SUMMARY

A review of the presentation and diagnosis of acute narrow-angle glaucoma and open-angle glaucoma is presented. Both medical and surgical treatment are discussed with an evaluation of operative techniques.

There is deranged hydrodynamics in the glaucomatous eye. The aim of treatment, be it medical or surgical, is to restore adequate circulation of the intra-ocular fluid, so that the intra-ocular tension is within a normal range which can be safely tolerated by that specific eye. The nature of the hydrodynamic imbalance must be determined and herein lies the essence of the treatment of glaucoma.

For the present purpose only the following types of glaucoma will be considered: narrow-angle glaucoma (acute glaucoma) and open-angle glaucoma (chronic simple glaucoma). This article is based on personal experience, on statistics gleaned from the patients treated in the Department of Ophthalmology at Groote Schuur Hospital, and on a review of the relevant ophthalmic literature.

ACUTE NARROW-ANGLE GLAUCOMA

The prodromal symptoms are blurred vision and rings of rainbow tints are seen around lights. These haloes are distinctive and significant and indicate a transient rise of intra-ocular pressure. Not infrequently, there is a history of emotional upset. The prodromal symptoms present in subdued light, for instance at a cinema or in the evenings.

The stage of acute glaucoma then follows. There is a sudden onset of severe pain, associated with either an emotional or a physical crisis. The pain is in the distribution of the trigeminal nerve, and is associated with severe and violent headaches. This pain may be so severe as to cause constitutional symptoms such as nausea, vomiting

and even collapse and has been confused with an acute abdomen or an acute attack of ischaemic heart disease.

Local examination shows a depressed visual acuity and a raised tension; in fact, the eye is 'stony' hard to palpation. There is marked ciliary tenderness. The bulbar conjunctiva is congested and may be chemotic, there is marked limbal injection and the episcleral veins are prominent and the cornea is cloudy due to epithelial oedema. The corneal sensation is depressed and there is a greenish tinge in the pupil (this has given rise to the name of glaucoma, which in Greek means pale green). There may be punctate corneal staining and the pupil is semi-dilated, oval and immobile. The iris is congested, discoloured and dull.

The most important sign is the presence of a shallow anterior chamber.

The lens and the iris may lie immediately posterior to the cornea. Often the eyelids are swollen and oedematous. The disc is rarely observed because of the corneal oedema.

Treatment

The regimen of treatment at Groote Schuur Hospital is as follows:

1. *Sedation of patient.* The patient is sedated, the medication used depending on the degree of systemic upset. Morphine has been found to be a very satisfactory drug. The dose depends on the weight of the patient.

2. *Local treatment* consists of pilocarpine 2% and eserine ¼% drops (this is a mixture made in the dispensary at Groote Schuur Hospital). The regimen is as follows: The drops every minute for 5 minutes, then every 5 minutes for ½ hour, then every ½ hour for 4 hours and thereafter every 4 hours for 24 hours.

3. *Systemic treatment.* Diamox 500 mg is given by intravenous injection.

A useful supplement to Diamox for reduction of intra-ocular tension in acute glaucoma is glycerol. The dose is 1 ml/kg in an equal volume of fruit juice. Some patients, however, are made nauseous or even vomit because of the glycerol.

During this treatment the patient is reassessed at ½-hourly intervals to ascertain the response of the eye to the treatment. In the majority of cases this regimen reduces the intra-ocular tension within a few hours, otherwise we prefer to use mannitol by intravenous injection. The dose of mannitol is 2·5 - 10 ml/kg of a 20% solution and this is given at 60 drops per minute intravenously.

Once the tension is reduced substantially, the eye is examined with a gonioscope, to ascertain the patency of the filtration angle. The use of the gonioscope and the interpretation of the results are important. One may imagine that the angle is occluded, but by tilting the gonioscope lens, pressure shifts aqueous to the opposite side and may actually open the angle.[1]

The diagnosis of angle-closure glaucoma is a clear-cut indication for operation. Medical treatment is temporary. The big danger is compression of the filtration angle by persistent touch of iris against the trabecular meshwork, thus causing peripheral anterior synechiae. An iridectomy is the operation of choice. This bypass allows for the passage of aqueous into the anterior chamber, the iris diaphragm recedes posteriorly and the anterior chamber deepens, thus re-establishing normal intra-ocular hydrodynamics.

Peripheral iridectomy.[2,3] Once the eye is relatively quiet, the tension controlled, and the iris not congested, the patient is taken to theatre and a peripheral iridectomy is performed. Even if peripheral anterior synechiae are observed, a peripheral iridectomy should first be done.

A peripheral iridectomy in the opposite eye is mandatory and usually done either while waiting for the affected eye to settle and become less congested, or soon after the peripheral iridectomy to the first eye.

It is most important that once a clear view of the fundus is obtained in the affected eye, the presence of an intra-ocular neoplasm be excluded.

1. A superior rectus suture is placed in position.
2. I prefer to place the iridectomy incision in the upper temporal quadrant. A subsequent drainage operation can then be sited in the upper nasal quadrant.
3. A limbal-based conjunctival flap is made, the flap folded back onto the limbus and any bleeding controlled with light cautery.
4. The limbus is identified by using a light placed in the 6-o'clock position and with a Bard Barker (No. 15) blade a scratch incision is made half-way down at the limbus before the incision is completed with a keratome.
5. Black silk (6 - 0) is passed through the conjunctiva through the lips of the wound and through the conjunctiva on the opposite side and the central loop retracted.[3]
6. The posterior lip is depressed and usually the peripheral iris presents in the wound. The tip of the tented iris is excised with De Wecker's scissors. It is important that the iridectomy is complete: note whether or not there is pigment on the excised iris.
7. The iris is replaced by gently stroking the cornea with an iris repositor.
8. The sclero-limbal suture is tied and a continuous suture is placed through the conjunctival wound.

OPEN-ANGLE GLAUCOMA

Rather than become mentally entwined in figures, statistics and mysticism with which the glaucomatous literature is cursed, my approach to the problem is tempered by the judicious remark of Professor Goldman, who said 'Blindness, not increased pressure, haunts people suffering from glaucoma'.[4] The saga of open-angle glaucoma is to me rather depressing.

The problems in open-angle glaucoma lie with the definition, diagnosis and treatment.

Definition

I regard glaucoma as a chronic ocular condition characterized by sustained raised intra-ocular tension (with applanation tonometer) of over 21 mmHg which is associated with field loss and/or optic atrophy.

Diagnosis

Ocular hypertension. Ocular hypertension and chronic simple glaucoma are not synonymous. While the figure of 21 mmHg (applanation tonometry)[5] is regarded as abnormally high for the average eye, one must realize that the intra-ocular tension varies over every 24-hour period—Drance has shown that one-third of medically treated glau-

comatous eyes with an intra-ocular pressure tension of 19 mmHg (applanation tonometry) or less during visits, had peaks of 24 mmHg or more, some up to 37 mmHg, and half of these peaks occurred at 10 p.m. or 6 a.m.[6]

There is, however, a long safe interval before optic nerve damage and field loss occur in open-angle glaucoma.[7-9]

Elevated intra-ocular tension does not necessarily mean progressive field loss. Goldman,[7] reviewing Leydhecker's survey of 19 000 normal individuals, showed that the number of ocular-hypertensives increased with age and that the number of field defects increased markedly with advancing years. The two linear graphs were approximately parallel, but separated by an interval representing 18 years. This would suggest with ocular hypertension that it takes 18 years to develop field changes. Hollows and Graham,[8] however, found very few cases of field loss with ocular hypertension. It has been suggested that a 'hypertensive' eye can tolerate increases of intra-ocular tension for a longer time than was previously thought.

This long 'safe' period influences the ophthalmologist's decision to defer treatment in patients with only a raised intra-ocular tension. This in turn places more of a responsibility on the ophthalmologists because of the adequate follow-up which has become mandatory.

Cupping. Although it is frequently stated that with glaucoma definite loss of visual field is associated with cupping of the disc, in one series[10] of examinations 34% of the eyes with apparently normal discs had appreciable field defects. In 12% of eyes with apparently pathological cupped discs there was a normal visual field. Cupping and atrophy of the optic discs should not be equated immediately with glaucoma. Leopold[11] has made a disquietening statement concerning 12 patients in whom the diagnosis of chronic open-angle glaucoma was made because of the cupping and atrophy of the optic discs. In fact, with some of the eyes glaucomatous procedures had been done. Further investigations, however, showed the cause of the optic atrophy to be either pituitary tumour or sphenoidal ridge meningioma.

Field loss. The location and nature of the initial visual field changes are also in dispute. Drance[12] suggested that the visual field loss in chronic simple glaucoma is most probably due to embarrassment of the blood supply to the optic nerve head.

It should be emphasized that for the practising ophthalmologist central visual fields are far more important than the peripheral fields, because once one is able to demonstrate peripheral field loss, one is then dealing with an advanced condition.[13] The ophthalmologist must detect early visual field loss.

Filtration angle depths. Van Herick and Schaffer[14] use a simplified method for assessing the filtration angle depths. A slit-lamp light is focused at the limbus, and the anterior chamber depth is assessed by comparing the width of the corneal section with that of the distance between the anterior surface of the iris and the posterior surface of the cornea. If this distance is equal to half the width of the cornea then the angle is incapable of closure. A width equal to one-quarter of the corneal section width, requires a gonioscopic examination of the eye. Less than one-quarter implies a narrow filtration angle. These figures are for emmetropic eyes because with age and hypermetropia the depth of the anterior chamber decreases. Myopes have deep anterior chambers.

The coefficient of outflow. This is a useful ancillary investigation but should be done with an electronic tonometer and following a water-drinking test. It is unlikely that the average ophthalmologist in practice will have this instrument and for this figure to be of value it is suggested that the patient be referred to a glaucoma unit.

Treatment

A more realistic approach to the treatment of open-angle glaucoma is necessary.

Medical therapy. The general trend of thought concerning the treatment of ocular hypertension is towards conservatism.[15,16] An eye with ocular hypertension should have a trial of therapy using the safest drugs which are probably pilocarpine and epinephrine. The most diluted concentration of pilocarpine which controls the intra-ocular tension and which can be tolerated by the patient is prescribed. If there are annoying side-effects or if there is no substantial reduction of intra-ocular tension, the treatment is discontinued.

Epinephrine is most useful in reducing the intra-ocular tension, but allergies to the drops are common.[17]

In ocular hypertension it is unwise to use anticholinesterase inhibitors[18-22] or carbonic anhydrase inhibitors, because of their potential dangerous side-effects (Table I).

An increased responsibility now rests on the ophthalmologist to assess the patient at regular and frequent intervals, when the fields of vision should be re-evaluated and

TABLE I. SIDE-EFFECTS OF DRUGS USED IN GLAUCOMA

Parasympathetic Agents (e.g. Pilocarpine)
1. Decreased pupil aperture
2. Pupil-block glaucoma
3. Lens subluxation
4. Peripheral retinal tears
5. Posterior synechiae

Anticholinesterase Agents (e.g. Phospholine Iodide)
1. Cataracts 20 - 80%
2. Decreased serum cholinesterase—succinylcholine (Scoline) apnoea
3. Parasympathetic stimulation—Sweating, Salivation, Nausea, Diarrhoea, Abdominal cramp

Sympathomimetic Drugs (e.g. Epinephrine)
1. Raised blood pressure, Extrasystoles
2. Aching of eyes, eyebrow—Ischaemic Dilator/Spasm
3. Adrenochrome / Melanin } Deposits in conjunctiva
4. Reactive hyperaemia
5. Allergic conjunctivitis and dermatitis
6. Aphakia—central scotoma ?due to macular ischaemia
7. Angle-closure glaucoma

Carbonic Anhydrase Inhibiting Agents (e.g. Acetazolamide (Diamox))
1. Nervous system— Paraesthesia: hands, feet; Depression and mental confusion
2. Gastro-intestinal system—Epigastric pain, Decreased appetite, Diarrhoea, Constipation
3. Genito-urinary tract— Diuresis, Stone formation

the state of the optic nerve head reassessed and the tensions measured.

Surgical Treatment

Surgical treatment is only considered if a concerted regimen of medical treatment has failed or if the patient is incapable of obtaining or instilling the drops into his eyes.

A galaxy of operations are described by different authors who claim varying successes with each operation. The literature on the subject is intoxicating, for authors tend to compare dissimilar or modified operations and draw unusual and confusing conclusions.[23-25]

The essential principles of these glaucoma operations are either to increase the facility of outflow of the aqueous or decrease its production. The operations to decrease the production of aqueous, e.g. cyclodiathermy or cyclodialysis, are not used for open-angle glaucoma.

The filtering operations aim at producing a subconjunctival fistula through the sclero-limbus. The operations to be considered briefly are the trephine operations (which may be either at the corneo-limbus or at the sclero-limbus); secondly, sclerectomy/sclerotomy operations; and thirdly, iridencleisis. Gonio-puncture trabeculotomy operations and fistula operations where various plastic materials are used to maintain the fistula patent, will not be considered.

Trephine operations. I subscribe to the view of Sugar[23,25] who recommends sclero-limbal trephination. Wide conjunctival and Tenon's flaps are reflected and dissected as far as the limbus. A 1·5 - 2-mm trephine is used and is sited about 0·3 mm from the corneo-limbal junction on the scleral side. The trephine is tilted forward until the anterior chamber is entered, and the posterior hinge of sclera is then cut with scissors. The advantages of this operation are firstly, there is no button of the conjunctiva because there is no corneal splitting and, secondly, there is less possibility of infection because of the adequate cover by the conjunctiva and the Tenon's capsule. Antagonists of this operation maintain that there may be a danger of injury to the ciliary body but this in fact rarely occurs if the technique as described by Sugar[23,25] is used.

Sclerotomy. There are two sclerotomies which I have found to be satisfactory—they are the cautery sclerotomy of Scheie[24] and the modified sclerotomy as described by Stallard.

With the cautery sclerotomy, conjunctiva and Tenon's capsule are reflected independently as far as the sclero-limbus where a line of thermocautery is applied. A perpendicular incision is made and cautery applied to the lips of the wound, especially to the posterior lip. The anterior chamber is entered and a peripheral iridectomy performed. The conjunctival/Tenon's flap is resutured in layers. The complications of this procedure are delayed formation of the anterior chamber associated with hypotony. In addition there is an increased incidence of secondary infection. Cataract may result because of the heat which is applied to the wound edge.

Stallard's modification of an anterior flap sclerotomy with a basal iridencleisis is of particular use because the sphincter pupillae remains intact. Again a conjunctival and a Tenon's flap is drawn over the limbus. A perpendicular incision about 5 mm long and 2 mm above the limbus is made. A cyclodialysis spatula is introduced into the scleral wound and enters the anterior chamber. The wound is enlarged with scissors at each end, the extension passing towards the limbus. The peripheral iris is incarcerated in the wound.

Iridencleisis. A conjunctival and a Tenon's flap is drawn over the limbus. A perpendicular incision over the scleral limbus is made and both limbs of the iris are included in the wound. We have found this latter procedure to be particularly useful in the Coloured and Bantu patient where there is an increased tendency for scarring of the wound.

Scheie,[24] in reviewing the advantages of an iridencleisis, peripheral iridectomy with scleral cautery and scleral limbal trephine, found that the pressure was controlled in 83% of the iridencleisis patients, 88% of the Scheie procedures and 97% where scleral limbal trephine was used. Hypotony occurred in 3·5% iridencleisis cases, 12% where Scheie procedures had been done and in 30% where sclero-limbal trephination was done. Sugar,[25] in reviewing this article, felt that the incidence of 30% hypotony was far above the incidence that he had found.

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