GLAUCOMA.

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Glaucoma is a term used to denote a pathological increase of intraocular pressure.

The factors concerned in the maintenance of normal intraocular pressure and the alterations, both chemical, physical and biochemical, which take place when the normal mechanism is disturbed are not yet fully understood. In spite of years of research the cause of primary glaucoma has not yet been elucidated.

The problem is exceedingly complex and involves the following:—the formation and drainage of aqueous; the pressure in the intraocular capillaries and the factors controlling it; the relation between the blood in the capillaries and the intraocular fluid and the passage of chemical substances between these two; the part played by the sympathetic system in controlling the amount of blood in the eye; abnormal substances in the blood; and psychical influences, which also play a part.

The immediate cause of a rise of intraocular pressure is obstruction to the out-flow of aqueous. We must consider, therefore, how the aqueous is produced and how it leaves the eye.

Formation of aqueous. The aqueous is formed in the ciliary body. Precisely how we do not yet know. At one time it was thought to be secreted from the cells of the ciliary processes. The researches of Duke-Elder during the last ten years seemed to establish, however, that the aqueous was formed by a simple process of dialysis from the capillaries in the ciliary body. Recently the work of Douglas Robertson (1937) has provided evidence that the aqueous is not a dialysate. He says "There is evidence to show that there is no chemical equilibrium between the blood and the aqueous humour. . . . It is suggested that the evidence chemical, hydrostatic and osmotic, points to the formation of the aqueous humour by a process of secretion, and in addition there is evidence of the necessity for the expenditure of energy in its formation."

Probably the truth lies with both these theories, and that aqueous is formed by dialysis and also by secretion.

Circulation of aqueous. The aqueous is produced in the ciliary body; it circulates in the posterior chamber of the eye, passes forward through the pupil into the anterior chamber, and finally leaves the eye by the canal of Schlemm and the spaces of Fontana which are situated in the angle of the anterior chamber (filtration angle). Hence it reaches the venous system. Drainage is probably brought about chiefly by osmosis.

Etiology.

It is possible that an abnormal production of aqueous might raise the intraocular pressure, but when this occurs it is presumably compensated for by increased drainage. By far the most important cause of a rise of pressure is obstruction to aqueous circulation.

Obstruction. The aqueous circulation may be interfered with

1. In the posterior chamber by swelling of the ciliary body or vitreous.
2. In passing through the pupil, by adhesions between the iris and lens capsule.
3. In the anterior chamber and Schlemm's canal by several factors:—
 - (a) pushing forward of the lens (or swelling of it) making the anterior chamber shallow and narrowing the filtration angle.
 - (b) adhesions between the root of the iris and the back of the cornea narrowing the filtration angle.
 - (c) malformation of Schlemm's canal (infantile glaucoma), blocking of Schlemm's canal and the spaces of Fontana by products of inflammation (cells, fibrin, pigment).

Increase in viscosity of aqueous fluid as a result of intraocular inflammation will retard its flow.

A small eye with a shallow anterior chamber (e.g. hypermetropia) must be regarded as a predisposing cause of the disease. In this connection it must be remembered that the lens increases in size with age.

Clinically there are all stages between a slight rise of tension lasting only a few minutes, and probably not appreciated by the patient, and an eye with absolute glaucoma, i.e. one in which the tension is permanently raised and the sight lost.

It will be convenient to consider glaucoma under the following headings:

1. Infantile glaucoma.
2. Secondary glaucoma.
3. Acute primary glaucoma.
4. Chronic primary glaucoma.
5. Glaucoma capsulo-cuticulare.

Infantile glaucoma (Buphthalmos).

Most cases are seen shortly after birth and may be brought with the complaint that one or both eyes seem too large.

Owing to some congenital defect—adherence of the root of the iris to the cornea, absence or obliteration of Schlemm's canal—the exit of aqueous is interfered with. At this stage of development the cornea and sclera possess elasticity and are able to stretch in response to the increased intraocular pressure. Thus the eye increases considerably in size (hence the name buphthalmos, i.e. ox eye). The nutrition of the whole eye becomes impaired. Opacities develop in the cornea, ruptures occur in Descemet's membrane. The anterior chamber deepens as pressure increases. Lens and vitreous opacities are common. Owing to the large globe, axial myopia develops, myopic fundus changes appear and the optic disc becomes cupped from increased pressure. The disease is sometimes unilateral. The *prognosis* is bad.

Treatment is of little avail. Conservative treatment such as is employed in other forms of glaucoma (*vide infra*) is quite useless.

Some form of operation is usually tried, either a broad iridectomy or a filtration operation, e.g. corneoscleral trephine or sclerectomy.

The disease is rare, but much more rare it is to see an adult with buphthalmos who has any useful vision. My own feeling is that in those patients in whom vision is preserved to adult age it is because the disease has become self-arrested and not by reason of a successful operation.

Secondary glaucoma.

This is a rise of tension occurring during the course of some other ocular disease and resulting directly from it.

Thus it may be caused by:—

(A) **Injury.**

1. Perforating wounds with resulting anterior synechia. This narrows the filtration angle and makes shallow the anterior chamber.
2. Wounds of the lens.
 - (a) in perforating injuries, soft lens matter coming forward into the anterior chamber, swelling up and blocking the filtration angle.
 - (b) dislocation of the lens into the anterior chamber.
 - (c) injury causing serious intraocular hæmorrhage. In this case the lens would be pushed forward.

(B) **Inflammation.**

Iridocyclitis. This is the commonest cause of secondary glaucoma and may produce obstruction in several ways.

- (a) adhesion of the swollen iris to the back of the cornea near the angle.
- (b) adhesion of the iris to the anterior capsule of the lens in the pupil area (ring synechiæ). Aqueous is thus unable to pass through the pupil from the posterior to the anterior chamber. That part of the iris which is free is ballooned forward into the anterior chamber which may thus become little more than a funnel-shaped space in the centre. This condition of the iris is known as iris bombé.
- (c) blocking the angle of the anterior chamber, also the canal of Schlemm by inflammatory products (cells, fibrin, etc.).

(C) **New growth.**

Intraocular tumour (e.g. choroidal sarcoma) may cause a rise of tension either by occupying space or by interference with venous channels.

(D) **Thrombosis of the central retinal vein.**

This may be followed some weeks later by an attack of acute or subacute glaucoma. The cause of the glaucoma is obstruction to the exit of blood from the eye.

(E) **Aphakia.**

Following the extraction of cataract chronic glaucoma is not unknown. Why it should occur is not clear. It has been suggested that there is obstruction to the flow of aqueous by the presence of vitreous in the pupil. Another possibility is the downgrowth of epithelium into the anterior chamber. This might occur in a case in which lens capsule or iris becomes caught in the wound preventing healing.

Symptoms. The symptoms and signs are those of the primary disease accompanied by a rise of tension. If the patient has been under observation for the primary disease, signs of secondary glaucoma will have been carefully searched for.

Treatment is that of the primary disease. Other measures are:

1. *In a perforating injury with anterior synechia*, it may be possible to divide the adhesion. If not, and the eye is useful, an iridectomy or trephine operation may be successful. In wounds of the lens the soft opaque lens matter may be let out of the anterior chamber by paracentesis and curette evacuation. In dislocation of the lens into the anterior chamber it is best to extract the lens. The pupil should be contracted with a miotic in order to deter the lens from slipping back into the vitreous during the operation. In intra-ocular hæmorrhage operative treatment is contraindicated.
2. *Iridocyclitis.* Atropine is essential. By dilating the pupil it will prevent ring synechiæ and iris bombé. Operative procedures may be necessary. The rule is to do as little as possible if there is much active iritis. If the eye settles down a more extensive operation can be better undertaken.

The following operations are employed:—

- (a) (Where there is active iritis). *Paracentesis of the anterior chamber.* The albuminous aqueous is let out and this may favourably influence progress. Too much must not be expected of this operation.
- (b) In ring synechiæ and iris bombé. *Injection of mydricine sub-conjunctivally* to free adhesions. *Iridotomy.* Making a hole in the iris and so establishing communication between posterior and anterior chambers. *Broad Iridectomy.* The object is to open up the filtration angle by removing a piece of iris root. This operation should be done only if the eye is free from inflammation. Some surgeons prefer a trephine operation instead of broad iridectomy.
- (c) Intraocular new growth. *The eye should be removed.*
- (d) In thrombosis of the central vein, *operation is contraindicated*, as any procedure is likely to be accompanied by severe intra-ocular hæmorrhage. If the eye is very painful it should be removed.
- (e) In glaucoma after cataract extraction. *Cyclodialysis* may be tried. Failing this *corneoscleral trephine* sometimes is successful, but treatment is unsatisfactory.

Acute primary glaucoma.

This is one of the few surgical emergencies with which the ophthalmic surgeon is called upon to deal.

Etiology. The cause is unknown. It is more common in women than men; patients are usually beyond middle life. Jews are susceptible to the disease. Small eyes with shallow anterior chambers predispose to glaucoma. There is some connection between the ductless glands, the emotions and acute glaucoma,

probably due to disturbance of the sympathetic control of the vasomotor system of the eye. A local rise in capillary blood pressure can influence intraocular pressure. A rise in general blood pressure does not.

Signs and symptoms. The onset is usually sudden. There is severe pain in the eye and temple. The pain may be so great as to cause collapse and vomiting. Vision is very much diminished. The conjunctiva is congested, there may be circumcorneal injection; the cornea appears misty due to oedema in the substantia propria; the anterior chamber is shallow. Owing to corneal oedema it is difficult to see the iris and for this same reason fundus examination is usually impossible. The pupil is semi-dilated and inactive. Intraocular tension, tested by the fingers, is found to be very much raised compared to the other eye, and the eye-ball may feel stony hard.

It may be possible to get a history of previous attacks of mistiness lasting a few hours.

An attack, if untreated, may subside only to recur later. Each time an attack occurs vision is permanently affected. It is very important, therefore, in considering treatment not only to restore normal tension as soon as possible but to prevent subsequent attacks.

Differential diagnosis. It is most essential to distinguish between *acute glaucoma*, *acute iritis* and *acute conjunctivitis*. The differences are best compared in tabular form.

	ACUTE CONJUNCTIVITIS.	ACUTE IRITIS.	ACUTE PRIMARY GLAUCOMA.
<i>Age and Sex</i> ..	Any age, either sex.	Any age, either sex.	Elderly. Commoner in females.
<i>Symptoms</i> ..	Pricking. Little pain. Discharge. Lids stuck together in the morning. Sight not affected.	Neuralgic pain, radiating to temporal region. No discharge or sticking of lids. Sight misty.	Very acute pain radiating to temporal region, causing collapse and vomiting. No discharge. Sight seriously impaired.
<i>Conjunctiva</i> ..	Scarlet colour. The injection more marked in the fornices, fading towards the cornea. Presence of mucus or mucus.	Red colour. Generally injected. Circumcorneal injection always.	Red colour. Generally injected. May be circumcorneal injection.
<i>Cornea</i> ..	Clear.	Folds in Descemet's membrane. May be K.P.	Edematous, appears like ground glass. No K.P.
<i>Pupil</i> ..	Briskly active.	Contracted. May be inactive.	Semi-dilated. Inactive.
<i>Iris</i> ..	Normal.	Swollen, discoloured. May have new blood vessels in it.	Indistinct owing to corneal oedema.
<i>Tension</i> ..	Normal.	Normal.	Very much raised.

Treatment. It is obvious that an eye with acute glaucoma is likely to react badly to operative interference. We must therefore endeavour to lower the intraocular pressure before undertaking an operation. There must, however, be no long delay if the eye does not respond to conservative measures or sight will be seriously impaired.

The patient is put to bed. A saline purge is given. Morphia may be necessary for the pain. Frequent hot bathing or the Maddox dry heater is comforting. Medical diathermy relieves pain and lowers tension temporarily. Eserine salicylate 1 per cent. every quarter hour for about two hours is instilled into the eye, then $\frac{1}{2}$ per cent. every two hours. A few drops should be put into the other eye, as the emotional stress of an attack of acute glaucoma in one eye has been known to excite an attack in the other. Two or three leeches should be placed on the temple of the side affected. Intravenous injection of hypertonic saline (30 c.c. of 30 per cent.) or glucose (100 c.c. of 50 per cent.) may be of value in increasing the osmotic pressure of the blood and temporarily lowering intraocular pressure.

If there is a response to this treatment, if the eye becomes less congested and the cornea less hazy and the tension lower, operation may be postponed until the eye quiets down. Operation must then be performed to prevent another attack.

If, however, the eye does not respond to conservative treatment it is unwise to wait longer than 6—12 hours before operation is undertaken.

Choice of operation.

(A) *A hard eye which has not responded to non-operative treatment.*

1. A preliminary posterior sclerotomy is justified. It may lower the tension sufficiently to assist in the success of a bigger procedure which should be carried out immediately afterwards.
2. Broad iridectomy. This operation is found to give better results than a corneoscleral trephine. In the latter operation the trephine hole becomes occluded with fibrin and may quite soon get blocked.

Choice of anæsthetic.—My own preference is for evipan. The administration of it worries the patient very little, and there is a fall in intraocular pressure when it is given, which is an advantage. 4—5 c.c. intravenously with preliminary medication of $\frac{1}{8}$ gr. omnopon and $\frac{1}{600}$ gr. scopolamine is an average dose in an elderly person. Retrobulbar novocaine and adrenalin may be employed or a general anæsthetic. Cocaine drops 2 per cent.—4 per cent. are instilled into the conjunctival sac if evipan or retrobulbar anæsthesia are used. A few drops of adrenalin, 1 in 1,000, instilled into the conjunctival sac just before operation minimizes bleeding from the congested conjunctiva.

A speculum is inserted and the eye fixed with toothed forceps. The conjunctiva is often friable in these cases so that it is advisable to grasp the sheath of the internal rectus through the conjunctiva, in order to keep the eye steady while making the section.

The section is best made with a ground down Von Graefe knife. Some surgeons use a keratome.

The section should extend from “10—2 o'clock” and be placed 2 mm. on the scleral side of the limbus. With a section so placed the iris root can be separated and removed. Iris forceps are gently inserted at one extremity of the section and the iris grasped near the pupillary margin. The iris is drawn out of the wound and the pillar cut with iris scissors. The iris is then dragged across the wound to the other extremity of the section and the pillar is drawn out and cut. The lateral pillars are gently replaced with an iris repositor, atropine is instilled and the eye bandaged. Dark glasses may be worn after 48 hours.

It is very important that the iris base should be removed, that is why the section is made slightly scleral to the limbus.

Steps of the operation may be interfered with by hæmorrhage into the anterior chamber.

At a later date lens opacities may appear at the site of the iridectomy, and this is not necessarily due to injury to the lens capsule at the time of operation.

(B) *An eye the tension of which has been lowered to reasonable limits by treatment.*

1. Broad iridectomy. This operation is described above. Local anæsthesia is recommended, i.e. retrobulbar injection of novocaine and adrenalin, and cocaine drops in the eye.
2. Corneoscleral trephine. Some surgeons prefer this operation in a quiet eye. The operation will be described under chronic glaucoma.

Chronic glaucoma.

It is important to realise that this disease is frequently discovered by the oculist at a routine examination, and that it may have progressed to a serious degree without the patient being aware of anything amiss. In other words, chronic glaucoma may be, in early stages, symptomless. Sometimes, on the other hand, a history may be obtained of attacks of misty vision soon passing off, difficulty in reading with glasses which are at other times satisfactory (due to weakness of accommodation) or the patient may notice coloured rings round artificial lights (haloes). The latter are due to oedema of the cornea and must not be confused with a similar subjective symptom caused by mucus on the corneal surface. Differentiation is easy, because the "mucus" halo disappears on blinking.

Signs. A small hypermetropic eye with a shallow anterior chamber predisposes to the disease. The signs of chronic glaucoma are best considered under the following headings:—

- (a) *Fundus.*
- (b) *Fields of vision.*
- (c) *Intraocular tension.*

(A) **Fundus.** The weakest part of the adult eye is at the exit of the optic nerve fibres from the globe. The nerve fibres pierce the sclerotic at the lamina cribrosa. In the normal eye the lamina cribrosa is usually situated near the centre of the optic disc and is always bordered by a circular rim, pinkish in colour. Sometimes the rim is only visible on the nasal side of the disc, but it can always be seen by the ophthalmoscope. The retinal vessels pass across the disc margin and are not deflected until they reach the margin of the lamina cribrosa, when they dip down and pass through with the nerve fibres into the nerve trunk outside the globe.

As a result of several transient attacks of increased intraocular pressure the appearance of the nerve head becomes altered. The normal cup becomes enlarged and deepened and the nerve fibres are pressed against the disc margin. In time the pink rim of the disc disappears and the cup extends right up to the margin. The retinal vessels are pushed to the edge also, and on reaching it they dip sharply and are lost to view until they again appear on the floor of the cup. The ophthalmoscopic picture thus produced is known as pathological cupping of the disc and is pathognomonic of chronic glaucoma.

Pulsation may be seen in the retinal arteries in glaucoma, a phenomenon only observed otherwise in aortic incompetence. If pulsation is not actually visible it is very easily elicited by gentle digital pressure on the globe through the upper lid.

(B) **Visual fields.** The testing of the visual fields forms the most important part of the examination of a person suspected of glaucoma. The first signs of the disease can be discovered from changes in the fields, before any ophthalmoscopic changes appear in the disc and before there are any subjective symptoms of loss of vision.

1. *Peripheral field.* Early in the course of the disease there is a defect in the nasal field, above or below, which gradually progresses and is later accompanied by a gradual contraction of the rest of the field. Ronnés step is a cone-shaped defect which is continuous with the nasal contraction. It usually has a straight horizontal edge.
2. *Scotomata.* (May occur before peripheral changes.) Seidel's arcuate scotoma is a prolongation upwards and nasalwards or downwards and nasalwards of the blind spot. This scotoma follows the curve of the course of the papillo-macular nerve fibres.

Bjerrum's scotoma is an enlargement of the blind spot which occurs in conjunction with Seidel's scotoma. The peripheral defects are easily demonstrable by a perimeter. For the revelation of scotomata a Bjerrum screen or Harman scotometer is best employed.

(C) **Tension.** In chronic glaucoma a rise of tension is transient until very late stages in the disease. Therefore it will usually be absent when the patient is examined and in any case may not be sufficiently high to be appreciated by the digital method of examination. There is a mechanical method of determining tension which has a real value if its limitations are understood. The instrument usually employed is the tonometer designed by Schiotz. Briefly it is used thus:—

About 3 drops of 2 per cent. holocaine are instilled into the eye. The patient is placed so that the head and eye look straight upwards. The surgeon separates the lids (carefully avoiding putting any pressure on the globe in so doing) and the instrument is applied to the cornea. A pointer at the top of the instrument will fluctuate according to the pressure of the eye and the amount of its excursion read off on a scale. By reference to a chart the intraocular pressure is determined. This test is repeated with different weights in the tonometer.

The use of this method is in comparison. Readings obtained at different times of day or on different days are compared and it is then seen whether the tension rises above physiological limits at any time. Normal tension varies considerably but can be regarded as being between 16—24 mm. Hg.

This test which at best only gives approximate results is of value only, therefore, after several readings. An instrument may show a reading of 30 mm. in an eye which in fact has not glaucoma. That reading happens to be within normal limits for that particular eye with that particular instrument. But if an eye has a tension of 30 mm. one day and 35 mm. another, and repeated readings reveal such increases, then it is probable that this eye is glaucomatous and exhaustive investigation must be carried out on the visual field.

A diagnosis of glaucoma should not be made with the tonometer test alone.

Treatment. The earlier diagnosis is made and the sooner treatment is begun the better. Loss of vision or of field is usually permanent and with treatment we can only hope to prevent further loss.

Treatment is non-operative, operative or both. Unless there are contra-indications operation is advised.

Non-operative treatment by drugs. This is directed chiefly against what we regard as the structural cause of glaucoma (i.e. shallow anterior chamber, root of iris blocking the filtration angle). By means of miotics the pupil is constricted. As a result the iris root is drawn away from the filtration angle and drainage of aqueous is facilitated. The drugs usually employed are eserine salicylate $\frac{1}{4}$ per cent.—1 per cent. and pilocarpine nitrate $\frac{1}{2}$ per cent.—2 per cent. These drugs are used in the form of drops, sufficiently often in any given case to keep the pupil pinpoint. This treatment does not cure glaucoma but it may keep the tension within physiological limits in cases in which operation is refused or inadvisable. Eserine is more powerful but may be more irritating than pilocarpine.

General health. Subjects of glaucoma should avoid, as far as possible, worry and excitement, especially the former. Any cardio-vascular disease should, as far as possible, be treated by the physician because disease of the intraocular vessels must influence intraocular fluid interchange.

Operative. The object of any operation is to make a channel from the interior of the eye to the exterior which will remain permanently open and allow aqueous to reach the subconjunctival tissues and be absorbed. (There is one operation—cyclodialysis—which does not come under this heading. See below.)

Contraindications to operation. Most surgeons to-day advise operation when the diagnosis of chronic glaucoma is made. Some go further and advise operation on the second eye even if this does not yet show any signs of the disease. If the patient is very old, if for reasons of general health and especially if there is gross vascular disease operation is considered inadvisable, or is refused, conservative treatment by drugs with frequent examinations to make sure the disease is not progressing, is justified. I think, also, conservative treatment is justified in the other eye even if there are very early signs of glaucoma, provided the patient is willing to use miotics regularly and to present himself every two or three months for examination. If in any of the above cases it is found that the disease is progressing, however slowly, in spite of miotics, operation must be urged as the only hope of saving sight.

Filtration operations.

1. *Corneoscleral trephine.*
2. *Flap sclerotomy.*
3. *Sclerectomy.* *Holth.*
 ,, *Lagrange.*
4. *Cyclodialysis.*

Corneoscleral trephine seems to give the best results in the hands of most surgeons, and the operation will be described in full. The other operations will be dealt with briefly.

Corneoscleral trephine. (Elliot).

I always use the reverse flap method (suggested by Foster Moore). The conjunctival flap is dissected up from the limbus instead of down from above.

The eye is cocainized. Adrenalin is not used at this stage unless the eye is red as it is inclined to shrink the conjunctiva.

With a small Beer's knife the conjunctiva is incised at the limbus at about "two o'clock." It is freed all round the limbus as far as "ten o'clock" and is

then separated from episcleral tissue above with scissors. The flap is made sufficiently large to be anchored down over the upper part of the cornea so covering the trephine hole. When the flap is mobile a silk stitch is inserted at A in the diagram. This stitch must grip the sclera so that it cannot cut out. If

attention is paid to this point the flap will not, later, ride up and lay bare the hole. The other end of the stitch is passed through the edge of the flap at B, so that when the stitch is tied B approximates to A.B, and the flap covers the cornea as shown. After the stitch is inserted it is left loose and the flap is retracted so as to expose the upper limbus. A few drops of adrenalin may now be instilled if there is a little bleeding. The trephine hole is made, using a 2 mm. trephine, and the hole can be placed exactly where the surgeon wishes, i.e. astride the limbus, or more forward in the cornea. The iris and disc are dealt with in the ordinary way and the stitch is then tied.

Atropine 1 per cent. is instilled in the operated eye and eserine $\frac{1}{2}$ per cent. in the non-operated eye. Both eyes are bandaged for forty-eight hours.

The advantages of the reverse flap are its simplicity; the trephine hole can be made exactly in the right place; there is no possibility of button-holing the flap; and I think the resulting bleb is more diffuse and less of a "lump" than that made by the ordinary method.

It has been said against this modification that there is sometimes a lot of bleeding; I can only say I have not experienced this. I think it is most important to make a small flap and only to separate conjunctiva. Another complication suggested is the riding up of the flap necessitating another operation to cover the hole. I have only seen this happen once, and I think it is important in this connection to make the fixation stitch grip well at A.

The results of this operation are not always encouraging. Sometimes the disease seems to be checked for years and in other cases the operation has to be done again, and even then the disease continues on its way.

Flap sclerotomy. (Herbert).

A keratome incision is made, subconjunctivally, about 2 mm. from the limbus and a rectangular flap is cut by incisions at right angles to the first incision, the corneal edge of the rectangle being left intact. Massage of the globe is commenced twenty-four hours after operation, as recommended by Cruise, to prevent the flap from healing up. This operation does not disturb the iris and can easily be repeated if necessary.

Sclerectomy.

1. *Holth*—a section is made 1 mm. from the limbus and a scleral flap removed by punch forceps. An iridectomy may be done.

2. *Lagrange*—a horizontal piece of sclera is removed after an incision as for iridectomy. There is a good deal of astigmatism after this operation.

Cyclodialysis.

This causes least disturbance of all the glaucoma operations. The ciliary region is exposed subconjunctivally 8 mm. behind the limbus, a spatula is gently inserted between ciliary body and sclera and made to pass forward until it appears in the anterior chamber. By sweeping the instrument from side to side the space between sclera and ciliary body is opened up and aqueous can reach this space and drain thence into the veins. The drawback to this operation is that the space may soon get obliterated. This operation is indicated in cases where as little as possible is to be done e.g. aphakia.

Glaucoma Capsulo-Cuticulare.

This disease is included under a separate heading because it is not yet clear where it belongs. It was described by Vogt in 1925. The essential features are that there is exfoliation of the lens capsule accompanied by signs of chronic glaucoma. By exfoliation is meant the formation of flakes at the pupil border which later become loose and separate from the lens capsule to float in the aqueous. There is also a central disc and a peripheral band on the surface of the capsule. The flakes block the filtration angle and this, according to Vogt, is the cause of the glaucoma. Vogt's figures indicate that seven per cent. of chronic glaucoma cases show exfoliation. A recent paper by Hörven reviews the literature and evidence of this disease. According to him no less than 80—90 per cent. of cases of chronic primary glaucoma (glaucoma simplex) show exfoliation. He explains the extraordinary difference between his figures and Vogt's by the fact that the latter may include "chronic inflammatory glaucoma" among his cases. Obviously more work is needed before the significance of exfoliation is understood.

In considering the **prognosis** of operation in chronic glaucoma, I have been impressed by the success of some filtration operations and the relative failure of others. It seems to me that a great deal depends on the state of the intraocular blood vessels. If these are diseased, the probable cause of the glaucoma is an upset of the intraocular fluid interchange (and all the biochemical problems which this involves) and we can therefore expect little from an operation which merely provides increased drainage for the aqueous fluid. As we cannot restore to normal diseased or degenerate blood vessels, it is not to be expected that we can do much to influence an intraocular pressure which has become raised because of the disturbance of function of these vessels.

On the other hand, in cases in which the rise of pressure results from mechanical obstruction in the region of the filtration angle, e.g. a hypermetropic eye, any operation which provides permanent exit for the aqueous should be successful.

These are only small points in one of the most difficult problems of ophthalmic surgery but they are important, I think, in advising the patient or his friends as to the ultimate outcome of the individual case.

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