

This ebook is for the use of anyone anywhere in the United States and most other parts of the world at no cost and with almost no restrictions whatsoever. You may copy it, give it away or re-use it under the terms of the Project Gutenberg License included with this ebook or online at [www.gutenberg.org](http://www.gutenberg.org). If you are not located in the United States, you'll have to check the laws of the country where you are located before using this eBook.

Title: Glaucoma

Editor: Willis O. Nance  
Editor: Wesley Hamilton Peck

Release date: November 19, 2007 [eBook #23544]

Language: English

Credits: Produced by Bryan Ness, Martin Pettit and the Online Distributed Proofreading Team at <http://www.pgdp.net> (This book was produced from scanned images of public domain material from the Google Print project.)

\*\*\* START OF THE PROJECT GUTENBERG EBOOK GLAUCOMA \*\*\*

[Pg 1]

# GLAUCOMA

A SYMPOSIUM PRESENTED AT A MEETING OF THE  
CHICAGO OPHTHALMOLOGICAL SOCIETY,  
NOVEMBER 17, 1913.

EDITED BY

**WILLIS O. NANCE, M.D.,**

**PRESIDENT CHICAGO OPHTHALMOLOGICAL SOCIETY (1913); OPHTHALMIC  
SURGEON, ILLINOIS CHARITABLE EYE AND EAR INFIRMARY; FORMER  
OCULIST AND AURIST, COOK COUNTY HOSPITAL; EDITOR  
JOURNAL OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY,**

AND

**WESLEY HAMILTON PECK, M.D.,**

**PRESIDENT CHICAGO OPHTHALMOLOGICAL SOCIETY (1914); FORMER  
PROFESSOR OPHTHALMOLOGY, CHICAGO EYE, EAR, NOSE AND  
THROAT COLLEGE; ASSISTANT SURGEON, ILLINOIS  
CHARITABLE EYE AND EAR INFIRMARY;  
OPHTHALMIC SURGEON, OAK  
PARK HOSPITAL.**

1914  
CHICAGO MEDICAL BOOK COMPANY  
CHICAGO

[Pg 2]

---

COPYRIGHT 1914  
BY  
CHICAGO MEDICAL BOOK COMPANY

---

## CONTRIBUTORS

[Pg 3]

EDWARD JACKSON, A.M., M.D.

Prof. Opth. Univ. Colo.; Emer. Prof. Opth. Phila. Polyclinic; Ex-Ch. Sec. Opth. A.M.A.; Ex-Pres. Am. Acad. Med. and Am. Acad. Opth. and Oto-Laryng.; Mem. Am. Opth. Soc. and Honorary Mem. Chicago Opth. Society.

JOHN ELMER WEEKS, M.D., D.Sc.

Prof. Opth. Univ. and Bellevue Hosp. Med. Coll., N.Y.; Opth. Surg. N.Y. Eye and Ear Inf.; Mem. Am. Opth. Soc.; Hon. Mem. Chicago Opth. Soc. and Royal Hungarian Med. Soc. Budapest.

GEORGE EDMUND DE SCHWEINITZ, A.M., LL.D., M.D.

Prof. Opth. Univ. Penn.; Opth. Surg. Univ. Hosp., Phila. Hosp., Orthop. Hosp. and Inf. for Nerv. Dis.; Consult. Opth. Surg. Phila. Polyclinic; Honorary Member Chicago Opth. Soc.

ROBERT HENRY ELLIOT, M.D., B.S. LOND., Sc.D. EDIN., F.R.C.S. ENG., ETC., LIEUT.-COLONEL, I.M.S.

Supt. Gov. Opth. Hosp., Madras. India; Prof. Opth. Med. Coll., Madras; Fellow Univ. of Madras; Honorary Member Chicago Ophthalmological Society, U.S.A.

CASEY A. WOOD, M.D., C.M., D.C.L.

Prof. Opth. Univ. Ill.; Late Prof. Opth. N. W. Univ.; Ex-Pres. Am. Acad. of Med.; Am. Acad. Opth. and of the Chicago Opth. Soc.; Ophthalmic Surg. St. Luke's Hosp.; Consulting Opth. Surg. St. Luke's and Cook County Hosp.; Ex-Ch. Opth. Sec. A.M.A.; Editor System Opth. Therapeutics. Sys. Opth. Operations and American Encyclopedia Ophthalmology.

FRANCIS LANE, A.B., M.D.

Pathologist and Asst. Ophthalmic Surgeon Ill. Char. Eye and Ear Inf.; Instructor in Opth. Rush Med. Coll.; Asst. Opth. Surg. Presbyterian Hospital.

E. V. L. BROWN, M.D.

Asst. Prof. Pathology of the Eye, Univ. Chicago; Asst. Prof. Opth. Rush Med. College; Opth. Surg. Ill. Eye and Ear Inf. and Cook County Hosp.; Mem. Am. Opth. Soc.

NELSON M. BLACK, Ph.G., M.D.

Author of The Development of the Fusion Center in the Treatment of Strabismus; Examination of the Eyes of Transportation Employes; Artificial illumination a Factor in Ocular Discomfort, etc.

FRANK C. TODD, M.D.

Prof. Opth. and Oto-Laryng., Univ. Minn.; Chairman Sec. Opth. A.M.A. and second Vice-Pres. A.M.A.; Opth. Surg. Univ. and Hill Crest Hospital.

ALBERT EUGENE BULSON, JR., B.S., M.D.

Prof. Opth. Ind. School Med.; Ex-Ch. Sec. Opth. A.M.A.; Opth. Surg. St. Joseph's Hospital; Editor Jour. Ind. Slate Med. Assn.

---

DEDICATED TO  
DR. EDWARD JACKSON  
DR. JOHN E. WEEKS  
DR. GEORGE EDMUND DE SCHWEINITZ  
LIEUTENANT COLONEL ROBERT HENRY ELLIOT  
HONORARY MEMBERS  
BY THE  
CHICAGO OPHTHALMOLOGICAL SOCIETY  
IN RECOGNITION OF  
THEIR SPLENDID ACHIEVEMENTS  
IN THE DOMAIN OF OPHTHALMOLOGY

---

[Pg 4]

## INDEX

### ABSTRACTS.

#### ETIOLOGY AND CLASSIFICATION OF GLAUCOMA.

*Edward Jackson, M.D.*

#### ETIOLOGY AND CLASSIFICATION OF GLAUCOMA.

Discussion, *Francis Lane, M.D.*

#### PATHOLOGY OF GLAUCOMA.

*John Elmer Weeks, M.D.*

PATHOLOGY OF GLAUCOMA.

Discussion, *E. V. L. Brown, M.D.*

CONCERNING NON-SURGICAL MEASURES FOR THE REDUCTION OF INCREASED INTRA-OCULAR TENSION.

*George Edmund de Schweinitz, M.D.*

CONCERNING NON-SURGICAL MEASURES FOR THE REDUCTION OF INCREASED INTRA-OCULAR TENSION.

Discussion, *Nelson Miles Black, M.D.*

TREPHINING FOR GLAUCOMA.

*Robert Henry Elliot, M.D.*

TREPHINING FOR GLAUCOMA.

Discussion, *Frank C. Todd, M.D.*

OPERATIONS OTHER THAN SCLERAL TREPHINING FOR THE RELIEF OF GLAUCOMA.

*Casey A. Wood, M.D.*

OPERATIONS OTHER THAN SCLERAL TREPHINING FOR THE RELIEF OF GLAUCOMA.

Discussion, *Albert E. Bulson, Jr., M.D.*

---

## ABSTRACTS.

[Pg 5]

### I. Etiology and Classification of Glaucoma.

Abstract:—

Etiologic factors include: obstruction of lymph spaces, especially the angle of the anterior chamber; blood pressure, arterial, capillary and venous; affinity of tissues for fluids; alterations of the intra-ocular fluids; inflammations in the eye ball; and failure of a nerve apparatus to control fluid in the globe. Classification: various types of glaucoma constituting clinical entities must be recognised, as: simple glaucoma, recurring exacerbations, congestive, mechanical, and increased tension arising during uveal inflammations.

DR. EDWARD JACKSON, Denver.

Discussion by DR. FRANCIS LANE, Chicago.

### II. Pathology of Glaucoma.

Abstract:—

(a) Changes taking place in corneal tissue.

(b) Iris angle with particular reference to the ligamentum pectinatum.

(c) Variations in the condition of the ciliary body.

(d) Consideration of the anatomical changes that take place in glaucoma secondary to retinal and chorioidal hemorrhage.

DR. JOHN E. WEEKS, New York City.

Discussion by DR. E. V. L. BROWN, Chicago.

### III. Concerning Non-surgical Measures for the Reduction of Increased Intra-ocular Tension.

[Pg 6]

Abstract:—

(a) The use of myotics; their preparation, method of administration, and explanation of their action.

(b) Reduction of increased intra-ocular tension by means of various mechanical measures, notably massage, vibration massage, suction massage, electricity and diathermy.

(c) Indirect reduction of increased intra-ocular tension, brought about by lowering the general vascular pressure.

(d) The relation of osmosis, lymphagogue activity, the absorption of edema, the stimulation of capillary contractility, and the lowering of the affinity of

ocular colloids for water in their relation to the reduction of increased intra-ocular tension.

DR. GEORGE EDMUND DE SCHWEINITZ, Philadelphia.

Discussion by DR. NELSON M. BLACK, Milwaukee.

#### IV. Trephining for Glaucoma.

Abstract:—

- (a) The aim of the operation is the formation of a foreign-body-free fistula.
- (b) It is most important to leave uveal tissue untouched.
- (c) Method of doing this explained.
- (d) The area available for trephining.
- (e) Method of increasing that area.
- (f) Cornea splitting.
- (g) Placing of trephine.
- (h) Technique of using trephine.
- (i) The operation is not difficult.
- (j) The operation valuable as a prophylactic measure.

DR. ROBERT H. ELLIOT, F.R.C.S., Lieut.-Col. I.M.S., Madras, India.

Discussion by DR. FRANK C. TODD, Minneapolis.

#### V. Operations Other than Scleral Trephining for the Relief of Glaucoma.

[Pg 7]

Abstract:—

Most of the ordinary surgical procedures employed for lowering intra-ocular tension furnish a permanent cure of certain fairly well defined varieties of glaucoma. They also relieve the symptoms and retard the progress of other varieties of the disease, even if they do not perform a cure. In a third class of cases, they either have no effect whatever in arresting the disease or they hasten its march towards blindness.

What operative procedure gives, on the whole, the best results? In other words, what operation is the easiest of performance, is the least likely to be attended by serious complications and is available for the largest number of cases? Reasons for believing that of the better known procedures simple iridectomy is the least effective, while those interventions producing a large, thin, scleral filtration-cicatrix are the most valuable.

DR. CASEY A. WOOD, Chicago.

Discussion by DR. A. E. BULSON, JR., Fort Wayne

[Pg 8]

---

## **Etiology and Classification of Glaucoma**

[Pg 9]

**BY**

**EDWARD JACKSON, M.D.,  
Denver.**

It is convenient to start with the conception that glaucoma is increased tension of the eyeball, plus the causes and effects of such increase; although a broad survey of the facts may reveal a clinical entity to be called glaucoma, without increased tension constantly or necessarily present, and cases of increased intra-ocular tension not to be classed as glaucoma.

The physiologic tension of the eyeball is essential to ocular refraction, and closely related to ocular nutrition. Fully to understand the mechanism for its regulation would carry us far toward

an understanding of the causes of glaucoma. Normal tension is maintained with a continuous flow of fluid into the eye and a corresponding outflow. Complete interruption of the nutritional stream would be speedy death; partial interruption may be held responsible for most of the visual impairment and pain of glaucoma.

[Pg 10]

The balance of intra-ocular pressure is not maintained by the slight distensibility of the sclero-corneal coat. Increased pressure does not open new channels for the escape of intra-ocular fluid; if, indeed, it does not tend to close the normal channels.

The affinity of the tissues for water, or, as Fischer explains it, the affinity of the tissue colloids for water, seems too little related to the requirements of ocular function to furnish the needed regulation of tension. The lymph spaces and blood-channels of the eye are large, as compared with the mass of its tissue colloids. In these spaces and channels must be sought a means for rapid response to the need for regulation of intra-ocular tension. Fischer has shown, that when the enucleated eyeball is placed in a weak solution of hydrochloric acid, the swelling of the tissue colloids is sufficient in a few hours, to burst the sclero-corneal coat. But this is an eye in which all nutritional changes have ceased. He brings together many facts to support the view that in the living tissues impaired circulation, and especially diminished oxidation, are the chief causes of increased affinity of the colloids for water. Such affinity increased by the impairment of the intra-ocular circulation, may well constitute a factor making for malignancy in glaucoma. But it can hardly explain the original departure from a normal pressure balance.

[Pg 11]

We must assume that intra-ocular pressure is kept down to the normal limit, by the prompt response of a regulative mechanism, which diminishes the flow of fluid into the eye, or permits its more rapid escape, whenever fluid tends to accumulate in the eye and increase its tension.

Little has been done to show that increase of fluid entering into the eye is the cause of glaucoma. A normal, or even a low arterial blood pressure is sufficiently above the normal intra-ocular pressure to furnish a source of increased fluid in the eye. Increased arterial pressure has been found in a large proportion of cases of glaucoma; and may be necessary to the production of the highest intra-ocular tension. A sudden relaxation of the arterial walls, that would permit the arterial blood pressure to make itself felt in the eye, might cause an important rise of intra-ocular tension and may be a factor in the etiology of acute attacks. It affords a possible mechanism through which may be produced the recognized glaucomatous effects of certain nerve disturbances. But such attacks are not commonly associated with noticeable flushing of the head and face generally; and paralysis of the cervical sympathetic is known to lower the intra-ocular tension.

[Pg 12]

Capillary blood pressure must lie between the arterial blood pressure and the venous blood pressure. It must be closely associated with the nutritional processes like secretion or inflammation; beyond this we know little about it. The association of increased blood pressure with glaucoma seems to be generally an indirect one through vascular lesions and disturbances of nutrition.

[Pg 13]

### *Obstructed Outflow*

A reservoir with a free outlet can only fill during a flood; and then quickly empties itself again. The outflow channels in the normal eye provide for carrying away of the waste products of such an active nutrition, that it is hard to think they will become inadequate in glaucoma until there has been a marked decrease from their normal capacity. Priestley Smith has pointed out that the glaucomatous eye softens more slowly than the normal eye after enucleation, in spite of the fact that a greater force is operating to drive fluid out of the eye. In his recent tonometric studies Schoenberg noted that under manipulation the glaucomatous eye softened more slowly than the normal eye; and suggests this diminished drainage as an important evidence of glaucoma.

[Pg 14]

Obstructed outflow might begin in an abnormal tendency of the tissues to retain fluid, a tendency that Fischer might locate in the colloids. The increase of intra-ocular pressure noted in cases of uveal inflammation, to be presently referred to, may be due to some such tendency. But it is rational to ascribe to obstruction of the filtration angle of the anterior chamber, the important part it has been supposed to play in the pathology of glaucoma. However this obstruction may be brought about, whether by thickening of the iris root during dilatation of the pupil, pushing forward of the iris root by the larger ciliary processes of age, or the enlarged crystalline lens pressing on the ciliary processes; or by inflammatory adhesion of the iris to the filtration area; ballooning of the iris, or its displacement by traumatic cataract; or adhesion to the cornea after perforating ulcer in the secondary glaucomas; or whether the obstruction is due to the accumulation of experimental precipitates, as shown by Schreiber and Wengler, or possibly of pigment granules into Fontana's space; or a process of sclerosis closing the spaces by contraction of new-formed connective tissue, or the covering over with proliferating implanted epithelium following injury opening the anterior chamber; glaucoma follows impairment of this drainage space, and lessened outflow through it. This blocking of the angle of the anterior chamber must be regarded as an established fact in the etiology of glaucoma. But because it is so definitely established, and because so much work has been done with reference to it, we may attach to it an undue importance.

[Pg 15]

The escape of the outflow of fluid from the eye is ultimately through the veins. The general venous blood pressure is so low (often negative in the great veins of the neck during inspiration) that no obstacle can come from it to the ocular outflow. The venous blood pressure permits the

[Pg 16]

eyeball to become perfectly soft. We have all seen tension of 5 mm., or even less; and general venous pressure does not rise to the normal intra-ocular tension. Increased intra-ocular pressure requires that there must be some obstacle that keeps the intra-ocular fluid from reaching the general venous system. This may be in the lymph drainage system of the eye; but it may also be in the ocular veins themselves.

Experimentally the eyeball can be made to burst by tying all the venous outlets from it. I have seen very high intra-ocular tension develop in a few hours after general thrombosis of the orbital veins. The absence of the canal of Schlemm is noted in congenital buphthalmos. The enlargement of the anterior perforating veins is an old symptom of chronic glaucoma. Obstruction to outflow of blood through the vorticosae veins, by the increased intra-ocular pressure, has long been a recognized explanation of the malignant tendency of glaucoma—a part of the vicious circle established in this disease. There is reason that we should give careful attention to the views of Heerfordt and Zirm, that obstruction to the venous outflow may be the effective cause of the disease. Zirm believes the venous plexus of the choroid is an essential part of the mechanism for the regulation of intra-ocular tension, the necessary vaso-motor control depending on nerve centers situated in the iris.

[Pg 17]

### *Nerve Control*

The accurate control of normal intra-ocular pressure, by mutual adjustment of inflow and outflow of fluid, is scarcely conceivable without some highly specialized, extremely sensitive nerve mechanism to preside over it. This is suggested by analogy with the regulation of secretion in the lacrimal, salivary, or peptic glands, or the maintenance of blood pressure in the heart and arteries. Clinical observations point the same way. Many patients connect their attacks (especially their earlier ones of ocular discomfort, impaired vision, haloes around the light, and dilated pupil) with social excitement, anxiety, worry, anger or fatigue. A patient of mine gave up her card parties, because an exciting game generally ended in blurred vision, a rainbow around the light, and a dilated pupil, and sometimes an aching eye. Another woman watching beside her dying husband and exposed to extreme cold, had her first attack of glaucoma, so severe as to destroy the sight of one eye. The other eye, also affected at the time, recovered good vision, and has remained several years without a second attack and without treatment.

[Pg 18]

Laqueur's first attack occurred at the end of a long exhausting morning in the operating room, with luncheon delayed two hours. The connection of his later attacks with anger, worry, embarrassment, even the excitement of watching a play at the theatre, was noted again and again. In Javal's case, the attack fatal to one eye came at the culmination of an exciting electoral campaign. The other eye was stricken at the termination of the Dreyfus case, in which Javal was intensely interested. There seems to be a special liability to glaucoma among those residing at high altitudes, best explained by nerve influence. The frequency of glaucoma among Jews may be due to a small cornea, as suggested by Priestley Smith; but it is quite as reasonable to connect it with a racial excitability or nervous instability. More definite knowledge of the nervous mechanism concerned in the regulation of intra-ocular pressure and the production of glaucoma is much needed.

[Pg 19]

### *Alterations of Fluids and Tissues*

The influence of increased affinity of the tissues for fluid has already been referred to. That a similar obstacle to the escape of fluid from the eyeball might be due to a change of character in the fluid, is a conception that has been entertained as a working hypothesis, and much experimental and analytical work has been done to test its correctness. This work has been so slightly related to practical ophthalmology, and so contradictory in its results that alterations in the fluids can only be regarded as a possible etiologic factor. Glaucoma secondary to intra-ocular hemorrhage, operations on the lens or its capsule, or severe nutritional disturbance may be capable of such explanation.

[Pg 20]

### *Different Kinds of Glaucoma*

A better grasp of the etiology of glaucoma may be attained by considering separately various types of cases; although perfectly typical cases may be rare; and cases of mixed type and etiology much more frequent.

Simple glaucoma has been recognized as closely related to atrophy of the optic nerve with deep excavation. No line of demarcation can be drawn between them, except by reserving the term of glaucoma for cases that depart from the pure type, terminating in glaucoma of some other kind, which is no more significant than the passage of a conjunctivitis into a keratitis, or an iritis into a glaucoma. Cases of simple glaucoma do run their course of many years to complete blindness, or to death, without exacerbations, inflammation, or characteristic pain. In such cases the intra-ocular tension does not rise suddenly; and it may be little or not at all elevated above the usual normal limit.

[Pg 21]

For nine years I have watched the progress of such a glaucoma in a man now aged 87, with slow development of glaucomatous cupping of the optic disc, now more than 3 D. deep. The tension has never been noted at more than Plus T (?), and when taken with the tonometer varied from 9 to 32 mm. for the worse eye, and 13 to 24 mm. for the other. Similar cases in which the tension

In the eye there is probably a normal equilibrium between blood pressure, tissue activity, and intra-ocular tension. This may be destroyed either by increasing the intra-ocular tension, or lowering the tissue activity, or the blood pressure. Lowered blood pressure has been suggested by Paton as an explanation of symptoms usually ascribed to vascular obstruction. Rising blood pressure may be required in old age to compensate for diminished tissue activity; and it is conceivable, under normal intra-ocular tension, that diminished nutritional activity may result in the same symptoms as are produced in other eyes by increased tension. Glaucoma is probably not so much an increase of tension as a loss of balance between intra-ocular tension and nutritional activity.

In contrast with the above are the cases marked by sudden elevations of ocular tension recurring repeatedly over long periods without permanent visual impairment. Laqueur's case continued of this character for six years, under the use of miotics, and then was cured by iridectomy, the cure remaining permanent with normal vision until his death after 30 years. Millikin has reported the case of a patient who in five years had "many hundreds" of attacks, in which vision was impaired, haloes appeared about the light, the pupil dilated, the cornea became steamy, and tension rose to plus T. 1 or plus T. 2. After iridectomy the attacks ceased, leaving no pathological cupping of the disc, full vision, and a good field. I have seen cases of this type in women under middle age, and of marked nervous instability. [Pg 23]

A third type which will come to be more generally recognized, as the tonometer comes to be more widely used, includes cases in which there is little beside the increase of intra-ocular tension to justify their mention in a discussion on glaucoma. A patient, then aged 21, suffered three years ago from a scotoma almost central; and was first seen six months after that with a macular choroidal atrophy and abnormal pigmentation. She suffered, we afterwards concluded, from choroidal tuberculosis. A recurrence involving adjoining choroid occurred fourteen months ago. There was at the start pain, slight dilatation of the pupil, and slight general hyperemia of the globe. The tension of the eyeball rose to 60 mm., that of the fellow eye being 20 mm. Under miotics the tension fell at first but slightly. It was 55 mm. at the end of a week; but after two weeks came down to normal, 20 mm. A month later the tension rose to 28 mm., but for a year has continued normal; the eye did well under tuberculin treatment, and without any local treatment. In September of this year I had two cases of iritis in which the intra-ocular tension rose to 45 and 52 mm., respectively, and gradually returned to normal, with the cure of the iritis under atropine. In one of these cases, a lady of 70, I used atropine also in the other eye, but the tension of that eye remained normal, 22 to 24 mm., throughout. After needling the lens in young people I have seen a rise of intra-ocular tension to 50 and 60 mm., maintained for many days, with considerable general deep hyperemia, and soreness of the globe, followed by gradual return to normal tension, and no permanent impairment of vision or the visual field. [Pg 24]

One other type may be mentioned. That of an elderly patient with marked vascular disease, often renal involvement, and distinctly impaired nutrition. There may be renal retinitis or retinal hemorrhages. The case may easily become one of hemorrhagic glaucoma. It may run a very chronic course. But it may become suddenly worse, or go on to complete blindness with pain, demanding enucleation, after some temporary perturbation, as the performance of a glaucoma operation. It is pre-eminently the kind of a case you would prefer would go to some one else. [Pg 25]

Each of these types illustrate a distinct cause or group of causes. The first type brings us near to what may be the essential nature of glaucoma, impairment of ocular nutrition by the intra-ocular tension, which is generally elevated, but may not be above the usual normal. A special weakness in the nutrition of nerve tissue may be assumed. It would help to explain the cavernous atrophy of the optic nerve associated with simple glaucoma. The second type shows impairment of the regulative mechanism permitting rapid rise of the intra-ocular pressure. In persons of good nerve nutrition and strong recuperative power, it may exist for years without doing permanent damage. But joined to causes of the first type, lowered nutritive activity, it causes rapid and permanent loss of sight. The third group are cases associated with glaucoma only as causes. In eyes with low nutritive power, or subject to exacerbations of increased intra-ocular pressure, uveal inflammations may prove disastrous. The fourth type shows the results of the combination of the causes of the other types; with the elements of acute or slow malignancy added—the impaired circulation and lowered oxidation producing some degree of edema of the tissues that insures a fatal result. [Pg 26]

This is no complete presentation of my subject, but a selection of facts bearing on the etiology, to serve as a foundation for the discussion of those practical aspects of glaucoma which are to claim your attention through the papers and remarks of subsequent speakers. [Pg 27]

---

## **Dr. Edward Jackson's Paper on Etiology and Classification of Glaucoma** [Pg 28]

**Discussion,**  
**FRANCIS LANE, M.D.**  
**Chicago.**

Not one of the theories thus far propounded to explain the essential cause of increased intra-ocular tension is satisfactory. Our present day knowledge apparently ceases with a more or less incomplete understanding of the mere circumstance under which increase of tension in general depends.

The question of the source of the normal intra-ocular pressure must first be solved before any discussion of a pathological increase can be engaged in. This question primarily hinges on whether the corneo-sclera is to be regarded as an unelastic capsule with a fixed volume, or as a yielding envelope with an ever changing capacity.

[Pg 29]

This brings us at once to the consideration of that theory which probably has held our attention for the longest period of time, *i. e.*, the volumetric theory. According to it, the normal intra-ocular tension depends on the volume of fluids within the eyeball. Any variation in the quantity of the contents gives rise to a change in the pressure, therefore, the globe has been regarded as "an elastic capsule, whose capacity, form, and internal pressure depend on the balance struck between a constant inflow, or formation of aqueous, and a proportionate outflow or resorption." (Henderson.)

Hill has satisfactorily demonstrated that, under physiological conditions, the hydrostatic pressure within the eye and the skull is identical; it rises and falls simultaneously; it is the same as the cerebral venous pressure; it is constantly varying, depending directly on the general circulation. Upon these findings Henderson based his opinion that the physiological properties of the tunica fibrosa and the skull are identical, realizing at the same time, that the rigidity of the corneo-sclera, because of its fibrous nature, is not as firm as the cranium. In accepting this belief the inference was that the cubic capacity of both coverings is fixed. Applying these conclusions to the eye, it can be said that the pressure of the fixed intra-ocular volume varies with the venous tension within the bulb, which in turn is influenced by the general circulation. Such a conception, while not strictly in accord with recognized physiological teachings, proves that the normal intra-ocular pressure is not a question of volume content, but that it is purely a question of pressure of a fixed volume within an unyielding capsule. Dr. Jackson virtually puts aside the volumetric theory with his statement, that "the balance of intra-ocular pressure is not maintained by the slight distensibility of the sclero-corneal coat." Further discussion on the inadequacy of the volumetric theory need not detain us.

[Pg 30]

It is well to recall a few anatomical features because of their bearing on the theories herein considered.

[Pg 31]

1. The angle of the anterior chamber is a true angle and not an annular sinus.
2. The meshwork of the iris angle (ligamentum pectinatum), a cellular structure at birth, undergoes a progressive and physiological fibrosis with early subsequent sclerosis, until finally it becomes a fibrous structure. The individual strands of this meshwork are more than two times as large at advanced age as at birth, consequently the alveoli of the meshwork becomes markedly reduced in size.
3. The spongy nature of this meshwork affords free access of aqueous to the venous sinus of Schlemm, thence by tributaries into the supra-choroidal space and anterior uveal venous system.
4. Fuchs's iris cripts afford direct access of aqueous to the veins of the iris.

Furthermore, two simple principles are taught by physics: Fluids are incompressible and they seek the lowest hydrostatic level. The application of these perfectly obvious principles to the eyeball makes the intra-ocular pressure the same as that within the elastic venous walls, which is the lowest circulating pressure within the bulb.

[Pg 32]

To summarize: The aqueous has direct access to the anterior uveal venous system; the physiological thickening of the strands of the meshwork of the iris angle supplies a mechanical obstruction between the anterior chamber and the venous sinus of Schlemm; intra-ocular pressure stands at the same level as the intra-venous, consequently, the hydrostatic pressure is the same on both sides of the iris angle meshwork, because the canal of Schlemm is a secondary venous system; lastly, the outflow of aqueous into the venous sinus is by diffusion, not by filtration, because the pressure is the same on both sides of the meshwork.

These facts and deductions have given rise to the present day circulatory theory of intra-ocular pressure, so we now can approach the predisposing and exciting factors which determine glaucoma.

[Pg 33]

The central fact to be borne in mind is, if the physiological pressure is vascular in origin and nature, the pathological pressure must likewise be derived from the same source.

Sclerosis of the meshwork of the iris angle is the predisposing factor because it hinders free access of aqueous into the venous sinus of Schlemm. Sclerosis alone, however, will not cause glaucoma so long as access to the iris veins can keep the intra-ocular pressure at the intra-venous level, and, too, as long as the exciting cause is absent.

The exciting cause is vascular, maintained and influenced by the general circulatory pressure. A rise of the general vascular tension alone will not cause glaucoma, because any alteration in intra-ocular pressure resulting would be purely a temporary change, easily taken care of by the extensive access of aqueous to the intra-ocular venous system. When these two factors coexist in their varying combinations, pathological increase of pressure results—in short, glaucoma.

[Pg 34]



Syphilis, rheumatism, gout, auto-intoxication and many other constitutional disorders are well recognized agencies which induce sclerosis in body tissues, so there can be little doubt that these conditions produce pathological sclerosis of the meshwork of the iris angle. Psychic disturbances, congested portal or renal system, hard mental or muscular work, etc., etc., induce increased pressure of the general circulation, and so simultaneously the intra-ocular pressure.

According to the edema theory advanced by Fischer, glaucoma is "essentially an edema of the eyeball, and for its production we must hold responsible the same circumstances which are responsible for a state of edema in any other part of the body." The magnificent experimental work of this investigator has shown that edema is nothing more or less than an increased capacity of the protein colloid tissues for water; that the most important factor leading to this increased hydration capacity is an abnormal production or accumulation of acid content, effected by those agencies which are instrumental in causing sclerosis and an increase of blood pressure.

[Pg 35]

It seems that both of these theories afford an explanation for many of the secondary pathological manifestations which characterize the intra-ocular tissues during a glaucomatous onset.

Fischer criticizes the Henderson theory on the ground that increased blood pressure alone does not lead to edema—edema is thwarted by high blood pressure. On the other hand, if Fischer believes that sclerosis of the meshwork of the iris angle is a result and not a cause of glaucoma, then it would seem that Henderson has the better of the argument. The physiological changes in this structure, which take place with advancing age, can rightfully be looked upon as a predisposing factor in glaucoma.

Dr. Jackson has presented all other phases of this part of the symposium in such a comprehensive manner that nothing further remains to be said.

[Pg 36]

---

## Pathology of Glaucoma

[Pg 37]

BY

JOHN E. WEEKS, M.D.,  
New York City.

In reviewing the pathology of glaucoma it seems proper to consider the various structures and tissues of the eye in logical order.

*Lids and Conjunctiva.* "The only change observed in these tissues is a reflex edema, excited apparently by pressure on the ciliary nerves and, probably, irritation of the vaso-motor fibers of the sympathetic."

*Lachrymal Gland.* Hyper secretion due to reflex irritation.

*Cornea.* As has been shown by Priestley Smith, the cornea in glaucomatous eyes is, as a rule, smaller than in non-glaucomatous eyes, the mean of a series of measurements being 11.1 mm. horizontally and 10.3 mm. vertically in glaucomatous and 11.6 mm. horizontally and 11 mm. vertically in non-glaucomatous eyes. In cases of considerable increase of tension, particularly if the onset is sudden, the circulation of lymph in the cornea is interfered with, the anterior layers of the cornea become edematous, the spaces between the lamellae filled with albuminous fluid. Some of this fluid finds its way through Bowman's membrane, apparently by way of the minute channels which permit the passage of small nerve twigs, and enters the epithelial cell layer. The fluid finds its way between the epithelial cells in the deeper layers, apparently being taken into some of the superficial cells by imbibition. Some of the swollen surface cells open spontaneously and discharge their contents, others drop off. The process causes a roughening of the surface of the cornea and produces a faint haziness. There is another form of haziness that develops on sudden rise in tension and completely disappears on subsidence of the tension. This is due, as has been shown by V. Fleischl (Sitzungsberichle d. Wiener Akad. d. Wissensch, 1880) and others, to increased tension on the fibrillae of the cornea, a double refraction being induced. In cases of long continued increase of tension minute permanent vesicles form in the epithelial layers, particularly in the superficial portion. Anaesthesia of the cornea develops, due to pressure on the nerve fibers that are distributed to the epithelium, the compression probably occurring along the course of the long ciliary nerves, from which the corneal nerves are derived, as they pass between the choroid and the unyielding sclera (Collins & Mayou).

[Pg 38]

[Pg 39]

In advanced cases of glaucoma after the congestive period has subsided the cornea becomes somewhat condensed, the lymph spaces contracted; a condition of sclerosis obtains. Alteration in the shape of the cornea occurs only rarely in adult life. When it does occur it takes place in corneæ that have suffered from keratitis. The alteration is usually in the form of ectasiæ. In infancy and early youth (buphthalmia) the cornea may become uniformly enlarged and globular. Often, however, the enlargement of the cornea is irregular. Increase in tension may produce fissures in Descemet's membrane. These occur more frequently in the cornea that have suffered a change in shape, as in buphthalmos. Gaps occur in the elastic membrane which become covered by endothelium. Some cloudiness may be seen in the corneal lamellae adjacent to these fissures, in some cases due evidently to the filtration of aqueous humor through defective

[Pg 40]

endothelium. Prolonged high intra-ocular tension may be accompanied, particularly in cases of secondary glaucoma, by vesicular and bullous keratitis.

In acute glaucoma the sclera appears to be edematous and slightly thickened. As the disease progresses the sclera becomes denser than normal. The oblique openings—passages for the venae vorticosae—are said to be narrowed. The openings for the passage of the anterior ciliary vessels are enlarged in many, particularly in advanced cases. Minute herniae at these openings are sometimes present. Dilatation and tortuosity of the anterior ciliary veins are due apparently to excessive flow of blood through them on account of the abnormally small amount carried off by the venae vorticosae. In the stage of degeneration, ectasae of the sclera occur most frequently near the equator of the globe. Spontaneous rupture may take place.

[Pg 41]

*Anterior Chamber.* The anterior chamber is shallow, as a rule. This is almost without exception in primary glaucoma in adults. In secondary glaucoma in which occlusion of Fontana's spaces occurs as a result of the deposition of fibrin or other inflammatory products the anterior chamber may be of normal depth, or deeper than normal. Very deep anterior chamber may occur in glaucoma, due to retraction of lens and iris following fibrinous or plastic exudation into the vitreous, or when it occurs in congenital glaucoma, due to enlargement of the globe.

[Pg 42]

*Aqueous Humor.* The aqueous humor, as has been pointed out by Uribe-Troncoso (Pathoginie du Glauome 1903) contains a greatly increased quantity of albuminoids and inorganic salts in glaucoma. In acute glaucoma the increase of albuminoids (blood proteids) is greater than in chronic glaucoma. The aqueous humor becomes slightly turbid in acute attacks, coagulating more readily than the normal. The plastic principle contained in the aqueous is rarely sufficient to cause adhesion between the margin of the iris and the lens capsule, but the colloid nature of the aqueous, according to Troncoso, lessens its diffusibility and prevents its free passage into the lymph channels. The increase in albuminoids is a consequence of congestion and venous stasis and does not precede the attack.

*Filtration Angle.* The changes that occur in the filtration angle before it is encroached upon by iris tissue are sclerosis of the ligamentum pectinatum in adults to which Henderson (Trans. Ophth. Soc. U.K. Vol. xxviii) has called our attention; the accompanying sclerosis of the other tissues to the inner side of Schlemm's canal; and, in some cases, the deposition of pigmented cells derived from the iris and ciliary processes (Levinsohn) which serve to obstruct the lymph spaces. In many of the cases of acute glaucoma and almost all of the cases of chronic glaucoma of long standing the filtration angle becomes blocked by the advance of the root of the iris.

[Pg 43]

*Iris.* In acute glaucoma the iris is congested and thickened. It is pushed forward and may lie against the cornea at its periphery. When the attack subsides, the iris falls away from the cornea. Aside from the congestion, the primary changes that take place in the iris are indicative of paresis of the fibers of the motor oculi that supply the sphincter pupillae, and stimulation of the fibers from the sympathetic producing vasomotor spasm. The long diameter of the pupil apparently lies in the direction of the terminal vessels of the two principal branches of each long ciliary artery which form the circulus iridis major, where the vasomotor spasm would have the greatest effect in lessening the blood supply. The haziness of the cornea and slight turbidity of the aqueous contribute greatly to the apparent change in the color of the iris. In cases of simple chronic glaucoma there is but little evidence of edema of the iris. If the iris lies in contact with the sclera and cornea for some time, it becomes adherent (peripheral anterior synechia). As the disease progresses, the stroma of the iris atrophies and contracts. There is very little evidence of small-cell infiltration or the formation of cicatricial tissue. Numerous slits may develop in the iris through which the fundus of the eye may be seen (polycoria). The pigment layer does not atrophy in proportion to the stroma of the iris; by the contraction of the stroma of the pigment layer is doubled upon itself at the pupillary margin, forming a black ring of greater or less width (ectropian uveae). The iris becomes attached to the pectinate ligament and to the endothelium of Descemet's membrane. In a very few cases the closure of the angle is not complete at the apex, a small space remaining comparatively free for a long time. The adhesion of the iris to the pectinaform ligament and cornea is not uniform at all parts of the periphery; it varies in width. Portions of the iris angle may remain open while other parts are closed. Where the iris tissue lies in contact with the cornea, the stroma of the iris almost totally disappears. In some cases the iris becomes totally adherent to the cornea.

[Pg 44]

[Pg 45]

*Ciliary Body and Chorioid.* In acute glaucoma there is congestion of the entire uveal tract, the congestion partaking more of a venous stasis than of an active or arterial congestion. The vessels of the ciliary process, which are larger and more tortuous in adults of advanced years than in the young, become enormously distended, causing almost complete obliteration of the perilental space. They press against the root of the iris and the equator of the lens, forcing them forward. There is edema of the ureal tract, apparently from transudation of serum. Many small, and sometimes rather large hemorrhages may occur. There is but little small cell infiltration, indicating almost total absence of what is ordinarily recognized as true inflammation. It is probable that the secretion from the glandular zone of the ciliary body is increased.

[Pg 46]

On subsidence of the congestion, as after miotics or iridectomy, the tissues may return to very nearly a normal condition. The iris recedes from contact with the ligamentum pectinatum and cornea and the filtration angle is again open. In some cases the iris becomes adherent to the head of the ciliary processes and, when atrophy of the ciliary body occurs, is drawn backward at the base of the iris by the receding tissues. If the hypertension persists or is repeated at varying periods, a slow atrophy of the uveal tract sets in. Eventually the ciliary body becomes very much

[Pg 47]

reduced in thickness, is flattened out, the ciliary processes reduced in size and the blood vessels disappear or are reduced much in caliber. Those that persist possess walls that are much thickened. This is particularly true of hemorrhagic glaucoma.

In advanced absolute glaucoma the chorioid may become reduced to a very thin membrane consisting of connective tissue and pigmented cells, scarcely distinguishable even by moderate powers of the microscope. Atrophy is marked in the vicinity of the venae vorticosae. Czermak and Birnbacher describe proliferation of the endothelium of the large veins with contraction and obliteration of their lumen.

*Optic Nerve and Retina.* In the acute form the retina and optic nerve present the same condition that is present in the vascular tunic; namely, that of venous stasis with the consequent edema. Frequently minute hemorrhages occur in the retina, particularly in violent acute attacks. Cupping of the discs slowly develops, causing more or less stretching of the nerve fibers over the edge of the cup. The gradual diminution of the field of vision is due in greater part to death of peripheral nervous elements of the retina, those parts of the field farthest removed from the large arterial trunks suffering first. The arrangement of the arteries at the disc, passing out as they do from the nasal side, of necessity make the vessels that pass to the temporal part of the retina longest and of less caliber. These vessels and their terminals are first to suffer marked diminution in size; death of the perceptive elements supplied with nutrition by these vessels follows. For this reason the nasal part of the field of vision is more often the first to disappear. In congestive (inflammatory) glaucoma, the typical field of vision shows most marked contraction on the nasal side. The disturbance of the nutrition of the retina accounts in greater part for the various forms of visual field met with. [Pg 48] [Pg 49]

Death of all of the perceptive elements of the retina eventually occurs. The loss of nutrition is apparently not the whole cause of blindness. Atrophy of the nerve fibers follows death of retinal neurons, but atrophy of some of the nerve fibers may be, and probably is, due to the pressure and traction exerted upon them at the margin of the disc. It is probable that too much importance has been given to this mode of interference with the nerve fibers. However, the change in the position of the lamina cribrosa must exert a deleterious effect, particularly on those fibers which pass through the peripheral meshes, the shape of which must necessarily be much distorted. In glaucoma simplex, which is largely devoid of marked congestive periods (acute attacks), a surprisingly high degree of acuity of vision may exist with a deep excavation and pale nerve. Careful studies of the retinal vessels in glaucoma (Verhoeff Arch. of Ophth. XLII. p. 145; Opin. Soc. Française d'Ophth. 1908) disclose the fact that an increase in the elastic tissue and connective tissue elements occurs in *some cases*, also proliferation of the endothelial cells, which serve to irregularly narrow and, in some instances, obliterate the lumen of the vessel. Arteries and veins are both affected. Hyaline degeneration of the media also occurs. The process is not uniform. [Pg 50]

*Glaucomatous Cup.* The excavation of the disc progresses slowly and is due in part to stretching the fibers of the lamina cribrosa pressing this structure outward, and partly to atrophy and disappearance of the nerve tissue and much of the vascular tissues in the nerve head. The displacement backward of the lamina cribrosa may cause that structure to lie behind the outer surface of the sclera. Atrophy and cystic degeneration of the nerve trunk follows destruction of retinal neurons and cupping of the disc. Neuroglia remains in part. Connective tissue elements increase in the optic nerve as the nerve fibers disappear. [Pg 51]

*Glaucomatous Ring.* The development of the pale circle which surrounds the disc, particularly in glaucomatous eyes, is due to a very slight recession of the pigment layer of the retina and of the margin of the chorioid at this point with some atrophy, apparently consequent on the beginning retraction of the lamina cribrosa and slightly increased pressure of the nerve fiber layer on the underlying tissues at the margin of the disc. This permits the sclera to show through a very little at this part. In some eyes in which there is a beginning sclero-chorioiditis posterior, the condition is very similar to that presented by the glaucomatous ring.

*Field of Vision.* The two pathological processes that operate to destroy the function of the retina suffice to produce scotomata in the field of vision of varying shapes. The typical glaucomatous field in the acute cases shows a defect most pronounced to the nasal side. As has been shown by Bjeraum, the blind spot corresponding with the optic disc is enlarged in glaucoma, a relative scotoma often connecting it with the blind nasal portion of the field either above or below the horizontal meridian (Straub). The field in a simple glaucoma is apt to approach concentric limitation; namely, more like the field in simple atrophy. This is consistent with the fact that simple glaucoma in many cases possesses the characteristics of glaucoma plus atrophy of the optic nerve. [Pg 52]

*Vitreous.* During the acute attack, the vitreous may become slightly turbid by transudation of serum from the vessel of the ciliary body and the chorioid and may become filled with fibrin. In some chronic cases in which absolute glaucoma is reached the development of small blood vessels in convoluted loops springing from the vessels of the discs has been observed. Any process that increases the volume of the contents of the vitreous chamber, as hemorrhage, neoplasm, profuse serous or plastic exudation, may by pushing iris and lens forward produce an attack of acute glaucoma. [Pg 53]

*Buphthalmos.* Reis (Graefe's Arch. f. Ophth. V. LX. 1905) states that there is always obliteration of the anterior scleral venous channels (Schlemm's canal) in buphthalmos. Seefelder (Graefe's Arch. V. LXIII. 1906) mentions the abnormal position and abnormal narrowing of Schlemm's

canal and the imperfect and insufficient differentiation of the cornea-scleral junction. In all of the cases in which the eye has been examined microscopically obliteration of Schlemm's canal has been reported. This is thought to be a defect in development. Magitot (Ann. d'Oculis CXLVII) suggests that injury to mesoderm which pushes itself between the ectoderm and anterior surface of the lens would account for the failure in development of Schlemm's canal. The changes that occur in the tissues of the eye appear to be largely due to the stretching consequent on the more or less uniform distentions of the globe as a result of hypertension.

[Pg 54]

*Cornea.* This portion of the fibrous membrane is enlarged, globous or flattened, irregularly thinned, particularly at the periphery, where it may be as thin as tissue paper, nebulous because of the stretching of its fibers principally, but in some degree (differing in different cases) to edema of the epithelial layer. Fissures occur in Descemet's membrane.

*Anterior Chamber.* This is very deep in the greater number of cases. However, this rule has many exceptions.

The vascular tunic may be congested in young infants, but atrophy soon develops and may reach an extreme degree. The sclera ordinarily becomes quite thin throughout, but may retain almost a normal thickness at the equator of the globe and posteriorly. Posterior sclera ectasae may develop. The iris, as a rule, hangs free from the cornea, often tremulous because of retraction of the lens beyond the iris plane. In some cases the iris is partly or totally adherent to the posterior surface of the cornea.

[Pg 55]

The vascular membrane (iris, ciliary body and chorioid) and the retina become atrophic, the atrophy varying in degree in various parts. Detachment of the retina may occur, often preceded by or accompanied by subretinal hemorrhage. The optic disc becomes deeply cupped and the tissues of the optic disc and optic nerve extremely atrophied. The crystalline lens may become cataractous and shrunken. Spontaneous rupture of the suspensory ligament with consequent subluxation of the lens may follow.

*Secondary Glaucoma.* The pathological conditions that precede secondary glaucoma are many and differ widely. They may be briefly classified as:

[Pg 56]

1. Those that cause a partial or complete closure of the lymph spaces and Schlemm's canal by cicatricial contraction, as in sclero-keratitis.
2. Those that cause obstruction to the lymph spaces at the filtration angle by the deposition of fibrin or cellular elements, as in iritis, hemorrhage into the anterior chamber, etc.
3. Those that cause obstruction of the filtration angle by advancement of the iris and lens, as occurs when the volume of the contents of the vitreous chamber is increased, as from retinal or chorioidal hemorrhage or neoplasm.

The various changes are so numerous that they need not be described further here. The ultimate changes due to high tension resemble those already described.

---

## Dr. John E. Weeks' Paper on Pathology of Glaucoma

[Pg 57]

**Discussion,**  
**E. V. L. BROWN, M.D.,**  
**Chicago.**

I would like to emphasize one of the newer features of the pathologic anatomy of glaucoma, one which has received too little attention in this country: the *lacunar* or *cavernous atrophy* of the *optic nerve*.

The name accurately describes the condition. Tiny clear spaces form in the lamina cribrosa and in front and behind it in the nerve tissue. Their exact nature is unknown. Usually they are entirely empty, often they are traversed by fine glial fibers. They seem to be in no relation to the blood vessels. Adjoining lacunae are supposed to fuse to form larger cavernae and these finally merge and constitute the final glaucoma cup. The lamina may then bridge across the space like a cord, or lie back against the end of the nerve trunk.

[Pg 58]

Schnabel considered all glaucoma cups to be formed in this way, independent of tension. His views were strongly supported by Elschnig, but as vigorously opposed by others. Axenfeld cites the fact that the glaucoma cup may disappear after operation. (I myself have seen a cup of 7 D. reduced to 1 D. in the course of a year after the tension had been lowered from 62 to 12.) Stock found the same lacunae in eight cases of myopia. The last extended study of the subject was made by E. v. Hippel, who found lacunae in 20 of 33 cases (60 per cent); enough certainly to make one look for them carefully in every case. He publishes a large number of excellent photomicrographs, but none more typical than one I have in my possession.

I have been especially interested in this subject because I have met with a complete and total glaucoma cup, with the typical (ampulliform) undermining of the scleral ring, in a pair of eyes without increased tension. The (Schiotz) tonometer was used daily for 70 consecutive days and

[Pg 59]

never registered more than 12-14 mm. Hg. The man had been blinded by wood alcohol. At the time I could find no other report in the literature, but overlooked a publication by Lewin and Guillery. Friedenberg has since reported cases of the same nature.

If other conditions than increased tension can produce a typical (ampulliform) glaucomatous excavation of the disc, why may not the cavernous atrophy and cup in glaucoma be due in part at least to similar processes, possibly in the nature of a toxic oedema of the nerve, either in association with tension or independent of it, as contended for by Schnabel?

---

## Concerning Non-Surgical Measures for the Reduction of Increased Intra-ocular Tension

[Pg 61]

BY

GEORGE EDMUND DE SCHWEINITZ, M.D.,  
Philadelphia.

Only a few years ago the literature of glaucoma was big with discussions of the comparative value of the surgical and non-surgical treatment of glaucoma, and especially of the chronic types of this disease. Now, thanks to the achievements of Lagrange, Fergus, Herbert and Elliot, the value of a filtering cicatrix, although known for a long time, has attained increased importance, due to the improvement and elaboration of operative technic, and the medical journals of the day are weighted with opinions and experiences from all over the world as to these surgical measures. But true as this is, we are not yet in a position to discard non-surgical procedures (1) because operation is not always possible, (2) because operation is not always permitted, and (3) because in certain circumstances operation is not advisable. Hence a glance at the non-surgical methods of reducing increased intra-ocular tension is not out of place, and for convenience they may be catalogued as follows:

[Pg 62]

1. Myosis produced by means of solutions of various drugs, a myosis followed by reduction of intra-ocular tension.
2. Reduction of tension by means of various mechanical measures, notably massage, vibration massage and suction massage, and by means of electricity and diathermy.
3. Indirect reduction of intra-ocular tension, accomplished by lowering general vascular pressure.
4. Reduction of ocular tension by stimulation of osmosis, of lymphagog activity, of absorption of edema, and of capillary contractility, and by decreasing affinity of ocular colloids for water.

[Pg 63]

1. *The Myotics.* Of these, eserine (physostigmin) and pilocarpin, with their respective salts, the sulphate and the salicylate in the first instance, and the hydrochlorid and the nitrate in the second, are well established in favor and efficiency. Personally, it has always seemed to me that the salicylate of eserine is preferable to the sulphate, but I have not persuaded myself that the nitrate of pilocarpin possesses material advantages over the hydrochlorid, although some authors prefer it. With arecalin, the alkaloid of the Betel nut, I have no experience, nor have I used its mixture with eserine, recommended by Merck as more potent than either of the drugs in separate solution.

The substance isophysostigmin, found with eserine in Calabar bean, according to Ogiu, exceeds in its myotic activity the sulphate of eserine, *i. e.*, 1/80 of a grain of the drug is equal to 1/60 of a grain of the sulphate of eserine, but it is certainly not less irritating than physostigmin, and according to Stephenson's researches, is more so, and in this sense has no superiority over the usual alkaloid. In general terms, it may be said that the time has not arrived to make a preachment "on the passing of eserine and pilocarpin."

[Pg 64]

*Physiologic Action.* Concerning the ocular, physiologic action of the two chief alkaloids respectively of Calabar Bean and of Jaborandi, there still exists difference of opinion. It has always been easy to attribute the myotic action of these drugs, or at least, of eserine, to their stimulant action on the peripheral ends of the oculo-motor, thus causing sphincter contraction, and to a depressing action on the sympathetic fibers, thus causing removal of the action of the dilatator of the iris. But complete experimental proof of such action is wanting, and it is probable that myosis follows a direct stimulation of the sphincter muscle fibers, aided, perhaps, by contraction of the iris vessels, although the last named effect is denied by so competent an authority as Hobart Hare.

[Pg 65]

Exactly how the myotics reduce intra-ocular tension is not definitely proven. Usually it is taught that because of the myosis the base of the iris wedged in the angle of the anterior chamber is loosened and withdrawn, precisely as a fold in a coat is straightened by a tug on the fabric beneath it. Experiments, however, for example, by E. E. Henderson, have shown that the rate of filtration in an eye with artificially raised pressure is considerably larger when it is under the influence of eserine than it is when under the influence of atropin; that is by the contraction of the pupil the iris-surface filtration is increased and consequently the pressure is reduced. We all know that Thomas Henderson maintains that the results of iridectomy are beneficial because the

raw edges of the coloboma, which do not cicatrize, permit access of the aqueous to the iris veins, and that myotics, inasmuch as they contract the pupil, open the iris crypts and therefore act, less efficiently, perhaps, but act none the less like an iridectomy. The normal intra-ocular pressure is uninfluenced by myotics because this pressure represents the lowest circulatory pressure in the eye, and further contact between aqueous and veins cannot reduce it below this level, another point which is made by Thomas Henderson in support of his contention.

[Pg 66]

The clinical fact remains that either by mechanical means, as it were, in the liberation of a plugged filtering angle, or by the increasing of iris-surface filtration, the myotics markedly reduce the abnormal intra-ocular pressure.

*Methods of Administration and Indications.* With the methods of administration of the myotics we are all so familiar that time need not be wasted in their reiteration, except to refer to a few practical points. In acute glaucoma, and every one knows that in this disease their action is often prompt and sometimes curative, eserine in a strength of one to four grains to the ounce may be instilled with sufficient frequency to establish myosis, and its action in this respect is enhanced if the congestion of the eye is lowered by measures to which I shall refer later. There is a good deal of clinical evidence to indicate that in this type of glaucoma, as well as in the so-called sub-acute varieties, myotic activity is increased by a mixture of pilocarpin and eserine in the same solution, exactly as a mixture of arecalin and eserine is more potent than either of the drugs in separate solution.

[Pg 67]

Prior to the happy advent of technically correctly placed filtering cicatrices, a large number of surgeons depended almost exclusively on the use of myotics in so-called simple, chronic or non-inflammatory glaucoma. This is not the place to introduce a discussion of the comparative value of iridectomy and myotic treatment in simple glaucoma as based upon statistical records. We must wait now for a sufficient period of time and then compare the value of myotic treatment with that of operations by means of which satisfactory filtration is produced. We are somewhat in the position that general surgeons occupied when aseptic methods first became prevalent. We do not usually compare the statistics of early aseptic days with those of the pre-antiseptic period, and I do not think we ought to compare the statistics of myotic treatment with ordinary iridectomy any longer, but that we should wait until we can make a comparison between the results of prolonged myosis and those of an improved modern technic which establishes a permanent filtration. In the meantime the patients who will not or cannot submit to operation must be reckoned with. Doubtless many patients with chronic glaucoma can be satisfactorily managed with myotic treatment, although personally I have always advocated operation when this could be performed, but it cannot always be performed. This rule should guide us, namely, to begin with a comparatively weak solution of the selected drug, for example, as Posey has advocated a tenth of a grain of salicylate of eserine to the ounce, and the strength gradually increased so that at the end of some months the patient is using a solution 1 grain to the ounce; or if the pilocarpin is preferred, solutions in double these strengths. It is my own belief, and that of many who have studied this subject, that if, without eserine irritation, a myosis can be maintained, and if the treatment can be begun early enough, the chances of preserving vision and the field of vision are good. I believe that the two most important instillations during the twenty-four hours of the number necessary to maintain this myosis are on retiring and if possible in the very early morning, some time between two and four o'clock. Most patients can be taught to wake themselves at the proper period of time, and are little inconvenienced by this disturbance of their sleep. I believe that eserine irritation is most successfully avoided, not by preparations of the myotics in combination with the antiseptics, for example, tricresol, which has been so much advocated, but by ordering very small quantities of the solution, insisting that it shall be frequently renewed and sterilized at each preparation, and that a half an hour after its instillation, during the day time at least, the eye shall be thoroughly flushed with some mild antiseptic solution, for example, boric acid and sodium chlorid. Whether the action of the eserine on the choroidal circulation, which is maintained by Wahlfours, aids in this favorable action of the myotics remains to be proved. It has been maintained by this author and by others who have followed him.

[Pg 68]

[Pg 69]

[Pg 70]

The great trouble with myotic treatment is not its lack of efficiency, but the difficulty of carrying it out successfully on ambulant patients, even in the better walks of life. It is hard successfully to maintain in a patient with chronic glaucoma what I may call an eserine life, just as it is hard to maintain in a person with an enlarged prostate a catheter life and escape infection, resulting, if it occurs, in the one instance in a difficult and stubborn conjunctivitis, and in the other in a cystitis. Still, we are obliged to use myotics, and the way to employ them to the patients' best advantage, I have ventured to repeat in spite of the universal familiarity with the methods. Perhaps we may reach that happy day when, especially with improved tonometric methods, increased skill in measuring the rate of filtration and better instruments for determining the light sense, we can anticipate the advent of glaucoma and get ahead of the ocular and visual deterioration which increased tension produces, by performing preventive operations which shall aid nature's filtration channels in the establishment of an artificial one. But increased tension is not the whole story of glaucoma, and a filtering cicatrix is not the last word in surgical therapeutics, and there is much to learn.

[Pg 71]

[Pg 72]

2. *Reduction of tension by means of various mechanical measures, notably massage, and by means of electricity and diathermy.* Massage is of ancient lineage. In general terms, in so far as ocular massage is concerned, it may be applied to the eye with the finger tips (ordinary massage), by means of various instruments (vibration massage), and with the help of certain

suction cups (suction massage, which is indeed a form of vibratory massage). Many authors are satisfied with their results without the employment of any instrument, and prefer simple massage with the tip of the finger to any form of the instrumental variety, to quote the words of Casey Wood. At one time in my career I experimented very extensively with massage, not alone for the purpose of reducing intra-ocular tension, but in various diseases of the lid and cornea, and taught a trained nurse, who herself had a nebulous cornea, to make what I may call a specialty of this particular therapeutic procedure. She became exceedingly skillful and was quite faithful. We believed that the best results were obtained in a seance of two or three minutes, the finger tip being used over the lid, and the surface of the cornea lubricated with a drop of pure olive oil, although in glaucoma the addition of the oil is not necessary. Four movements were utilized, the first a stroking movement in lines radiating from the central pressure, very much as the spokes of a wheel radiate from the hub, second a circular movement, third a pressure movement, a little dipping motion, so that the cornea was slightly depressed, and finally, a gentle tapping movement, precisely the same, except that it was a diminutive one, as the tapping movement that the Swedish masseur makes. Usually each movement occupied from a half to one minute, according to the results desired. I agree with Casey Wood that such a technic furnishes just as good results as any one with the aid of an instrument.

[Pg 73]

[Pg 74]

Referring particularly to the reduction of intra-ocular tension, many surgeons have been impressed with the value of various instruments. Thus, Ohm, who has worked particularly in the reduction of the increased tension of secondary glaucoma, for example, after discussion of lamellar cataract, advocates the Piesbergen instrument, which makes 3,000 vibrations a minute, and is applied over the closed lids. I think the instrument best known is the one introduced by Malakow. For this purpose the point of an Edison electric pen is armed with a small ivory ball, and the vibration rate varies from 200 to several thousand a minute, the rapidly revolving ball being passed over the closed lids, in some instances directly upon the cornea itself. I am frankly afraid of these vibrating machines, and again make a plea for the finger tip, just as I am afraid of a Von Hippel trephine, and prefer one which is rotated with the fingers.

[Pg 75]

A special investigation of pressure massage according to the method of Domec has been made by Paul Knapp of Basel. This, as you know, consists in applying the thumb to the cornea through the closed lids, and making repeated pressures upon it at the rate of 60 to 100 a minute. He checked his results with the tonometer after 200, 500 and 1,000 pressures, and found that even in normal eyeballs such massage was followed by a fall of intra-ocular tension, the average being nearly 9 mm. after a thousand pressures. Within three-quarters of an hour the tension returns to the normal. In acute glaucoma such massage is not available, but it is of assistance in encouraging a reduction of the intra-ocular tension and keeping it at a normal grade after operative work, particularly after a filtering cicatrix has been made, as was well shown by Weeks in his study of glaucomatous eyes operated upon by the Lagrange method. It is interesting to remember that Paul Knapp, in the course of this investigation, observed reduction of the tension after the use of holocain.

[Pg 76]

Another method of reducing the intra-ocular tension is by the suction method, which consists in the use of certain cups from which the air is exhausted by means of a suction apparatus. Domec uses an elliptical eye cup, the concave margins of which fit closely about the globe. The air is exhausted with each respiration of the patient and from 50 to 200 tractions are made at each sitting. Domec is of the opinion that this method succeeds in two ways, namely, in producing analgesia by traction on the ciliary nerves, and in reducing intra-ocular tension.

Unfortunately, it is difficult for regular physicians to make reference to massage of the eyeball lest their words should be misquoted by irregular practitioners who employ this method, selling various instruments to trusting patients, and attributing to this simple and often beneficial procedure all sorts of marvelous influences. Doubtless all of us have seen eyes utterly ruined because the patient has trusted to the advertisements of these people, and has continued to use some foolish little suction pump, when what his eye needed was operative procedure or skilled therapeutics.

[Pg 77]

If I should sum up my opinion of massage in the reduction of intra-ocular tension, I would say that it is useful in enhancing the action of myotics, and particularly useful, as Domec, Knapp, Ohm, Weeks and many others have shown, after the filtering angle has been opened by a proper operative procedure. It seems to me that it is distinctly our duty to inform patients that it is no panacea, and that they must never trust themselves in the hands of irregular practitioners who pretend to cure all ocular ills with massage.

[Pg 78]

*Electricity.* The credit of first using high frequency currents in the treatment of glaucoma belongs to Truc, Imbert and Marques, and Roure's experiments indicate that this current suitably applied appears to have an influence not only in reducing the arterial tension, but also the ocular tension. Thus, in an interesting series of experiments he has been able to reduce an arterial pressure of 200 mm. to 140 mm., and an ocular tension of plus 2 to the normal after eighteen applications of the high frequency current. The current is applied for ten to fifteen minutes at a time twice a week. Some surgeons, for example, Würdemann, have suggested the use of electricity combined with massage, and have apparently achieved satisfactory results.

The constant current has also been much employed for the purpose of reducing intra-ocular tension. Coleman quotes Le Prince's observations, who applies the negative pole to the eye and the positive pole to the neck, gradually passing a current of 30 to 40 ma. during a quarter of an hour, and who reports notable diminution of tension. Coleman points out that in his own

[Pg 79]

experience he has not found any patient who would willingly tolerate more than 19 ma. of current with an ordinary sized electrode, although he grants that it is possible that Le Prince used a very large electrode. Unfortunately he does not mention its size. Ziegler of my own city, who has studied most scientifically and intelligently the use of electricity in diseases of the eye, announces this rule: The positive pole should be used in all inflammatory processes of the eye, glaucoma excepted, and with this rule Coleman agrees. Now, although the negative pole is a stimulant and therefore not generally indicated in inflammation, as Coleman points out, the object in view is to diminish the density of the ocular capsule and its tension, hence the negative rather than the positive pole should be used, inasmuch as the former, according to him, while it is a sedative, hardens tissue and would tend to increase intra-ocular tension by diminishing excretion. Moreover, in chronic glaucoma the ordinary inflammatory processes are not present, indeed, primary acute glaucoma itself is not an inflammation.

[Pg 80]

I have no personal experience in the use of the constant current with negative pole application to the eye in the reduction of increased intra-ocular tension, but quote for our general benefit the opinions of those who have employed it. I have always been very frankly pessimistic in regard to the therapeutic value of electricity in ocular disorders. Perhaps I am wrong; I am willing to be enlightened. There seems little doubt that Truc and Imbert's observations that high frequency currents can temporarily reduce intra-ocular tension is correct, that they are able to relieve the pain of primary and of secondary glaucoma would seem to be proved by many observations, some of which I have myself made, and other very accurate and excellent ones have been made by Risley in Philadelphia.

[Pg 81]

A word might be said in regard to *diathermy*. According to Zahn, the method of applying diathermy to the human eye is to take a layer of cotton wool 1 cm. thick soaked in a 2 per cent solution of sodium chlorid, which is applied close to the outside of the lids. On this is put an electrode 15 cm. in size with a large indifferent electrode applied to the back of the neck. It is not germane to the subject to name the various ocular diseases which were treated in this manner, but Clausnizer has made an investigation of the influence of diathermy on intra-ocular tension. In a number of diseases, for example, iridocyclitis, the method produced distinct rise of pressure. In one, a patient with secondary glaucoma, prior to the diathermic application the tension was 37½ mm., after the passage of the current it had fallen to 28 mm., but the next morning the tension rose to 45 mm. In a patient with chronic glaucoma no definite alteration of tension could be found. This observation is mentioned, not because it puts us in possession of a valuable therapeutic measure, but largely because it is a good example of how in this disease it is wise to investigate any method which furnishes a hope of relief.

[Pg 82]

In a few instances endeavor has been made to reduce the intra-ocular tension, or at least to relieve glaucomatous symptoms, by galvanism of the cervical sympathetic, for example, by placing one electrode along the whole length of this nerve in the neck and one on the back of the neck on the opposite side, 15 to 20 ma. of current being used. Good results have been reported by an observer named Allard. I confess that I am entirely faithless in regard to any results that may be reached in this manner. It is possible that as the positive pole is a sedative, if there were any influence, the influence of sedation would be present, but certainly it has over and over again been experimentally proved that irritation of the cervical sympathetic quite rapidly produces elevation of intra-ocular tension of 2 to 4 mm. In some experimental work the primary elevation of intra-ocular tension was followed by a secondary drop.

[Pg 83]

3. *Indirect reduction of increased intra-ocular tension brought about by lowering general vascular pressure.* Much has been written in regard to the association between increased vascular pressure and increased intra-ocular pressure. It is not my province to analyze observations often contradictory and not infrequently inaccurate. This much seems to be established: First, that at corresponding ages there is usually a higher average blood pressure in glaucomatous subjects than there is in non-glaucomatous subjects; second, that arteriosclerosis and therefore usually increased blood pressure, with all its concomitant conditions, is correctly classified as an exciting cause of glaucoma; and third, that the regulation of this increased blood pressure is part of the advantageous management of increased intra-ocular pressure, although it may be too much to say, as Gilbert has, that blood pressure and intra-ocular pressure rise and fall together. It may be true, as Thomas Henderson says, that the intra-ocular pressure is influenced by changes in the general arterial or general venous pressures, whereby a rise in general arterial pressure induces a proportionate rise in the intra-ocular pressure, but it would seem that future investigations must confirm this statement before it can be entirely accepted, as well as his further statement that the effect of an increased general venous pressure is a direct one, producing millimeter for millimeter a corresponding increase in the intra-ocular pressure.

[Pg 84]

Now, it goes without saying, if these data are correct, or even only partly correct, that part of the treatment of the increased intra-ocular pressure state must be constitutional in that the vascular pressures should be lowered in order that the beneficial effect of their relationship to the intra-ocular pressure shall be established. It is further a great mistake to drive down a high arterial pressure simply because that exists. In other words, it is often necessary from the general standpoint that a certain amount of plus pressure shall remain if the patient's general well-being is to be maintained. There must always be a differential diagnosis between plus pressure and what may be called over plus pressure. That is to say, a man may be perfectly comfortable and properly need, for example, a pressure of 160 or 165 mm., which is above the physiologic limit, but which is a plus pressure, while some disturbance in his general life may add to that 10, 15 or 20 mm. more of pressure, which is then the over plus amount. This over plus amount may be in

[Pg 85]

[Pg 86]



association with a rise of intra-ocular pressure, and must be eliminated if the latter is to be controlled by a non-operative procedure, or, indeed, by an operative one.

It is no easy matter to determine the presence of increased venous pressure, although there are tolerably accurate instrumental technics, and yet, as Henderson points out, it is just this increased general venous pressure which is often detrimental. Therefore the perfunctory use of such drugs as nitrite of amyl and the other nitrites may not be in the least indicated when, for example, the venous pressure depends upon inability of the right heart to perform its functions, and the drug needed may, for example, be digitalis. Far better than pressure-reducing drugs like nitrite of amyl, urgently indicated in some instances and for some purposes, is the regulation of life and the restoration to their normality of the metabolic processes, the elimination of the worry which is usually the exciting agent that brings about the over plus pressure, which may have as one of its expressions an acute rise of intra-ocular tension. I believe that in the management of a case of glaucoma, whether it be chronic or chronic with sub-acute exacerbations, the greatest care with the aid of an expert clinician must be exercised to find out exactly what mean pressure of the arterial and venous system best conforms with the patient's general welfare, and I am bitterly opposed, and I think with right, to the sudden reduction of tensions, except in emergencies, without a perfect understanding of the facts I have ventured to indicate. This does not for a moment mean that prior, for example, to operative work it is not necessary to get rid by means of drugs of an over plus tension, for surely the elimination of such an over plus tension may be the means of preventing, for example, an intra-ocular hemorrhage, and in this emergency we must not lose sight of Gilbert's recent investigation, who has found that blood withdrawn to the extent of 8 grams to each kilogram of the body weight always produces lowering of the intra-ocular tension, appearing in six to eight hours and lasting to the next day in simple glaucoma, and in inflammatory glaucoma commencing the day after the venesection and lasting two to three days. It is not necessary for me to point out the value of free purgation and diaphoresis in this respect.

[Pg 87]

[Pg 88]

In most instances the successful maintenance of a glaucomatous life, exclusive of operative interference, in addition to sustained myosis, demands the investigation of the patient's metabolism, which must be kept at the normal standard, the removal of the evil effects of auto-infection, as we are wont to call it, and especially the elimination of the cause which is responsible for the over plus tension of the arteries and of the veins. This is best secured by just such regulation of life as has been referred to, aided when necessary by the ordinary drugs which the patient's condition indicate, and the success of all treatments, be they operative or non-operative, is enhanced if such a happy state of affairs can be brought about.

[Pg 89]

I am firmly convinced that every glaucomatous patient, and I now refer to those who are the subjects of chronic progressive glaucoma, should be carefully studied from the general standpoint by the oculist with the aid of an expert internist, just as I am convinced that the modern expert internist should not study his cases of cardio-vascular disease without the help of the oculist. Perhaps I am going a little far afield, but in justification of my statement I want to quote the opinion of Dr. Hobart Hare, one of America's most expert clinicians, on blood pressure, because it seems to me much harm has been done by the more or less brutal knocking down of blood pressure simply because blood pressure above the normal existed. "Concerning the matter of high blood pressure," writes Hare, "independent of cerebral lesions, the longer I study the matter the more convinced I am that this blood pressure is devised by nature to compensate for fibroid changes in peripheral vessels, in order that tissues which would otherwise be cut off from adequate blood supply may receive plenty of blood, and I consider it one of the most vital points to ascertain whether a pressure is what may be called the patient's pathological norm, that is, the pressure which is required in the face of vascular changes, or whether this pressure is in excess of his pathological norm. If it is in excess, measures directed to bring it to the pathological norm should be instituted, but if the pressure found proves to be the pathological norm it is a bitter mistake to lower it, be the pressure what it may. If it is lowered below the pathological norm, all manner of disturbed cardiac action, etc., may result. There is no more reason for reducing a blood pressure below his pathological norm than there is for reducing it below his physiological norm. The adjustment of a man's blood pressure to his pathological norm often has to be as correctly done as the adjustment of a watch which is losing or gaining time."

[Pg 90]

[Pg 91]

I shall not quote Hare's elaborate methods for determining these various points because they do not belong to a paper of this character, but I quote his admirable advice because it emphasizes what I believe to be an essential in the treatment of chronic glaucoma, exclusive of operative work, that is, the intelligent co-operation of the oculist and the internist.

Some such thought was in the mind of Ibershoff, who quotes Sterling and Henderson's views that the rate of secretion depends upon and varies with the difference in the blood pressure and the tension of the eyeball, and that the specific gravity of the secretion increases directly with the blood pressure and inversely with the ocular tension. Should the blood pressure be very high, paracentesis, for example, would apparently not be the proper procedure, and the resulting difference produced between the blood pressure and the eye tension would cause a rapid reformation of fluid with higher specific gravity and higher osmotic coefficient. The proper procedure in these circumstances is first properly to reduce the blood pressure, or what I have, quoting Hare, ventured to call the over plus pressure.

[Pg 92]

4. *The relation of osmosis, lymphagogue activity, absorption of edema, capillary contractility and decreased affinity of ocular colloids for water to the reduction of increased intra-ocular tension.*  
We are all familiar with the attention which was directed some years ago to the statements

coming from French clinics that the treatment of glaucoma should include the administration of osmotic substances as adjuvants in the reduction of increased intra-ocular tension. Particularly was this treatment advocated by Cantonnet in the administration of daily doses of 3 grams of chlorid of sodium, preceded, of course, by a careful urinary examination and the estimation of the amount of urine and its contained chlorids. Carefully this dose was increased in proper circumstances to 15 grams per diem, and in Cantonnet's original paper good results were achieved in 12 of the 17 patients so treated. I have myself experimented somewhat, not with the administration of sodium chlorid by the mouth, but with the introduction by the bowel of fairly large quantities of physiologic salt solution in patients with glaucoma whose quantity of urinary secretion was markedly below the normal, and in one or two startling instances, which have been reported, achieved success in the rapid reduction of the intra-ocular tension when by this technic the urine secretion rose to the normal amount. To be sure, myotics were also used, but these myotics were insufficient, totally so in the two instances noted prior to the enteroclysis. [Pg 93]

Very interesting are the observations on the subconjunctival injections of various substances, notably the citrate of sodium, because of its power of decreasing the affinity of ocular colloids for water. This method of treating increased intra-ocular tension, introduced, as you know, by Thomas and Fischer, has met with confirmation from a number of sources in spite of the fact that Happe's experimental study failed to confirm Fischer's observations; indeed, he even reports in several instances a rise of tension. [Pg 94]

As you will remember, the strength of ordinary crystallized sodium citrate in water should be from 4.05 to 5.41 per cent. Of this five to fifteen minims are injected, the eye having been previously cocainized and adrenalinized. With frequent injections the weaker of the two solutions is mixed with 2 to 4 parts of physiologic salt solution. These authors in no sense claim to cure glaucoma, but to ameliorate it and reduce the tension. Weekers has used the salts of calcium, 3 grams a day, with success in so far as lowering of tension is concerned, although it must be stated, as a reviewer of his work has said, that his recommendation of this drug in these respects is poorly supported. On the other hand, Tristiano seems to have proved that calcium chlorid is capable of lowering ocular tension and clinically may be used as an adjuvant in the treatment of glaucoma for this purpose, largely because he believes that he has proven that it facilitates the absorption of edema. Darier has reported that a single subconjunctival injection of a milligram of iodate of sodium has cleared the cornea and lessened the intra-ocular pain in glaucoma. [Pg 95]

What shall be said in regard to certain medicinal agents which stimulate the lymphagogue activity of the eyeball in their relation to the reduction of intra-ocular tension, notably of dionin? Toczyski's experiments with this drug on the normal eye indicate that it produces first a rise of tension, which shortly falls to the normal and sometimes below it, the tension being high as long as the primary narrowing of the pupil is maintained, but more than one author, particularly A. Senn, holds an opposite view and reports acute glaucoma following its instillation into a chronic glaucomatous eye. He believes that dionin not only does not reduce the tension but hinders the filtration through the anterior lymph channels by the pressure of the edema which is produced on the veins and by the increased secretion of the ciliary processes. In spite of this statement, most of us must agree with Karl Grossman's observations that certainly in acute and particularly in chronic secondary glaucoma, this is a most valuable agent, especially if it is combined with holocain, which Paul Knapp in his well-known research has proved can reduce the tension even of the normal eye. I cannot think that anybody who has systematically used dionin with holocain, the former in gradually increasing strength, beginning with 2 per cent and going up to 8 per cent, in various types of acute glaucoma, particularly of the secondary variety, can fail to have noted a favorable influence. [Pg 96]

Many authors, for example, Darier, Grandclement and others, are strong in their recommendation of adrenalin, particularly if this drug is added to the various myotic mixtures, and yet adrenalin is certainly not without danger in the treatment of glaucoma. McCallan has seen a number of instances of striking increase of intra-ocular tension following this instillation in the conjunctival sac. Harmon has had a similar experience, as also has Senn. It is possible that in these circumstances the solution was too strong. Should the rise of tension occur, and I have seen it myself, it is doubtless due to the fact that this drug dilates the pupil, which would be especially dangerous if the dilatation should occur before contraction of the ciliary vessels; also the narrowing of the ciliary veins by the adrenalin might by virtue of this narrowing obstruct the gate of outflow. I have never been able to persuade myself that, except as an adjuvant to operative work, there was any real therapeutic value in the instillation of adrenalin. [Pg 97]

A word in regard to the effect of general narcosis on intra-ocular tension. Thus, Neuschuler has observed that narcosis causes an elevation of the intra-ocular tension of from 2 to 6 degrees as measured with Fick's tonometer. These observations were made while he was experimenting on irritation of the sympathetic as a method of producing increased intra-ocular tension. This is not in accord with Axenfeld's recent observations. It is well known, this observer points out, that after the period of excitation and muscular rigidity disappears, there is a lowering of blood pressure in chloroform narcosis and coincidentally a sinking of the intra-ocular pressure. Not only this, the intra-ocular tension of normal eyes during this narcosis drops several millimeters. Only such eyes as have high hypertony, for example, in absolute glaucoma, are unaffected during chloroform narcosis. In the light of this observation it will be interesting to measure the tension both of normal and glaucomatous eyes during narcosis in a large series of cases, and if it is confirmed there will be an additional reason why in many circumstances general narcosis is advantageous in glaucomatous patients. Formerly I thought it was essential, if iridectomy was to [Pg 98]

[Pg 99]

be performed, lest some sudden movement on the part of the patient might bring the point of the knife in contact with the lens. I have rarely employed it in corneo-scleral trephining, and yet if there is this temporary reduction of intra-ocular pressure, it is not without a certain therapeutic value, and the matter is mentioned as a suggestion that additional observations along this line shall be made.

[Pg 100]

---

## **Dr. George Edmund de Schweinitz' Paper on Concerning Non-Surgical Measures for the Reduction of Increased Intra-ocular Tension**

[Pg 101]

**Discussion,  
NELSON MILES BLACK, M.D.,  
Milwaukee.**

It seems almost useless to attempt any discussion of Dr. de Schweinitz' most terse and comprehensive paper. However, Dr. de Schweinitz mentioned the close relationship which should exist in the non-surgical treatment of increased intra-ocular tension between the internist and the ophthalmologist, but neglected to mention a corresponding relation which should exist between the rhinologist and the ophthalmologist, and possibly between the dental surgeon and the ophthalmologist.

I would like to refer to the *now* recognized close relationship which exists between disease of the nasal accessory sinuses and diseases of the eye. The definition of glaucoma found in Dr. Wood's system of therapeutics gives rise to an hypothesis as to why disease of the nasal accessory sinuses may be a factor in producing increased intra-ocular tension and why treatment directed toward obtaining free drainage from the sinuses gives good results in so many cases, especially if the relationship is recognized sufficiently early. "Glaucoma proper is essentially a damming or blocking of the drainage from the interior of the eye. The chief lymph stream flows from the posterior chamber past the margin of the lens, through the zonula of Zinn, beneath the iris, through the pupil into the anterior chamber, thence through the tissue at the junction of the iris and sclera into the circular canal of Schlemm and from this space into *the external lymph channels. Obstruction to the steady escape of the intra-ocular fluids at any point in this drainage system or any undue increase of the fluids themselves may produce glaucoma.* Probably the most important obstruction to the exosmosis is at the angle close to Schlemm's canal."

[Pg 102]

[Pg 103]

The following hypothesis is based upon Fischer's edema theory of glaucoma and the relation of the circulation of the eye and orbit and that of the nose and the accessory sinuses, the minute anatomy of which is not as yet thoroughly understood. However, sufficient work has been done to make it appear that the lymph channels which drain the eyes and orbits empty into the same main channel as do those which drain the sinuses. Admitted for sake of argument that such is the case, then disease either acute or chronic of one or more of the sinuses with the accompanying inflammatory reaction, congestion and stasis, will cause an increased amount of fluid to be taken care of by the lymph channels draining these sinuses. This will in turn cause flooding of the common lymph channel, producing a stagnation in the flow of fluid from the orbits and eyes at the junction with the main channel, with backing up of the fluid within these channels and retention of the waste products within the orbits and eyes; thus will be brought about conditions most favorable (to quote from Fischer's theory of glaucoma) "to an abnormal production or accumulation of acid in the eye. In consequence of this abnormal acid content the hydration capacity of the ocular colloids is raised and glaucoma results, not because water is pushed into the ocular colloids, but because these suffer changes which make them suck in water from any available source."

[Pg 104]

This hypothesis also might suggest why the subconjunctival injection of sodium citrate in addition to alkalizing the ocular contents, may be effective in reducing tension, *i. e.*, the amount of fluid injected beneath conjunctiva may overcome the stagnation in the lymph passages, flush out these channels and improve ocular elimination.

Fischer in a personal letter says:

[Pg 105]

"You have two possibilities for the production of glaucoma with sinus disease: A toxic factor due to poisons being carried into the eye; and second, interference with a proper blood supply to the eye through compression of the efferent or afferent blood vessels supplying the eye from edema of the tissues about the eye consequent upon the sinus infection. Either is associated with the production of substances which increase the hydration capacity of the ocular colloids."

If such is the case why could not the existence of pyorrhea and blind abscesses about the roots of the teeth be the source of the toxic factors mentioned by Fischer? Hence the suggested association of the dental surgeon with the ophthalmologist in these cases of apparently idiopathic increased intra-ocular tension.

It would be well to state here a cursory examination of the mouth will not discover root abscesses any more than such examination will discover non-suppurative sinus disease. A careful

[Pg 106]

examination of each tooth together with radiograms of the entire maxilla are absolutely essential to determine their presence or absence.

---

## Trephining for Glaucoma

[Pg 107]

BY

**ROBERT HENRY ELLIOT, M.D.,  
London, England.**

Mr. President and Members of The Chicago Ophthalmological Society:

As the hour is late I propose to take up only the principal points in connection with my subject and to deal with each one shortly.

First: The operation of trephining is suitable, not merely for chronic cases, but for sub-acute and acute cases of glaucoma as well. I would urge on your attention that, of all the operations dealing with glaucoma, this one involves the minimum of surgical violence, and should, therefore, in acute cases be the operation of choice. It is, moreover, much safer than any other operation I know of, and is no less certain in its results. I do not advise trephining in the secondary glaucoma following intumescent cataract, for in such cases the semi-fluid lens bulges into and blocks the trephine hole. Nor for obvious reasons do I recommend it in cases where there is reason to believe that a communication exists between the aqueous and vitreous chambers.

[Pg 108]

Second: The object of trephining is to tap and permanently drain the aqueous fluid from the anterior chamber of the eye into the sub-conjunctival space; in doing so it is essential to avoid as far as possible all interference with the uveal tissue. The purpose of an iridectomy is to avoid the danger of the iris in the neighborhood of the wound being drawn and impacted in the trephined hole. We have found in a large number of cases in which an iridectomy has been omitted, that the results have been in no way inferior to those in which a piece of iris has been removed, provided always that no subsequent iris prolapse takes place. In pursuance of our purpose to avoid uveal tissue, we split the cornea, and place the trephine as far forward as such splitting will allow, and we bear on the trephine in such a way that it cuts through on the corneal edge of the wound first. This insures establishing our fistula in the most anterior position possible, and, therefore, as far away as possible from the ciliary body and the angle of the chamber.

[Pg 109]

Third: The difficulties of the operation. Far too much stress has been laid on these. Trephining is an operation which can be performed by any surgeon who is used to ophthalmic manipulations, and who has good sight. It is essential that he should work in a good light. The necessary technique can be acquired from a written description. It is not for a moment necessary that the surgeon who wishes to learn trephining should see the originator of the operation at work. If, however, he feels diffident at undertaking the procedure until he has seen it done by another, there are many centers in this country where the operation is now being successfully performed. I would mention amongst those which I have visited New York, Minneapolis, St. Louis, Nashville, Louisville, Detroit and Chicago. I have seen results of trephining by American surgeons which could not be bettered anywhere.

[Pg 110]

Fourth: I am sure that everybody will recognize the difficulties of operating during such a tour as I am now making. I have so far in the last month performed over seventy trephinings in ten cities, and in twice as many clinics. To adapt one's self to different clinical methods, different assistants and different nurses is so difficult that, as you are aware, many distinguished surgeons refuse to work out of their own clinics. One cannot expect the results of such a tour to be on a par with those one obtains in one's own quiet daily surroundings. I am, however, confident that you will make a generous allowance for these difficulties, and I gladly welcome the suggestion that all the cases which I have operated on in America be collected together and reviewed as a whole.

[Pg 111]

Fifth: In conclusion I would like to express the pleasure with which I listened to Dr. de Schweinitz' paper. I believed from the title that there might be a wide divergence of opinion between us. I find to my great relief that we are in absolute accord. I know, however, that there are in America and elsewhere able men who consider that the medical treatment of glaucoma should be pushed as long as possible. I cannot but feel that this is a survival of the dread that most surgeons have felt in recommending one of the older operations for glaucoma. We have now in our hands a method so safe, so easy and so certain that I feel sure that this dread will ere long pass away, and that the diagnosis of glaucoma will then be followed by a very early operation. In India I have gone farther than this, and where one eye has shown high tension, I have frequently trephined both. The prophylactic use of the operation is more than justified in that land of long distances and scattered medical aid, and where the patient is not likely to return a second time for surgical help. This prophylactic trephining is a proposition that I put before you today for your consideration, reminding you at the same time that glaucoma is practically invariably a bi-lateral condition. I have seen even in America not a few people blind in both eyes who might have retained the sight of the second eye had the surgeon advised a double sclerectomy when he first saw the case, despite the fact that the second eye was then to all appearances non-glaucomatous.

[Pg 112]

# Dr. Robert Henry Elliot's Paper on Trephining for Glaucoma

[Pg 113]

## Discussion, FRANK C. TODD, M.D., Minneapolis.

It is very difficult for one of limited experience to discuss a subject presented so ably by Lieutenant Colonel Elliot to whom we are indebted for the sclero-corneal trephine operation. He has already over a period of a little over four years performed over 900 trephinings, and has made a most careful subsequent study of the results of those operations on as many cases as he had the opportunity to observe.

Anyone who has read Colonel Elliot's book on the sclera-corneal trephining operation will be struck with the fact that he has not only had a tremendous experience in ophthalmic surgery, but that he has made the best of that unusual opportunity, and that to a foundation of a careful training he has added the experience of twenty-two years of hard painstaking work.

[Pg 114]

I have recently had the privilege of entertaining Colonel Elliot in my own city, where I had the opportunity of assisting him and hence closely observing his technique in eighteen trephinings. It has since been my duty, and responsibility I may add, to care for those eighteen eyes. For two years I have been doing the Herbert tongue flap, or a similar operation. The results have been highly satisfactory thus far and similar to those following the trephining operation, which operation I have performed in a number of cases during the past ten months. My conclusions as to these two operations are favorable to the trephining operation because the Herbert tongue flap operation is much more difficult, and hence less certain than the Elliot trephining operation.

The time for discussion does not permit a detailed statement of the results nor experiences in the handling of these trephining cases. Of the entire number five totally blind eyes were trephined. Tension was reduced in all but one. In that one hemorrhage occurred at the time of the operation. One of these blind eyes had not been totally blind longer than a few weeks. Hand movement vision developed in this eye. Another eye totally blind one year has thus far developed perception of light. Of the cases with varying degrees of vision from hand movements to six-ninths all but one have either remained the same or shown some improvement. The one exception was an eye having six-ninths vision. A small button hole iridectomy was made; prolapse of the iris into the wound occurred four days later requiring incision. Upon incision of the prolapse intra-ocular hemorrhage occurred, causing nearly total blindness for two weeks. Vision is clearing fast and it remains yet to be seen what the final results may be. One buphthalmic eye trephined by myself gave good results.

[Pg 115]

I have as yet seen no cases of remote infection, but the report of Axenfeld and some others would indicate that this occurred following the Lagrange as well as the trephining operation, the then bulging conjunctiva having become eroded and infection having taken place through the eroded conjunctiva as shown when stained with flourescin.

[Pg 116]

The opinion, not yet conclusive, that I have thus far formed as a consequence of my experience and the information obtained from others of greater experience is as follows:

First: That in those cases of chronic glaucoma in which iridectomy has been of benefit in preventing or retarding the oncoming of blindness, the result has apparently been secured by reason of the fact that filtration has been produced, and not merely because a piece of iris has been removed.

Second: That in chronic glaucoma (in acute glaucoma iridectomy has proven a satisfactory operation) when the progress of the disease cannot be arrested by non-surgical treatment (an even in some of these, where, for instance the patient cannot be kept under observation or will not carry out the treatment) some form of operation intended to produce filtration should be performed.

[Pg 117]

Third: The Elliot sclero-corneal trephining operation carefully performed in accordance with the author's technique in the light of our present knowledge seems to be the best and safest operation to produce that result.

Fourth: That to glaucoma may be added buphthalmos and staphyloma, as diseases often capable of relief by trephining and indeed toward the relief of which trephining is the best form of operation yet presented.

Fifth: That the results secured when the operation is well done and the after care is properly followed out are satisfactory, in that the operation in a large proportion of cases apparently permanently lowers the tension to normal or below normal, relieves pain, prevents the oncoming blindness (otherwise inevitable) and in many cases causes an improvement in the acuity of vision, in the visual field. And in occasional cases of blindness of not too long duration, it restores some vision, occasionally to a marked degree.

[Pg 118]

Sixth: That it is not a simple nor easy operation and should, therefore, be performed only by an

operator well trained in ophthalmic surgery. The careful and skillful technique of the originator of the operation perhaps accounts for his greater success in its results and those who perform the operation should follow his technique and be capable of handling complications that may later arise.

In conclusion, Mr. President, I wish to say that we ophthalmologists the world over are indebted to Lieutenant Colonel Elliot not alone for his contributions to our knowledge, but for his persistence against precedent and criticism in establishing the facts upon which rest the foundation for the success of his operation, and for so emphasizing the great importance of this epoch-making achievement.

[Pg 119]

It is because we respect his wisdom gained by incessant study and experience in a country where climatic conditions are such that a man of ordinary energy would have failed to do even average work that we so readily welcome the teaching of this enthusiastic evangelist.

His pilgrimage to our country will be the means of starting many in this new field, and we shall soon be able to draw more definite and final conclusions from our own experiences.

---

## Operations Other than Scleral Trephining for the Relief of Glaucoma

[Pg 121]

BY

CASEY A. WOOD, M.D.,  
Chicago.

In this paper I shall say a few words about the large number of operative procedures that, apart from trephining, or, preferably, *trepanation*, have been urged in the treatment of the various forms of glaucoma. Their name is legion and among them we find peripheral iridectomy; anterior sclerotomy; irido-sclerotomy; scleriritomy; de Wecker's dialysis of the iris; Hancock's division of the ciliary muscle; the incision of the iridian angle of de Vincentiis; sclero-cyclo-iridic puncture; the Sterns-Semmereole *sclerotomy antero-posterior*; the *transfixio iridis* of Fuchs; Antonelli's peripheral iridectomy; Holth's formation of a cystoid cicatrix; Hern's operation; Terson's sclero-iridectomy; Abadie's ciliarotomy; Ballantyne's incarceration of iris method; Masselon's small equatorial sclerotomy; Simi's equatorial sclerotomy; Galezowski's sclero-choriotomy; excision of the cervical ganglion; removal of the ciliary ganglion; Querenghi's operation of sclero-choriotomy; Bettremieux's simple anterior sclerectomy; Heine's cyclodialysis; Herbert's wedge-isolation operation; Verhoeff's operation with a special sclerotome; Holth's sclerectomy with a punch-forceps; Walker's hyposcleral cyclotomy; posterior sclerotomy; T-shaped sclerotomy; and last but not least the Lagrange form of sclerectomy with its various modifications by Brooksbank James, myself and others.

[Pg 122]

In addition to the foregoing list—which is by no means complete—there are several combinations of operations, as, for example, the Fergus trephining operation, which is really a combination of a sclero-corneal trepanation and a cyclodialysis.

[Pg 123]

So far as it is practicable there is a certain amount of wisdom in comparing the results of an operative procedure with others with which it is brought in competition, and I believe we are even now in a position to form at least some idea of the comparative value of the three methods that comprise the great majority of interventions made use of by ophthalmic surgeons at the present time. I refer to *iridectomy*, the *Lagrange operation*, and the *Elliot operation*. So far as regards the last named procedure, I congratulate this Society that it has had an opportunity of seeing a demonstration and hearing a discussion by the famous ophthalmic surgeon who perfected it.

As regards the others let me recommend to you the complete description of them given by Posey in *A System of Ophthalmic Operations*.

Let us consider the first of the three procedures just mentioned—*iridectomy*—introduced by von Graefe. The mechanism of its mode of cure is best studied in cases of acute primary glaucoma, when there is apposition of the periphery of the iris to the cornea. In these acute cases there is probably only a mere *apposition*, and the blocking up of the sclero-iridian angle is largely mechanical. Here the root of the iris is readily removed in its entirety and a really peripheral iridectomy is easily done. When, however, a true *adhesion* between corneal and iridic tissue takes place the filtration angle is not so easily opened. True peripheral adhesions are not readily broken up or separated, and the iridectomy is, for that reason at least, not effective. Moreover, this form of anterior synechia (resulting from a true union of iris and cornea) is so intimate that the iris root is, by the iridectomy, torn away only at the sclero-iridian angle at the anterior border of the adhesion—and does not open up a channel into Schlemm's canal. It is not, therefore, difficult to understand why iridectomy alone in any of the forms of chronic glaucoma fails to open up the true filtration spaces and does not provide a drain that permits of an escape of fluid from the posterior chamber through the loose tissue that surrounds it into the canal of Schlemm. Treacher Collins found, after a careful examination of eyes upon which iridectomy had been

[Pg 124]

[Pg 125]

performed for glaucoma, that it is extremely rare for the initial section to pass through the pectinate ligament, while Schlemm's canal invariably escapes. Moreover, since the sclero-corneal incision is uniformly oblique, the position and extent of the external wound does not always furnish evidence of the character of the internal wound. In all likelihood many cases of relief or cure following iridectomy are those due to the formation of cystoid scars or minute fistulae, rather than as a result of the removal of a portion of the iris periphery.

The best brief tabulation of the results obtained by iridectomy, in glaucoma, is to be found in Weeks' textbook on *Diseases of the Eye*, page 417: "Sulzer reports as follows: Acute glaucoma, 149 cases; improved, 72.5 per cent; serviceable vision preserved, 11.3 per cent; vision impaired at once, 4.08 per cent; very little vision, 12.12 per cent. [Pg 126]

"Zentmeyer and Posey: In simple glaucoma central vision increased in 60 per cent; remained the same in 20 per cent; diminished in 20 per cent.

"Wygodski: Inflammatory glaucoma, 37 cases; improvement, 76 per cent; unimproved, 5 per cent; deterioration, 19 per cent. Sub-acute (chronic inflammatory), 147 cases; improvement 10 per cent; unimproved (condition the same as before iridectomy), 40 per cent; deterioration, 30 per cent; blindness, 20 per cent. Cases operated on at an early stage gave 85 per cent of good results. Simple glaucoma, 104 cases; improvement, 0.96 per cent; condition as before, 10.5 per cent; deterioration, 52 per cent; amaurosis, 36.5 per cent. [Pg 127]

"Hahnloser and Sidler: One hundred seventy-two eyes observed not less than ten years after operation; acute inflammatory, 31 eyes; good results, 64 per cent; relatively good, 13 per cent; blind 23 per cent; chronic inflammatory, 37 eyes; good result, 29.9 per cent; relatively good, 27 per cent; blind, 43 per cent; simple glaucoma, 76 eyes; good results, 42 per cent; relatively good, 28.9 per cent; blind, 28.9 per cent."

As far as the *Lagrange procedure* is concerned, you will remember that after eserization an oblique incision is made through the sclera by means of a narrow Graefe knife and a large conjunctival flap secured. This is obtained by making a peripheral section of the sclero-corneal margin with the knife and, as soon as the edge of the knife reaches the upper limit of the anterior chamber, it is turned backward and brought out through the sclera obliquely. The conjunctival flap thus formed is turned back over the cornea, and the fragment of sclera that is left attached to the cornea is removed by means of a fine pair of delicate curved scissors. Following this an iridectomy is performed. The conjunctival flap is now replaced and a bandage applied. [Pg 128]

This operation opens a large filtration passage for the intra-ocular fluids and the prompt healing of the wound with its mucous covering prevents prolapse of the iris.

Under no circumstances must iris be left between the lips of the wound.

Although Lagrange advocated iridectomy in all cases in his first communication, he no longer judges the procedure to be necessary in all instances, reserving it for cases in which for any reason, such as hypertension, prolapse is to be feared.

While Lagrange holds that it is necessary to open the anterior chamber, Bettremieux thinks that a removal of but a portion of the thickness of the sclera suffices. His procedure is as follows: After raising a flap of conjunctiva from the neighborhood of the limbus a medium sized needle, curved and flattened towards its point and firmly grasped in a needle holder, is thrust superficially into the sclera tangentially to the upper edge of the cornea, so as to become fixed in the capsule of the eyeball. A small shaving of the sclera, about ½ mm. thick, 1½ to 2 mm. broad and from 2 to 3 mm. long, is then excised by means of a narrow Graefe knife. The scleral slip is then freed from the conjunctiva at each end and the mucous membrane brought together over the wound by fine catgut sutures. [Pg 129]

As you are well aware, numerous operators regard the Lagrange operation as superior to the iridectomy of von Graefe because they believe there is filtration through the newly formed tissue between the lips of the operative wound. Among those of many observers the conclusions of Ballantyne may be quoted: "The results of sclerectomy vary according to the degree of hypertension of the eye operated on. Three varieties of cicatrix are distinguishable according to the amount of sclera excised: (1) that in which there is mere thinning of the sclera owing to the excised portion not reaching the posterior surface of the cornea (conjunctiva smoothly covers the cicatrix); (2) that represented by a subconjunctival fistulette, due to excision of the whole thickness of the sclera, in an eye with moderate tension (the conjunctiva lies smoothly over the cicatrix); (3) the fistulous cicatrix with an ampulliform elevation of the overlying conjunctiva, resulting from excision of the whole thickness of the sclera in an eye the seat of high tension. In cases of high tension, even a simple sclerectomy will allow ample filtration, owing to the gaping of the wound, while in cases without elevation of the tension, sclerectomy will be quite ineffectual. Lagrange therefore proposes the following rules of procedure: (a) If tensions is normal to +1, do sclerectomy without iridectomy, the amount of sclera excised being inversely proportionate to the degree of hypertension. (b) If tension is +1 to +3, do sclerotomy-iridectomy, the iridectomy being added to avoid entanglement of the iris. Lagrange does not recommend his operation for acute glaucoma. It is especially adapted for cases of chronic simple glaucoma." [Pg 130]

During the past ten years or more I have been doing a modification of the Lagrange operation, the details of which (The Operative Treatment of Glaucoma with Special Reference to the Lagrange Method, *The Canadian Medical Association Journal*, November, 1911) I have elsewhere published. [Pg 131]

As stated in this paper I have modified the procedure to the extent of removing *all* the conjunctiva attached to the borders of the operative wound. I admit that this intervention exposes the root of the iris and the ciliary body, but I have never yet had the slightest infection of the wound. I attribute this freedom from sepsis to careful cleansing of the conjunctival sac and to other pre-operative precautions, but especially to the use, before and after the operation, of White's ointment—a preparation of 1-3000 mercuric chloride in sterile vaseline. One cannot use sublimate in such a strong *watery* solution, but the vaseline seems to modify it and to allow of such slow absorption that it is not only a non-irritant but a most excellent antiseptic application in operations on the eye.

[Pg 132]

In any event the result of the Lagrange operation proper, as well as my modification of it, is to produce a drainage-oedema about the incisional wound which persists almost indefinitely. In many cases this swelling amounts to a bleb which may be increased by massage of or pressure upon the eyeball. The efficacy of the operation in lowering intra-ocular tension is to some extent measured by the degree and the constancy of this epibulbar oedema; indeed, I suspect that the most successful examples are those in which sclera fistulae, minute or otherwise, form as a sequel of the operation.

[Pg 133]

My object in excising the conjunctiva about the sclero-corneal flap, is to delay union of the wound edges, to widen the bridge of loose cicatricial tissue between them, to prevent such a complete growth of the endothelium as would cover the wound and block the exit of fluids, and to insure intra-ocular rest.

In cases of *chronic* increase of intra-ocular tension associated with a quiet uveitis or an iridokeratitis, when the patient exhibits traces of old synechiae, or where there is danger of their re-formation, I do not hesitate to use atropia as long as the wound of operation has not healed.

To the present time I have done 72 operations of the sort and have seen no reason to alter the opinion of it expressed in the article mentioned. Whatever objection may in the future arise—and I freely confess that it *seems* to be fraught with the dangers that many of my colleagues have pointed out as probable—I have so far not seen a single case of infection of the wound of operation. While I believe the anti-glaucomatous results to be excellent, I may also claim that the operation is of the simplest character; and it is easy of performance and the resulting filtration-scar is large and (perhaps) more permeable to the changed intra-ocular fluids than the quicker healing wounds of the usual Lagrange and Elliot procedures.

[Pg 134]

It is regarded by most operators as desirable that there should not be long delayed healing of the operative wound, and the fact that the conjunctiva covers the incision is often spoken of as an advantage, partly because it shields the large open area produced by the Lagrange incision from infection.

My experience of this modified operation continues to be that it is necessary to clear the neighborhood of the operation wound entirely of conjunctiva. If the down-growth of epithelium into the operative wound is permitted the effects are by no means as pronounced, and the eventual lowering of tension is not as permanent as they otherwise would be.

[Pg 135]

Another matter: I am satisfied that the delayed filling of the wound by connective tissue is desirable in most cases of *chronic* glaucoma. A complete drainage of the intra-ocular fluids that results from long delayed union of the wound edges, allows the interior of the eye to regain, as far as possible, the *status quo ante*. On the other hand the comparatively early closure of the wound (or the termination of *free* drainage and minus tension) tends to re-establish the *status glaucamatosus*. Whether these desirable results are to be realized or not will, of course, depend upon a future experience larger than I have yet had. This modification of the Lagrange operation seems to be a radical one and I do not expect its adoption until the results of an extended trial are carefully recorded and reported.

[Pg 136]

Quite recently several operators, who have been in a position to do so, have contrasted the results obtained by the Elliot method and those following the Lagrange procedure. Probably the most important of these observations is the experience of Meller (Die Sklerektomie nach Lagrange und die Trepanation nach Elliot) set forth in a paper read by him at the last meeting of the *Deutsche Naturforscher und Aertze*. In this report Meller gives an account of 389 sclerectomies following the usual Lagrange procedure. Twelve per cent of the cases were of acute glaucoma; 61.5 per cent of chronic inflammatory glaucoma, and 9 per cent of simple glaucoma. The rest of the operations were done in other forms of the disease. In more than half the cases the usual iridectomy was performed; in 30 per cent the procedure was peripheral; in 4 per cent there was no iridectomy. The patients were studied during a period of five years. In more than half the instances there was a pale, cystic, oedematous cicatrix; in 11 per cent the scar was ectatic, and in the remainder the field of operation was quite flat. The form of the scar was described in most instances, but it was not noticed that there was a definite relation between the cicatricial formation and the intra-ocular tension. In 70 per cent of the cases a good result followed the operation, but in 10 per cent the result was decidedly unsatisfactory. Cloudiness of the lens set in in 4 per cent of the cases, while posterior synechiae developed in the great majority of them. In 2.3 per cent the eye was attacked by iridocyclitis and in 3.4 per cent enucleation was found to be necessary. Six eyes became atrophic but were not, for various reasons, removed. One and three-tenths per cent of the eyes operated on were lost from late infection. Vitreous was lost in 6.2 per cent. Two eyes became blind from expulsive hemorrhage. The large majority of these complications arose in the eyes operated on for chronic glaucoma. There were fewer eyes lost following the operation for glaucoma simplex than in the other forms

[Pg 137]

[Pg 138]



of the disease. Recurrences were noticed in 11.3 per cent of all the cases; in simple glaucoma 14.3 per cent as against the acute and chronic forms with 6 per cent. A return of the glaucoma was noticed in 7 per cent of the pale, oedematous, post-operative scars, in 16 per cent of the flat cicatrices, and in 24 per cent of the ectatic variety. Considerable stress is laid upon the fact of the marked softness of the eyes after each operation. There were histological examinations made of the eyeballs in 11 cases, in which the position of the incision and excision, the development of the scar tissue, and the appearance of the complications were duly set forth. The operator then gave a history of over 178 trepanations after the Elliot method and compares them with the procedure of Lagrange. He concludes that the Elliot trephining operation is less dangerous, is more likely to be followed by the development of a cystic scar, and leads to loss of the eye in only 2.4 per cent of the eyes operated on. In Elliot's cases the percentage of relapse was more noticeable than in the Lagrange cases where no iridectomy was done. This observer concludes that the method of Elliot is to be preferred to that of Lagrange, and that in the former case iridectomy is an important factor in obtaining a favorable result. This being the case one cannot truthfully say that trephining alone can take the place of the old Graefe iridectomy. On the other hand, trephining may with advantage be employed instead of iridectomy for cases difficult or dangerous under the latter method.

[Pg 139]

Whatever difference of opinion was noticeable at the Vienna meeting, all of those present, especially Meller, the reader of the paper just quoted, were decidedly of the opinion that the Elliot operation is in every respect the one best adapted to buphthalmia, or congenital glaucoma.

[Pg 140]

In conclusion let me say that the acceptance or rejection of Colonel Elliot's procedure or any other operation is not to be decided by the percentage of iritis, secondary cataract, relapses, lost eyes, etc., but by deciding whether or not his procedure in the various forms of glaucoma gives the best results, including the preservation of comfortable eyes. In other words, we are seeking not the operation that will cure *every* case of glaucoma but the one which is capable, *in the hands of the average ophthalmic surgeon*, of relieving or curing *most* cases of that affection.

---

## **Dr. Casey A. Wood's Paper on Operations Other than Scleral Trephining for the Relief of Glaucoma**

[Pg 141]

**Discussion,**  
**ALBERT E. BULSON, JR., M.D.,**  
**Fort Wayne.**

Increasing belief in Colonel Elliot's view that trephining should be the operation of choice in any form of glaucoma, makes it difficult to consider operations other than trephining in anything but a spirit of disfavor.

Until recently the decision as to the kind of operative procedure to be employed for the relief of glaucoma has depended on the form and stage of the disease, and the amount and character of the vision of the affected eye. Many operators still hold that an iridectomy is the most valuable of all operations for acute inflammatory glaucoma, and not a few hold that the operation has a decided place in the treatment of simple glaucoma. The operation is not without difficulties, and one is inclined to agree with Elliot who says that "The man who can make a 'finished iridectomy' quietly and cleanly has graduated as an ophthalmic operator." The difficulties of an iridectomy are especially pronounced in those cases in which the anterior chamber is extremely shallow and the iris is pressed against the cornea. It is in such cases that the success of the operation is increased by the addition of posterior sclerotomy and the intelligent use of miotics prior to the performance of the iridectomy. Even then the permanent results of the iridectomy will be modified in proportion to the success secured in freeing the filtration angle and opening Schlemm's canal by thorough removal of the root of the iris.

[Pg 142]

The failure of many apparently well executed iridectomies may be attributed to the fact that the iris is not removed to the extreme root, and the remaining stump is sufficient to block the drainage. This is especially apt to be the case in chronic glaucoma where the iris is adherent to the cornea, and in efforts to free the filtration angle by an iridectomy the iris is torn off in front of the adhesion and the filtration angle is not opened.

[Pg 143]

As Elliot has pointed out, iridectomy is most open to attack on the ground of safety. We have to take into account the large scleral wound made, and the fact that this lies close to the ciliary body. The sudden release of all tension and the simultaneous weakening of the supports of the lens and vitreous body create very unfavorable conditions under which to make the crucial step of the operation.

The poor results following an iridectomy in chronic glaucoma have led to the devising of many substitute operations, of which those tending to the production of a filtering scar are now preferred, and, experience shows, hold out the most hope of bringing about long continued relief. It even is considered probable that the effects of an iridectomy which brings about more or less permanent reduction in the intra-ocular pressure is due to the formation of a filtering scar which augments whatever results may have been secured in the attempt to open up the drainage into the canal of Schlemm.

[Pg 144]

Dr. Wood has referred to several of the many substitutes for iridectomy that have been proposed, and it is unnecessary to enumerate them again or to attempt to point out their good or bad features. It is sufficient to say that for the average operator and the larger per cent of cases, the operation which is easiest to perform, is attended with the least risk and offers the best hope of permanent results should be the one of choice. Sympathectomy has failed to secure a place in ophthalmic surgery, sclerotomy has not been found adequate, and cyclodialysis is not sufficiently simple of execution or permanently beneficial in its results to give it prominence.

[Pg 145]

Of the operations proposed for the formation of a filtering cicatrix, those of Elliot and Lagrange are justifiably the most popular. Those of us who have had the pleasure of seeing the trephining operation done by Col. Elliot are impressed with the fact that the operation, even in the hands of its originator, is not, when properly done, uniformly easy of performance. It does, however, offer the advantage of carrying with it the minimum amount of risk, and the apparently permanent results secured justify the ophthalmologist in acquainting himself with the technique of the operation, for, as pointed out by Sydney Stephenson and others, "the technique is responsible for success or failure." Furthermore, there is no sufficient reason why the field of usefulness of the operation should be confined to the chronic forms of glaucoma, and Col. Elliot unhesitatingly recommends trephining as safer and more efficient than any other operative procedures at present employed for the relief of acute glaucoma.

[Pg 146]

The success of the Lagrange operation, which, like the Elliot operation, aims to produce a fistulous communication between the anterior chamber and the sub-conjunctival area, depends upon securing the removal of a relatively large section of all of the layers of the scleral and corneal lip of the wound, so that a permanent opening, covered by the replaced conjunctival flap, is made. Unlike the trephine operation which was evolved from it, the Lagrange operation requires the same kind of an opening of the eyeball as required for a well executed iridectomy, and a properly placed section entirely in scleral tissue, with a good sized conjunctival flap, are elements which enter into the ultimate success or failure of the procedure.

Aside from the dangers incident to a wide incision in the neighborhood of the ciliary body and the possibility of accident to the lens or vitreous body, or of intra-ocular hemorrhage, there is for the average operator the added difficulty and danger in removing a piece of sclera of the exact size required. The technique of the operation is even more difficult and exacting than in the performance of the trephine operation, and it also compares unfavorably in safety.

[Pg 147]

The advisability of removing the conjunctival flap, as advocated by Dr. Wood, as a modification of the Lagrange operation, may be seriously questioned, for aside from the fact that apparently no advantages in aiding permanent filtration are added, there is, added to the objections to the Lagrange operation already mentioned, the very serious disadvantage of subjecting the area at the root of the iris to infection for a prolonged period of time. The advantages of the protection afforded by a conjunctival flap far outweigh the disadvantages of a remotely possible interference of drainage by the blocking of the open wound with conjunctival tissue. The fortunate experience of Dr. Wood in not having infection in a wound which remains open and unprotected for variable lengths of time is not likely to be the experience of any considerable number of operators, and probably will not always be the experience of Dr. Wood. Furthermore, the possibilities of damage by hemorrhage from the choroidal or retinal vessels, delayed formation of the anterior chamber and adhesion of the capsule of the lens to the wound, and the injurious effects of even slight trauma subsequent to the operation, including loss of vitreous, are increased by omitting the conjunctival flap.

[Pg 148]

The modern operation for the relief of glaucoma, by which a filtering scar is produced which permits escape of liquid from the anterior chamber, is the one which apparently holds out the most hope of permanently relieving the condition. While success will depend always to a certain extent upon the personal equation, yet it seems now that for a large majority if not all of the cases we are justified in abandoning all other operations than trephining, notwithstanding the verdict of Elschmig and others that fistula forming operations eventually will be discarded in favor of iridectomy and cyclodialysis.

[Pg 149]

Late or secondary infection, not unknown following iridectomy, may follow the trephine operation, and already some fifteen or sixteen cases have been reported. But while this possibility is a real danger, which improved technique may greatly minimize (Col. Elliot has not seen a case of secondary infection in an experience of over 1200 trephining cases of his own and a large number of others performed by his assistants and pupils) the ultimate verdict must rest with results as compared with other measures. At present, as pointed out by Meller, whose statistics Dr. Wood has cited, trephining heads the list of remedial measures for the relief of glaucoma, and it has the advantage of being applicable to any form of the disease, to be relatively free from danger, either immediate or remote, and to produce the highest percentage of favorable results. The addition of an iridectomy in every case of trephining does not unduly complicate the operation and has much to commend it in offering the patient every possibility of relief.

[Pg 150]

Transcriber's Note:

The index has been moved to the beginning of the text and has been used as a table of contents.

Updated editions will replace the previous one—the old editions will be renamed.

Creating the works from print editions not protected by U.S. copyright law means that no one owns a United States copyright in these works, so the Foundation (and you!) can copy and distribute it in the United States without permission and without paying copyright royalties. Special rules, set forth in the General Terms of Use part of this license, apply to copying and distributing Project Gutenberg™ electronic works to protect the PROJECT GUTENBERG™ concept and trademark. Project Gutenberg is a registered trademark, and may not be used if you charge for an eBook, except by following the terms of the trademark license, including paying royalties for use of the Project Gutenberg trademark. If you do not charge anything for copies of this eBook, complying with the trademark license is very easy. You may use this eBook for nearly any purpose such as creation of derivative works, reports, performances and research. Project Gutenberg eBooks may be modified and printed and given away—you may do practically ANYTHING in the United States with eBooks not protected by U.S. copyright law. Redistribution is subject to the trademark license, especially commercial redistribution.

START: FULL LICENSE  
THE FULL PROJECT GUTENBERG LICENSE  
PLEASE READ THIS BEFORE YOU DISTRIBUTE OR USE THIS WORK

To protect the Project Gutenberg™ mission of promoting the free distribution of electronic works, by using or distributing this work (or any other work associated in any way with the phrase “Project Gutenberg”), you agree to comply with all the terms of the Full Project Gutenberg™ License available with this file or online at [www.gutenberg.org/license](http://www.gutenberg.org/license).

**Section 1. General Terms of Use and Redistributing Project Gutenberg™ electronic works**

1.A. By reading or using any part of this Project Gutenberg™ electronic work, you indicate that you have read, understand, agree to and accept all the terms of this license and intellectual property (trademark/copyright) agreement. If you do not agree to abide by all the terms of this agreement, you must cease using and return or destroy all copies of Project Gutenberg™ electronic works in your possession. If you paid a fee for obtaining a copy of or access to a Project Gutenberg™ electronic work and you do not agree to be bound by the terms of this agreement, you may obtain a refund from the person or entity to whom you paid the fee as set forth in paragraph 1.E.8.

1.B. “Project Gutenberg” is a registered trademark. It may only be used on or associated in any way with an electronic work by people who agree to be bound by the terms of this agreement. There are a few things that you can do with most Project Gutenberg™ electronic works even without complying with the full terms of this agreement. See paragraph 1.C below. There are a lot of things you can do with Project Gutenberg™ electronic works if you follow the terms of this agreement and help preserve free future access to Project Gutenberg™ electronic works. See paragraph 1.E below.

1.C. The Project Gutenberg Literary Archive Foundation (“the Foundation” or PGLAF), owns a compilation copyright in the collection of Project Gutenberg™ electronic works. Nearly all the individual works in the collection are in the public domain in the United States. If an individual work is unprotected by copyright law in the United States and you are located in the United States, we do not claim a right to prevent you from copying, distributing, performing, displaying or creating derivative works based on the work as long as all references to Project Gutenberg are removed. Of course, we hope that you will support the Project Gutenberg™ mission of promoting free access to electronic works by freely sharing Project Gutenberg™ works in compliance with the terms of this agreement for keeping the Project Gutenberg™ name associated with the work. You can easily comply with the terms of this agreement by keeping this work in the same format with its attached full Project Gutenberg™ License when you share it without charge with others.

1.D. The copyright laws of the place where you are located also govern what you can do with this work. Copyright laws in most countries are in a constant state of change. If you are outside the United States, check the laws of your country in addition to the terms of this agreement before downloading, copying, displaying, performing, distributing or creating derivative works based on this work or any other Project Gutenberg™ work. The Foundation makes no representations concerning the copyright status of any work in any country other than the United States.

1.E. Unless you have removed all references to Project Gutenberg:

1.E.1. The following sentence, with active links to, or other immediate access to, the full Project Gutenberg™ License must appear prominently whenever any copy of a Project Gutenberg™ work (any work on which the phrase “Project Gutenberg” appears, or with which the phrase “Project Gutenberg” is associated) is accessed, displayed, performed,

viewed, copied or distributed:

This eBook is for the use of anyone anywhere in the United States and most other parts of the world at no cost and with almost no restrictions whatsoever. You may copy it, give it away or re-use it under the terms of the Project Gutenberg License included with this eBook or online at [www.gutenberg.org](http://www.gutenberg.org). If you are not located in the United States, you will have to check the laws of the country where you are located before using this eBook.

1.E.2. If an individual Project Gutenberg™ electronic work is derived from texts not protected by U.S. copyright law (does not contain a notice indicating that it is posted with permission of the copyright holder), the work can be copied and distributed to anyone in the United States without paying any fees or charges. If you are redistributing or providing access to a work with the phrase “Project Gutenberg” associated with or appearing on the work, you must comply either with the requirements of paragraphs 1.E.1 through 1.E.7 or obtain permission for the use of the work and the Project Gutenberg™ trademark as set forth in paragraphs 1.E.8 or 1.E.9.

1.E.3. If an individual Project Gutenberg™ electronic work is posted with the permission of the copyright holder, your use and distribution must comply with both paragraphs 1.E.1 through 1.E.7 and any additional terms imposed by the copyright holder. Additional terms will be linked to the Project Gutenberg™ License for all works posted with the permission of the copyright holder found at the beginning of this work.

1.E.4. Do not unlink or detach or remove the full Project Gutenberg™ License terms from this work, or any files containing a part of this work or any other work associated with Project Gutenberg™.

1.E.5. Do not copy, display, perform, distribute or redistribute this electronic work, or any part of this electronic work, without prominently displaying the sentence set forth in paragraph 1.E.1 with active links or immediate access to the full terms of the Project Gutenberg™ License.

1.E.6. You may convert to and distribute this work in any binary, compressed, marked up, nonproprietary or proprietary form, including any word processing or hypertext form. However, if you provide access to or distribute copies of a Project Gutenberg™ work in a format other than “Plain Vanilla ASCII” or other format used in the official version posted on the official Project Gutenberg™ website ([www.gutenberg.org](http://www.gutenberg.org)), you must, at no additional cost, fee or expense to the user, provide a copy, a means of exporting a copy, or a means of obtaining a copy upon request, of the work in its original “Plain Vanilla ASCII” or other form. Any alternate format must include the full Project Gutenberg™ License as specified in paragraph 1.E.1.

1.E.7. Do not charge a fee for access to, viewing, displaying, performing, copying or distributing any Project Gutenberg™ works unless you comply with paragraph 1.E.8 or 1.E.9.

1.E.8. You may charge a reasonable fee for copies of or providing access to or distributing Project Gutenberg™ electronic works provided that:

- You pay a royalty fee of 20% of the gross profits you derive from the use of Project Gutenberg™ works calculated using the method you already use to calculate your applicable taxes. The fee is owed to the owner of the Project Gutenberg™ trademark, but he has agreed to donate royalties under this paragraph to the Project Gutenberg Literary Archive Foundation. Royalty payments must be paid within 60 days following each date on which you prepare (or are legally required to prepare) your periodic tax returns. Royalty payments should be clearly marked as such and sent to the Project Gutenberg Literary Archive Foundation at the address specified in Section 4, “Information about donations to the Project Gutenberg Literary Archive Foundation.”
- You provide a full refund of any money paid by a user who notifies you in writing (or by e-mail) within 30 days of receipt that s/he does not agree to the terms of the full Project Gutenberg™ License. You must require such a user to return or destroy all copies of the works possessed in a physical medium and discontinue all use of and all access to other copies of Project Gutenberg™ works.
- You provide, in accordance with paragraph 1.F.3, a full refund of any money paid for a work or a replacement copy, if a defect in the electronic work is discovered and reported to you within 90 days of receipt of the work.
- You comply with all other terms of this agreement for free distribution of Project Gutenberg™ works.

1.E.9. If you wish to charge a fee or distribute a Project Gutenberg™ electronic work or group of works on different terms than are set forth in this agreement, you must obtain permission in writing from the Project Gutenberg Literary Archive Foundation, the manager of the Project Gutenberg™ trademark. Contact the Foundation as set forth in Section 3 below.

1.F.

1.F.1. Project Gutenberg volunteers and employees expend considerable effort to identify, do copyright research on, transcribe and proofread works not protected by U.S. copyright law in creating the Project Gutenberg™ collection. Despite these efforts, Project Gutenberg™ electronic works, and the medium on which they may be stored, may contain “Defects,” such as, but not limited to, incomplete, inaccurate or corrupt data, transcription errors, a copyright or other intellectual property infringement, a defective or damaged disk or other medium, a computer virus, or computer codes that damage or cannot be read by your equipment.

1.F.2. LIMITED WARRANTY, DISCLAIMER OF DAMAGES - Except for the “Right of Replacement or Refund” described in paragraph 1.F.3, the Project Gutenberg Literary Archive Foundation, the owner of the Project Gutenberg™ trademark, and any other party distributing a Project Gutenberg™ electronic work under this agreement, disclaim all liability to you for damages, costs and expenses, including legal fees. YOU AGREE THAT YOU HAVE NO REMEDIES FOR NEGLIGENCE, STRICT LIABILITY, BREACH OF WARRANTY OR BREACH OF CONTRACT EXCEPT THOSE PROVIDED IN PARAGRAPH 1.F.3. YOU AGREE THAT THE FOUNDATION, THE TRADEMARK OWNER, AND ANY DISTRIBUTOR UNDER THIS AGREEMENT WILL NOT BE LIABLE TO YOU FOR ACTUAL, DIRECT, INDIRECT, CONSEQUENTIAL, PUNITIVE OR INCIDENTAL DAMAGES EVEN IF YOU GIVE NOTICE OF THE POSSIBILITY OF SUCH DAMAGE.

1.F.3. LIMITED RIGHT OF REPLACEMENT OR REFUND - If you discover a defect in this electronic work within 90 days of receiving it, you can receive a refund of the money (if any) you paid for it by sending a written explanation to the person you received the work from. If you received the work on a physical medium, you must return the medium with your written explanation. The person or entity that provided you with the defective work may elect to provide a replacement copy in lieu of a refund. If you received the work electronically, the person or entity providing it to you may choose to give you a second opportunity to receive the work electronically in lieu of a refund. If the second copy is also defective, you may demand a refund in writing without further opportunities to fix the problem.

1.F.4. Except for the limited right of replacement or refund set forth in paragraph 1.F.3, this work is provided to you ‘AS-IS’, WITH NO OTHER WARRANTIES OF ANY KIND, EXPRESS OR IMPLIED, INCLUDING BUT NOT LIMITED TO WARRANTIES OF MERCHANTABILITY OR FITNESS FOR ANY PURPOSE.

1.F.5. Some states do not allow disclaimers of certain implied warranties or the exclusion or limitation of certain types of damages. If any disclaimer or limitation set forth in this agreement violates the law of the state applicable to this agreement, the agreement shall be interpreted to make the maximum disclaimer or limitation permitted by the applicable state law. The invalidity or unenforceability of any provision of this agreement shall not void the remaining provisions.

1.F.6. INDEMNITY - You agree to indemnify and hold the Foundation, the trademark owner, any agent or employee of the Foundation, anyone providing copies of Project Gutenberg™ electronic works in accordance with this agreement, and any volunteers associated with the production, promotion and distribution of Project Gutenberg™ electronic works, harmless from all liability, costs and expenses, including legal fees, that arise directly or indirectly from any of the following which you do or cause to occur: (a) distribution of this or any Project Gutenberg™ work, (b) alteration, modification, or additions or deletions to any Project Gutenberg™ work, and (c) any Defect you cause.

## **Section 2. Information about the Mission of Project Gutenberg™**

Project Gutenberg™ is synonymous with the free distribution of electronic works in formats readable by the widest variety of computers including obsolete, old, middle-aged and new computers. It exists because of the efforts of hundreds of volunteers and donations from people in all walks of life.

Volunteers and financial support to provide volunteers with the assistance they need are critical to reaching Project Gutenberg™’s goals and ensuring that the Project Gutenberg™ collection will remain freely available for generations to come. In 2001, the Project Gutenberg Literary Archive Foundation was created to provide a secure and permanent future for Project Gutenberg™ and future generations. To learn more about the Project Gutenberg Literary Archive Foundation and how your efforts and donations can help, see Sections 3 and 4 and the Foundation information page at [www.gutenberg.org](http://www.gutenberg.org).

## **Section 3. Information about the Project Gutenberg Literary Archive Foundation**

The Project Gutenberg Literary Archive Foundation is a non-profit 501(c)(3) educational corporation organized under the laws of the state of Mississippi and granted tax exempt status by the Internal Revenue Service. The Foundation’s EIN or federal tax identification

number is 64-6221541. Contributions to the Project Gutenberg Literary Archive Foundation are tax deductible to the full extent permitted by U.S. federal laws and your state's laws.

The Foundation's business office is located at 809 North 1500 West, Salt Lake City, UT 84116, (801) 596-1887. Email contact links and up to date contact information can be found at the Foundation's website and official page at [www.gutenberg.org/contact](http://www.gutenberg.org/contact)

#### **Section 4. Information about Donations to the Project Gutenberg Literary Archive Foundation**

Project Gutenberg™ depends upon and cannot survive without widespread public support and donations to carry out its mission of increasing the number of public domain and licensed works that can be freely distributed in machine-readable form accessible by the widest array of equipment including outdated equipment. Many small donations (\$1 to \$5,000) are particularly important to maintaining tax exempt status with the IRS.

The Foundation is committed to complying with the laws regulating charities and charitable donations in all 50 states of the United States. Compliance requirements are not uniform and it takes a considerable effort, much paperwork and many fees to meet and keep up with these requirements. We do not solicit donations in locations where we have not received written confirmation of compliance. To SEND DONATIONS or determine the status of compliance for any particular state visit [www.gutenberg.org/donate](http://www.gutenberg.org/donate).

While we cannot and do not solicit contributions from states where we have not met the solicitation requirements, we know of no prohibition against accepting unsolicited donations from donors in such states who approach us with offers to donate.

International donations are gratefully accepted, but we cannot make any statements concerning tax treatment of donations received from outside the United States. U.S. laws alone swamp our small staff.

Please check the Project Gutenberg web pages for current donation methods and addresses. Donations are accepted in a number of other ways including checks, online payments and credit card donations. To donate, please visit: [www.gutenberg.org/donate](http://www.gutenberg.org/donate)

#### **Section 5. General Information About Project Gutenberg™ electronic works**

Professor Michael S. Hart was the originator of the Project Gutenberg™ concept of a library of electronic works that could be freely shared with anyone. For forty years, he produced and distributed Project Gutenberg™ eBooks with only a loose network of volunteer support.

Project Gutenberg™ eBooks are often created from several printed editions, all of which are confirmed as not protected by copyright in the U.S. unless a copyright notice is included. Thus, we do not necessarily keep eBooks in compliance with any particular paper edition.

Most people start at our website which has the main PG search facility: [www.gutenberg.org](http://www.gutenberg.org).

This website includes information about Project Gutenberg™, including how to make donations to the Project Gutenberg Literary Archive Foundation, how to help produce our new eBooks, and how to subscribe to our email newsletter to hear about new eBooks.