woman, 46 years of age, came to a well known ophthalmologist with inflammatory glaucoma in the right eye. Her vision was reduced to the moving hand. He made an iridectomy and the trouble stopped there. It is her history which is of special significance. She had been ill at home for nearly a month with headache, neuralgia, nausea and vomiting. It was known that vision was impaired, but little or no attention was paid to it. She was thought too sick to come to the city. She was warned, after operation, of the waste of important time and urged to seek advice at once if similar symptoms appeared in the left eye. It was ten years before they did. She came to me one afternoon after a dreadful trip on a bay boat. Nausea, head pains, and blurred vision were her symptoms. She came because of the warning given her and against the advice of her home physician. She had learned the lesson if her doctor had not. It was impossible to get her vision before operation. The condition present was acute inflammatory glaucoma of three days' standing. Iridectomy under ether was done the day she came to me and restored her vision to 20/30. Now, nine years afterwards, it is 20/40. The difference in the result of the two operations was merely the difference in time between onset and operation.

INFLAMMATORY GLAUCOMA

The diagnosis of the second variety of glaucoma to which I wish to call attention from the standpoint of this paper, is that in which ocular inflammation is present. Pain in and about the eye, ocular injection, etc., are enough to focus attention here, whether or not there are other symptoms. There are two possible errors of diagnosis unless a physician is warned about them. One is mistaking the injection for an ordinary conjunctival hyperemia; in other words, overlooking the filling of the circum-corneal and episcleral vessels. The other, which is of the gravest importance, is mistaking the case for iritis. The danger here is enhanced by the fact that atropia, necessary for iritis, plays havoc with glaucoma. The reverse is equally true. Miotics, needed in glaucoma, increase iritis. I have seen two such cases of mistaken diagnosis in which atropia was used. As a rule, the glaucomatous dilatation of the pupil should prevent such a mistake. This dilatation is not always

present and, so, only a test for tension can clear the diagnosis. Such primary mistakes as these can not be excused. Any one who undertakes to pass judgment upon a congested eye ought to know the difference between conjunctival and circumcorneal injection and the meaning of each. He ought to know how to test the size and action of the pupil and be able to draw correct inferences. He should be able to appreciate by digital touch any increased tension. If he is deficient in either, he will do less harm by refraining from any treatment than by acting upon an examination with insufficient knowledge. He should, however, master such primary essentials, for many physicians are so situated that reliable advice can not be readily obtained.

It is not a matter of wonder that a physician, suddenly called to a case of severe eye trouble, should make a mistake. Unless he was one of the few men who, during undergraduate life, was attracted to ophthalmology and unless his teacher so linked his eye instruction with pathological processes that the student associated the two in his thinking, he soon forgets the little ophthalmology he picked up in undergraduate days. As I tried to point out in a paper written for this Section two years ago,* the average student leaves the medical school with the impression that ophthalmology is a sort of artificial addendum to, and not an integral part of, general medicine. There is another reason. When a physician is so environed that he can not command skilled advice he must do the best he can. He may have been in general practice many years. He may not own an eye book and if he does, after brushing off the dust, he does not know where to find what he wants. But there are few men who do not take and read some journal. If you want to know why most of them skip the ophthalmic articles, go over the titles and read some of the highly technical and special papers appearing in the current journals. Most of them are very valuable to the specialist, but they do not touch the wants of a man who, through circumstances, must only

now and then take the responsibility of an eye diagnosis. We write over, not their heads, but their wants. If our chairman is right in thinking that the physicians who take the JOURNAL of this Association regularly read the special articles, it is our duty to furnish something which may be of real use. It is this conviction, plus Dr. Stuckey's request, which has led me to inflict so primary a paper upon an ophthalmic audience. I trust that our general men may find something in it which will help.
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DISCUSSION

Dr. J. A. Stuckey, Lexington, Ky.—I am quite sure I express the sentiment of the majority of the men present when I say that I am very grateful to Dr. Woods for having told you some things for me. My thought has been directed to this subject because of a large number of cases of glaucoma I have met with in my clinics in the mountains. We have been seeing entirely too many cases in Central Kentucky, cases of eye trouble that have been treated for weeks and months at intervals as neuralgia or rheumatism, all needing correction of refractive error. Many of these cases "have gone the rounds" in the use of home remedies and "eye drops," and when they have come to me the vision has been permanently impaired. I have had many operations to do because these cases were not seen early enough. In the mountains we found an excuse for this condition, and I believe the reason is the large amount of focal infection due to the teeth, submerged tonsils and intestinal autointoxication. There is more need of dentistry in the mountains than any other one thing I know of. The majority of cases I saw had high blood pressure. I believe when the general practitioner is better taught, when he better appreciates his relation to his patient from the ophthalmological standpoint, and when we do not talk so much of so-called "high brow stuff," the general practitioner will feel just a little bit different towards us. I have seen some very humiliating instances where the general practitioner apologized for not having suspected the real eye condition—when he treated the eye for redness that he thought was a conjunctivitis, and treated the pain because he thought the patient had rheumatism in the joints as well as in the eye. It never occurred to him to take the reflexes, test the ocular tension and he did not examine the cornea carefully. The most important man in the medical profession today is the general practitioner, and I am sure if we give him the aid that he desires of us as specialists he will appreciate it.

My reason for asking Dr. Woods to write this paper was because I wanted some plain language and real help to come from some one just about as big as he is. I think he is just about as big as anybody we have in the South.

Last Thursday I saw a patient who had gone the rounds of what Dr. Woods calls the "pseudo-scientists." He had had his glasses changed seven

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times in eight years, and when he came to me I found him with a dilated pupil, a hazy cornea, and wearing a minus-9. And that man was only forty-seven years of age. I have seen other cases where patients had been told they had incipient cataract, as mentioned by the essayist, and were waiting for the development of the cataracts. I fully agree that we ought to leave out that word "incipient." The patient has enough trouble without anticipating more. I want to endorse every word Dr. Woods said in his paper.

Dr. J. W. Jervey, Greenville, S. C.—I think we will all agree that while it is highly necessary that these conditions should be recognized early in order to save the eyesight of the victim, yet it is rather difficult in many of these cases for the ophthalmologist to determine the existence of a simple glaucoma. I must say that it seems almost a hopeless undertaking to try to teach the general practitioner the different tensions which would warrant the diagnosis of glaucoma, or even a suspicion of glaucoma. There is of course one way to get the matter before the general profession, and that is through the media of various general medical organizations; to have each ophthalmologist who is a member of his state medical society, or his county medical society, present the matter before the profession. It so often happens that the only complaint of a case of simple glaucoma is some apparent functional disturbance of the vision. These patients come in time and time again with no objective symptoms whatever—no peripheral injection, no dilatation, absolutely no glaucomatous haze, but in the course of the routine refraction if your ophthalmoscope is invariably used some of these cases will be detected. However, some of these cases of simple glaucoma show a sporadic tendency. They may show tension at one time, and at another time the tension is normal, even with the use of the tonometer, and these cases in many instances show not the slightest sign of cupping of the disc. It is not until the disease is in a dangerous stage that the patient notices a gradual diminution in the field, perhaps of one eye, perhaps of both, and then we are confronted with the fact that we have a case of glaucoma. But if a systematic examination with the ophthalmoscope is made in every case of refraction I have no doubt many cases of glaucoma will be uncovered which heretofore have been passed by unrecognized. I certainly hope that some method can be devised which would put the general practitioner on his guard in these cases.

Dr. Meyer Wiener, St. Louis, Mo.—There is very little to add to what Dr. Woods has said, except regarding the information to the patient as to whether the disease is cataract or not. Dr. Woods stated that he tells the patient or some one of the family that he has cataract. I infer from that that he does not himself tell the patient that he has cataract. I believe in truth in medicine and I believe the patient should always be told what is wrong with him, regardless of what the consequences may be immediately, because the patient will find out some time or other that he has cataract, and it will be a dreadful shock when he does find it out. He probably feels that you did not know what was the matter before, and it reflects discredit on the medical profession. I have had two or three instances in my practice, but I do not

now let the patient get away without telling him what the condition is.

Dr. Woods mentioned the fields as most important in making a diagnosis of glaucoma. I do not think this can be emphasized too strongly—that we should look in the peripheral fields for glaucoma. We cannot always find defects in the field in glaucoma with an ordinary perimeter, but we must search for it with some such instrument as Dr. Peters' campimeter, or the Lloyds stereocampimeter; something that will find the blind spots in the field of vision. I agree that it is not only the general practitioner who needs to be educated, but ourselves. We should be careful about judging too hastily whether the patient has or has not glaucoma. I recall a case that came to us about eighteen years ago that had an acute attack of glaucoma, but refused operation. We treated her with eserine and the inflammation subsided and the tension became normal. She went to New York and consulted a gentleman who was considered the leading ophthalmic surgeon there. After many tests this man told her she did not have glaucoma; that he did not believe she ever had had glaucoma. I am not criticising this man, because I think he ranks among the three or four best men in ophthalmology in the United States; but I think he made a mistake in this case. The patient went home, and on the way had another acute attack, with the result that the vision of one eye is practically gone and that of the other greatly reduced. There is bare perception of light in one eye and about 20/40 in the other. Now if a man like that can make such a mistake, how can we expect the general practitioner to always recognize glaucoma?

Another thing, I think most ophthalmologists think they must find cupping of the disc in glaucoma, whereas in the acute stage there is no cupping; there may even be a blurring of the edges and hyperemia. It is only when we have prolonged increase of tension that we have cupping.

Dr. J. A. White, Richmond, Va.—I agree with Dr. Jervey that trying to educate the general profession seems a hopeless task. Through a long experience I have many and many a time had people hopelessly blind from glaucoma sent to me to have cataracts extracted. I have written several papers along the same line and made addresses on the subject before medical societies, trying to educate some of the medical profession to our point of view, but I doubt whether I have ever made many converts.

Dr. Woods' paper, properly speaking, should have been read before the Section on Medicine if he expected to convert anybody or to educate them so that some of the many blind people in this country might be saved from that blindness. It is my experience that notwithstanding the enormous advance in ophthalmology in the last twenty-five years, the general practitioner of medicine takes no interest whatever in eye diseases, and I have myself noticed that the profession in our largest cities have not the slightest knowledge of eye disease, not even a rudimentary knowledge. That is my experience among the general profession where I live, and I believe it is the experience of all of you. So I repeat that this paper should have been read to the people we want to educate, not to us, not one of whom will

take issue, with Dr. Woods in a single word he has said. I hope the paper will be published in such a way as to come under the observation of, at any rate, the members of this Association, who, if they read it carefully, may apply its lesson to a good end.

Dr. H. H. Briggs, Asheville, N. C.—I want to emphasize what the last two speakers have said about our responsibility, the ophthalmologist's responsibility, in this matter. It is true that the optometrist has done a great deal—or rather, his lack of knowledge of pathology has caused a great many cases of simple chronic glaucoma to go on to the stage where operation is futile. But he is not to blame so much because of his lack of knowledge of a trouble which is not optical, but pathological—a disease of the eye. If the general practitioner's knowledge of disease is not adequate he can refer the patient to the ophthalmologist, but we as ophthalmologists have often been remiss and have not carefully examined and diagnosed these cases. We are prone to refract cases that come to us with diminution of vision, and fail for weeks or months to detect glaucomic symptoms which a proper examination would have revealed in the beginning.

Another point is that every case of diminished vision which can not be corrected by refraction, and which can not be accounted for in any other way, should be suspected of being a symptom of simple glaucoma. We should never use a cycloplegic in any case until we are reasonably sure there is no increased tension, and that beginning glaucoma is not present.

Dr. E. H. Cary, Dallas, Tex.—The point we seem to be trying to make is that no greater amount of progress has been made in educating the medical profession. I agree with the Doctor that a paper of this kind is not much appreciated and has not much effect on the man who is practicing who has not had a correct conception of this when he was a student. My observation through years of experience has been that we have an ever-increasing list of mistakes, and I have been led to believe that the worst cases are those who have been deterred through bad service from going to some one who might have been capable of making a real diagnosis, this delay brought about by advice from the general practitioner who himself did not have in his early training a correct conception of the subject nor the fear of God instilled into him. I can recall distinctly that the question of ophthalmology and these other subjects as taught by Dr. Noyes did not impress me, and none of us passed the examinations. We were not required to pass the examination. The teaching for many years has put the fear of God into the heart of the student, but it is up to the teachers, to those men who actually teach. If the teacher has a right finally to require an examination that examines and will actually exclude the student from getting his diploma unless he passes, then he can put the fear of God into the student's heart and he will go out thoroughly convinced that there are one or two things he must know. One is the differentiation between iritis and glaucoma—he must know that in detail, and there is reason in it. I have been watching this for years, and I find these men do not make mistakes. Do not try to

get your general practitioner so well educated that he will be able to make a diagnosis; but he can be afraid of making mistakes, and he will do something to avoid making mistakes. If you have done that you have made progress. I think the progress is marked in recent years and will continue, but mainly through the students now being taught.

Dr. Hilliard Wood, Nashville, Tenn.—We have all had those sad cases come to us in which, through an error in diagnosis by the general practitioner, the vision was practically totally lost. The evil of an error in diagnosis is twofold: in the first place, no good is accomplished by treatment; and in the second place, positive harm has been done by treatment, which is usually atropine or nitrate of silver, depending upon whether it was called an iritis or a conjunctivitis, but alas! the time of election for operation has been allowed to pass.

I rise more especially to call attention, in the early diagnosis of glaucoma, to a syndrome laid down by Colonel Elliot on his visit here in 1913: three symptoms of the earliest stages of developing glaucoma. These three symptoms are: plus tension, as taken by the tonometer, and not with the finger; reduced central vision; and contraction of the field, as taken by the perimeter. These three are the best guides in the early stages, where there is still a question in one's mind as to the correct diagnosis. But glaucoma, especially the more chronic types in which a diagnosis is most doubtful even in your mind and mine, develops more or less intermittently, and in the intervals between the attacks the patient will not even show this syndrome of Colonel Elliot. It has been my custom, where the patient lives within reach, to have him come to me during the attack and then examine for the symptoms to which I have referred. I believe if we wait to make a diagnosis until cupping of the disc and dilated pupils appear, we will let go by the time when we could have done something for our patient.

Dr. Hiram Woods (closing).—I agree with what Dr. Jervey and Dr. White have said about the almost "hopelessness" of educating general practitioners, especially those in long practice. At the same time, I think there are a few bright spots on the horizon. One of them is the education of students, spoken of by Dr. Cary. They should not be burdened with instruction about matters we do not understand ourselves. Our efforts should follow the recognized lines of diagnosis. A teacher should not be satisfied until he has drummed and drummed, and drummed and drummed into students the meaning of certain signs and symptoms of iritis and glaucoma as differentiated from those of conjunctivitis. The insufficiency of such diagnosis as facial neuralgia, chronic rheumatism, etc., with treatment by cold tar products, should be pushed home.

This brings us to another bright spot. When these extraocular symptoms (gastric, neuralgic, rheumatic, etc.) appear, they now make the general practitioner look for a focus of infection or peripheral irritant and, if in this search, he can be made to realize that the eyes ought to be taken into account, such a trouble as chronic glaucoma may be found earlier. Before he got

to looking into these matters, when he was content with a diagnosis of "rheumatism," when he asked a man if he had ever had a chancre and, if he said "No," let it go at that—conditions were hopeless. Now many practitioners are going further and want an eye examination. What should the oculist do? Mere routine study of vision, refraction error, etc., is insufficient. Perimetry and tonometry should be parts of our work.

With regard to Dr. Weiner's criticism: he thinks a patient ought to be "told the truth" when he has a "cataract." Of course, in so far as misstatements are concerned; but what is meant by "cataract" and what false impressions can be conveyed by the term "cataract" to you may mean a peripheral, non-progressive opacity in the lens; to the average layman it means slowly progressive blindness to an operation which may or may not succeed. We all know there are hundreds and hundreds of cases of peripheral striations which never come to an operative stage. These opacities do no harm; they do not get worse. What is the use of frightening a man with the idea that he is going blind? Call it "peripheral striations," tell him there is a little cloud at the edge of his lens and it should be watched; but while he has useful vision do not torment him with the idea that he is going blind. You are telling him the truth; you are simply avoiding that particular presentation of his condition which will scare him and make him think everything else is due to "cataract." To some extent you can dangerously mislead him. See quotation from Fuchs and case narrated.

What Dr. Weiner said about the campimeter is true. You can get a small scotoma with the campimeter that you can not get with the ordinary perimeter.

Dr. Woods has called attention to a very important matter. If we wait, in diagnosis, for the cupping of the disc, we are often too late. This brings me to another thing that I will take the liberty of speaking about for just a minute. Oculists should compel themselves to make perimetric examinations. A short time ago a man under examination before the American Board of Ophthalmological Examiners was asked to take a field of vision. He replied that he owned a perimeter, but used it as a hat rack; that he had not time to take fields because he was too busy with routine cases; it took him too long to get through. He knew nothing about perimetry. If this was his point of view, certainly many of his patients over forty got away without the sort of examination they needed. I have discovered cases of early glaucoma simply by making myself go through this routine. It is a branch of ophthalmology that has been sadly neglected.

After all, the thing gets down to what Dr. Cary has said: we must make the average young man afraid to make mistakes, and if we accomplish that we will do much.

AN ATYPICAL MASTOIDITIS FOLLOWING A STAPHYLOCOCCEMIA*

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It is the intention of the writer in the preparation of this paper to discuss the hematogenous infection of that type of mastoid disease which has been classified as primary mastoiditis or idiopathic mastoid abscess and to report a case of more than passing interest.

While the subject of primary mastoiditis is not a new one, in searching the literature on this subject the writer is surprised to find how little definite information has been thrown on those of hematogenous origin.

In the review of 40 cases of primary mastoiditis only 7 of these are stated to be of hematogenous origin. Plisson reports 1 case, Humblet 1, Labarre 1, and Chauveau 4. In only 1 of these cases is the affecting organism stated.

Plisson reports a case of general infection (perhaps influenza) in a man, age 22, with mastoiditis without involvement of the middle ear. Mastoid pus showed streptococcus in pure culture. He believes this to be a hematogenous infection. He states that primitive mastoiditis without middle ear involvement occurs most frequently in influenza, but also in other general infections.

Humblet reports one case in a woman, age 27, history of a broken and sensitive tooth. Redness and tenderness over the mastoid. No sign of middle ear involvement; tympanum absolutely normal. Infection was probably hematogenous. No bacteriological examination.

Labarre, in 1912, reported one case of primitive mastoiditis in which there was no sign of middle ear involvement and no previous history of ear disease. This case was reported as of hematogenous origin. The infecting organism was not mentioned. Chauveau reports 4 cases of primary mastoiditis. The original article was not attainable.

*Read in Section on Eye, Ear, Nose and Throat, Southern Medical Association, Fourteenth Annual Meeting, Louisville, Ky., Nov. 15-18, 1920.