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by Douglas R. ANDERSON, M.D.
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IN CHRONIC OPEN ANGLE GLAUCOMA

by Gordon R. DOUGLAS
Stephen M. DRANCE
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TENSION TOLERANCE

by Wolfgang LEYDHECKER

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(Director: Prof. Dr. W. Leydhecker)
TONOMETERS - NEW DEVELOPMENTS

by Robert A. MOSES, M.D.

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This work supported in part by Grant EY 000256, National Eye Institute, National Institutes of Health, Bethesda, Maryland, U.S.A.
BIOMETRY AND THE CLINICIAN

Introduction* to the paper by R.F. Lowe
Primary angle-closure glaucoma: Biometry and the Clinician

Y DELMARCELLE (Liège)

"Car les quantités furent créées au commencement en même temps que la substance."
J. KEPLER, 1596

* Not presented at the Albi's Symposium
PRIMARY ANGLE-CLOSURE GLAUCOMA:
BIOMETRY AND THE CLINICIAN

by Ronald F. LOWE, M.D.
ELECTRON MICROSCOPIC EXAMINATION OF THE TRABECULAR MESHWORK IN CAPSULAR GLAUCOMA

by Erick LINNER
and Johannes W. ROHEN

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HYPERSENSITIVITY TO GLUCOCORTICOSTEROIDS
AND PRIMARY OPEN-ANGLE GLAUCOMA

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Bernard Becker
Steven M. Podos
OCULAR MICROThERAPY
ZERO ORDER DRUG RELEASE

by Kenneth T. RICHARDSON, M.D.
THE PHARMACOLOGY OF THE ADRENERGIC THERAPY OF GLAUCOMA

by Maurice E. LANGHAM

The W.K. Kellog Research Laboratories
The Wilmer Institute
The Johns Hopkins University School of Medicine
Baltimore
THE NON-MIOTIC THERAPY OF OPEN ANGLE GLAUCOMA

by G.D. PATERSON, G. PATERSON
and S.J.H. MILLER

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THE 1974 SURGERY OF GLAUCOMAS

by Raymond ETIENNE
Jacques CHARLEUX
and Jean-Claude VILLON

Raymond Etienne
LASER TREATMENT OF GLAUCOMA

by E.S. PERKINS, M.D., Ph. D., F.R.C.S.
and N.A.P. BROWN, F.R.C.S.

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LASER-TRABECULO-PUNCTURE (LTP)

by Prof. Dr. Med. Hugo HAGER

The most remarkable fact regarding the reactions to a high-power laser trabeculotomy is that in a very short period of time, usually before the start of treatment, there is an immediate increase in the pressure. This is due to a combination of factors: the laser energy is absorbed by the tissue, causing a rapid rise in temperature and pressure. The high-pressure wave generated by this process can cause structural changes in the trabecular meshwork, leading to a decrease in resistance and an increase in outflow facility. These effects might be a result of the laser energy disrupting the cell membranes, causing cell death and tissue damage. As a result, the trabecular meshwork becomes more permeable, allowing increased outflow of aqueous humor and a decrease in intraocular pressure.
TRABECULOTOMY VERSUS MEDICAL TREATMENT IN CHRONIC OPEN ANGLE GLAUCOMA
First results after 16 months follow up

by Ph. DEMAILLY
L. PAPOZ
and F. VALTOT
SUMMARY

R.N. Shaffer — The continued interest in the vascular supply of the optic disc is shown by the excellent papers of Anderson, Bill, Hayreh, Galin, Best and Drance. Our knowledge of the disc’s anatomy and physiology increases but we still have no definite explanation of the specific disorder responsible for the characteristic nerve-fibre defect seen in glaucoma. Ischaemia certainly seems to be the basic cause of damage. How this ischaemia is produced at the relatively low pressures found in open angle glaucoma is not yet understood.

To prevent damage to the optic nerve therapy should ideally be directed to improving its blood supply. The systemic blood flow can only be influenced by improving the general circulation. Usually this is not possible. Consequently we are left with control of intraocular pressure as our best method of improving nourishment of the optic papilla. This accounts for our apparent preoccupation with intraocular pressure. Dr. Leydenheker summarizes the six significant factors, height of tension, duration of tension elevation, the stage of the glaucoma, the size of the excavation, the condition of the disc vessels, and the systemic blood pressure. Dr. Moses points out that none of the new tonometers are superior to the Goldmann tonometer.

The mechanisms by which resistance is increased in the exfoliation syndrome (glaucoma capsular) is described by Linner. In all cases with tensions above 30 mm Hg large amounts of exfoliative material is found in phagocytes in the trabeculum and in the inner wall of Schlemm’s canal. The endothelial cells are thinner and the juxtacanalicular zone is thicker than in normals. The glaucoma is more severe than in chronic simple open angle glaucoma.

The mechanism of pressure elevation in angle-closure glaucoma is described by Lowe. His careful measurements confirm the forward position of the lens and the shallow anterior chamber characteristic of these eyes. He states that lens growth with ageing only, changes the chamber depth of 0.5 mm on the average.

The rapid cupping of the optic disc seen in anterior ischaemic optic neuropathy is discussed by Hayreh. It is questionable, in my opinion if the physiopathology of this cupping is the same as that seen in open angle glaucoma. It is probably analogous to the cavernous degeneration described by Schnabel following attacks of intraocular angle closure glaucoma. A good animal model has been described by
Hamasaki and Zimmerman. Alphachymotrypsin injected into monkey eyes gives a similar high rise of pressure with liquefaction of the nerve behind the lamina cribrosa which bows back producing a marked cupping. This seems different from the loss of glial elements producing cupping in open angle glaucoma. Bowing back of the lamina cribrosa is a late occurrence according to Goldmann.

In infants this loss of substance can produce cupping in a few weeks as shown by Hetherington et al. Normalization of pressure can completely reverse the cupping. There is no loss of neurons. This can be proven by normal fields performed when the child is old enough. Similar but less dramatic findings are found in older patients. This refutes the contention that the ophthalmoscope shows only late damage to the nerve. In truth, as stressed by Armaly both ophthalmoscopy and perimetry are important. Sometimes one, sometimes the other may demonstrate earlier changes. Personally I believe there is usually some increase in cupping before field loss can be demonstrated. The ophthalmologist must use tonometry, ophthalmoscopy, perimetry and good clinical judgement in his care of the glaucoma patient.

Documentation of changes in cupping is most important. It may be that Krakau's method of stereogrammetry will be the most sensitive way to document changes in cup volume. More available methods are serial stereophotography, and contact lens ophthalmoscopy with careful examination of the cup.

Fascinating differences are seen between the corticosteroid response of open angle glaucoma patients and normal controls. Becker and Podo's studies on the hyper-sensitivity of primary open angle glaucoma patients to glucocorticosteroids are fascinating. The significance of these findings in the etiology and treatment of the condition remains obscure.

The use of catecholamines in the medical treatment of open angle glaucoma is discussed by Langham. The action of a new \( \beta_2 \) agonist, salbutamol, is described. Pressure is lowered but tachyphylaxis and hyperaemia discourages long term use. Similar findings were reported by Paterson and Paterson. They reported more promising results with the topical use of a sympathetic post-ganglionic blocking agent, guanethidine combined with epinephrine. Others in the group reported tachyphylaxis, hyperaemia and decreasing effectiveness with time.

An interesting long-term prospective comparison of the success of medical therapy of open angle glaucoma versus the use of trabeculectomy is being conducted by Demailly. The 16 months of follow-up to date is insufficient to present definite conclusions. His future findings will be of great importance. New surgical methods are presented. Hager describes the use of the laser to punch openings in the trabecular meshwork. This interesting method is still experimental. Most reports suggest that pressure reduction by this method is transient. Both Hager and Perkins and Brown agree that the laser can produce iridectomies. At present surgical iridectomy remains the preferred method.

Etienne and Charleux describe several relatively new surgical methods for control of open angle glaucoma. Their greatest enthusiasm is for trabeculectomy in which their statistical results have been outstanding. Trabeculectomized eyes are surprisingly quiet post-operatively and seldom have flat anterior chambers occurred. Time will tell if the success rate is higher and complication rate lower, with this or the other
operations described. They are also enthusiastic about iridectomy-cycloretractions as advocated by Krasnov.

The total drainage of aqueous from the eye can be accomplished by one 12 micron opening. A 1 mm trephine opening is 1.000 micron; a trabeculectomy is some 5,000 microns, but covered by one scleral flap. It is hard for me to call this "microsurgery" even though performed through a microscope. Many operations have been devised for control of open angle glaucoma. High enthusiasm is expressed at first followed by disillusionment as long term results become available. I shall be both surprised and pleased if this is not true of many of these new "micro-surgical" methods. The quotation on wound healing remains true, "it is not so much the quality of the wound as the quality of the wounded".

With the exception of angle closure glaucoma, the basic mechanism of the pressure elevation and of the optic nerve vulnerability remains somewhat mysterious. It was agreed that this symposium had problems unsolved. However it had demonstrated areas of agreement and of disagreement among the experts gathered at Albi. It had pointed out new area for investigation. Most important, it had led to the cementing of international friendships and prepared the way for future cooperation in studying that fascinating disease... glaucoma.
Addenda

- Stereophotographies, 469
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- Pneumatonography: A new methodological approach to the analysis of intraocular pressure and aqueous humor dynamics in human eyes, 475
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