development of hypotony. He had found that this had greatly reduced the incidence of flat anterior chamber and choroidal separation and also gave a very diffuse bleb. Tension control over a prolonged period also seemed to be very much better than with conventional operations, probably because the aqueous was spread out over a broad area, preventing adhesion between the conjunctiva and underlying tissue, so that the bleb did not become localized there during the early stages following surgery.

Dr. Simmons questioned whether trabeculectomy or other microsurgery might be dangerous in the hands of a surgeon who undertook glaucoma surgery only occasionally. Dr. Anderson expressed the opinion that recently trained surgeons who used a microscope routinely (e.g. for cataract surgery) would find trabecular surgery easy and safe, but he agreed that these were difficult operations for surgeons who did not use the operating microscope frequently.

**Summary**

**PROFESSOR HANS GOLDMANN**

*Berne, Switzerland*

Certain points from this Symposium are particularly memorable. The first concerns the changes which occur in glaucomatous atrophy of the disc. They are characterized by the combined disappearance of nerve fibres and glial tissue in contradistinction to simple atrophy in which only the nerve fibres disappear. Paradoxically, in juvenile glaucoma, restoration of pressure, not hypotony, may restore the normal disc appearance, a phenomenon not yet understood. There is no doubt that there is a pressure-dependent circulation sensitivity which is different in different eye tissues. The circulation at the disc and its surroundings shows the greatest sensitivity to a rise in pressure which differs in different sections of the disc and of the adjacent uveal tissue. The rest of the uvea is less sensitive and the retina still less so. The significance of the fluorescein appearance-time of the disc is still a matter of controversy. The influence on function when the papillary circulation is damaged is particularly impressive in cases of true low tension glaucoma in which general vascular and blood factors have been shown to play an important part. The axonal flow in the nerve fibres may be of some importance in the development of nerve fibre degeneration and should be further examined.

The indication is becoming stronger that, at least in the majority of cases, the obstruction to aqueous flow lies between the anterior chamber and Schlemm's canal. There is still uncertainty about the role of the trabecular meshwork and the canal endothelium. The finding of intracellular vacuoles and canals in the endothelium poses new problems. Is active transport involved? The seemingly good relationship between pressure-head and flow speaks rather against it. In patients with simple glaucoma the number of vacuoles is diminished. This may be due to a pathological state of the endothelial cells or to a diminished supply of aqueous, or it may be an artefact caused by therapy. When hypertension follows trauma the changes in the trabecular meshwork are the cause of the high pressure, even though there may be other structural changes in the eye such as angle recession.

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Summary

In impression tonometry much of what is called scleral rigidity is caused by the displacement of blood out of the uvea. The volume displaced is uncertain and in consequence there is often uncertainty about how to extrapolate from \( P_t \) to \( P_o \) in the single case and whether the readings given by tonography include a combined effect of blood elimination from the uvea and aqueous outflow. All the so-called provocative tests for open-angle glaucoma have a limited value. Different tests often do no more than say the same in another way and therefore do not increase the amount of information, e.g. an increase in pressure with water drinking from 15 to 20 mmHg, that is 5 mmHg, is percentage-wise the same as an increase from 27 to 36 mmHg. Therefore, in reality, the experiment proves nothing, except that the tension was 15 in the first case and 27 in the second. Or the real relation between the test and what it should prove is not clear, e.g. steroid sensitivity does not always indicate a disposition to simple glaucoma. The presence of an abnormal cup/disc relationship does not of itself indicate the presence of simple glaucoma. The diagnosis of simple glaucoma is confirmed when the characteristic deterioration of the visual field is found together with a raised intraocular pressure or an abnormal cup. But to make an essential distinction between ocular hypertension and simple glaucoma makes a virtue of our ignorance and reverts to a state of affairs very much like that of 50 years ago. It is true that we have, in the methods of static and automatic perimetry, ways of detecting very early changes in the visual field, but it is not satisfactory to have to wait until these changes have taken place. Even then the question remains how much the intraocular pressure should be diminished to avoid further damage. It is therefore clear that our next task must be to develop a clinically useful quantitative method to determine the relationship between the tension and the visual field which might be applied to the individual patient. Until then, I am convinced that it is necessary to keep under observation those whose tension is found, after repeated examination, to lie between 22 and 25 mmHg applanation without a visual field defect, and to begin medical treatment in cases with a persistently higher pressure, even in the absence of any field defect.

Goniography is important for the diagnosis of the disposition to angle-block glaucoma. Predisposition to angle-closure is caused by the increasing thickness of the ageing lens which moves the lens iris diaphragm forwards. This is not the only factor, for not every old person develops angle-closure glaucoma! In some cases loosening of the zonule is involved; a special thickening of the equatorial parts of the lens may contribute. In any case, an anteposition of the sphincter iridis increases the axial component of the force of the sphincter and the difference in pressure between the posterior and the anterior chamber, thus contributing to the bulging of the iris. This bulging is eliminated by a peripheral iridectomy even if the iridectomy is not basal.

Concerning medical therapy, it is worth remembering that a combination of Eppy and Guanethidine is quite efficient and seems to be remarkably free from side-effects. The goal of therapy is the normalization of tension during the 24 hours of the day; hence the necessity for the diurnal curve to include a pressure reading taken in the morning in bed. Actually "normalization" means that there is no deterioration of the visual field over a period of time.

Another point which has emerged is that Ketamine is very useful to enable the pressures to be measured in babies. These children who have congenital glaucoma easily develop anisometropia followed by amblyopia and it is important to look for this as soon as possible in order to try to forestall its development. Goniotomy is very satisfactory for the treatment of congenital glaucoma, and also appears to be the only satisfactory operation for juvenile glaucoma. Although the mechanism of malignant glaucoma is not yet known, the
vitreous does seem to be especially involved. The operation of massive aspiration of the vitreous and re-formation of the anterior chamber with air has been successful in this serious complication.

Further points are raised by the new surgical procedures of trabeculotomy and trabeculectomy. The first point concerns the theoretical question of whether the results of these operations decide where the pathological resistance to the outflow of aqueous humour is situated in simple glaucoma: in the trabecular meshwork or in the outlets of Schlemm's canal. The answer is not yet known because conjunctival oedema or a bleb so often develops some time after the operation. The second point is of great importance. One of the dangers of the use of filtering procedures is the formation of a scar-rimmed avascular and poorly vascularized polycystic bleb which easily becomes necrotic in conjunctival infection and so causes late infections. This dreaded complication is practically avoided when no bleb at all or a diffuse conjunctival oedema develops, as frequently happens—not immediately, and this to me seems important—but a certain time after trabeculectomy. Similar diffuse blebs can often be induced, if after a filtering procedure such as Scheie's operation, the conjunctival flap is covered by a contact glass for a time following the operation. The common denominator of both procedures with the same final results seems to be that, in the first instance, the opening of Schlemm's canal during the operation improves its function at least for a certain time, which combined with the diminished secretion of aqueous common after any intraocular intervention, is long enough for normal healing of the conjunctival wound without the intervention of the influence of aqueous. At a later stage aqueous is eliminated increasingly under normal conjunctiva. In the second case there is no improvement of outflow through Schlemm's canal, but the subconjunctival leakage is hindered by the contact glass until this is removed. Again, normal healing of the conjunctival flap is promoted. The factor common to these two operations is that there is little or no subconjunctival aqueous while the conjunctiva heals, and this absence of aqueous is necessary in order to prevent the formation of a dangerous avascular bleb. It is most important to make a deliberate effort to prevent the formation of this bleb, and not to leave it to chance. Success in establishing aqueous drainage without the formation of an avascular bleb is an important advance in glaucoma surgery.
Summary: glaucoma: Cambridge Ophthalmological symposium, September 1971.

H Goldmann

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