GLAUCOMA.*

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Glaucoma is not the title of a single disease. It is rather a convenient label for a large and very varied group of pathological conditions, the distinctive feature common to all of which is a rise in the pressure within the eye.

The causes of these conditions are most diverse, and the difference in the symptoms presented is extraordinary. When we speak of the hardness of a glaucomatous eye, or of its rise in tension, we are referring to the outward manifestation of an increase of the fluid pressure within the globe. To this increase all the causes of glaucoma lead up ; on it every sign and symptom of the condition depend.

If the rise in pressure can be traced to the action of some antecedent local disease we speak of the glaucoma as *secondary* ; failing this, we term it *primary*.

The presence of an increase in intraocular pressure necessarily brings about some measure of interference with the escape of blood from the interior of the eye to the outside. So long as such interference does not give rise to obvious congestion of the eye or of the tissues overlying it, we speak of the condition as *simple* or *non-congestive* glaucoma. When evidence of interference with the venous return appears the disease is said to be *congestive*. The term "inflammatory", though often used, is erroneous and should be discarded.

The classification of all cases of glaucoma into the three groups, acute, subacute and chronic has led to much confusion owing to the difference in the way in which these terms have been used by writers on the subject.

A case which presents the signs and symptoms of severe congestion, as a result of a steep rise in intraocular pressure, may be termed acute ; similarly, with eyes presenting the signs of subacute congestion. To define a chronic case is more difficult. All that the term really implies is that the condition has lasted for some time, but in the accepted use of the term it is also understood that the case is not in an acute or subacute stage. Any glaucomatous eye may be in an acute, a subacute, or a chronic stage ; and may readily pass from one to another of these stages and back again into that from which it sprang ; but to speak of acute, subacute and chronic glaucoma as if we were dealing with so many clinical entities is most misleading.

The Anatomy of the parts concerned.

The ciliary body and its surroundings are the cockpit of glaucoma.

Notice that the subconjunctival tissue is continued well in advance of the sclero-corneal junction. This is a very important point in connection with trephining. It is along this line that we work in "splitting the cornea", which is an essential step in the operation (Fig. 1).

Schlemm's Canal lies at the junction of the cornea and sclera, close to the inner surface of the corneo-scleral envelope. It is separated from the aqueous

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chamber only by the loose open network of the pectinate ligament. Fluid can pass readily from the chamber to the canal through this open meshwork.

FIG. 1.

Meridional section through the anterior portion of the eye. (Modified from Fuchs).

- schl. = Schlemm's canal.
- sp. = Scleral spur.
- a.c. = Anterior chamber.
- p.l. = Pectinate ligament.
- cr. = Crypt of iris.
- c.m. = Ciliary muscle.
- c.p. = Ciliary process.
- c.ep. = Ciliary epithelium.
- s.l. = Suspensory ligament.

The scleral spur lies between the pectinate ligament anteriorly, and some fibres of the ciliary muscle posteriorly. A contraction of these fibres will pull back the spur and open wide the Canal of Schlemm. As soon as muscular contraction ceases, the elastic pectinate ligament will draw the scleral spur back into place and so help to close the Canal of Schlemm. This pump action is constantly taking place during waking life, and helps to draw the fluid from the anterior chamber into the Canal of Schlemm and then to sweep it on again from the Canal into the neighbouring veins. This is a most important point clinically. *It is wrong to shut up the eyes of a glaucoma patient or to stop him from reading and writing within reasonable limits, for by so doing we are depriving him of this valuable pump action.* I mention this because I so often find patients made miserable by the cutting down of their activities, and this too, in a way which adds to, instead of taking from, their risks. My strong advice to all glaucoma patients—of course in the absence of congestion—is “use your eyes, short of over-fatigue; lead a normal life and try to forget your trouble.” The less we interfere with these patients, as a general rule, the better.

The angle of the chamber is at the best of times a narrow space. I have always believed, and I still believe, that interference with the outflow of the aqueous fluid around this ring is the essential element in the production of glaucoma. I am fully aware that there is another school of thought which lays little stress on filtration at the angle and great stress on the difference in physical constitution between the blood and the aqueous fluid, and between the blood of different patients. Recently, a very generous patient placed £750 at my disposal for a research into any subject I chose to select. I handed it over to Professor Dodds who has employed it for the investigation of a research on this subject. The results will shortly be published and will, I think, lead many ophthalmologists back to our old ideas to which I have always been faithful, on the importance of the angle of the chamber. (Fig. 2.)

FIG. 2.

The filtration angle.

a, Canal of Schlemm; *b*, trabecular tissue of pectinate ligament; *c*, scleral vein; *d*, dense scleral tissue; *e*, scleral process or spur projecting inwards and forwards from the sclera close behind the canal of Schlemm; *f*, radial muscular fibres of the iris extending outwards below the iridial angle; *g*, iridial angle; *h*, meridional fibres of the ciliary muscle; *i*, circular fibres of the ciliary muscle.

The angle can be blocked, (1) by extreme dilatation of the iris (Fig. 2). It is like the effect produced when a curtain is drawn back from its night to its day position. This tends to fill up the angle and so to impede the passage of fluid through it. (2) The ciliary body may become congested and swollen; its apices move forward, press upon the base of the iris, push the latter against the cornea and close the

angle. Simultaneously with this, the attachments of the suspensory ligament move forward and so allow the lens to advance and to press on the iris base. (3) The lens enlarges as life advances, and the swollen lens presses the iris forward and blocks the angle just as in the previous case.

When the iris and cornea are brought into close contact in any of the ways above described, their adjacent surfaces tend to be glued together by the exudate they throw out and so the angle becomes permanently obliterated. Clinically, this is a most important fact for it makes the establishment of filtration by any operation very difficult to bring about (Fig. 3, A. & B.).

FIG. 3A.

FIG. 3B.

(From original photos by E.E.)

Angle of chamber :

A, Open ; B, Closed. S, Sclera ; l, limbus ; c, cornea ; sch, canal of Schlemm ; p, pectinate ligament ; a, angle of chamber ; c.b, ciliary body ; i, iris.

Note that in A the angle is widely open, and the pectinate ligament is freely bathed in the fluid of the chamber ; whilst in B the base of the iris is adherent to the pectinate ligament, thus shutting the canal of Schlemm off from the aqueous fluid.

The crypts on the surface of the iris lead to the depth of the iris stroma and help to carry fluid away in the early stages of glaucoma. This finds its way along the iris stroma to the pectinate ligament and into Schlemm's Canal.

The ciliary body has been a fierce subject of contention, some believing that it was definitely secretory in function while others hold that fluid is transuded from its lining cells. I shall not carry you into this discussion.

There is a free communication between the vascular system within the eye and that on its surface through the perforating vessels which pierce the sclera close behind the cornea. Both here and in the neighbourhood of the vasa vorticosa, the evidence of congestion within the eye is to be looked for when present.

The whole of the choroidal and retinal circulation lies between the fluid contents of the eye within, and the unyielding sclera without, and must suffer whenever the pressure within the eye is increased. The arterial and venous circulations react differently. The pressure diminishes the amount of blood entering

the eye through the arteries and also that leaving it through the veins. The consequences are diminution of the arterial supply and the production of venous congestion. If the pressure rises slowly the circulation can adapt itself to the change of conditions and the glaucoma remains simple; but if the increase in pressure is rapid, such adaptation is impossible and congestive glaucoma supervenes.

(Photo by Mr. W. Chesterman.)

FIG. 4.

Cupping of the optic disc (glaucomatous).

Note (1) the sharp scleral spur on the right side; (2) the overhanging edges; and (3) the retinal vessels hiding under the latter.

The optic nerve entrance, being the weakest part of the eye, is the first to yield under a rise of pressure; hence the cupping of the optic disc (Fig. 4). The whole nerve-head, including the lamina cribrosa is pushed backward. Owing to the fact that the fibres of the optic nerve lose their medullary sheath as they pass through the scleral coat, the bulk of the nerve diminishes from without inward; consequently, a section through this part shows the nerve like a truncated cone with its base at the external surface of the eye and its truncated end at the fundus. Naturally the channel in which it lies has the same form. This explains the shape of an advanced glaucoma cup with its overhanging edges and with the apparent interruption in the continuity of the bloodvessels emerging from it.

Intraocular Pressure and Ocular Tension.

The tension of an eye is a measure of the state of distension of its tunic. It can be roughly estimated by the fingers, but much more accurately by a tonometer. This is essentially a specialist's instrument. You might as well estimate a patient's temperature by placing your hand on his skin as take an intraocular pressure with the fingers alone. There are plenty of cases in which the eye is obviously subnormal or hard, but there are a large number in which even a skilled ophthalmic surgeon might be mistaken as to the presence of increased pressure, for the tension of the globe depends on other factors beside the intraocular pressure, the most important of these being the thickness and distensibility of the ocular tunic. The

only way that we can accurately estimate the intraocular pressure is by introducing the needle of a manometer into the globe. This for clinical purposes is manifestly ruled out.

The sclero-corneal coat has only a slight degree of distensibility or of elasticity, and these properties are best marked in the young. I am going to avoid controversial subjects, as for one thing, time would not permit of taking them up. The major part of the contents of the globe consists of the intraocular fluid which is being constantly changed. We will not discuss the method of that change, but it is essential that as much fluid should leave the eye as passes into it out of the vessels. Otherwise, the globe becomes over-distended and hard, just as a football does when it is blown up. You can blow a football up till it feels as hard as stone; an eye over-distended with fluid has a similar hardness. There is another element in the case. The blood vessels in the eye are full of fluid; the volume of this blood must be added to that of the clear intraocular fluid. Each systole drives an extra quantity of fluid into the eye. This escapes again during diastole. If you watch an eye on the verge of glaucoma you will observe an arterial pulse. This is quite a different thing from the venous pulse which is a sign of little significance and which may be disregarded. An arterial pulse is a *very important danger sign*. It means that the intraocular pressure is so high that it can only be overcome at the height of the systole, and that during diastole it suffices to close the lumen of the arteries and to stop the flow of blood through them. It is a striking sign. To detect it, look for the lighter coloured arteries and you will notice that through the whole length of these where they lie on the disc, there is a flash-pulse, very different from the limited pulse often seen at the proximal ends of the veins just before they disappear into the nervehead. If there is no arterial pulse, press gently with the finger-tip on the globe of the eye whilst directing the patient to fix his vision on a light on the opposite wall. If glaucoma is threatening, the arterial flash-pulse will at once be seen. I never put any mydriatic into an elderly eye without first testing in this way for the possible danger of threatening glaucoma. Let me pause here to warn you most earnestly against ever allowing an elderly patient to escape from your observation with a pupil dilated by a mydriatic you have used, unless, of course, you are deliberately producing mydriasis and cycloplegia for iritis or some similar condition. I have seen glaucoma follow the use of even the mildest mydriatics, and at least three times after injections by dental surgeons to facilitate extractions. On the other hand, I meet with not infrequent cases in which homatropin or even atropin are being used to obtain temporary mydriasis in cases of central nuclear cataract. *I consider such a practice indefensible* and I think it should be completely discontinued. An iridectomy upward is, in skilled hands, a very simple operation, and produces permanent and satisfactory mydriasis usually attended with great improvement of vision and with no risks such as drugs involve. Indeed, it adds to the patient's safety from future high tension.

I said a little while back that a tonometer is a specialist's instrument. I should be sorry if that led any of you to think that it is a difficult one to use. This is far from being the case. What I meant was that it is one that you seldom feel the need of in general practice. In case you wish to use it I offer you a few suggestions. (1) Stand the base of it in absolute alcohol for a couple of minutes beforehand, then dip it under freshly boiled water and work the piston up and down from above a few times to get rid of the alcohol; next dip the base into sterilised castor oil before applying it to the eye. This is a great protection against injury. (2) Have the patient either lying down across a bed or seated in an armchair, the head-end of which can

be lowered. Standing behind the patient, you can manipulate the instrument easily. (3) Having used a local anæsthetic, direct the patient's attention to a spot, preferably a mark, on the ceiling straight above him. (4) While he is watching this mark with the other eye, get an assistant to draw the lower lid down gently, and holding the upper lid up with your left hand, lower the base of the instrument on to the cornea, slowly, steadily and without a jerk. (5) Whilst taking the reading, notice if there is an arterial pulse, as the latter is a sure sign that a true reading has been taken. Be careful that the instrument is at right-angles to the patient's eye. If he is nervous, talk to him reassuringly and explain what is being done. Violent movements on his part may easily cause an abrasion of the cornea which is most undesirable. (6) Don't let him out of sight until the mydriasis has been neutralised by miotics. This may take some time, but it would be a terrible disaster to allow the patient to go home and contract an attack of glaucoma due to the mydriatic you have employed.

The Pathological Anatomy of Glaucoma.

All I wish to do is to draw attention to a few points of outstanding interest. How is it that we meet glaucoma with increasing frequency as age advances? I am going to suggest to you a rough analogy. A new area is built in a town. The surface drainage is provided for on ordinary expectations. For years all goes well. Then a very heavy storm strikes the town and the drains are overflooded. The town has perhaps grown and the drains are now scarcely sufficient, or at least the margin of safety is so small as to be untrustworthy. The drains have not been kept in proper repair and their lumen is partly blocked, hence the flooding. There is one factor more to take account of, namely, a sudden and very heavy rainfall.

Now to apply all this to the glaucoma case. We can leave aside all dispute as to exactly how the mechanism acts and simply grasp the main facts. The majority of people are provided at the start with a drainage system equal to any demands that will be put upon it in a normal lifetime. In others, the drainage system has always been a bit on the skimpy side; in yet others, skimpier still, whilst in a few, the buphthalmic children, it has been too scanty from the first. Even with the more happily placed ones, the arrangements for disposing of superfluous fluid become less favourable as life advances, and the dangers inseparable from a flush of fluid are steadily increasing. Then again, in early life the cardio-vascular system is elastic, adaptable and resourceful. As years go by, these properties lessen, and the danger of vaso-motor storms increases, as also does their severity. The consequence is that the eye is faced from time to time, more frequently and more strongly, with the task of having to dispose of an increased bulk of fluid which threatens a dangerous rise in the intraocular pressure. With all this there are connected some interesting points. The anterior chamber of the eye is shallow, lessening the channel of excretion to which I drew your attention when speaking of the anatomy of the part. As a result of long continued glaucoma the angle becomes not merely closed, but sealed, so intensifying the obstruction just alluded to.

A warning signal is the small size of glaucomatous eyes. I look upon this as very important, and when a patient comes to me with a family history of glaucoma I measure the diameter of the cornea very carefully. In the event of it being large, I give a comparatively happy prognosis, whereas, when it is below the average, I feel distinctly anxious as to the future.

Causes of Glaucoma.

First among these I would place *age*. Every decade in the advance of life brings increasing risks up to at least the sixth. *Age* acts by altering the anatomical condition of the parts; (2) by the increase of degenerative changes; (3) by rendering the eye more liable to auto-intoxication; and (4) by interfering with the smooth working of the cardio-vascular system. *Sex*. Congestive glaucoma is more frequent in females, probably as a result of the unstable condition of their nervous system associated with the climateric. Non-congestive glaucoma is rather more frequent in men than in women. *Heredity* is a very important influence and should be carefully borne in mind when dealing with the descendants or relations of glaucoma cases. Of mydriasis I have already spoken. Nerve shock and strain are powerful exciting causes. Bereavement, haunting fear, business anxiety, long fatiguing night-watches, prolonged mental overwork, sleeplessness and other similar factors loom large in the excitation of congestive attacks. I would like you also to bear in mind the influence of injuries, for very slight trauma to the eye will undoubtedly sometimes bring on an attack.

The Diagnosis of Glaucoma.

I have no time to deal with this at length. It would demand a small book to itself. What I do want to do is to try and be practically helpful to you, for glaucoma is still today one of the pitfalls of practice. I do not believe that any one of you need ever be "let down" if you will accept a little simple advice. (1) Never forget the possibility of glaucoma. (2) When in doubt shelter yourself behind an expert's opinion.

There are plenty of cases in which no one living can give an absolute opinion after one examination; but there are none which, if carefully watched, do not unfold themselves and make the diagnosis certain.

Two problems constantly present themselves. (1) Is this a case of glaucoma? And (2) when should surgical interference be undertaken?

There is no such thing as "prodromal glaucoma". The disease can be divided into three stages, the early, the established and the late, only too often the "too late". Over 30 years ago I urged that if operation is to be of any use in glaucoma it should be undertaken as early as possible. I did not then know that over 30 years earlier Von Graefe and De Wecker had laid stress on the same point. I could show a large number of case-sheets in which years had been wasted and vision had been allowed to deteriorate very seriously before any ophthalmic surgeon first saw the patient.

What signs and symptoms should put the practitioner on his guard? All depends on whether the case is of the simple or of the congestive type. Let us take the last named first. The mention of coloured rings round lights should at once attract your attention for it may be the only symptom or sign that the patient is aware of in the very early stages. He will notice that when he is tired, as for instance on his way home from office on a winter night, every lamp has a rainbow halo round it. Don't get confused over the many-coloured sparkling haloes cast by the beam of a motor car's headlight or by a bright street-lamp (Fig. 5). The true halo

is like a rainbow, blue inside and orange and red on the outside, with a green or yellow in between; only it is a complete ring instead of a half one. You can get a

(Morax.)

FIG. 5.

The corona of light round a flame; this is sometimes confused with the coloured haloes of glaucoma. It is not a pathologic phenomenon.

fair idea of a halo by dusting some lycopodium between two glass plates and binding them together like a magic lantern slide. A very vivid halo is got by pricking your finger and putting a drop of blood into your conjunctival sac. Then look at a candle or match-light at the other end of a dark room. When in doubt, show these tests to a patient and tell him to be on the look out in the future.

Headache. A frequently repeated headache when a patient gets tired always deserves suspicion, especially if one-sided. Enquire for coloured rings round lights. *Make a point of seeing the patient during an attack* and look out for two things. (1) A dilatation of the pupil on that side; and (2) a zone of circumcorneal congestion. Lose no time in shifting the responsibility on to an ophthalmic expert. Don't let vomiting mislead you into thinking it is a case of migraine.

A very severe headache, especially if one-sided, should always raise the possibility of glaucoma in your mind. The accompanying halo will be brilliant, the anterior chamber very shallow, the pupil widely dilated and the circumcorneal congestion well marked. You can't make a mistake if you remember the possibility of glaucoma. The effect of eserin will clinch the diagnosis.

Then as to simple or non-congestive glaucoma. Here the possibility of overlooking the condition is very much greater, for all that serves to furnish a diagnosis may be limited to what, many years ago, I spoke of as the essential triad of glaucoma signs. (1) Contraction of the field; (2) Cupping and atrophy of the disc; and (3) Failure of visual acuity. Even the last named sign may largely fail us,

for central visual acuity sometimes remains quite reasonably good until very late in the disease. What the patient does notice, however, is the restriction of his visual fields. This may come on so slowly that his attention is not drawn to it until quite a late stage. It is, however, accompanied by a symptom which should always make the doctor anxious and suspicious. I refer to a marked diminution of the light sense. The patient notices that when he comes out of his brightly lit office on a winter night it takes him far longer than formerly to adapt himself to the sudden change of illumination. Again, when he goes from shadow into bright light he is similarly troubled, though perhaps not quite so severely. Not a few of the patients will have noticed that their distant vision as well as that for reading, is not quite so good as it used to be. Of course, there are many other more likely explanations of these troubles than that of glaucoma, and yet I would urge upon you the necessity of bearing it in mind and of excluding it. It is so easy to do this. The typical cupping of the disc which even a student can recognise, places the diagnosis beyond doubt. The whole area of the nerve-head is depressed *en bloc*; the dead whiteness is unmistakable and the alteration in the apparent direction of the vessels as they pass from the disc on to the surrounding fundus is equally so. If the pressure is high an arterial pulse can be very easily elicited by gentle digital pressure on the globe. The arteries are on the small side and the veins on the large.

It was my good fortune to work under Dr. Samuel Jones Gee, at St. Bartholomew's Hospital. He insisted on every one of us students using the ophthalmoscope as a routine procedure. To-day, with the self-lit instrument, ophthalmoscopy has been made very easy and very simple. Cases of glaucoma are missed, not because the practitioner cannot diagnose them, but because he *sometimes forgets entirely that there is such a disease.*

From this we go on to consider for a moment the decision as to whether and when operative interference should be substituted for medical treatment. One must always bear in mind the age and the state of health of the patient. Will his sight outlast his life? If not, and unless there is some very strong contra-indication, operation should be undertaken. Great age, the presence of glycosuria and other conditions which are often considered decisive contra-indications, are by no means to be invariably accepted as such. If the pressure remains high; if the fields are diminishing; if the blind spot is enlarging, and especially if the central visual acuity is going down, operation is urgently called for. That is a point, however, which you must leave to your consultant. I personally always welcome the advent of a practitioner into the case, because I can explain every element of it so easily to him, can put both sides before him and can then leave it to him as the friend of the patient, to advise him and to help him to make up his mind as to what should be done.

In the meantime, there are certain pieces of advice which one gives the patient and which one asks the doctor to see are carried out. (1) The regular use of a weak solution of pilocarpin should be undertaken. Instil the drops in the early morning, last thing at night, and about 2 or 3 o'clock in the afternoon. I will give you my reasons for specifying these times. During sleep, the pump action of the intraocular musculature is in abeyance; therefore instil a drop as late as possible and again as soon as the patient wakes. As the day goes on and the patient gets tired, his vasomotor mechanism is apt to be upset. Then is the very time when we want to make escape of fluid at the angle of the eye as easy as possible. Whatever theorists may have to say about it, the advice is good. (2) The patient should avoid stooping down with the head low and thereby

throwing an extra strain on the cerebral and intraocular circulations. He should also avoid lifting weights, pulling on straps and ropes, straining due to constipation, and all such incitements to cerebral congestion. (3) Hurrying for trains and 'buses, and getting fussed over anything are bad for the glaucomatous patient. Worry, anxiety, sorrow and all depressing emotions are very harmful to him and he should endeavour to cultivate a philosophic spirit and to cut out the influence of such factors as far as he possibly can. It is easier advice to give than to take, but it is practical and a good deal can be done by it. (4) I am averse from any interference with the patient's habits which can possibly be avoided. The attitude of "Go out and see what Tommy's doing and tell him not to", is bad. Within moderation and unless there is some strong contra-indication, I would not only not stop him working or amusing himself, but I would encourage him to do so, subject to the obvious limitation implied by the words "short of fatigue". (5) Massage of the eye is of great value. The patient can be taught to do it for himself and to employ it five or six times a day for a few minutes each time. If he is intelligent he will soon learn to recognise when the eye is hard and when it has become softened by the massage. The best method is with the two middle or two index fingers placed as far apart on the eye as possible and each pressed in turn toward the centre of the eye; while one finger is pressing, the other should oppose a moderate amount of counterpressure, and *vice versa*. (6) Lay great stress on regular meals and regular sleep.

Operation.

When, in spite of all other treatment, the eye is going downhill, it is the surgeon's duty to advise and even to urge operation. Unchecked glaucoma ends in absolute blindness. If a man is being taken out to be shot, it is worth his while jumping into the river and swimming for it or trying a scrap with his escort, or anything else. If he doesn't, death is certain. No operation for glaucoma is free of risks, but in early cases these are very small and in late ones it is often surprising how good are the results obtained. The eye is condemned; the risks are small compared to the certainty of blindness if nothing is done. In the old days in Madras Hospital, the poor results of iridectomy filled me with despair. Lagrange's operation brought a rift in the darkness, but it was very dangerous. The idea of trephining the eye occurred to me as an alternative, but it was two or three years before I plucked up courage to try it, and that on a practically blind eye. It went so well that I tried several more the same morning. That was in August, 1909. Since then I have done no other operation, for it has fulfilled what, to my mind, are the essentials of a glaucoma operation, namely, the establishment of a fistula between the anterior chamber and the sub-conjunctival space with a minimum of trauma, a minimum of risk and the maximum of ease and certainty. It has taken time for it to make its way, but the reports I get justify me in saying that today it is absolutely world-wide. The more I do it, the more I like it. It is complete; it has a purpose in every detail; and it has given me and the many who use it delightful results. It is true that some people have had a large percentage of failures with it, but I do not think that this can be fairly ascribed to the operation, for there are a very great number who have found it all I claim for it. When I had fully described it and given it a good start I left it with confidence to the judgment and experience of the profession. I felt sure what the result would be and I have not been disappointed. No operation for so serious a condition as glaucoma can be claimed to be free from risk, but I feel that I can advise a patient to submit to sclerocorneal trephining with very great confidence.