GLAUCOMA*

BY

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THE term glaucoma is used by some as synonymous with a raised intra-ocular pressure, but it seems more correct to apply it to a definite pathological entity, a symptom complex in which raised tension is generally present. There are cases of glaucoma without increase of intra-ocular pressure, and I think that I am right in saying that there are hard eyes which are not strictly glaucomatous. We can classify glaucoma as primary and secondary, if indeed we are correct in designating cases of cyclitis with high tension as truly glaucomatous.

Primary glaucoma can again be sub-divided into two categories: *congestive* glaucoma, and *glaucoma simplex*. I regard these two conditions as completely separate diseases, having but one thing in common—a raised intra-ocular pressure.

Congestive glaucoma is like a strangulated hernia, and is due to the formation of a vicious cycle. It may attack an eye which is, as far as one can discover, perfectly normal. Thirty-five years ago I performed my first operation in England at a private house in Coventry. It was an immediate success. I saw the patient many years later. He had never had any further trouble with the eye, and I could find no sign of glaucoma. Although I hold that the two classes of glaucoma that I have mentioned have little if anything in common, yet a case of congestive glaucoma may pass into a chronic stage which is quite unlike glaucoma simplex, and glaucoma simplex can develop congestive signs, and pass into a stage similar to that of a chronic congestive case. There is at present no certain knowledge of the cause of congestive glaucoma, but it is probably due to a physico-chemical derangement which leads to absorption of water by the vitreous, which swells, pushes the lens forward, and so blocks the iridic angle. This closure of the exits leads to more congestion and further advance of the lens, till the aqueous circulation ceases and the vicious cycle is complete. It is obvious that whereas there is free communication between the retro-lental space and the posterior chamber, no increase of the intra-ocular pressure can of itself alter the position of the lens. This can be attained by three conditions: an accumulation of fluid in the supra-choroidal space; general swelling of the choroid from vascular engorgement; and swelling of the vitreous. There

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is no evidence that fluid accumulates outside the choroid, nor can I think that hyperaemia of the choroid can attain such proportion that it can thrust the lens forward. We are left with a swollen vitreous. This seems to me to be the most probable cause of the initial rise of intra-ocular pressure in congestive glaucoma. There is no relationship between the general blood pressure and the ocular tension.

The signs of congestive glaucoma are mainly those of congestion, which may be moderate or intense. The general appearance in a typical well-developed case is pathognomonic. The conjunctiva is engorged and there is ciliary injection. The cornea is hazy, the pupil semi-dilated and irregular. The anterior chamber is shallow, and the pupil area greyish rather than black. Examination of the cornea with lateral illumination and the loupe shows that it is oedematous and that in the general steamy surface there are clear lacunae. This haze of the cornea may be partly due to polarization of the light reflected from the corneal lamellae. The late George Young, who used to give the lectures in optics for this course, showed that if a pig's eye be squeezed the cornea at once becomes milky, and he suggested that the increased intra-ocular pressure made the corneal lamellae slide over each other and induce polarization. Often the fundus is invisible, but when the media are sufficiently clear "flashing" of the arteries may be seen. I avoid the usual term, "visible pulsation," for this gives an entirely wrong impression of the actual picture. The whole arterial system flashes like the beam of a French lighthouse. It is not at all like the visible pulsation of a retinal vein, which is quite a slow process and has no relationship to glaucoma, although Elliot held a contrary view. It is possible to make the retinal arteries flash in this way in a normal eye by pressure on the globe. If an eye is predisposed to an attack of congestive glaucoma this flashing may be induced by quite gentle pressure, and this is a most valuable diagnostic method. In a very acute case the iris vessels may be injected. The mesoblastic part of the iris consists mainly of thick-walled blood vessels, and some of these at any rate are capable of resuming their embryonic function of carrying blood.

There are often definite prodromal attacks of congestion which are characterised by pain, slight or severe; by obscurations and dimness of vision; often by vomiting; and by coloured haloes round lights. The attacks may become more and more severe till one day the vicious cycle closes and glaucoma is present. Congestive glaucoma is accompanied by severe, often agonizing, pain in the eye and brow, there is dimness of vision, sometimes blindness, and severe constitutional symptoms. There is a rise of body temperature, and often *severe vomiting*. This most

characteristic symptom is of supreme importance because these general symptoms lead to a diagnosis of "influenza," with accompanying ocular neuralgia. Much valuable time may be lost and treatment of the most detrimental kind may be applied to the eye. One well-known ophthalmologist of the past said that he would like to rent permanently a whole page of the *British Medical Journal* and on it print, in leaded type: "*Vomiting is a common symptom of congestive glaucoma.*" A child who develops a rise of ocular tension after a dissection of the lens does not complain of pain—he vomits.

The diagnosis of congestive glaucoma is generally easy, but may be very difficult. The only disease that ought to be confused with it is irido-cyclitis with rise of tension. We ask ourselves: Is this a case of glaucoma or of irido-cyclitis? Am I to use atropine or eserine? There is a simple answer: You are not compelled to use either; just order normal saline irrigation, and perhaps diathermy, and wait and see. The diagnosis may be made by careful use of the loupe and of the slit-lamp. This may reveal keratic precipitates and an albuminous aqueous. One does not find "K.P." in a pure glaucoma, and I think that a highly albuminous aqueous is strong evidence of cyclitis. Some think that there is no real distinction and that many acute glaucomas are secondary to cyclitis. Be this as it may, whether or no the basic causes are identical, the treatment is widely different and a clinical diagnosis is imperative. But in some cases the cornea is so hazy that the slit-lamp is useless and the diagnosis may be impossible. In such cases, after a short period of expectant treatment, I would use eserine, and if this appears to be harmful, then try homatropine rather than atropine. If this treatment is obviously wrong, then the dilatation of the pupil caused by homatropine is more readily overcome by eserine than the action of atropine.

The old idea was that once glaucoma was diagnosed, an operation must be undertaken at once. Every hour was precious. This is not the case, and I think that to-day few surgeons rush to an immediate operation. Once, at The Warneford Hospital, some 25 or more years ago, I saw a case of violently acute glaucoma. Both eyes had been treated, I think, for a fortnight with atropine; both were stone-hard with widely dilated pupils, and perception of light was abolished—a truly desperate case. I trephined both eyes and the result was incredible. The eyes became soft and lost all inflammation, and eventually both recovered, with a final acuity of 6/12. This astonishing outcome cannot be taken as an example for it is most unlikely to happen again, but it shows that it is not necessary in the ordinary case to rush to operation upon a highly inflamed eye when all the conditions are unfavourable for an operation. However violent the glaucoma,

I would always try the effect of eserine instilled every two hours till the pupils contract, and use diathermy. Hot pads and the wooden spoon are relics of a past age, for we now know that however hot we make the lids, short of actual scalding none of the heat reaches the eye. This has been proved by placing a thermopile under the lids.

There is no reason for not trying diathermy, but if the patient does not like it I would not continue its use. I rather tend to regard diathermy as a passing fashion, and have seen little, if any, good follow its use in any ocular condition; but others are most enthusiastic and so I must not dogmatize. The patient is so very unhappy that I see no reason to add to his discomforts by free purging. This has come down to us from the middle ages, but there is not an atom of evidence that purgation has any action in lowering intra-ocular tension. Rest in bed and eserine is the proper treatment. Injections of hypertonic solutions into the veins might be useful, but I have not tried it. I have seen no good effect from the use of dextro or laevo-glaucon.

If this expectant treatment results in the pupil becoming small and the eye getting white, and if the tonometer shows that the tension is normal, then I think that the correct procedure is to do nothing active. The glaucoma has been overcome. It may never return, but it probably will. Let the patient have a 1 per cent. solution of pilocarpine to use every night, with instructions to push it should he note any recurrence of his original symptoms (pain and misty vision), and at once obtain the advice of a specialist. He must remain under constant observation. If he is the sort of man who will forget his troubles and let things slide, or if he will be living in a place where he is far from any ophthalmic surgeon, then an operation must be considered, and this must be undertaken if the tension does not return to normal.

Having decided that an operation is necessary, we must consider the best method. It is usual to perform an iridectomy, and many still adhere to what is called "the classical iridectomy." This is a horrible mutilation in which two-fifths of the iris, or even more, is removed. In London the section used to be made with a cataract knife, and some still adopt this instrument. I have always used a keratome, and I think that this is the instrument chosen in most clinics of the world. The chief object of the operation is to clear the iridic angle; the mere size of the coloboma has no merit. In order to effect this with ease and safety I, many years ago, devised my "trap-door" operation. I have described this elsewhere,* but in the main it is a trephine in which the disc is not removed, but replaced. The operation begins as an ordinary trephine operation, except that it is not necessary to cut a wide

* *Brit. J. of Ophthalm.*, December, 1932. *Trans. Ophthalm. Soc. U.K.*, Vol. LVI, 1936.

conjunctival flap. The hole is cut so that the disc remains attached at the upper aspect as described by Elliot. The iris almost invariably prolapses and is excised. The disc is now replaced and the conjunctiva sutured in the usual way. There are many advantages in this operation and, as far as I know, no disadvantages. There is no danger of wounding the lens. There is no post-operative astigmatism which may be present if a cataract knife is used. In many cases there will be slight filtration, but rarely enough to cause an ectatic scar. I have come to the conclusion that generally, if eserine has failed to bring the glaucomatous attack to an end, then mere iridectomy will be insufficient to prevent recurrences. This is where the slight filtration that may follow the "trap-door" method is so valuable. I do not care for iridencleisis in acute cases, and if the anterior chamber is shallow it is almost impossible to perform it without danger to the lens capsule. I have seen cataract with a tear in the capsule follow the "trap-door" operation on two occasions: I think that the capsule becomes very friable in acute glaucoma, and the mere removal of the slight support of the iris may lead to a rupture. In neither of these cases had any instrument been introduced into the anterior chamber, and yet there was, in one case, a triangular tear in the capsule just as though it had been wounded by a keratome.

If an operation is unsuccessful, then one must think of the presence of a tumour in the eye, or of thrombosis of the central retinal vein. If the other eye shows no sign of glaucoma, and if the vessels are healthy, then it is very probable that the glaucomatous eye contains a tumour. I have seen two cases of this nature recently. If operation fails to cure the glaucoma, remove the eye.

Recurrence is not uncommon after a simple iridectomy, and in this case it is wise to trephine the eye. If, however, the operation is successful, the cure may be permanent, and one does not see that slow deterioration that we shall have to describe in cases of glaucoma simplex.

In the absence of more certain knowledge, we can say only that glaucoma simplex is the result of senile changes in the eye and, I think, mainly affecting the optic nerve. Priestley Smith thought that it was caused by enlargement of the lens, and those who read his book (as all should) will almost be persuaded by the logic of his facts and the theories he deduces from them. But is there any real evidence that the lens is unduly large in the majority of eyes suffering from this disease? I am very sceptical whether there really is such a thing as a glaucoma caused by an intumescent lens, or whether the lens ever is intumescent. Does the lens, in fact, ever "swell"? Probably in an eye otherwise predisposed to glaucoma an unduly large lens may be the final act in that train

of events that end with a glaucoma simplex with raised tension. Close attachment of the iris root to the back of the cornea, the so-called *soudure de Knies*, is another suggested cause. In many cases of glaucoma the iridic angle has been found on section to be widely open, and the gonioscope shows the same condition. The *soudure* is an effect—not a cause. Increased production of lymph due to changes in the cervical sympathetic is a mere speculative cause. There is no evidence that arterio-sclerosis has any aetiological significance. We must content ourselves with the conclusion that we do not know the cause of glaucoma simplex, but we believe that, like cataract, it is a senile degeneration. In the majority of cases glaucoma simplex has no symptoms till it has been developing for a long time — perhaps for years. Many of our patients are not aware, till we tell them, that there is anything wrong with their eyes. Sometimes, indeed, we are the first to draw their attention to the fact that one eye is actually blind, or that they have a very contracted field of vision. Pain is unusual and is present only in some of those cases where the tension is raised or when congestive signs have been super-added to the simple state.

I wish to emphasise the fact that there are only three important and characteristic signs: changes in the optic disc; alterations in the fields of vision; and increase in the intra-ocular pressure. The last is the least important as a diagnostic sign, and may be absent.

Glaucomatous cupping of the optic disc when fully developed is so typical that it ought not to be mistaken for anything else. The cup comes close up to the edge of the disc; its edges are sharp, and are often undercut so that the path of the vessels is lost as they dip over the edge; the vessels reappear in a different plane and seem to have lost their continuity. But in its early stage the glaucoma cup may be difficult to diagnose. There is a shallow, sharp-edged, senile, saucer-like cup identical in appearance with the nascent glaucoma cup, and it may show the same pallor. It is never as white as the advanced glaucoma cup, which often shows what the French call a *nacré* hue—in plain English, mother-of-pearl. In some cases of tabetic atrophy there is a definite cup. Once I was completely deceived and spent a long time trying in vain to obtain confirmatory signs of glaucoma.

Finally, there is a congenital deformity which copies faithfully a fully developed glaucoma cup. In my young days I once confidently diagnosed a glaucoma cup in a young male private patient, but my view was negatived by an older and more experienced consultant. I lost *kudos* but gained knowledge. I saw the man many years later. There was no alteration in the appearance of the cups, and no other signs of glaucoma. If you will make a practice of examining with the ophthalmoscope

every patient that you see in clinic or hospital you will occasionally pick up this kind of oddity. In a school clinic it is easy to have a peep by the indirect method—one rather neglected in this country. May I remind you that if you are emmetropic or hypermetropic you will not be able to get a clear fundus picture unless you put up a +2.0 D. sphere behind your concave mirror. The indirect method is of great value in assessing the comparative tint of the two discs; it will show up a small difference in pallor that is not so easily recognised by the direct method. Again, the colour value of the observer's two eyes is not always the same, so if you are in doubt, examine both of the patient's eyes with one of your own. Again, the illumination of the instrument has much to do with the colour of the disc. If you have just had a new battery you will be apt to see pale discs. All these factors must be taken into account in estimating the colour of a glaucomatous disc. To become proficient, never miss any chance of examining normal fundi. There is a very wide margin between the normal and the abnormal, and this is important when you have to decide whether papilloedema is present or absent.

What is the cause of cupping? I am sure that most of you will say that there is an obvious answer. It is, of course, due to the raised intra-ocular pressure driving back the lamina cribrosa. This view was taken in the pre-tonometric era, and is, I imagine, still current. Is it true? I think that I can advance evidence that will at any rate make you begin to wonder, to think and, I hope, to investigate. Some years ago a man came to the Coventry Hospital who had been struck in the eye by a fragment of steel that had flown from a fractured drill. An X-ray picture happened to show the optic nerve quite clearly, and it was seen to contain the large mass of metal that I am handing round for your inspection. When the eye had been excised I tried to remove the steel with artery forceps—in vain. I had to get a pair of pliers to extract it. This is the structure that has to be cupped by the intra-ocular pressure, and yet the eye does not bulge at the intercalary region where it is most vulnerable. Then I find that cupping is not present in cases of congestive glaucoma when the eyes are stone-hard, even if this enormous pressure has been acting upon the optic nerve for quite a long time. On the other hand, I find deep 100 per cent. cups in eyes that have never shown any rise of tension as determined with the tonometer. But I am told this is true if the tension is estimated in the day-time, but if you try at night you will find hyper-tension. This argument seems to me to be mere casuistry. Schnabel, some thirty-five years ago, published an article pointing out that eyes which he had operated upon for glaucoma simplex might, during the few weeks that

they were in the ward after the operation, and with a now normal tension, develop a glaucoma cup. I have myself seen this happen more than once. In one case at the Coventry Hospital I made a note, "No cup." A month or so later I noted, "Shallow cup," and after a few more weeks, "Deep cup." This case had only a moderate rise of tension—I think about 30 Schiötz units. I am unable to give you the exact dates and figures because the Coventry Hospital has been practically destroyed by enemy action, and I doubt whether the records could be found. Schnabel described a condition which he called *cavernous atrophy*, in which atrophy of the glial tissue in the nerve head causes large spaces to develop, causing a cup.

Pickard, of Exeter, has made a careful study of glaucoma without rise of tension, and has written several papers on the subject. I have seen many of these cases and have followed up a few of them for years. Some of them have shown no deterioration of function, but in others there is a slow, but unfortunately sure, downward course. Professor Enroth, of Helsingfors, told me, in a private communication, that in his experience the majority of cases of glaucoma simplex ended in total blindness whatever treatment was adopted. This has not been my own experience. I have seen many cases fifteen and twenty years after a successful operation who have maintained their visual functions unaltered. On the other hand I have not infrequently been asked to examine patients for blind pensions upon whom I had operated apparently with success in the past, and many of them have been blind. My own view is that the glaucoma cup is caused by a senile degenerative process in the nerve head which softens it and allows it to become cupped by very slightly raised tension, or even by the normal intra-ocular pressure. In secondary glaucoma deep cups are often present, so we must assume that the inflammatory process has the same action upon the nerve tissues that we have assumed in glaucoma simplex. In some cases the depth of the cup appears to diminish after operation, but in others, in spite of a normal or even a sub-normal tension, the process goes steadily on; the cup becomes total and the fields small, till eventually complete blindness results.

The alterations in the fields of vision that are seen in glaucoma may affect either the peripheral or the central part of the field, or both. The typical peripheral change is the formation of a nasal step. This quadrantal notch deepens till it approaches the fixation spot. Here it tends to halt for a time and the patient alternately sees and loses the object he looks at. But one day the step reaches the centre and then central vision is lost. In my own opinion no time should be lost in a case of this kind; an immediate operation is called for. It has been taught that it is dangerous to operate—

that the intervention may just push the eye over into central blindness. My colleague at the Birmingham Eye Hospital, Mr. Charles Rudd, has just told me that he has recently experienced this misfortune with one of his patients, and there is no doubt that it occasionally happens. On the other hand, if nothing is done the end is certain, and generally the operation is successful. I have operated upon cases of this kind and have never seen immediate blindness follow. I had one case of this kind at Leamington—a private patient. She complained that objects kept going and coming, and she had the typical step impinging upon the central fixation spot. I performed a trephing operation, and followed up the case till the lady died. There was no further deterioration, and to the end she kept losing her central fixation and finding it again. In many cases the end stage of the field is a small central area all, say, within the five degree circle. These are not so dangerous as the deep steps.

The changes in the central part of the field are most important, and an examination with the tangent screen, the Bjerrum screen, must never be omitted, using small test discs and working at a distance of two metres. The first step should be to pass the test object along a circular path, about half way between the blind spot and the fixation spot, using the fixation spot as a centre. If there are any horn-like prolongations of the blind spot this path will intercept them. The common scotoma in this region is the Siedl. It consists of a horn-like extension of the blind spot along a course roughly concentric with the meridians on the chart. These horns may be above or below the central raphe, or in both situations. They may extend to embrace the fixation spot and implicate central vision. I have seen this happen when the peripheral field showed no contraction. We may find peripheral contraction, central scotomata, or both. These central scotomata are not very common, but when present their effect may be devastating and quite unexpected—in fact, loss of central vision from this cause may be the first symptom of glaucoma simplex. Never omit the Bjerrum test. It takes a very short time to demonstrate the absence of a Siedl scotoma, but a long time to map it out properly. You will find full details of all these field changes in Traquair's Book on Perimetry.

Rise of intra-ocular pressure is, from a diagnostic standpoint, the least important of the signs of glaucoma simplex. In general it is not high, averaging from 30 to 35 Schiötz units on the old scale—say at most 40 units. Often it is much lower, and, as we have seen, may not be present. In the past there was much muddled thinking. Congestive glaucoma and its variants had not been clearly differentiated from the simple variety, and the tonometer was not in general use. I have made a most careful

study of this instrument from the time it was introduced by Schiötz thirty-five years ago. I tried the Sydney Stephenson instrument and found that it was unreliable. But I have not the slightest doubt that the Schiötz model is a thoroughly reliable instrument. This is, I think, the opinion held outside the British Commonwealth of Nations, but there are still some in the country who think that they can form a better judgment with their fingers. One still hears of the *tactus eruditus*, a term that used to impress medical students (special fingers given to special men). My own fingers are most untrustworthy. Without the tonometer I should have operated upon eyes not glaucomatous, and eyes calling for operation would have been left alone. At two meetings of the Midland Ophthalmological Society I asked the members for opinions on the tension of an eye and I got the most extraordinary answers: "Normal," "A little up," "Softish," "Hard." The tonometer at once gave the true reading. And yet I have been told by my colleagues that the tonometer is quite unreliable, and that "educated fingers" are far better.

Sometimes we get a case of glaucoma simplex with a very high tension. This is often a long-standing neglected case with a deep cup, small field, with shallow anterior chamber, and other signs indicating that a simple case has become complicated by congestive features. These cases call for immediate operation, probably more than a mere iridectomy, and they generally do badly.

Apart from the three signs that I have mentioned of glaucoma simplex, there are no others of any importance. It is said that changes take place in refraction and accommodation. I have never noted such. In many old persons the anterior chamber tends to get smaller, probably because the lens is larger than normal. The same may be found in glaucoma simplex, but a shallow anterior chamber is not a typical sign of the simple variety. These vague signs are those rather of senility than of disease.

The differential diagnosis is generally not difficult. I have already mentioned some of the conditions which may lead to difficulty. Certain cases of chiasmal lesion cause fields very similar to those of glaucoma, but they tend to be bilateral, and only a deep senile cup could lead to confusion with those forms of glaucoma which show no rise of tension. In all doubtful cases an X-ray picture of the sella turcica will make the diagnosis clear. It should be a universal rule to photograph the skull in any obscure change in the optic disc and fields. A slowly growing cataract may mask a glaucoma simplex, especially if bilateral, and no doubt mistakes have been and will be made; but the routine use of the tonometer will obviate many of them. The modern tendency to quick refraction, caused by the unsatisfactory staffing of our hospitals, by

the N.O.T.B., by rapid examination of recruits, and by the difficulties of the present time, are liable to lead to early cases of glaucoma simplex being overlooked, and this is more likely to occur to those who carry out refraction without a general knowledge of ophthalmology.

The most important question that we have to decide is whether we ought to operate, and when. I think that one may say that as soon as it has become obvious that, in spite of treatment with meiotics, there is a definite deterioration in function, then an operation is necessary. The age and general physical condition of the patient must be considered.

Many procedures have been employed to cure glaucoma simplex. I have tried iridectomy, Herbert's small flap operations, and punch operations. These have not been satisfactory in my hands. Trephining has so many disadvantages that I reserve it for special cases. The drawbacks are: the frequency of an ectatic scar that is unsightly, often uncomfortable, and apt to be a channel of destructive late infections; and late formation of the anterior chamber, with consequent development of a nuclear cataract, a serious and not very uncommon event. Apart from this form of cataract, my own statistics show that 10 per cent. of all trephine operations are followed by cataract, and this figure agrees with that published by others. For the past fifteen years or more I have chosen iridencleisis as my usual operation. I have now done about one hundred-and-fifty cases, and I am satisfied that as far as I am concerned it is superior to trephining. The operation is not easy, but the results are very good and there are few complications. Cataract is a rare sequel and late infection has not been observed. I have seen many cases up to fifteen years after operation with eyes still functioning normally. Only this month I have examined two. One man, aged seventy-one years, had an iridencleisis ten years ago. He has only one functioning eye, but this has an acuity 6/12. There is a shallow pale glaucoma cup and the field is very small. But he does good work for the country, making fine tools in a munitions factory. He has no cataract. The other eye is blind. There had been two trephining operations and the lens is opaque. In the other case a trephine operation had been done on one eye. There is a nuclear cataract in this eye. The other had an iridencleisis at the same time. The lens is clear and the vision good. I have recently had one case of sympathetic ophthalmitis in a very decrepid old man. He never came up after the operation till the inflammation was advanced and past treatment. Of course, similar conditions have followed trephining and any other operation upon the globe. I have had no experience of cyclodialysis. I used to see many cases of Herbert's wedge isolation performed by a colleague, but I was not impressed with the results.

Time does not allow me to deal with secondary glaucoma, with juvenile glaucoma, or with buphthalmos.

Many of the views that I have expressed in this lecture are heterodox. I hope that you will regard them as my own opinions, but that you will keep them in mind and try to ascertain in your own future practices to what extent they are true or false.

SOME REMARKS ON FINCHAM'S CAPSULAR THEORY OF ACCOMMODATION

BY

J. W. NORDENSON

STOCKHOLM

SOME years ago a new theory of accommodation, called the capsular theory, was presented by Fincham.* It was an attempt to conciliate the two diverging theories of v. Helmholtz and Tscherning. Fincham agreed with v. Helmholtz so far as to accept the idea that the alteration of the curvature of the lens was due to a slackening of the zonula, allowing the elasticity of the lens-capsule to exert its influence; on the other hand he accepted the idea of Tscherning that the lens in accommodation takes a conoidal shape, forming a lenticonus anterior. According to him this conoidal form is due to the fact that the capsule of the lens is of varying thickness in different parts. As this capsular theory, which has attracted a certain amount of interest, is incompatible with certain facts about accommodation, I here allow myself to advance some critical remarks on it.

In forming an opinion on this new theory we have to consider two questions. The first is whether the lens really assumes a conoidal form in accommodation; the second is whether a conoidal form can be caused by the varying thickness of the lens-capsule in different parts.

Before entering on the first of these questions—whether the lens assumes a conoidal shape or not—it is necessary to remark that Fincham's conception of a conoid does not coincide with the generally accepted scientific definition of this word. Thus the ancient mathematicians used it to denote a surface, engendered by an element of a parabola or a hyperbola rotating round its axis, in other words a truncated rotation—paraboloid or hyperboloid. In later times it has been used to denote the surface engendered by a straight line—generatrix—running both along a

* FINCHAM, E.—The Mechanism of Accommodation. Monograph Supplement VIII. *Brit. Jl. of Ophthal.*, 1937.