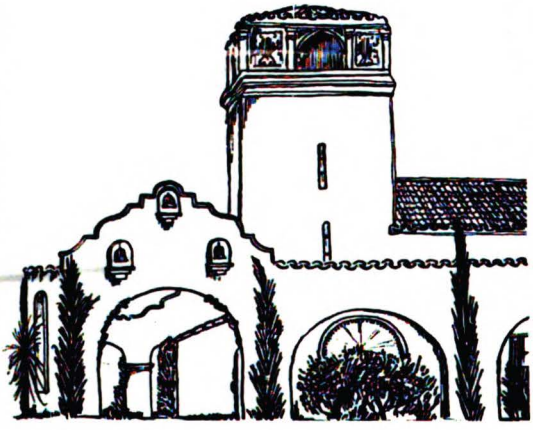
See 'paper' page 45 for Dr. Bates
PDF pg 45 for the Other docotor's article

Presented by

Thomas J. Dailey, D. O.



CALIFORNIA COLLEGE OF MEDICINE

LOS ANGELES, CALIFORNIA

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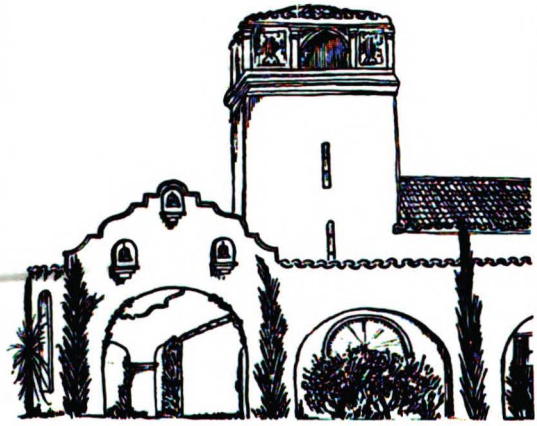

CALIFORNIA COLLEGE OF MEDICINE

LOS ANGELES, CALIFORNIA

TRANSACTIONS OF THE

SECTION ON

Ophthalmology

of the
American Medical Association
at the Sixty-Eighth Annual
Session, held at New York
City, June 5 to 8, 1917

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1903-04	Chairman, R. L. Randolph, Baltimore. Secretary, Albert E. Bulson, Jr., Fort Wayne, Ind.
1904-05	Chairman, Christian R. Holmes, Cincinnati. Secretary, Albert E. Bulson, Jr., Fort Wayne, Ind.

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1912-13 Chairman, Hiram Woods, Baltimore.
Vice-Chairman, Edgar S. Thomson, New York.
Secretary, George S. Derby, Boston.

1913-14 Chairman, Frank C. Todd, Minneapolis.
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Secretary, George S. Derby, Boston.

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1916-17 Chairman, William Zentmayer, Philadelphia.
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1917-18 Chairman, Alexander Duane, New York.
Vice Chairman, F. Phinizy Calhoun, Atlanta, Ga.
Secretary, George S. Derby, Boston.
Delegate, Thomas B. Holloway, Philadelphia.

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PROCEEDINGS OF THE SECTION

WEDNESDAY, JUNE 6—MORNING

The section was called to order at 9 o'clock by the chairman, Dr. William Zentmayer, Philadelphia.

The chairman announced that Dr. Edgar S. Thomson, New York, would act as secretary of the section in the absence of Dr. George S. Derby, who is abroad.

Dr. William Zentmayer, Philadelphia, read the chairman's address, the subject of the scientific portion of which was "The Eye and the Endocrine Organs." No discussion.

On motion the chairman's address was referred for action to the executive committee.

The secretary announced that Major Theodore C. Lyster of the United States Army would deliver an address in the meeting room at 9 p. m. on the visual tests required in the aviation service of the army.

Dr. Samuel Theobald, Baltimore, read a paper on "Some Observations Which Seem to Lend Support to a Long Discarded Theory of the Chief Function of the Oblique Muscles of the Eye." Discussed by Drs. Lucien Howe, Buffalo; Edward Jackson, Denver; Francis Valk, New York; Alexander Duane, New York; W. H. Bates, New York; Walter B. Lancaster, Boston; Will P. Walter, Evanston, Ill.; F. Park Lewis, Buffalo, and Samuel Theobald, Baltimore.

Dr. Edmond E. Blaauw, Buffalo, read a paper on "Binocular Single Vision: Leaving Out the Consideration of the Color Perception and the Problems of Strabismus." A stereoscopic test apparatus was exhibited. Discussed by Drs. Walter B. Lancaster, Boston; F. H. Verhoeff, Boston; George T. Stevens, New York; Francis Valk, New York; Linn Emerson, Orange, N. J.; Elmer G. Starr, Buffalo; Lucien Howe, Buffalo, and Edmond E. Blaauw, Buffalo.

Dr. Frederick H. Verhoeff, Boston, read a paper on "The Treatment of Hypopyon Keratitis."

Dr. William E. Shahan, St. Louis, read a paper on "Further Study of the Effects of Heat on the Eye" (with exhibition of instrument for application of heat to the eye).

These two papers were discussed by Drs. William C. Posey, Philadelphia; Edgar S. Thomson, New York; Peter N. K. Schwenk, Philadelphia; John E. Weeks, New York; Elmer G. Starr, Buffalo; J. A. Donovan, Butte, Mont.; Melville Black, Denver; Arthur G. Bennett, Buffalo; R. L. Randolph, Baltimore; S. Lewis Ziegler, Philadelphia; F. Park Lewis,

Buffalo; L. Webster Fox, Philadelphia; William Zentmayer, Philadelphia; Frederick H. Verhoeff, Boston, and William E. Shahan, St. Louis.

Dr. F. Park Lewis, Buffalo, read a paper on "Crystalline Deposits in the Eye." Discussed by Drs. Burton Chance, Philadelphia, and Melville Black, Denver.

WEDNESDAY, JUNE 6—AFTERNOON

The section was called to order at 2 o'clock by the chairman, Dr. William Zentmayer, Philadelphia.

On motion the section went into executive session.

Dr. Lucien Howe, Buffalo, chairman, presented the report of the Committee on Collective Investigation Concerning the Ocular Muscles. Dr. W. L. Pyle, Philadelphia, moved that the report of the committee be adopted. Seconded by Dr. C. D. Wescott, Chicago. Carried.

The scientific session was resumed and the following instruments and appliances were exhibited:

Dr. Norman W. Price, Niagara Falls, N. Y., exhibited a new phorometer which may be used comfortably by the patient at the bedside, and which measures accurately. The instrument consists of a dial graduated from 0 to 90 degrees, with a long arm and a mirror at the end in which the patient watches his own eye. The patient sits with his back to the light and the oculist turns the dial in front of the patient's eye. When it is directly in front the dial registers 0, and as it is turned down it will register anywhere up to 90 degrees. The dial turns on itself at all angles so that the angle can be obtained at any point. It can be used rapidly, taking only one quarter of the time of the ordinary phorometer.

Dr. Walter L. Pyle, Philadelphia, exhibited a model of a new trial frame for test lenses, the advantage of which is the ability to place it in firm position on the sides of the nose and the adjacent cheeks approximately in the position that the prospective spectacles or eye glasses will be worn. The frame can be lowered or raised by a screw; the two sides of the frame can be moved independently, of advantage in the numerous cases in which the eyes are unequally distant from the nose. A special construction permits the turning of the screw for lateral motion, over the eye opposite to the one being tested, thus avoiding obstruction by the examiner's hand. The frame is rigid without being heavy.

Dr. Henry H. Briggs, Asheville, N. C., exhibited a hook and link with which he demonstrated his method of shortening an ocular muscle, described in his paper in the program on "Tendon Shortening."

Dr. E. R. Crossley, Chicago, exhibited a model of his apparatus for immobilization of the head and eyelids during operations on the eyeball.

Dr. Edmond E. Blaauw, Buffalo, exhibited with his paper at the morning session an apparatus for determining stereoscopic vision.

Dr. Francis Valk, New York, exhibited, in the discussion on Dr. Blaauw's paper, apparatus for determining stereoscopic vision.

Dr. Alan C. Woods, Philadelphia, read a paper on "The Anaphylactic Basis of Sympathetic Ophthalmia: An Experimental Study." Discussed by Drs. George E. de Schweinitz, Philadelphia; J. W. Jervey, Greenville, S. C.; F. H. Verhoeff, Boston; Stuart B. Muncaster, Washington, D. C.; Mark J. Schoenberg, New York, and Alan C. Woods, Philadelphia.

Dr. Arthur J. Bedell, Albany, N. Y., read a paper on "Oxycephalia: Report of Three Cases, with Operation in One." No Discussion.

Dr. William B. Chamberlin, Cleveland, read a paper on "The Endonasal Operation on the Lacrimal Sac." Discussed by Drs. Harris P. Mosher, Boston, and William B. Chamberlin, Cleveland.

Dr. Frank C. Todd, Minneapolis, chairman of the executive committee, presented a resolution for the action of the section that had the approval of the executive committee, requesting the House of Delegates to permit the section to begin its sessions on Tuesday afternoon of the meeting instead of Wednesday morning, as at the present session.

On motion of Dr. G. E. de Schweinitz, Philadelphia, duly seconded, the action of the executive committee was approved, and the committee was instructed to present the matter to the House of Delegates. [Not transmitted to the House of Delegates.]

Dr. Clifford B. Walker, Boston, read a paper on "Neurologic Perimetry and a Method of Imitating Daylight with Electrical Illumination." Discussed by Dr. Luther C. Peter, Philadelphia.

Dr. Walter R. Parker, Detroit, gave a "Demonstration of Pathologic Changes in the Optic Nerve in Cases of Artificially Induced Intracranial Pressure." No discussion.

THURSDAY, JUNE 7—MORNING

The meeting was called to order at 9 o'clock by the chairman, Dr. William Zentmayer, Philadelphia.

The executive committee reported that it desired to endorse the suggestion of the chairman of the section concerning the appointment of a committee on social service as related to ophthalmology, and recommended that the present chairman appoint that committee.

The committee also recommended the appointment of Dr. Peter A. Callan, New York, as memorialist for Dr. W. B.

Marple, New York, deceased, and Dr. Luther C. Peter, Philadelphia, as memorialist for Dr. Wendell Reber, Philadelphia, deceased.

On account of the absence of the secretary, Dr. George S. Derby, Boston, in Europe in military hospital medical service, the committee reported that Dr. Edgar S. Thomson, New York, had consented to act as secretary of the section during Dr. Derby's absence.

The term of Dr. Hiram Woods, Baltimore, as a member of the Board on Ophthalmic Examinations, had expired. It seemed desirable, in this initial period, that he be continued for another period of three years.

On motion, duly seconded, the report of the executive committee was adopted.

The following officers were elected: chairman, Dr. Alexander Duane, New York; vice chairman, Dr. F. Phinizy Calhoun, Atlanta, Ga.; acting secretary, Dr. Edgar S. Thomson, New York; delegate, Dr. Thomas B. Holloway, Philadelphia; alternate, Dr. Archibald L. MacLeish, Los Angeles.

Dr. Edward Jackson, Denver, chairman, read the report of the Committee on Standardizing Test Cards.

On motion, duly seconded, the report was adopted and the committee continued. The report will be found in the printed transactions of the section.

Dr. Edward Jackson, Denver, read the annual report of the American Board for Ophthalmic Examinations.

Dr. S. D. Risley moved the adoption of the report with its recommendations. Seconded and carried.

Dr. Walter R. Parker, Detroit, made a verbal report of the Committee on the Elliot Trephine Operation, which will be found in the transactions of the section, in which a request was made for funds for continuing the work of the committee.

Dr. S. D. Risley, Philadelphia, moved the continuance of the committee and the furnishing of funds. Dr. A. E. Bulson, Jr., Fort Wayne, Ind., suggested that the plea for funds be referred to the Knapp Testimonial Committee, as the section had no funds. He made this as an amendment to Dr. Risley's motion, and the amendment was accepted by Dr. Risley. The motion as amended then carried.

Dr. A. E. Bulson, Jr., Fort Wayne, Ind., read the Report of the Committee on the Knapp Testimonial Fund.

Dr. John E. Weeks, New York, moved that the report be accepted and filed. There were no objections and it was so ordered.

Dr. John E. Weeks, New York, read the report of the Committee on Award of the Knapp Medal, which will be found in the transactions. The committee awarded the medal

to Dr. Walter R. Parker, Detroit, in recognition of the work described in 1916 in his paper on "The Relation of Choked Disk to the Tension of the Eyeball, an Experimental Study."

Dr. A. E. Bulson, Jr., moved that the report be accepted. It was seconded by Dr. S. D. Risley and the motion was carried.

Dr. A. E. Bulson, Jr., by direction of the chairman, presented the medal to Dr. Parker, who expressed his appreciation.

Dr. Frank Allport, Chicago, chairman of the Committee on Conservation of Vision was not present. Dr. Hiram Woods, Baltimore, a member of the committee, made a report which will be found in the transactions, incorporating a letter from Dr. Allport offering his resignation, and also a letter from Edward N. Van Cleve, managing director of the executive staff of the National Committee for the Prevention of Blindness, suggesting the continuance of the committee and the appointment of a New York member of the section to act as local chairman for Dr. Allport, and that it act with the national committee under the name of "The Committee on Conservation of Vision, American Medical Association, Cooperating with the National Committee for the Prevention of Blindness." The name of Dr. Walter B. Weidler of New York was suggested as the local acting chairman. Dr. Woods recommended that the suggestion of Mr. Van Cleve be accepted.

Dr. A. E. Bulson, Jr., moved that the section approve the plan as proposed and that the committee, as far as the section is concerned, continue the work as outlined. Seconded by Dr. S. D. Risley, and carried.

Nominations were made for members of the Committee on Award of the Knapp Medal. The following were selected: Drs. Walter R. Parker, Detroit; William H. Wilder, Chicago, and Arnold Knapp, New York.

Dr. Hiram Woods, Baltimore, presented a resolution which, after discussion and amendment, was adopted in the following form:

WHEREAS, It is officially stated that in the present crisis of our country the government will need the services of every available medical man; and

WHEREAS, The present number of ophthalmologists in the regular medical department and Officers Reserve Corps is too small to meet requirements; and further,

WHEREAS, The present classification of ophthalmologists as surgeons has a tendency to prevent volunteering because it is apt to assign to men of special training work for which they are unfitted, and to remove them from work of great importance which they can do, therefore be it

***Resolved*, By the Section on Ophthalmology of the American Medical Association, in session in New York, June, 1917, that the section respectfully makes the following recommendations:**

1. That ophthalmologists be classed as a separate division of the medical department.

2. That a survey be made of the ophthalmologists of the United States for duty in various lines of work (examination of visual qualifications of recruits, aviation corps, signal corps, detection of malingerers, hospitals, etc.).

3. That the proper persons to make this survey are the members of the subcommittee on ophthalmology of the General Medical Board of the Council of, National Defense.

4. That the section respectfully petitions the surgeons-general of United States military forces to appoint this subcommittee on ophthalmology to make a review of the ophthalmic profession of the country and submit to the departments lists of those best qualified to fulfil the various duties falling on ophthalmologists.

5. That the surgeons-general be further petitioned to follow the recommendations of this board in the assignment of work as closely as is consistent with the good of the service, requesting those selected by the board, but not members of the Officers' Reserve Corps, to volunteer for such service as the board may designate. [Not transmitted to the House of Delegates.]

Dr. Edward Jackson, Denver, read a paper on "Conical Cornea, or Anterior Myopia."

Dr. Meyer Wiener, St. Louis, read a paper on "A New Operative Method for the Relief of Advanced Cases of Keratoconus, with Report of Two Cases."

These two papers were discussed by Drs. S. D. Risley, Philadelphia; Edward C. Ellett, Memphis, Tenn.; W. H. Wilder, Chicago; E. E. Blaauw, Buffalo; Hunter H. Turner, Pittsburgh; L. Webster Fox, Philadelphia; Edgar S. Thomson, New York; Edward Jackson, Denver, and Meyer Wiener, St. Louis.

Dr. George S. Derby's paper on "Interstitial Keratitis, with Special Reference to the End-Result," was, on motion, in the absence of Dr. Derby in Europe, read in abstract by the secretary, Dr. Edgar S. Thomson, New York. Discussed by Arnold Knapp, New York; J. A. Fordyce, New York; F. Park Lewis, Buffalo; R. L. Randolph, Baltimore; Hiram Woods, Baltimore; E. E. Holt, Portland, Me.; F. H. Verhoeff, Boston; H. F. Stoll, Hartford, Conn., and W. H. Wilder, Chicago.

Dr. Sydney Walker, Jr., Chicago, read a paper on "Present Status of Corneal Transplantation and Experimental Data." Discussed by Drs. John E. Weeks, New York; L. Webster Fox, Philadelphia, and Sydney Walker, Chicago.

Dr. George E. de Schweinitz, Philadelphia, moved that the secretary be requested to convey to Dr. George S. Derby, for this section, cordial greeting, and to express the hope that the opportunity which has fallen to him and his colleagues abroad shall meet abundant success and that he and they shall escape the harm and the hurt which would necessarily threaten them in their stimulating and patriotic line of duty.

Dr. John E. Weeks, New York, seconded the motion and it carried unamimously.

THURSDAY, JUNE 7—AFTERNOON

Dr. Henry H. Briggs, Asheville, N. C., read a paper on "Tendon Shortening." Discussed by Drs. Will P. Walter, Evanston, Ill.; J. W. Jervey, Greenville, S. C.; David W. Wells, Boston; Frank C. Todd, Minneapolis; Archibald L. MacLeish, Los Angeles; George A. Moore, Palmer, Mass., and Henry H. Briggs, Asheville, N. C.

Dr. Harry W. Woodruff, Joliet, Ill., read a paper on "Tendon Transplantation of the Eye Muscles." Discussed by Frank C. Todd, Minneapolis; Arthur S. Tenner, New York; Meyer Wiener, St. Louis, and Harry W. Woodruff, Joliet, Ill.

Dr. Leo J. Goldbach, Baltimore, read a paper on "Lymphatic Nodular Keratoconjunctivitis (Phlyctenules)."

Dr. Michael Goldenburg, Chicago, read a paper on "The Pathogenesis of Ophthalmia Eczematosa: A Preliminary Report."

These two papers were discussed by Drs. Richard J. Tivnen, Chicago; Hunter H. Turner, Pittsburgh; George F. Sullivan, Hoboken; H. W. Woodruff, Joliet, Ill.; A. E. Davis, New York; Leo J. Goldbach, Baltimore, and Michael Goldenburg, Chicago.

Drs. Aaron S. Green and Louis D. Green, San Francisco, presented a paper on "An Analysis of One Hundred and Forty-Six Consecutive Cases of Intracapsular Extraction of Senile Cataract." Discussed by Drs. H. F. Hansell, Philadelphia; Henry D. Thornburg, Chicago, and L. D. Green, San Francisco.

Dr. F. Park Lewis, Buffalo, introduced the following resolution:

Resolved, That the Section on Ophthalmology of the American Medical Association call the attention of the surgeons-general of the army and navy to the fact that conscripted men from regions infected with trachoma might be the source of a widespread epidemic of the disease, and that strict measures be adopted for the prevention of such a disaster. [Not transmitted to the House of Delegates.]

On motion the resolution was adopted.

Dr. William Zentmayer, the retiring chairman, made a few remarks thanking the section for assistance rendered and courtesies shown him.

Dr. Alexander Duane, the chairman elect, was inducted into office.

On motion of Dr. W. H. Wilder, Chicago, a vote of thanks was tendered to the retiring officers.

THE EYE AND THE ENDOCRINE ORGANS *

WILLIAM ZENTMAYER, M.D.
PHILADELPHIA

Members of the Section on Ophthalmology of the American Medical Association: Permit me again to express to you my deep appreciation of the honor of being chosen to preside over this, the thirty-ninth session of this body.

As was to be expected, the declaration by Congress that a state of war exists between the Imperial Government of Germany and the United States has been followed by a call to service of physicians from all parts of our country, and among these are many members of this section. Some have already gone. As you probably all know, Dr. Derby, our secretary, is now on the other side. Fortunately for the outcome of this meeting, most of the work preliminary to the meeting itself was finished before he left. We shall all miss his genial presence, and I his guiding hand. To him and to all others on his mission we wish success and an early return.

I wish to express to Dr. Thomson our appreciation of his kindness in consenting to act as secretary for the meeting.

Your chairman, and our secretary, Dr. Derby, are convinced that the new order of having the first session of the sections convene on Wednesday morning and the last on Friday morning is distinctly less desirable than the old plan of having the first session on Tuesday afternoon and the final session Thursday afternoon, and they would therefore recommend that the delegate of the section be instructed to use his influence to have a return to the former plan.

* Chairman's address.

That ophthalmologists in general have failed to utilize social service to anything like the extent that its value justifies is, I believe, in a large measure due to the fact that they have not had pointed out to them by those who have profited by its use the possible scope of this service. In order that we may have placed before us in concrete form what is and can be done along this line, I would suggest the appointment of a committee on collective investigation into the application of social service to ophthalmology.

It has long been in my mind, as perhaps it has been in the minds of many of you, that much energy, time and money are being wasted in our country in the publication of several ophthalmic journals whose activities are the same, and on one occasion, three years ago, I publicly voiced the desire that there might be brought about a consolidation of these journals and the publication of a single journal by our parent body, and I had hoped to initiate such a movement at this time. Circumstances have fortunately placed it earlier in more forcible hands. I can at least urge the passage of a resolution commending Dr. Jackson for his efforts in bringing this about, and ask the support of such a journal by the profession.

In the intersessional period our society has lost two members who were very active in its work.

In August of 1916, Dr. Wilbur Marple of New York died suddenly. Dr. Marple was an active participant in our proceedings. His contributions were numerous and varied, evidencing keen observation and originality. His discussions were always of value, and were presented in a fluent and impressive manner and with a confidence born of a thorough knowledge of ophthalmology.

Dec. 30, 1916, ophthalmology suffered a severe loss in the death of Dr. Wendell Reber after a brief illness of pneumonia contracted while in attendance on the annual meeting of the Academy of Ophthalmology and Oto-Laryngology. Dr. Reber's grasp of ophthal-

mology was broad, as his publications show, but it was in the elucidation of the obscure problems associated with the ocular muscles that he was particularly concerned and with which his name will always be associated. Dr. Reber was a brilliant man, highly accomplished and of broad culture. His death came in his prime, making his loss doubly great.

It would seem fitting that our *Transactions* should contain some record of the work of the men whose efforts and sacrifices have gone to the making of the character and influence of this body. If it meets with the approval of the House of Delegates, and if the executive committee so please, the Chair would consider it a privilege to appoint memorialists for these deceased members.

I have chosen as the subject of my address, "The Eye and the Endocrine Organs."

Few subjects have claimed more of the attention of the research worker, the internist and the anatomist in the past decade than the function, anatomy, pathology and interrelation of the ductless glands. As a result, our knowledge of these matters has been greatly increased and the literature has now reached voluminous proportions.

The increasing interest in these organs by the ophthalmologist is well shown by the fact that since I began collating the literature for this address two other essayists have anticipated me. In January of this year there appeared an admirable summary of the established facts in the relation between the internal secretions and the eye by the late Dr. Schirmer, and a less comprehensive article by Lisser in September, 1916, from both of which I have freely drawn.

It is my purpose to bring to your attention not only facts but also what is still within the domain of speculation, hoping thereby to stimulate research to determine the value of such theories. I shall consider the subject from the aspects of experimental and indirect evidence, clinical evidence, toxic effects from the

internal administration of the glands, indirect pathologic relation, suggestive etiology, and therapeutic uses of the glands in diseases of the eye.

EXPERIMENTAL AND INDIRECT EVIDENCE

The close topographic relation of the parathyroids to the thyroid and sympathetic nerves makes it difficult both in human surgery and in animal experimentation in operating on one structure to keep the other inviolate, and it is therefore difficult to decide in what relative degree the involvement of these structures is responsible for the resulting symptoms.

Thyroid Gland.—Gley and Rochon Duvigneaud found, from experiments on dogs, that in some instances after extirpation of the thyroid gland the cornea became porcelain white, leading at times to ectasia and superficial ulceration. Microscopically leukocytic infiltration was found. Leber, nevertheless, suggests that the appearances indicate, as the cause, a toxic endothelial necrosis. These investigators also saw produced an acute blepharitis with abundant lacrimation. Halstead noted conjunctivitis, and as a further complication partial blindness without ophthalmoscopic changes.

Following complete excision of the thyroid and parathyroids, in several instances Edmunds also encountered keratitis resembling syphilitic interstitial keratitis, in some cases unilateral and in others bilateral; but as its occurrence was usually associated with tetany leading to death, he believes it would in all cases have been bilateral had the animal lived. As a result of a similar operation, this author saw double cataract develop. Coats, who examined the lenses, was of the opinion that it was undoubtedly due to degenerative changes connected with the removal of the thyroids and the convulsions which followed.

While Edmunds believes that the altered secretion of the thyroid and parathyroids, produced by interference with the nerve supply, is more inimical to the

nutrition of the cornea than the toxins produced by the complete removal of the glands, de Quervain is convinced that tetany thyropriva, as produced experimentally in animals, is the result of a toxemia of the entire nervous system, especially the brain, and is certainly due neither to injury to the neighboring tissues in the neck nor to the operation per se.

In tetany thyropriva, Falta and Kahn found spasm of the ciliary muscle and hypersecretion of tears.

Parathyroids.—There is some experimental evidence to indicate that disturbed function of the parathyroids alone can cause cataract. While the experiments of Erdheim showing that in parathyroidectomized animals there is a diminished calcification of the dentin and a hyperplasia of the enamel which frequently leads to fracture of the teeth, and those of MacCallum, Voegtlein, Leopold and von Reusz, which demonstrated a decrease in the lime secretion and a diminution of the bone salts, do not have a direct bearing on established relationship between the parathyroids and the eye, they do direct one's thoughts to the possible bearing of these findings on the syndrome of blue scleras and friability of the bones.

The view of Jeandelize that many convulsive disorders of man (convulsions of childhood, epilepsy and eclampsia) are due to parathyroid insufficiency, together with the experimental evidence adduced, strengthens the assumption that relative insufficiency of the secretion of these glands is an important factor in the etiology of zonular cataract.

Suprarenals—In from five to ten seconds after the intravenous injection of epinephrin in rabbits and cats, the pupil begins to dilate, the nictitating membrane to retract, and slight ptosis to occur. These phenomena reach their height in from five to twenty seconds, and last about two and a half minutes longer. In dogs, after medium doses, there is miosis, enophthalmos and adduction of the eye, probably through oculomotor stimulation from increased intracranial

tension. Toxic doses, however, produce dilatation of the pupil. In man, in the presence of paralysis of the sympathetic, the conjunctival instillation of epinephrin produces mydriasis. From observation of the constant use of epinephrin in daily practice for a number of years Santos-Fernandez finds that it only infrequently produces mydriasis. According to Nieden, the local use of epinephrin diminishes lacrimal secretion, and we are all familiar with the contraction of the vessels which results.

When a 1 per cent. solution of epinephrin is instilled into the conjunctival sac of animals deprived of the thymus gland, epithelial bodies or thyroid gland, the pupil becomes dilated in all, but with this difference: that where the thymus has been extirpated, it occurs only after the lapse of weeks, whereas after extirpation of the thyroid it occurs within a few hours. In the presence of pancreatic lesions, repeated instillations produce dilatation of the pupil. In exophthalmic goiter and during the administration of thyroid extract dilatation of the pupil may occur when epinephrin is instilled into the eye. This is due probably to the increased irritability of the sympathetic.

According to Bayer, the contradictory observations on the effects of epinephrin on intra-ocular tension have been brought to agreement by the studies of Ruberts which have shown that when epinephrin of the usually applied strength is instilled into the normal eye it causes no fluctuation of the tension, while in the glaucomatous eye this is very marked. First there is a diminution, then an increase, and finally a decrease. In glaucomatous eyes it must therefore be used with caution.

Pineal Gland—There is no evidence to show that perversion of the secretion of this body is capable of producing any disturbance of the functions of the eye, nor is it at all likely that the ocular symptoms associated with teratomas of this organ are due to other than the pressure exerted on neighboring structures.

It is of sufficient interest to note that the epiphysis cerebri is, in the words of Lord, "a rudimentary structure, developmentally the representation of the median eye present in some animals." This is also the view of Barratt, who says it is not correct to speak of the whole diverticulum, or even any part of it still existing, as the "remains of a median eye or as a vestigial eye."

Both Ogles and Campbell found, in cysts of the pineal gland, cells of varying shape filled with coarsely granular dark brown pigment, and think that they may represent the pigment cells of the retina.

Pituitary Body.—Without more definite experimental data we must consider the ocular phenomena associated with disease of the pituitary body as due to direct pressure and increased intracranial tension; but there are symptoms, notably the "antecedent amblyopia" of de Schweinitz, the frequent variability, and at times transient nature of the disturbed function, and the beneficial effects of glandular therapy on the amblyopia in some cases of undoubted hypophysial disease, that suggest the possibility that in some cases disturbed secretion is a factor. Furthermore, Uhthoff points out that cases have been recorded in which the ocular condition, particularly the disturbance of motility, more especially the oculomotor, was induced by changes in the basal nerves not the result of pressure.

Schirmer directs attention to the fact that the symptoms in the syndrome to which Oppenheim first called attention and to which he has given the name "tabes pituitaria" are attributed to degenerative changes in the lumbar spinal cord due to the action of pituitary secretion, and comments that if this be admitted, a similar influence on the optic fibers is just as likely, and he prophesies that the time will come when certain cases of optic atrophy will be found to be of hypophysial etiology.

CLINICAL EVIDENCE

Time does not permit, nor would it be appropriate on this occasion, to give a careful analysis of the many eye symptoms and their variations dependent on the nature and extent of the disease of the pituitary body, yet we cannot be contented with their mere enumeration. According to de Schweinitz, one of the earliest ocular symptoms is "antecedent amblyopia" a form of blurred vision which antedates by a long period of time the objectively determinable amblyopia or any failure to read normally the ordinary test types, although the patient is conscious of a definite mist often in the central area. It may be unilateral and is probably by no means constant. Cushing and Parsons have also observed this symptom.

Visual Field Defects.—Coming now to the more definite phenomena, I shall first consider the visual field defects. As is well known, the typical field defect is a bitemporal hemianopsia, which is present in at least 40 per cent. of all cases; but we should give great weight to the assertion of Cushing that homonymous hemianopsia is by no means uncommon. According to Uhthoff, however, in the absence of acromegaly it is very rare. The development of the heteronymous defect from a superior temporal slant of the peripheral field, and the precedence of a hemichromatopsia are always to be borne in mind. As has been pointed out by several authors, notably by Weeks, Cushing and Evans, and emphasized by de Schweinitz, the boundary line between the seeing and the blind fields is not usually regular or vertical, and as Wilbrand and Sanger assert, there is probably always a preservation of the macula, but in some cases it is so limited as to be overlooked in the field taking.

While the frequency with which scotomas are present has been probably underestimated, yet, as will later be brought out, the very fact of their occurrence has doubtless led to mistakes in diagnosis. De Schweinitz and Holloway have classified these scotomas thus:

1. Small and paracentral with the possibility of their expanding into complete hemianopsia. 2. An absolute or relative defect up and out, forming a quadrant. 3. Scotomas varying in size and position with a possibility of their disappearing. 4. Large paracentral absolute scotomas in the outer halves of the visual fields. 5. Temporal field scotomas at some distance from fixation, manifested at some considerable time before the entire field is obliterated. 6. An antecedent amblyopia prior to the development of the central scotoma.

The amblyopia and the amaurosis usually develop slowly, though sudden blindness, just as sudden recovery of central vision or the sudden restoration of vision in the formerly blind field, is not unknown. Variations in visual acuity as well as in the extent of the visual field, from day to day, or even within shorter periods of time, are somewhat characteristic. According to Uhthoff, the dictum of Marie and others that the reduction of vision to blindness is a fundamental symptom of acromegaly is untenable.

Ophthalmoscopic Findings.—The preponderance of primary pressure atrophy over other disturbances of the optic nerve is only what is to be expected from the nature and position of the intracranial lesion. The relative frequency of the occurrence of optic atrophy, papilledema and optic neuritis with and without acromegaly, according to Uhthoff's tables, is 20, 9 and 8.5 per cent., optic neuritis, including papilledema, occurring twice as frequently when acromegaly is present than when it is absent.

Disturbances of Musculature.—Oculomotor palsy is by far the most frequent type, and was present in Uhthoff's tabulation in 15 per cent., whereas all other forms of paralyses were seen in but 6.5 per cent. Only rarely is the superior oblique involved. The state of the pupils is dependent on the degree of intracranial pressure and the amblyopia. The occurrence of the

hemianopic pupillary inaction sign is still in question. Nystagmus occurs in only a small percentage of cases.

Occasional Phenomena. — Visual hallucinations, which have been noted in a few instances, are not to be considered as of localizing importance, as they may occur with tumors situated almost anywhere within the cranium, as pointed out by Spiller. Evans alone speaks of cyanopsia. Exophthalmos has been observed as a phenomenon of congestion. Persistent photophobia has been occasionally seen, especially after operative interference (de Schweinitz and Holloway). The pigmentation of the skin of the lids is probably due to associated disturbance of the suprarenals. Difficulty of eversion of the lids and thickening of the palpebral glands are mentioned by de Schweinitz.

Tumors of the Pineal Gland.—According to Oppenheim, tumors of the pineal gland cause almost the same symptoms as tumors of the corpora quadrigemina; in the former, however, the oculomotor, trochlear, and abducent nerves are not so often affected, but nystagmus is more common. Frankl-Hochwart believes that when the foregoing symptoms are associated with an abnormal increase in stature, an unaccustomed growth of hair, obesity, drowsiness, and a premature genital and sexual development with evident precocity of adolescence, we must think of tumor of the pineal gland.

AFFECTIONS OF THE THYROID

Exophthalmic Goiter.—In view of the fact that some of the gravest symptoms of this affection may exist in the absence of either or even both of the phenomena from which it has received its most commonly given name, and that its pathogenesis is now known, it would seem more appropriate to speak always of this syndrome as "hyperthyroidism."

In the majority of the cases of hyperthyroidism, at some period, the eye symptoms dominate the clinical picture. The retraction of the upper lid, to which the

striking and pathetic facies is due, is often the symptom which first calls attention to the existence of the disease. Associated with this we usually find loss of coordination between the downward movement of the globe and the upper lid, and occasionally the difficulty of eversion of the upper lid, and Kocher has observed that when an object fixed by the eye is moved rapidly up and down, it causes a convulsive momentary contraction of the upper lid, while Joffroy has noted a failure of the skin of the forehead to wrinkle when the patient looks up.

Under the term "deficient complementary fixation in lateral eye rotation," Suker has recently described an ocular symptom of hyperthyroidism. After extreme lateral rotation of the eyes either to the right or to the left with the head fixed and with fixation of an object at this point maintained for a second or two, on attempting to follow this fixation point as it is rapidly swung into the median line, one of the eyes — it may be either — fails to follow the other in a complementary manner into proper convergence and for this point when it is brought into the median plane. Either the right or the left eye makes a sudden rotation into the fixation with its fellow, but before it does so, an apparent divergent strabismus is manifested. According to Suker, it is no doubt due to a dissociation in the functions of the sympathetic and the extraocular motor nerves of the eye, and perhaps also to exhaustion on extreme lateral rotation of the eyes.

Periodicity and incompleteness in the act of involuntary winking comprise a less constant lid sign. Exophthalmos may precede or appear coincidentally with the foregoing symptoms. Rarely it is unilateral and then may be accompanied by dilatation of the pupil. In some cases the proptosis indirectly leads to a destructive keratitis. A subjective accompanying symptom is a feeling of pressure behind the eyeball. Along with alopecia, which is not an uncommon symptom, there is falling out of the eyebrows and eyelashes.

Less frequently observed symptoms are: epiphora, or, in some cases, deficient lacrimation; pigmentation of the upper lid, which is probably due to suprarenal insufficiency; tremor of the eyeball, and weak convergence and paresis of the ocular muscles, in the form of bilateral ophthalmoplegia externa, of the associated muscles and individual muscles. Nystagmus is a rare symptom. The occasional existence of an ocular bruit has been much discussed pro and con. Riesman has recently revived interest in the question, having heard it in two cases. He states that it was first described by Snellen and Schonfeld. Carrington and Drummond also give clear descriptions of it. Riesman speaks of it as a rhythmic murmur keeping time with the pulse and not to be confounded with a more or less continuous hum evidently due to the muscular movements of the eyeball. Roemer, in discussing the symptom, states that Hering has proved it to be due to activity of the orbicularis.

Becker has observed lateral displacement of the light streak and pulsation of the retinal arteries synchronously with the radial pulse. Rarely optic neuritis and optic atrophy are observed. In view of the observations of Coppez, Standish and others that these symptoms can result from the ingestion of thyroid extract, and the experimental demonstration by Birch-Hirschfeld and also by Nabuo-Inouye that ganglion degeneration of the retina, and optic atrophy may follow thyroid toxemia in dogs, it seems strange that affections of the optic nerve are not encountered more frequently.

ATHYROIDISM

Myxedema.—In this condition, in which the changes in the thyroid are chiefly atrophic, the edema of the eyelids with the consequent narrowing of the palpebral fissures is the most marked ocular phenomenon. The edges of the eyelids are hyperemic; the eyebrows are elevated and the hairs and cilia are sparse and brittle. Subconjunctival hemorrhages may occur. Other

symptoms are lacrimation, asthenopia, neuroretinitis (Wagner) and superior-temporal contraction of the visual field (Ottolenghi).

Petzetakis studied the oculocardiac reflex in six persons with myxedema and found it enormously intensified over what is observed in normal persons. He believes that the hypothyroidism leaves the sympathetic without the normal stimulation of the thyroid, and as a consequence vagotonus results. In one case the normal balance was restored by thyroid feeding.

Cretinism.—In this condition, which has been called infantile or juvenile myxedema, besides the conditions just enumerated, there is a wide spacing of the eyes. A conjunctivitis which is sometimes present has been attributed by Hitschmann to the interference with drainage, the result of the saddle bridge.

In both of the foregoing conditions it has been noted that the mydriatic effect of homatropin and similar drugs persists beyond the usual time.

Mongolian Idiocy.—Because of a slight outward resemblance with the foregoing two affections and because of the improvement resulting from the use of thyroid extract, some modern clinicians suspect a thyroid origin for the Mongolian type of idiocy, in which the eye symptoms of epicanthus and convergence of the palpebral fissures have originated the terminology of this affection.

Papilledema Due to Thyroidectomy.—Krauss has reported a case of papilledema in a man, aged 23, coming on after thyroidectomy and first observed eight weeks after the operation. Although the parathyroids had been carefully avoided, the operation was followed by a toxemia inducing tetany. Central vision was about normal and the fields showed partial color reversal with peripheral transient scotomas; but the distinctive feature was said to be the slight cutting of the form field with abnormally large color fields. There were prodromal attacks of absolute blindness imme-

diately after the operation. The retinal edema extended from the peripapillary region along the course of the vessels.

Cataract Following Thyroidectomy.—After thyroidectomy in a woman, in which the operation was followed by tetany, Westphal saw double cataract going to maturity in five years; and Schiller observed bilateral cataract develop within six months after partial removal of a goiter in which the operation was followed by tetany.

The occurrence of tetany in all of these cases suggests an insult to the parathyroids as the probable essential factor in the postoperative symptoms.

The influence of the parathyroids in the causation of senile cataract has engaged the attention of Fischer and Triebenstein and also of Heschler; but the conflicting evidence prevents definite conclusions at this time. The former claim to have found signs of latent tetany in 82 per cent. of sixty-eight patients with senile cataract, whereas in control patients of the same age the percentage of tetany was less than 20. Heschler, however, found but 2 per cent. of tetany in fifty cases of presenile and senile cataract.

THYMUS GLAND

Garre found that in 95 per cent. of fatal cases of exophthalmic goiter there was a persistent hyperplastic thymus. An interesting case showing the influence of persistent thymus on the production of the symptoms of exophthalmic goiter is that of von Haberer. Notwithstanding that the patient had had part of the thyroid removed and the thyroid artery going to the remaining thyroid tissue ligated, his condition became desperate. Removal of a fragment of the thymus produced an amazing improvement with a return to the normal both subjectively and objectively, in three months' time.

Halstead records cases of exophthalmic goiter in which ligation of the thyroid arteries and thyroidec-

tomy failed to check the disease, but thymus feeding, radium and Roentgen-ray applications caused remarkable improvement.

TOXIC EFFECTS OF THE INTERNAL ADMINISTRATION OF THE GLANDS

Hyperthyroidism from Use of Thyroid Extract.—Standish, writing in 1916, found recorded in literature eight cases of retrobulbar optic neuritis due to the ingestion of thyroid extract, and added three cases, one of acute development, of his own. The ophthalmoscopic appearances varied from the normal to a slight papilledema, turgid veins and retinal hemorrhages. Of the eleven patients, seven were females, three males, and in one the sex was not stated. The preponderance of the female sex is to be attributed to the fact that thyroid extract is one of the ingredients of many of the proprietary remedies for obesity.

SUGGESTIVE ETIOLOGY

There are many conditions of obscure etiology exhibiting changes which have been ascribed to altered metabolism, autotoxemia and abiotrophy. In view of this, as our knowledge of the function of the ductless glands enlarges, many are suspecting that a fault in one or more of these, reacting on the others, may be the cause of some of these conditions.

Hereditary Optic Nerve Atrophy.—In support of his contention that Leber's disease is primarily due to an inherited temporary disorder of the pituitary body, J. H. Fisher brings forward the following facts: that Leber noticed a great tendency for the visual defects to appear at or about the age of puberty and that evidence of a neuropathic type was afforded by such symptoms as headache, vertigo, tremors, numbness of all the limbs, or even epileptic fits; that in several reported cases there were similar symmetrical field defects; that patients with Leber's disease, as also those with rapidly developing pituitary body growths,

often complain of subjective phenomena of light and colors often as "seen through a blue mist;" that variation in the degree of central amblyopia which occurred in Leber's disease is more consistent with an outside influence on the visual pathways than with primary changes in the nerve fibers; that in both conditions there is an epochal relation between the onset and the period of puberty and the climacteric and that in both, frequently in early stages of the disease, a very mild papillitis can be detected. In one of two affected children of a family, roentgenoscopy of the skull was negative, while the other showed a cellular or honeycombed shadow in the depression of the sella turcica. That similar changes were not found in the two cases he attributes to the fact that the negative finding was in a case of two years' standing. As the visual symptoms in Leber's disease are progressive up to a certain point and then come to rest, he argues that if the lesion which gives rise to these symptoms is due to some disorder of the pituitary body it also must needs be temporary and transient. He also makes the practical suggestion that cases of tobacco amblyopia with glycosuria be subjected to a more thorough critical investigation than we are accustomed to give them because of cases that are on record in which Leber's disease was complicated by glycosuria. Climenko, however, in two cases of Leber's disease failed to find symptoms pointing to disturbed internal secretion.

Hypothyroidism and Optic Atrophy.—The possibility of hypothyroidism producing optic neuritis terminating in atrophy is indicated by the occurrence of consecutive atrophy in two brothers and a sister who with other members of the family had general symptoms of hypothyroidism in a case reported by van Lint and Klesfeld. The visual fields showed peripheral contraction without scotoma. The roentgenograms revealed no lesions of the sella turcica.

Ocular Syndrome of Disturbance of the Internal Secretions.—Lamb describes a syndrome which he believes to be the initial group of ocular symptoms dependent on disturbance of the internal secretions. They are dilated and unequal pupils responding promptly to light and accommodation, eye ache, severe headache, chorioretinitis affecting more markedly the macula region and worse in the eye with the more dilated pupil. This is associated with a low grade ciliary congestion also in the eye with the larger pupil, usually the left one. Lacrimation and dryness of the conjunctiva may be present. There is a marked tendency to exophthalmos. The majority of these symptoms Lamb would explain by supposing an oversensitization of the tissues by the thyroid hypersecretion appearing in the blood, and in the presence of epinephrin producing a very definite excitation of the sympathetic nerve endings or "plates of Langley." The exophthalmos, insufficiency of convergence, asthenopia and ciliary congestion are due to fatigue following overstimulation. The chorioretinitis may be due to some direct effect of epinephrin on the pigment cells, or the result of exposure because of dilatation of the pupil over a long period of time, or to both.

Pigmentary Degeneration of the Retina.—Jones sees in many of the symptoms, general as well as ocular, of pigmentary degeneration of the retina, indication of disturbance of the ductless glands as an etiologic factor. Therapeutic measures based on this assumption have yielded him good results.

Osteitis Deformans.—The failure to find a causative factor for Paget's disease and because the pathology indicates a disturbance of metabolism which brings about the changes in the bones which resemble, clinically and experimentally, those encountered in disturbances of the ductless glands, have led to the belief on the part of some that the cause will be proved to be due to perverted action of these glands. The only records of ocular findings in this disease available are

those of Copez, who found in four cases pin point yellowish-white spots, presumably degenerative, in the retina; in two cases retinal hemorrhages, and in one, diplopia from displacement of the pulley of the superior oblique muscle.

Amaurotic Family Idiocy—The myasthenia, the periodic convulsions, the fact that a toxemia which has usually been assumed to be the cause of Sach's disease can scarcely be a direct one because the disease is familial, and finally that lesions have been found in the suprarenals by Church, in the thymus by McKee, and that in one case Gordon found enlargement of the thyroid and in another atrophy, and that the symptoms of the disease are similar to those arising from epinephrin insufficiency have led Sajous to the belief that this disease is produced by a toxin which provokes organic lesions in the ductless glands, particularly in the most vulnerable of these, the suprarenals, when there is in these a congenital vulnerability which inhibits their power to sustain metabolism and nutrition of the central nervous system, and the muscular and cardiovascular systems.

INDIRECT PATHOLOGIC RELATION

Secondary Hypernephroma of the Iris and Ciliary Body.—An association of one of the endocrine organs and the eye, of interest pathologically, is to be found in the case reported by Chance of secondary hypernephroma of the iris and ciliary body in which the primary tumor developed from a suprarenal rest in the left kidney. There were secondary growths elsewhere than in the eye.

Verhoeff calls attention to a case of epithelial tumor of the ciliary body, reported by Schlipp, and described by the latter as an unusual form of endothelioma, as probably one of secondary hypernephroma.

Keratoconus.—Based on the Abderhalden dialysis method, von Hippel found disturbance of metabolism of the internal secretory glands in two typical cases

of keratoconus, and in a corneal condition closely allied to it. The thymus was found involved in all three, and in one solely. In two, the suprarenals gave a strong positive reaction. In one the thyroid gave a negative reaction, in one, a doubtful reaction and in one a weakly positive reaction.

GLANDULAR THERAPY

Glandular therapy has its justification in an attempt to meet two indications — a deficient or altered glandular secretion and a profound inanition due to disturbed metabolism in the absence of any direct evidence of disease of the ductless glands.

In combating the ocular manifestation of disorders of the hypophysis, pituitary extract of either or both portions of the gland and thyroid extract have been employed apparently with success.

The following brief summary is taken from de Schweinitz's report of cases of pituitary body disease and the results of treatment: 1. Tumor of the pituitary body; complete blindness lasting twelve days in the right eye and six weeks in the left; complete restoration of the vision of the left eye under the influence of large doses of thyroid extract associated with inunctions of mercury. 2. Moderate choked disk with complete blindness of the right eye lasting one week, and partial blindness of the left eye with loss of the nasal field; complete restoration of the visual functions under the influence of the administration of thyroid extract associated with inunctions of mercury.

The same author jointly with How reports an interesting case of atrophic pallor of the optic nerves with paracentral scotoma in the right eye and later bitemporal hemianopsia with marked impairment of central vision, marked enlargement of the pituitary fossa, with deepening of the pituitary pit and decided thinning of the posterior clinoids. After three weeks' administration of anterior lobe extract and thyroid extract, decided improvement in vision resulted. Later, vision

of the right eye steadily improved, and that of the left steadily declined over a period of seven months. At the end of two and one-half years, visual acuity equaled $\frac{6}{5}$ and the fields were normal in each eye. During that period the patient consumed 2,100 tablets of 2½ grains each of thyroid extract and extract of the anterior lobe of the pituitary body.

Elsberg and King record a case of dyspituitarism with glandular insufficiency and cyst formation in which there were bitemporal hemianopsia, great impairment of central vision and pallid disks. One year later the field had changed to right homonymous hemianopsia, but subsequent to a decompression operation again became heteronymous. Four months' treatment with pituitary extract resulted in marked enlargement of both fields in the nasal area.

Tumme's case was also one of dyspituitarism with limitation of the visual fields, failure of central vision and pallor of the optic disks. Under the use of thyroid extract alternating daily with whole gland pituitary extract, fields and visual acuity were normal two years later.

A convincing case of the value of organotherapy is that of Clothier and Devitt, quoted by Reber. A man, aged 39, had bitemporal hemianopsia and hyperpituitarism with enlargement of the pituitary body as determined by the Roentgen ray. Under the administration of thyroid and pituitary gland, in four months there was reestablishment of full form and color fields. After an interval of two years of nontreatment, incomplete vertical hemianopsia in the right eye, and marked contraction of form and color in the left eye were present. After twelve months of the glandular treatment, vision and fields returned to normal. In this case, the administration of either gland alone was without result.

The employment of organotherapy in the treatment of hyperthyroidism is of questionable justification, although there are those who assert that in the later

stages of the disease the onset of symptoms of myxedema is an indication for the use of thyroid extract. Risley lends the weight of his authority to the use of thyroid extract in the treatment of interstitial keratitis in subjects whose inanition and mental turpitude indicate a lowered metabolism. He argues that the drug does good by improving the action of the thyroid gland, whose secretion has been diminished by the general lowered vitality.

In the treatment of a condition to which he gives the name "malignant uveitis," Bordley has employed thyroid extract with brilliant results. In all, eight cases were treated, four with marked success, two doubtful and two failures. He does not wish to infer from this that this disease is necessarily a symptom of cretinism or myxedema.

Muncaster has found thyroid extract in combination with epinephrin and sodium cacodylate of benefit in cases of pigmentary degeneration of the retina and in old pigment deposits in the fundus.

More recently, Jones has suggested organotherapy in pigmentary degeneration of the retina.

Phillips reports favorable results from the transplantation of epithelial bodies in the treatment of cataract.

Along with the general symptoms of cretinism and myxedema the ocular lesions also undergo improvement with thyroid feeding.

The local use of epinephrin for congestion of the conjunctival and ciliary vessels is no longer promiscuous and its employment in glaucoma is of doubtful value. Its use for the prevention and control of corneal staphyloma is recommended by Pontius. It is of material assistance in the prevention and control of bleeding in ophthalmic surgery.

CHIEF FUNCTION OF THE OBLIQUE MUSCLES OF THE EYE

SOME OBSERVATIONS WHICH SEEM TO LEND SUPPORT TO A LONG DISCARDED THEORY

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BALTIMORE

In endeavoring to find an answer to the question what is the chief function of the oblique muscles of the eye, it is important that we should take into account the fact that these muscles are not peculiar to man, but that, on the contrary, they are found almost without exception, in all vertebrates — the only exception being the myxinoids or lampreys. This fact, which shows that they were evolved many thousands of years before even the anthropoids made their appearance, seems to indicate that they have a much more important function to serve than that which is commonly attributed to them in man — that their chief office is to counteract the faulty action of the superior and inferior rectus muscles, owing to the fact that the direction of the pull of these muscles does not correspond with the sagittal axis of the eyeball. Surely these conditions do not exist generally in vertebrates, and, therefore, could have had no bearing on the evolution of the oblique muscles.

In the early part of the last century, the commonly accepted view, which, according to Prof. Joseph Pancoast, dates back to the time of Boerhaave, was that the oblique muscles were the antagonists of the recti and, especially, that they prevented the latter, when acting, from retracting the eyeball into the orbit. This

view, however, was not without its opponent, and the first instance I have found of its rejection occurs in an article by Green,[1] in which the author says:

To the favorite doctrine of most anatomical writers, we cannot subscribe, viz., that the obliqui are the *antagonists* of the recti. This error is based on another, viz., that there is a *necessity* for antagonism. i. e., that the recti *retract* the ball from its ordinary position in the socket.

It is to be regretted that, at least in the published abstract of his paper, the writer adduces no facts or observations to sustain these very positive assertions.

Coming down, now, to a more recent time, we find that so careful an observer as Fuchs held that some provision *is* necessary to prevent the eyeball, when performing its movements, from leaving its place in the orbit, and, according to his view, it is the bulbo-orbital fascia which provides against this misadventure.[2] He says:

By means of this system of fascia pervading the orbit, the contents of the latter are fixed in place. It is owing to them that the eye does not leave its place when performing its movements, but turns about a fixed center.[3]

This view of the action of the bulbo-orbital fascia, I may add, is held, also, by Duane,[4] another high authority on the ocular muscles and movements, who recently said:

The position of the eye in the orbit seems to me to be governed mainly by the fascial bands (including the cheek ligaments) that connect the eye and the muscles with the orbital walls.

It requires no little temerity on my part to call in question the competence of the bulbo-orbital fascia to

1. Green, C.: On the Functions of the Oblique Muscles of the Eye, abstr., Boston Med. and Surg. Jour., 1845, **32**, 191.

2. Motais, discussing the fixity of the eyeball in the orbit, in his treatise on the "Anatomie de l'appareil moteur de l'œil de l'homme et des vertébrés" (1887, pp. 123-124), answers the question: How can an organ with such yielding walls, immersed in a soft mass, and subject to such rapid movements, maintain a fixed position, by saying that it is due to the combined action of several anatomic elements, which he mentions in this order: the antagonism of the rectus and oblique muscles, the bulbo-orbital fascia, with its ligamentous wings, and the cushion of adipose tissue which occupies the deeper portion of the orbit.

3. Fuchs: Text-Book of Ophthalmology, fourth American edition, p. 689.

4. Personal communication to the author.

do what these authorities agree in holding it does do — to prevent the eye from leaving its place in the orbit, when the rectus muscles contract, and to cause it to turn about a fixed point.

To effect this end, it would seem there must be tense, inelastic bands of fascia connecting the sclera or Tenon's capsule with points on the orbital walls anterior to their attachments to the eyeball. That these bands must be, as I have described them, *tense* and *inelastic,* appears obvious; otherwise, having no power of contraction, they could not effectually oppose the very real backward pull of the several rectus muscles.

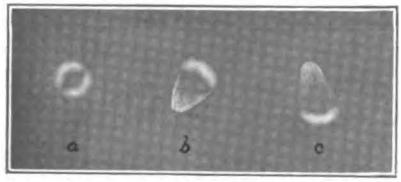
Do such fascial bands exist? And, if they did, would they not, acting as check ligaments, necessarily interfere with the movements of the eye?

These questions, it seems to me, admit of but one answer: No such bands do exist, and, if they did, the rotational movements of the eyeball would certainly be seriously curtailed.

Accepting the view of Fuchs, which I understand that Duane endorses, that *some* provision is necessary to prevent the eye being pulled back into the orbit by contraction of the rectus muscles, and thus enabled to rotate about a fixed point, it would appear that we must fall back on the discarded theory of the supporting or antagonistic action of the oblique muscles for a solution of the problem.

If this view is correct, it would seem to follow that the oblique muscles must take part in all movements of the eye.

Observations which I have recently made of certain subjective light sensations, manifestly caused by contraction of these muscles, appear to show that such, indeed, is the case, with the possible exception of the conjoint action of the interni in effecting convergence. On awakening in the early morning, while the room is still dark, I have observed, projected somewhat into the upper visual field of each eye and in juxtaposition,



Subjective light sensations seen in left half of visual field by right eye: *a*, produced by lateral rotation of the eye; *b*, by downward rotation; *c*, by upward rotation. The upper light sensation, in each instance, is caused by contraction of the inferior oblique, and the lower by contraction of the superior oblique.

always one above the other, two subjective light sensations, one decidedly sharper than the other, on rotating the eyes widely in any direction. On converging the eyes strongly, which I found not so easy to do in the dark, I have thought I obtained similar light sensations, but they were less well defined and more evanescent than those produced by rotating the eyes vertically or laterally.

When the eyes are turned upward, in which movement the inferior oblique should act more energetically than the superior, the brighter and better defined light sensation, as we should expect, is, as shown in the illustration above, the less defined one, produced by the feebler action of the superior oblique, below. When the eyes are turned downward, the superior oblique being then called into more energetic action, the brighter sensation is below, the feebler one above. When the eyes are rotated laterally, the upper and lower light sensations do not differ appreciably in intensity or in form, indicating, it would seem, that in these movements the obliques act with approximately equal energy; and the same is true of those which, I believe, I observed on converging the eyes. The phosphenes seen in the left half of the visual field are, of course, projected from the right eye, and vice versa.

I may add that the light sensations described rapidly decline in brilliance, and, with the exception of those caused by rotating the eyes upward, are best observed with the lids closed, and, further, that repeated attempts to produce them, by energetically rotating the eyes, presently give rise to an ill-defined aching sensation, most noticeable, perhaps, in the region of the trochlea of the superior oblique.

If further observation should show that these subjective sensations do not occur in convergence of the eyes for a near point, a possible explanation might be that in regarding near objects a certain amount of displacement of the eyes backward and inward, in addi-

tion to their inward rotation, may not be harmful, may, indeed, even be helpful, so that conjoint action on the part of the obliques under such circumstances would not be called for.

As to the competence of the oblique muscles to counteract the backward pull of the recti, and to maintain the eyeball in its proper position in the orbit, it would seem that they are fully equal to the task. With their fixed attachments close to the orbital border — that of the inferior oblique actually and that of the superior oblique potentially, because of its trochlea — and passing from these points to grasp the eyeball near its posterior pole, one from above, the other from below, and, moreover, unlike the bulbo-orbital fascia, having the power to contract, so as to oppose with exactness the backward pull of the recti, the oblique muscles appear to be entirely capable of causing the eye to rotate, with precision, about a fixed center, a provision so essential to accurate vision, and, especially, to perfect binocular vision.

If it be objected that to do this the direction of the pull of these muscles would have to be straight forward, it may be pointed out that the pull of the recti, especially the superior and inferior recti, is not straight backward, and that the direction in which the energy of the obliques is exerted seems really to be that best adapted to accomplish the end in view.

That the oblique muscles serve other important ends, especially in preventing the superior and inferior recti from tilting the vertical axis of the eye, and in assisting those muscles to rotate the eyeball upward and downward, is, of course, not to be denied; but that their *chief* function is that which I have set forth seems, to me, at least, to admit of but little doubt.

Cathedral and Howard Streets.

ABSTRACT OF DISCUSSION

DR. LUCIEN HOWE, Buffalo: I hesitated to open this discussion for the reason that I am ignorant of one phase of the question, namely, the metaphysical or psychometaphysical

factor. But the fundamental question in this discussion is simply one of anatomy. Or, in reality, two anatomical points are involved. The first question concerns the origin and insertion of the two obliques. As the inferior oblique is directed backward from its origin at an angle of 5 or 10 degrees, and as the superior oblique is also directed backward from the pulley at an angle of from 50 to 56 degrees, therefore, whenever these muscles contract at all, they must tend to draw the eye somewhat forward. I can see no escape from that conclusion.

The second point is whether the capsule of Tenon, or, more exactly, the capsule of Bonnet, does or does not continue backward, covering the posterior part of the globe. The sections which I have brought here demonstrate that it does. The capsule forms a sort of cup in which the eye rotates.

As for the third point, I have nothing to say. We must take the testimony of those who have studied after-images. Whenever I have experimented with them, I have only succeeded in accumulating a considerable amount of ignorance.

In a word, we must conclude, first, the two obliques do tend to draw the eye forward; second, the capsule does help to hold the eye in place, preventing its being drawn back by the recti; and third, as to the testimony obtainable from after-images, that varies with the individual.

Dr. Edward Jackson, Denver: As Dr. Howe has said, there can be no question about the tendency of these muscles to act on the eyeball if unopposed, the recti muscles tending in a general way to draw it back and the obliques to draw it forward. As to the necessity of the one to oppose the other in this particular respect, the uncertainty comes in through the other attachments of the eyeball. Not only do the recti draw the eyeball backward, but they draw it toward the nasal side of the orbit. Not only do the obliques draw the eyeball forward, they draw it toward the nasal side of the orbit; and there are no muscles that oppose the effect of these two sets of muscles to draw the eyeball toward the ethmoid bone. This suggests the importance of the other structures.

The eyeball is spoken of as rotating about a fixed center. Laboratory measurements show that it does rotate about a fixed center, but that fixed center is not the geometrical center of the eyeball. The fixed center of rotation is behind the geometrical center of the eyeball. The eyeball is not fixed in a certain position in the orbit, but it rolls from side to side. I think a better conception of the eyeball and its surroundings is that of a ball suspended in a hammock. Drawn to one side, it not only turns the anterior pole toward that side, but the the whole globe rolls slightly in the other direction. The eyeball is not held in an absolutely fixed position in the orbit, but is retained with such fixity as is necessary by the comparatively loose connective tissue hammock in which it rolls

up and down and from side to side. Undoubtedly there are actual movements of the eyeball with the contraction of particular muscles acting on it. Such actual movement I have studied, more particularly the projection of the eyeball, which needs more widely to be studied. I have measured in a good many hundred patients the position of the eyeball with reference to the external angle of the orbit when the eyes are widely opened. Sometimes it protrudes slightly, and sometimes as much as 1 mm. In connection with other muscular actions I have no doubt there are other movements of the eyeball which I have not been able to study and which may require exact observations to determine. But the point we must not lose sight of is that the eyeball is not fixed in the orbit and that it rotates around a certain center which is back of the center of the eyeball; and that the eyeball moves from side to side and up and down under the influence of the different muscles.

Dr. Francis Valk, New York: I like to look at the movements of the eyeball mechanically only. In Dr. Theobald's paper he speaks of the functions of the oblique muscles, and that is just what I want to speak of. The chief functions of the oblique muscles are simply to keep the vertical meridians parallel with one another, and I think if you will keep that in mind you will find that the results of your muscle work are better. I do not believe that the oblique muscles actually have any action in the movements of the eyeball. I know this is against the teaching of every man in the profession. It is well known that in every movement of the eyeball every muscle takes part, and the eyeball cannot move in any direction without the action of the oblique muscles; but do the obliques actually turn the eyeballs, or only act to steady the vertical meridians? Whether that is true or not I would like to have the younger men find out, as the action of the oblique muscles has not been fully understood. Now when we operate on the eye muscles, one may perform a tenotomy on the proximal origin of the inferior oblique, but I simply cannot see the reason for it or any suggestion why it should be done. Perhaps I am wrong. Dr. Duane has done the operation over and over again and others have reported cases, but I never have done it and never will do it because from a mechanical standpoint I cannot appreciate its usefulness. I want to put myself on record in reference to that fact.

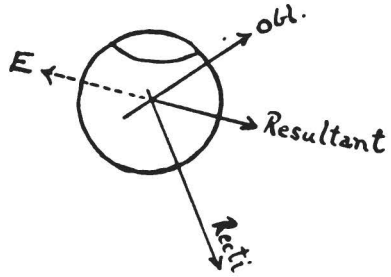
Dr. Alexander Duane, New York: I do not altogether agree with Dr. Theobald's contention that the main action of the obliques is to maintain the position of the eye in the orbit. Still less do I agree with Dr. Valk's idea that the sole, or even main, action of these muscles is to act on the vertical meridian of the eye. Among other facts that speak against Dr. Theobald's view is the absence of any very great protrusion of the eye in cases of ophthalmoplegia. In cases of

this sort that I have seen there has been little or no protrusion. Again, when we do a tenotomy of the inferior oblique and make traction on the tendon in order to test our effect, there is little if any movement of the eye forward, but the eye very distinctly moves up. Furthermore, the very oblique direction of both oblique tendons and the way in which they envelop the eye as they pass backward seem to negative the idea that any marked movement forward of the eye can be produced by their action. On the other hand, it does seem to me that the anatomic disposition of the fascial bands, and especially of the check ligaments, is such as to afford an adequate support to the eye and prevent any great displacement when rotated. According to Dr. Jackson's felicitous simile, the eye is swung in a sort of hammock, the arrangement permitting slight displacements, especially laterally, while the general position remains unchanged. On one point I agree heartily with Dr. Theobald. I do think that one or the other of the obliques takes part in every movement of the eye. I have long maintained that this holds for every eye muscle. Indeed, all the physiologic and pathologic data that we have indicate that in every movement of the eye every muscle except the one or ones directly antagonizing this movement participate. Thus, in abduction, the external rectus, to be sure, does the main work, but the two obliques certainly aid in carrying on the outward movement, while they and the superior and inferior recti act to steady the eye in its course and keep it from deviating up or down. The like is true of each of the other eye movements.

Dr. W. H. Bates, New York: The paper of Dr. Theobald deals with a subject which I have studied during the past five years, and before, and it is one of great importance. I published two years ago a statement that the principal function of the oblique muscles is that of accommodation. This fact I demonstrated to the satisfaction of many scientific men; and, when a scientific man accepts a demonstration of the truth you have got to do something more than talk. I had to conduct experiments on animals of all kinds and prove a great many things before they would accept the fact that the oblique muscles were muscles of accommodation. The oblique muscles by their contraction, I also discovered produce myopia. All people with good eyes do not have perfect sight all the time. I have discovered that you can prevent the pain of an operation when you have perfect sight, when you have the memory of perfect sight or when you have the imagination of perfect sight. (A personal experience was reported by the discussant of a painless operation without the use of ether, cocain or any other anesthetic, by the aid of the memory of perfect sight.)

Dr. Walter B. Lancaster, Boston: As Dr. Jackson has pointed out, the recti and obliques do not completely antago-

nize each other. We must have a force acting in the direction marked E in the figure. As a matter of fact, the external check ligament is located at this point and is a very firm structure, as is shown by the great resistance encountered when one tries to push a hypodermic needle through it in making a deep injection into the orbit. It is obvious, however, that a firm band stretching from the eyeball straight to the outer angle of the orbit at this point in the direction E could not act as the antagonist desired, for then it would be impossible for the eyeball to rotate more than a few degrees. The function of the external check ligament is entirely different. It is not the force we are seeking. This force is to be found in the combined effect of the fascia and connective tissue bands that envelop the eye in the manner Dr. Jackson has compared to a hammock, not in any single band. We must conceive of the eyeball as poised under the influence of



E dotted line shows the direction of pull needed to balance the resultant of the combined action of the recti and oblique muscles.

a multitude of balanced forces. These include the recti and oblique muscles and all the connective tissue bands and fascial network which envelops the globe as a web or hammock, further supported by the orbital fat. All of these are yielding, not rigid supports, as shown by the ease with which exophthalmos, often of very large amount, enophthalmos and retraction movements of the globe occur. These permit a certain amount of rotation, extending perhaps to 50 or 60 degrees in each direction; but the recti muscles could move the eye a good deal more than that if there were no opposing force. The muscles themselves could contract perhaps one third more than that.

When we measure the amplitude of rotation of the eyeball with a tropometer we do not measure the power or strength of the rotating muscle, but what we really measure is the limit of motion caused by these opposing forces which restrict the rotation of the eyeball. The limits of motion are set by the antagonist muscles, by the fascia and check ligaments, and also by the optic nerve, the ciliary nerve and

vessels, the conjunctiva, everything that is attached to the globe, and only in exceptional cases by the lack of power of the rotating muscle, to wit in cases of paresis.

When we have a clear conception of the globe as poised under the influence of all these balanced forces, we see that any motion of the eyeball requires the coordinated cooperation of all the external ocular muscles. It is not simply true, as Dr. Duane has just said, that all the muscles (except the antagonists) are concerned in any motion of the eyeball. All the muscles, *including the antagonists,* are concerned. If the external rectus contracts to rotate the eye outward, the internal rectus must and does relax in equal amount. Thus, the sum total of the forces acting on the eyeball remains the same, the extra pull of one muscle or set of muscles being exactly balanced by the equal relaxation of the antagonists. In extreme rotation this rule no longer holds. Then one muscle or set of muscles contracts as forcibly as it can be made to contract, and is not balanced by an equally excessive relaxation of the antagonists. Under these conditions we get a retraction movement of the globe. It is not uncommon to get such a retraction when all the external ocular muscles are suddenly contracted in the act of "squeezing." The intraocular tension is increased by the pressure of these muscles, even if the lids are prevented from pressing on the eye, and one can sometimes see the eyeball pulled back into the orbit a perceptible amount.

DR. WILL WALTER, Evanston, Ill.: We should emphasize what Dr. Duane has brought out, that in paralytic conditions of the recti we do not have protraction of the globe to any great degree. Dr. Todd has recently reported a case of inferior oblique paresis and he tells me that there was little or no retraction. If the dominant pull of the oblique muscles is forward, we would expect to find a considerable degree of protraction in the presence of paralysis of the recti, and we do not.

A second thought which militates against the view as to their dominance as proctractors is furnished by Fuchs' researches in which it was found that whereas in the normal eye the insertion is in the posterior outer quadrant, and the pull will be represented by an oblique angle, in the myopic eye which has a longer globe the insertion is anteroposterior and the pull is more abverting and less a protractor, and still this is a more prominent globe. In the hyperopic eye the insertion of the obliques is more horizontal—straddles the axis of the globe, and the pull is more directly forward, and still this is not a protracted, but a sunken eye.

With regard to the point which the essayist makes as to sensing the images on rotation, I think if they prove anything it is that the obliques take part in all motions of the eye, and with that we do not take issue.

DR. F. PARKE LEWIS, Buffalo: Sometimes a clinical fact will illuminate what otherwise might be obscure. A few years ago I saw a case of enophthalmos resulting from a blow which had produced a retracted eye. There was simply a sinking of the eye, indicating that there must have been a stretching of the fascia in the hammock band, as described by Dr. Jackson, rather than an involvement simply of the oblique muscles. I mention this as having a rather definite bearing on this point.

DR. SAMUEL THEOBALD, Baltimore: Dr. Duane, in speaking of his belief that all the muscles take part in all the movements of the eye, said that this was true, at least as to one or the other oblique muscle. I would call attention to the fact that the subjective observation which I made showed that both the oblique muscles took part in all movements of the eyes, with the probable exception of the conjoined action of the two interni.

BINOCULAR SINGLE VISION

LEAVING OUT THE CONSIDERATION OF THE COLOR PERCEPTION AND THE PROBLEMS OF STRABISMUS

E. E. BLAAUW, M.D.
BUFFALO

When I take the liberty of asking your interest for a few moments for a subject on which every one of us has pondered some time in his life, I do it because the subject has not been approached in these meetings since 1903, when Dr. N. M. Black spoke about the development of the fusion center in the treatment of strabismus. I have found only three papers since that date in the American ophthalmologic literature, one in 1903 by Dr. E. R. Lewis, and two during 1916 by Drs. A. Brav and G. A. Shepard. Looking over the American textbooks of ophthalmology since that date, I find that the theoretical treatment of the subject is not without some points about which we may talk for a moment, and that the examination for binocular single vision (B. S. V.) in its highest development, namely, the perception of depth, is hardly touched on. The result is that this examination is seldom done, although the perception of depth is of the highest value and can be expressed in terms as accurate as the visual acuity, if we can come to a general understanding. It is high time that more stress should be laid on this function of the binocular organ.

To substantiate what I have said about the textbooks, I may be pardoned if I briefly communicate what I have found:

Fox[1] says:

Perfect binocular vision, or the blending of two images into one, occurs only when the two images fall on correspond-

1. Fox, L. Webster: A Practical Treatise on Ophthalmology, 1910, p. 678.

ing points of the two retinae. These points are as follows. . . . Any disturbance of the harmonious action of the ocular muscles causes the images to be formed on noncorresponding points, and binocular vision is destroyed. This also occurs occasionally after cataract extraction due to malposition of the pupil. Diplopia does not always occur, because the image formed in the nonfixing eye is blurred by the dioptric system, and its perception is retarded by being thrown upon a portion of the retina less sensitive than the fovea centralis. Usually, however, by careful testing diplopia can be demonstrated in imperfect muscle balance.

Ball[2] says:

Binocular vision means the union in one single impression of images received simultaneously on both retinae. . . . Two chief theories have been proposed for the solution of the problem: (1) the theory of identical points, and (2) the theory of projection. The latter theory assumes that the retinocerebral apparatus, by a process of mental projection of the images into space, has the power of appreciating the shape and size of an image, as well as the direction of the rays of light, which form it. While the limits of this treatise will not permit an extended discussion of the subject of binocular single vision, it will be necessary to consider the first theory more extensively.

The theory of identical points assumes a correspondence of each point of one retina to a similarly situated point on the retina of the other eye. When the visual axes are directed toward a far distant object, the visual axes then being parallel (Fox states, p. 678: "The position of rest of the ocular muscles is probably divergence of the visual axes about 8 to 10 degrees from parallelism"), a correspondence actually exists; but when the visual axes converge, the points do not converge. Furthermore, a part of each retina has no corresponding points in the other. This is due to the fact that the actual center of the retina is not at the fovea centralis, but lies nearer the nasal side. So long as the images of a point are within the horopteric circle they fall on corresponding parts of the retina. Images of a point outside this circle do not fall on corresponding points.

[Then the description of the horopter of Johannes Mueller follows. He continues:]

Since the doctrine of identical points is true for only some visual acts, an attempt must be made to explain binocular single vision without the horopter. Such objects as are situated outside the horopter are seen double, but it is possible to obtain only a single cerebral impression of them. Thus the expert ophthalmologist keeps both eyes open while examining the fundus. The image seen by one eye in this

2. Ball, James Moores: Modern Ophthalmology, 1913, p. 75.

case is ignored. Under some circumstances a new mental picture of two combined dissimilar retinal impressions is made. Thus, ideas of solidity and depth are obtained in binocular vision by the mental combination of dissimilar retinal impressions, as in using the stereoscope. . . . One reason why nonidentical points yield good vision is that vision becomes less distinct as we pass from the center of the retina, and the observer learns to neglect the blurred peripheric images, while giving attention to those formed on the fovea.

Stereoscopic Vision: This is another name for binocular vision, or the sense of depth. The law of identical points was supposed to be absolute up to the time that Wheatstone constructed his stereoscope. This shows that the perception of depth is caused by a slight nonidentity of the two retinal images. An object appears to us to be solid, when each eye views it from a different point, as in normal vision.

G. E. deSchweinitz[3] (1910, p, 674) says:

If the associated movements of the eyes were not thus regulated by equal impulses from the coordinating center single vision would not be possible, because the image of any object would not fall upon corresponding points of the two retinas. . . . If, for any reason, the movements of the eyes become disarranged so that the images do not fall upon corresponding or identical areas, the images become double. . . . [Page 50:] The images are projected in different positions, when they are not formed on identical points of the two retinas. [Page 718:] It is often difficult to ascertain whether true binocular vision exists . . . and successful bar reading, usually quoted as a sufficient test, is, according to Priestley Smith, not without its fallacies. The author tests as follows: . . . if he can see all three at once and in a line he is *probably* using both eyes, . . . if with each eye alone he sees two, but with both eyes three, the proof is *fairly* positive. [Italics mine.]

Weeks[4] says:

This condition in which both eyes are accurately directed at the object at which one of the two is looking is called binocular fixation, i. e., binocular fixation means that both eyes are straight.

Corresponding Points: Binocular single vision: . . . The act of thus compounding two retinal impressions so that but one mental concept is formed out of them is called fusion. . . . And any image which forms its retinal images on the corresponding points in the two eyes will always appear single (law of binocular single vision).

3. DeSchweinitz, G. E.: Diseases of the Eye, ed. 6, 1910.
4. Weeks, J. E.: A Treatise of Diseases of the Eye, 1910, p. 709.

The usual schematic picture representation is then given, when the author continues:

While C. will appear double, because its images are formed on noncorresponding points.

Diplopia: The converse of this proposition is also true; that is, any object whose retinal images are not formed on corresponding points in the two eyes appear double.

When the two eyes are both fixing the same object that object and a large number of outlying objects, too, will appear single, because their respective images fall on corresponding points. All other objects, however, will appear double, and this will be particularly the case with objects either nearer or more remote than the object looked at. This physiologic diplopia is regarded as one of the most important of the factors enabling us to appreciate depth and relief (stereoscopic vision). We are not, however, usually aware that these outlying objects are really double, so that, in general, when we use binocular vision everything is apparently single to us.

Exceptions to Law of Diplopia: It must be noted that while binocular fixation is regularly associated with binocular single vision and absence of binocular fixation with diplopia, this association does not hold for all cases. A man may fix an object with both eyes, and yet see it with but one (uniocular vision) . . . or, because, as often happens in a cured squint, the image formed by that eye is mentally suppressed. In this case, then, there is binocular fixation, but not binocular single vision.

Anomalous Diplopia: In other rather rare cases there may be binocular single vision, but not binocular fixation.

Under the test of binocular vision on page 732 is described:

2. Maddox rod. In case he has binocular vision pat. will see both the streak and the flame. If he has also binocular single vision the streak and the flame will be superimposed.

—as if binocular single vision cannot be present, when the streak does not go through the light!

In the "American Encyclopedia of Ophthalmology" the subject of binocular single vision on page 970 is identical with the one referred to above by J. M. Ball. It is followed by the "ingenious illustration of the single character of binocular vision by Savage, in his 'Ophthalmic Myology,'" which to me appears nothing else than a mere obscure description of the correspondence of the two retinas. Just as his isogonal circle (p. 2253) is simply another name for horopter.

My objection chiefly is to the following sentence: "In obedience to the law of projection the one eye sees the line located in space as the other eye sees it; that is definitely related to the median plane of the head, and there is but one object." . . .

Then comes another paragraph, page 973: "Binocular vision. This is the faculty of using both eyes so that they see together without diplopia," which is followed with a discussion about binocular single vision; and — to show the kind of work — a reprint is given from Hill[5] from the "System of Diseases of the Eye," which appeared in the nineteenth century: "In man stereoscopic vision reaches a high state of development, although, owing to the prominence of the bridge of the nose, it probably does not take such complete possession of the retina as it does in the monkey"! Where in the world have ever experiments on monkeys' sense of depth been performed?

Gibbons[6] says:

> Each eye projecting its image in a different direction in space causes the two eyes to see a single object as two. There is binocular diplopia, or *double binocular vision.* [Italics mine.]

In the two volumes, about 900 pages devoted to the muscles of the eye, Dr. Lucien Howe devotes two thirds of one page to this most fundamental point of the highest practical interest (p. 133).

> Suppression of diplopia is physiological: When both eyes are fixed upon an object in front, evidently all other objects lying in that plane—or indeed anywhere else except in the circle of the horopter—are focused on parts of the retina in the two eyes which do not correspond with each other. This of course produces double vision, and if we were accustomed to take cognizance of all these double images the result would be confusing in the extreme. That is easily seen by pressing upon one eye in any direction, so as to produce diplopia. The fact is, therefore, that the normal eye is accustomed to suppress those images which do not fall on the fovea, and to such an extent that we are practically unconscious of it.

5. Hill: System of Diseases of the Eye, **1**, 390.
6. Gibbons, Edward E.: The Eye, Its Refraction and Diseases, 1904, p. 140.

This is accounted for in various ways, and perhaps no better explanation has been given than what Javal calls "the antagonism of the visual fields." Or, as Tscherning says: "It is sometimes the images of one eye that prodominate, sometimes those of the other, and as long as we see in a part of the visual field images with one eye, those of the other eye are completely suppressed." Javal considers that this has an important bearing on some forms of deviation with which we will have to deal later.

The author is not very much at ease with the working of the usual stereoscope, as is shown on page 306, Volume I:

Most of the refracting stereoscopes are made so that the object is viewed only with *considerable convergence.* [On page 86, Volume II:] In the latter [the stereoscope] the convergence is *increased* while the accommodation is diminished, and moreover, with the latter there is a fusion of two different images. [Italics mine.]

The same misunderstanding of this instrument is expressed by the writer of the Hogg's test for simulated blindness:[7]

A person who has normal vision in the two eyes and who looks through the prisms of a stereoscope fuses and involuntarily superimposes the two *lateral* images in a single combined image. [Italics mine.]

To show that outside the United States also no consensus of opinion has been reached, I will quote from the American translation of Roemer's book:[8]

Certainly when two corresponding places on the retinae are stimulated by one and the same object, a single sensation is always produced. When two corresponding places are stimulated dissimilarly the different sensations appear in one and the same line of vision, and all objects that delineate themselves on corresponding places in the retina appear to be at the same distance as the fixation point. On the other hand, stimulations of not corresponding, or disparate, places on the two retinae produce double images of the object in question. This is shown from an old experiment. . . . We learn from this experiment that all points nearer the retina than the point of fixation create retinal images with crossed diplopia. . . . This is because most objects are delineated on disparate places on the retina during vision with converging

7. American Encyclopedia of Ophthalmology, p. 1181.
8. Roemer, Paul: Text-Book of Ophthalmology, trans. by M. L. Foster, New York.

visual lines. There are several causes why we do not notice these double images ordinarily when looking at near objects, under normal conditions: 1. The images of objects produced on the periphery of the retina are much more indistinct than those of the central portion. These images do not make as strong an impression on our consciousness as those on the fovea. We do not notice the things pictured on disparate places of the retinae because our attention is directed wholly to the fixation point. In addition to this, even images which lie on corresponding places in the retinae by no means always appear simultaneously in the proper line of direction. The images conflict with each other, so that we have what is known as the antagonism of the visual fields. One or the other image may appear alternately in the line of direction appertaining to the identical points. Finally, it has been proved that, within certain limits, objects may be seen with binocular single vision that lie on disparate places in the retinae, but such objects appear to be nearer, or farther away than the point of fixation. A physiological phenomenon, however, claims our chief interest at this time. We do not notice the double images because at the instant when our attention is directed to a disparately delineated object our eyes immediately and quite automatically execute such a movement as will bring the image upon the center of the retina. . . . The movements of the eyes that displace the retinal images from disparate to corresponding places are called fusion movements.

It is as true now as in the days of Joseph Le Conte that a "discussion of binocular (single) vision is by far the most fascinating portion of the whole subject of vision."

Following the development along historical lines, it must be acknowledged that the mathematical treatment as well as the psychologic interpretation has done more harm than good. The mathematical treatment has made its understanding needlessly complicated, while the psychologic explanations were mostly nothing else than disguised metaphysics — or "the filling of the belly with east wind" (Book of Job).

The times are ripe to treat the subject of binocular single vision according to the rules of the science to which the subject belongs. It is essentially a physiologic process, which must be interpreted from the evolutionist standpoint.

We find in the lower vertebrates the two eyes on the sides of the head; this position changes to a more forward one with an ever enlarging common field of vision until in the primates we see the highest development. Every animal which has two eyes has binocular vision, as this term means only "vision with two eyes." We should therefore not use this term indiscriminately for binocular single vision. We cannot know how these animals see, just as we do not know whether or not those animals which have their eyes in the front part of their head can appreciate depth and solidity, as we do. A part of this lower form of vision, from the human side, or rather fundamental form of vision, is still found in man, namely, we have a simultaneous impression in the temporal parts of both fields independent from each other. In man we find a common field of vision, a part of which possesses a new faculty, which is absent in the single eye — anyhow to the perfection as it exists in both — namely, of appreciating depth and solidity, sometimes called "stereoscopic vision." It will therefore not be strange to find different degress of vision, from simultaneous vision with alternating macular perception and slight or no appreciation of depth to the highest acuteness of the appreciation of the distance of two points, a quarter of a minute being the size of the angle under which the two eyes can separate the position of two points, under favorable conditions. The great superiority of man over the lower animals is the potential faculty of a continuous development during his individual life. His newly acquired faculties are the least stable, are more apt to develop under individual effort, are more vulnerable in the upgoing line as well as in the downgoing one, with other words, a small obstacle may prevent the full development, as well as the fully developed acquisition can be more easily damaged. From this it results that the apparatus for binocular single vision can be perfected through exercise, and also that it can more easily change independently of

the apparatus of simultaneous vision, which is the older and the more stable through heredity.

When we see, we react to a stimulus of the outside world. Electromagnetic vibrations, which go out from some outside object, stimulate the retina, which stimulation is propagated to the brain. Here a double process occurs, a sensory and a neuromotor. The sensory process travels through cerebral paths, reaching the stimulated part of the retina, and then projects into the outside world its image, which under normal circumstances covers the object with mathematical precision. The neuromotor part directs the position of the visual lines. We know not the external world as it is, we see because we have eyes. "The eye creates the light." This does not mean that no objective world exists; it means that our appreciation of the world is entirely subjective. This conception presupposes that the line of impression (the line from the luminous object toward the corresponding point in the retina) is not the same as the line of projection (the line from the retinal point toward the corresponding point of the image). This difference becomes clear for the condition, when, for instance, the eye is displaced by mechanical means, or when one looks through a prism: we see then the image where nothing exists and where the object is we see nothing. We should not speak of a prism which "deflects the rays of light toward its base," but a prism which moves the image in the direction of its apex. The difference of the line of impression and the line of projection becomes especially valuable for the understanding of what happens, when we see stereoscopically. The projection of the visual image is not an "act of judgment," but the product of a sensory reaction and the result of an organic property of the nervous elements. The projection is inherent to the function of the visual apparatus; it follows with every luminous stimulation or mechanical excitation of the retina; it follows by necessity, automatically.

Wheatstone gave all the essential facts of the stereoscopic process when in 1838 he published in the *Philosophical Transactions* his "Contributions to the Physiology of Vision."

Euclid, more than 2,000 years ago, knew that the two eyes see two dissimilar pictures or portions of bodies. Galen has described with great minuteness in his work, "De Usu partium corporis humani," the various phenomena which are seen when we look at bodies with both eyes, and alternately with the right and left. We also find this dissimilarity mentioned by Leonardo da Vinci (after Brewster).

On account of its importance I will quote now Wheatstone (his entire essay should be reprinted as a classic):

If the optic axes of both eyes are parallel, the perspective projections of it, seen by each eye separately, are similar, and the appearance to the two eyes is precisely the same as when the object is seen by one eye only. There is, in such case, no difference between the visual appearance of an object in relief and its perspective projection on a plane surface. But this similarity no longer exists, when the object is placed so near the eyes that to view it the optic axes must converge; under these conditions a different perspective projection of it is seen by each eye, and these perspectives are more dissimilar as the convergence of the optic axes becomes greater. It being thus established, that the mind perceives an object of three dimensions by means of two dissimilar pictures projected by it on the two retinae.

I have given ample proof that objects whose pictures do not fall on corresponding points of the two retinae may still appear single (Fig. 1 *A*). Similar pictures falling on corresponding points of the two retinae may appear double and in different places (Fig. 1 *B*). The two strong lines, each seen by a different eye, will coincide, and the resultant perspective line will appear to occupy the same place as before; but the faint line which now falls on a line of the left retina, which corresponds with one of the coinciding strong lines, viz., the vertical one appears in a different place. . . . This experiment affords another proof that there is no necessary physiological connection between the corresponding points of the two retinae—a doctrine which has been maintained by so many authors. . . . Many of the supporters of the theory of corresponding points have thought, or rather have admitted, *without thinking,* that it was not inconsistent with the law of Aquilonius. . . . The same reasons, founded on the experi-

ments in this memoir, which disprove the theory of Aquilonius, induce me to reject the law of corresponding points as an accurate expression of the phenomena of single vision. According to the former, objects can appear single only in the plane of the horopter: according to the latter, only when they are in the circle of single vision, both positions are inconsistent with the binocular vision of objects in relief, the points of which they consist appearing single though they are at different distances before the eyes. I have already proved that the assumption made by all the maintainers of the theory of corresponding points, namely, that the two pictures pro-

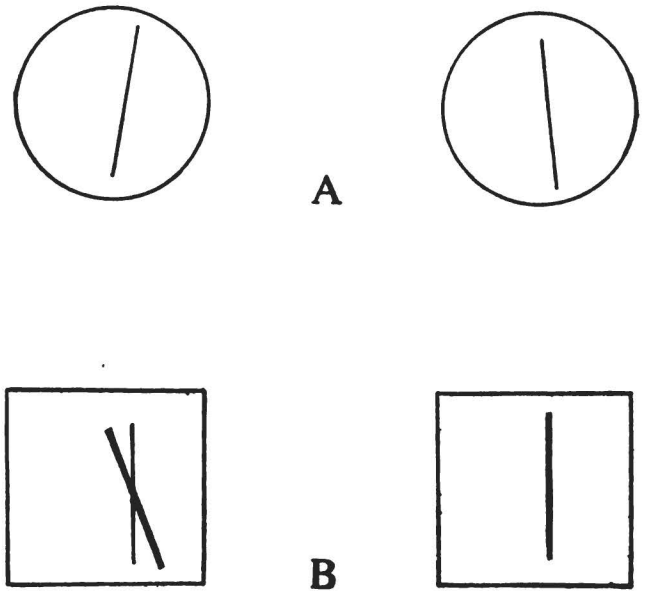


Fig. 1.

jected by any object in the retinae are exactly similar, is quite contrary to fact in every case, except that in which the optic axes are parallel.

It now remains to examine *why* two dissimilar pictures on the two retinae give rise to the perception of an object in relief. . . . The complete solution is far from being so easy as at a first glance it may appear to be, and is indeed one of great complexity. I shall merely show the most obvious explanations which might be offered, and show their insufficiency to explain the whole of the phenomena.

It may be supposed that we see but one point of a field of view distinctly at the same instant, the one, namely, to which

the optic axes are directed, while all other points are seen indistinctly, that the mind does not recognize them to be either single or double, and that the figure is appreciated by successively directing the point of convergence of the optic axes successively to a sufficient number of its points to enable us to judge accurately of its form. . . . Were it entirely so, no appearance of relief should present itself when the eyes remain intently fixed on one point of a binocular image in the stereoscope ("the instrument which has the property of representing solid figures"). . . . Were the theory of corresponding points true, the appearance should be that of superposition of the two drawings, to which, however, it has not the slightest similitude.

Another and a beautiful proof that the appearance of relief in binocular vision is an effect independent of the motions of the eye, may be obtained by impressing on the retinae ocular spectra of the component figures. The drawings should be made of broad colored lines on a ground of the complementary color, for instance, red lines on a green ground, viewed with the eyes fixed only to a single point of the compound figure; the drawings strongly illuminated and after a sufficient time the eyes must be carefully covered to exclude all external light. A spectrum in relief will then appear before the closed eyes: sometimes the right eye spectrum will be seen alone, sometimes that of the left eye, and at those moments when the two appear together, the binocular spectrum will present itself in bold relief. As in this case the pictures cannot shift their places on the retinae in whatever manner the eyes be moved about, the optic axes can during the experiment only correspond with a single point of each. When an object, or a part of it, thus appears in relief while the optic axes are directed to a single binocular point, it is easy to see that each point of the figure that appears single is seen at the intersection of the two lines of visible direction in which it is seen by each eye separately, whether these lines of visible direction terminate at corresponding points of the two retinae or not. But if we were to infer the converse of this, viz., that every point of an object in relief is seen by a single glance at the intersection of the lines of visible direction in which it is seen by each eye singly, we should be in error. On this supposition, objects before or beyond the intersection of the optic axes should never appear double, and we have abundant evidence that they do.

Wheatstone showed, therefore, that corresponding points, which are points lying on the horopter of Johannes Mueller, are seen singly, but also points within or without are seen singly, only they are appreciated at a different place in space. He even puts

down the law of intersection of the secondary axes, elaborated later by Giraud-Teulon and Parinaud. For better understanding we need consider what happens when we see stereoscopically. This should be done without an instrument. However, the instrument as such does not produce the phenomenon; it only enlightens the procedure. We need a reproduction of an object as seen with the right eye and as seen with the left one; these pictures are slightly different. If we now relax our convergence so that we see with as much as possible parallel visual axes, four images will appear in crossed double images; those of the left eye are projected to the right, those of the right eye to the left. When the two middle images come together (fuse), we receive the sensation of depth quite involuntarily, automatically, independent from any muscular action, what Dove (1841) demonstrated by using the electric spark. The two lateral images play no part in the process, and they are the ones which are shut out by the partition of the stereoscope, which also by its prisms with the apexes inward facilitate the parallelism of the visual axes and through their positive lenses enlarge the impressions.

There is a reality in the unreality of these pictures. They are virtual images in the sense that they do not respond to an objective reality; they can be compared with refractive or reflected images; they can be measured experimentally and the conditions calculated which vary their localization in space, their size and their depth. If, for instance, we take the two pictures of a fulcrum, the distance at which the smaller circle is seen from the base depends entirely on the degree of eccentricity (stereoscopic parallax), their distance from each other. We need only to accept the fact, to which we have agreed regarding the chief axes of projection, that we see the object in space where they intersect, that this also exists for all secondary axes of projection (law of Giraud-Teulon and Parinaud). The production of the stereoscopic relief follows from

this law. If the larger circles fall on corresponding points, the smaller will before fusion form homonymous or heteronymous images and after fusion be projected behind or before the large circle. The position in space is determined by the intersection of the principal axes.

One difficulty remains. It is the well known phenomenon, that if, for example, two pins in the median line are looked at, which are behind each other, only one pin can be seen distinctly at the time, while the one in front of the fixed pin is seen in heteronymous double images and the one behind the fixed pin in homonymous double images. In general, if the second point lies before or behind the fixed point in such a way that it comes between both axes of projection or lies on one of these, then always double images are seen. The double images, which form from incitation of two disparate points in the two retinas are located on both sides of the macula in the cyclopean eye, while in all other cases, when disparate points are excited they belong to corresponding halves of the retinas and would be located on the same side of the fovea in the cyclopean eye. However, this cannot be the sole cause, for if the second point is brought out, by lifting from out the plane which goes through the visual axes and the fixed point, the stereoscopic effect appears directly.

The fusion of the retinal images is chiefly connected with the sensation of relief. To produce this sensation it is necessary that the retinal excitation solicits it, determining the cerebral reaction which causes it. This happens during natural seeing, and is particularly apparent when stereoscopic relief is artificially produced. Perhaps in the above mentioned experiment of the three pins we see the double images because the brain does not receive the excitation for corporeal relief.

Stereoscopic vision and normal binocular single vision are not the same. The stereoscopic relief is obtained with virtual images. The distinction between

the vision truly subjective, as is the stereoscopic vision and the natural vision, is the noncoincidence or the coincidence of the image of projection with object. When this coincidence is absent, when the image of projection is localized in a spot of space in which the object does not exist and the vision has no more reality, the difference is so little appreciated that all students of stereoscopic vision have identified it with the natural vision of an object.

It is thus the image of protection which makes the reality of the visual sensation. Where it is we have the perception of the object even if it is not there; where it is not we see nothing even if the object is there.

The apparatus of binocular single vision possesses two qualities, which are the cause of the production of stereoscopic relief, namely, the faculty to fuse the exteriorated images of disparate retinal points and the localization of the binocular images where the axes of projection meet, for the axes which start from disparate points, as well as from those which originate in corresponding points.

The identity of the retinas remains the fundamental property of the binocular single vision, but it must be separated from the conception of the horopter. The fusion of double images exteriorated by disparate points is the fundamental cause of the binocular perception of relief. Stereoscopic vision is an abnormal binocular vision, obtained through virtual images with false projection; that is, they do not correspond to the position in space of the objects which give rise to these images. It can be produced without the help of the convergence, which function is indispensable for normal binocular single vision.

Stereoscopic vision implies two distinct actions, namely, the relaxation of the convergence accompanied by a synergetic relaxation of the accommodation, which is an adaptation chiefly of the muscular appara-

tus and the fusion of disparate retinal points, which produces the sense of relief, and is a sensory property.

The connections which give rise to the production of identical points do not exist over the entire retina; they become less marked on moving away from the fovea. Schoeler found by fusing very simple stereoscopic pictures during instantaneous illumination this region 2.85 mm. in the horizontal meridian at each side of the macula — 6.20 mm. above and 3.10 mm. below it.

In that part of the retina in which there can be question of identity, the faculty to fuse the projections from disparate points is not evenly developed. Ole Bull found that in the horizontal meridian two points of the binocular visual field with a distance of 30 minutes can be fused, while in the vertical meridian this distance cannot exceed 20 minutes.

The horopter is an objective representation of the identity of both retinas — a name and condition for the first time used by François d'Aguilon or Aguilonius. He was a Jesuit priest and published in 1613 at Antwerp his "Opticorum libri six." He defines the horopter as a line drawn through the point of intersection of the optic axes, and parallel to the line joining the centers of the two eyes: the plane of the horopter being a plane passing through the line at right angles to that of the optic axes. He concluded *a priori* that all points which lie in this line are seen singly (*uno loco*), all external to this double *geminis locis*). The mathematical development of the horopter has made its understanding needlessly intricate; it should only facilitate the general comprehension by the exposition of facts. It should not be given too much importance, especially a physiologic significance, which has resulted in a wrong interpretation of the identity of the retinas by giving it an inflexible geometric formula.

The apparatus of binocular single vision has as its chief function the more precise localization in space of the visual sensation; combined herewith is the

appreciation of the third dimension, depth and distance. This can be done without any muscular movement, thanks to the different localization in space of the binocular images, according to their projection from corresponding or disparate points, which is the function of the sensory apparatus.

Only for vertical contours do we possess a faculty for distinguishing distances; for horizontal contours we do not possess with motionless view the possibility to discern distance, with the reservation, of course, that of three evenly thick bars, the one which is nearest appears the thickest. At a distance of 2.5 meters this does not have much influence. The depth perception as described is therefore a function of the vertical meridians — of the double eye (Heine).

In this description of facts which lead to an understanding of binocular single vision I have not given anything original. If you can agree to accept the explanation the honor should be due to Wheatstone and Parinaud. Mine has been the pleasure of working out the problem to my own satisfaction.

Habent sua fata libelli — and *autores!* Parinaud has not been appreciated as he should be. His dualistic theory of vision has been appropriated by the Germans, and I have found no trace of influence from his two books: "La vision" (1898) and "Stéreoscopie et projection visuelle" (1904), in this country. For me Parinaud possessed the distinct spark of genius; he left his chosen science richer than he found it; he was a noble representative of the true French scientist.

When we now come to examine the sense of depth we find that all tests given in the textbooks are only qualitative ones, no one quantitative test is presented, although it is possible to measure this sense just as accurately as the acuity of vision. A new field still lies fallow.

There are many factors which enter into consideration in our appreciation of solidity. Our appreciation of distance *qua talis* is far below our judgment of

reciprocal distance. In the immediate neighborhood accommodation may be of some value, and convergence for a little larger distance, say about 4 meters. However, of the greatest importance is the binocular parallax, the expression of the stimulation of disparate retinal points. It is the difference of the angles under which a distinct external point is distant from the binocular fixed point to the right or left for the right or left eye. Each one of the images projected by both eyes goes out from partly disparate retinal points. Even if the limit is transgressed wherein no double images are formed, we are able, although with less security, to perceive depth by the help of double images. The difference in identity of both retinal images is observed much more accurately as the eye is able to appreciate the distance between two points (form sense). With a basal line of 65 mm. and a binocular parallax of one minute we can judge of the distance of two points, one at a distance of 225 meters, and the other at infinity. No constant relation exists between the observed differences in depth and their physiologic substratum (the binocular parallax), so that a distinct value of the latter should produce a distinct difference in depth, even if all other influences for appreciation of depth are absent. The impression of the size which we receive from an observed distance depends next on the binocular parallax on circumstances which determine our impression of distance.

Another factor in the appreciation of depth is the length of the basal line; the more the eyes are removed from each other, the finer the appreciation of the third dimension, all other factors remaining the same.

Stereoscopic parallax is the apparent displacement for each eye of the objects in different planes, and the deformity of the perspective images which result from it.

On examining patients we find that the sense of depth is very differently developed in man. It can be absent with perfect visual acuity of each eye, or hardly

developed. As the appreciation of depth is a faculty developed on the condition of simultaneous vision with alternating macular perception, we shall better understand the different degrees of its acuteness. It can be compared with the appreciation of colors; here we find total absence of color perception, then the appreciation of only two colors, and between this condition and the full appreciation of all colors many different variations. Or it suggests a condition as is found in congenital word blindness, as first described by James Hinshelwood. Word blindness may be due to a congenital defect or deficiency in the brain center or to some pathologic process usually occurring in later life destructive to that center (J. C. Clemesha).

With the stereoscope we can only examine if "fusion" is present, that is, if the patient is able to blend two somewhat unequal pictures, as, for instance, F. and L. to E. But we can only guess as to the presence of appreciation of depth, when we have given two pictures, which by blending should produce this sensation. We cannot prove that the patient sees depth, as also we cannot prove that the patient sees the same color as we by giving the same name.

Hering's fall experiment, considered by nearly all as an infallible test, gives also at the utmost a qualitative appreciation. The principle seems not to be so simple, as on page 877 in the "American Encyclopedia of Ophthalmology" and in J. M. Ball's textbook it is stated that "the patient looks with one eye" through a long tube, etc. Whatever the value of the test may be, we should not forget that it can be positive when the patient cannot fuse in the stereoscope, and that one needs the cooperation of the patient as well as the control of both eyes with each eye separately.

The scientists who explored this field with modern means, Volkmann and others, used three objects (pins and threads) and looked at the displacement of the middle one or at two objects. This simple device can

be easily applied; we examine then the stereoscopic parallax. Heine used the three threads, and Brooksbank James made a portable apparatus. He uses two threads or rather wooden pins, puts them in a dark box with a front opening, and asks the patient to look at a distance of 6 meters and to state when he sees the one object back of the other. He and Schoute have published observations. These should be repeated and we should try to find the relationship between visual acuity, length of the basal line and appreciation of the third dimension.

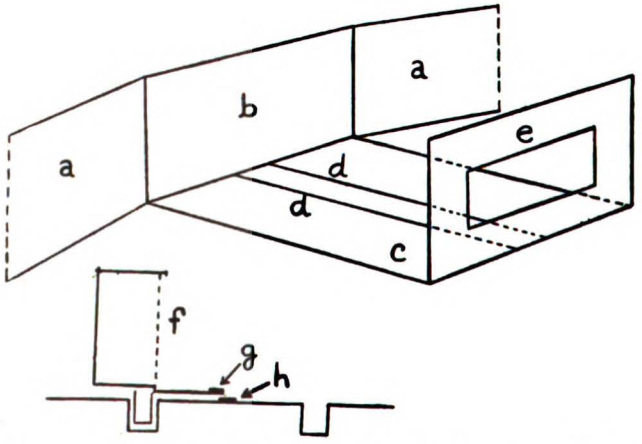


Fig. 2.—Box for examination of stereoscopic parallax: *a*, *a*, wings, *b*, back; *c*, base; *d*, *d*, grooves; *e*, front; *f*, thread; *g*, indicator; *h*, tape measure.

The binocular perception of depth or stereoscopic vision results from the inequality of both retinal images, which are received from a three dimensional object. If these two pictures should cover each other only points in one plane will cover each other exactly, all others will be separated by a distance, which Helmholtz called the stereoscopic parallax. To examine the perception of depth one can put three objects in the same frontal plane and determine how much the middle one must be moved forward or backward to be distinctly perceived in front or behind this plane.

The stereoscopic parallax can also be examined by taking two objects. G. T. Brooksbank James took a box, of which he removed the sides and roof (Fig. 2). In the front wall he

makes a good sized opening; the whole inside is made black. In the floor are two longitudinal grooves in which run two carriers to support the test objects; the grooves are placed at equal distances from the margins of the window and there is an interval of 50 mm. between them. At the suggestion of Schoute the test objects are white threads.

For clinical observations the box is placed at a distance of 6 meters from the patient. The box is lighted uniformly, slight movements of the head are without consequence; the accommodation is completely excluded; the perception of the relative position of the threads depends entirely on the binocular parallax.

L and R (Fig. 3) are the nodal points of both eyes. The distance La = Rd = 6 meters; cd is the chord of the angle of the stereoscopic parallax bLd.

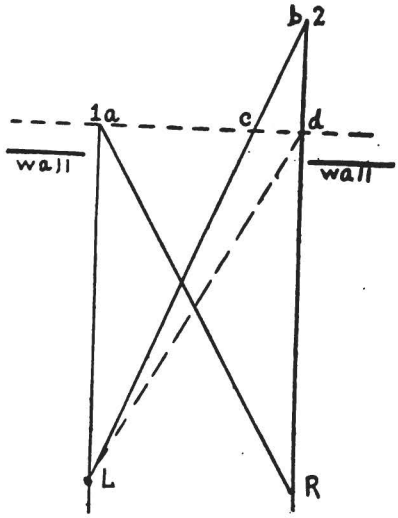


Fig. 3.

Method of Examining.—The patient sits at 6 meters. One eye is occluded by a blinder. He is asked how he sees the two threads, in one level or one closer than the other. He should be told not to guess, but to state what he sees. Then the question is repeated with both eyes open. The head is kept steady.

cd is the stereoscopic parallax:

$$\frac{ac}{aL} = \frac{cd}{bd}. \quad \frac{ad-cd}{6{,}000} = \frac{cd}{bd}.$$

$$cd = \frac{bd \times ad}{6{,}000-bd}.$$

CONCLUSIONS

1. The term "binocular vision" should not be used for binocular single vision indiscriminately. We must make a sharp distinction between simultaneous vision with alternating macular perception, with absence of fusion in the stereoscope, and binocular single vision.

2. A closer study of the presence of the faculty for appreciation of the third dimension may change somewhat the percentage of hereditary cases in squint; we shall probably find in the ascending line patients who do not squint but in whom this sense for depth is not or imperfectly developed.

3. Its presence or absence may explain the different results after correction of both eyes in anisometropia. It also will explain why some patients with important muscle imbalance have no pathologic symptoms. Operations on the external muscles should not be done unless the "wish" for binocular single vision can be demonstrated. The indication for cosmetic effect is excluded.

4. The appreciation of the third dimension has a high practical value; hence its loss should be compensated for whenever possible; but it is necessary, therefore, to know before the accident that the man had binocular single vision and its degree, so that later the loss can be expressed mathematically. The appreciation of the third dimension is present with different visual acuity, in different degrees. Good visual acuity does not mean also good appreciation of depth.

5. We shall have to reconsider the tests for malingering, as they start mainly from the supposition that binocular single vision has been present before.

6. Every thorough examination should include the examination for binocular single vision.

7. It would be laudable if the section could come to an understanding as to the kind of apparatus to be used.

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ABSTRACT OF DISCUSSION

DR. WALTER B. LANCASTER, Boston: Dr. Blaauw has opened up a large field of debatable ground. It is doubtful if his criticism of the statements in standard textbooks gives a fair impression. In most cases he has not specified in just what respect the author is at fault. On the whole, it seems that the chief fault found is in a different use of terms. However desirable it is to have uniformity and logical consistency, we know that these are not possible with human nature as it is, and we should be satisfied if an author gives a correct idea even if he does not express it as we should like to have him. In other words, I think Dr. Blaauw has tried to prove too much in assailing so large a majority of standard authors.

His suggestion that we approach the problems of binocular vision from the point of view of physiology and evolution rather than mathematics, is a good one. To be sure, we have to depend on our imagination when we try to picture the visual functions of other animals, but if we keep in mind the limitations and do not attempt to found a rigid proof on wholly unproved assumptions, we can derive valuable aid from this use of our imaginations.

The early forms of eye in vertebrates probably did not have any definite macular region. Vision was much the same in all parts of the field. Gradually certain differences in sensitivity developed and it became possible for the animal to fix his gaze, that is, to so rotate the eye or head as to bring the image of the object which he wished to see on the most sensitive part of the retina. During these early stages of development a wide range of vision—a large field—was the most important function, and the situation of the two eyes on opposite sides of the head favors this wideness of field.

It is not difficult to see how, as the macular vision developed, it was necessary for the animal to concentrate his attention on one or the other eye, and keep the sensations of the macula of the other eye and the peripheral parts of the fields of both eyes in abeyance to suppress them in the interest of a more vivid consciousness of the sensations from the macula of the fixing eye. We have two hands, and if we plunge them both into a bag of sawdust we can easily concentrate our attention on the sensations of one or the other, as some object is touched which we wish to investigate. The sensations from the other hand are suppressed but not extinguished. Amplify the analogy for yourselves.

By and by, as the eyes become more anteriorly located, their visual fields overlap more and more, and it is as if we were feeling of the same object with both hands. It is necessary to avoid the mistake of interpreting the two images as two separate objects, and this faculty develops. Meantime, when in doubt, the uncertainty is solved by fixating the object,

that is, causing its image to fall on the most sensitive part of the retina, the most highly developed part for clear vision.

As the eyes are placed more and more around toward the front of the head the overlapping portion of the fields increases until the larger part of the field is common to both eyes—is binocular.

It is possible now for both eyes to fix the same object, and the consciousness can receive two distinct or macular images of the same object at the same time. At first, no doubt, the habit of suppressing one or the other prevailed, as we now see it in the phenomena of antagonism or conflict of the visual fields. As the two images do not necessarily conflict in most cases, it became unnecessary to suppress one as it formerly was, when they were always different and so did conflict. The faculty of binocular vision was advanced a step. Two clear and vivid simultaneous sensations of the same identical character would seem to offer no difficulty to the consciousness which the animal could not learn to overcome. And so the fusion faculty arose.

But to speak figuratively, the consciousness soon detected slight differences in the two images under certain conditions, namely, when the object looked at was not too far off. For example, in looking at two sticks placed one in front of the other, it is noticed that with the right eye A is to the right of B, while with the left eye A is to the left of B. It is noticed that this always occurs when B is nearer the eye than A. This is the basis of the perception of depth. It is not a function of the eyes. This fusion of two sensations with a third, distinct from either, is a matter of the consciousness—is psychic, not ocular. It has been called by various names. When discussing this subject at the 1914 meeting, I called it "binocular fusion for depth." The best name proposed is that of McNab, "psychical synthesis." (See Transactions of Lohman, "Disturbances of the Visual Functions," page 145.) A firm grasp of this conception is all that is necessary to solve most of the puzzles of binocular vision. In so far as the two images on the two retinas are identical, as is the case when the visual axes are parallel, as in looking at a distance or as is the case when looking at a flat plane surface, such as the page of a book held at right angles to the line of vision, binocular vision simply gives a more intense sensation than would be received if one eye were suppressed or occluded. But when the images are different in the two eyes because they are not looking at the objects from the same point, that is, when the object is near enough so that the visual areas converge and one eye looks at the object from one angle and the other from a slightly different angle, the consciousness is able by a synthetic process to combine the two images into a single mental image with the added property of depth or third dimension. The sensitiveness to differences of the two images showing differences of

depth is very great. It is the most delicate of our space perceptions. Whereas the standard of visual acuity is taken as a resolving power of 1′, angular differences as small as 15″ or even less are detected by stereoscopic methods.

As to the great value of stereoscopic vision, I think it can be overestimated and often is. If you take the testimony of those who lack it, you certainly would not rate the function very high, for they are not conscious of any deprivation in the vast majority of cases. This is not a fair test, however. What we should do is to take the testimony of those who, having possessed and used it, have lost stereoscopic vision (binocular single vision, as Dr. Blaauw would prefer to have us say) through loss of one eye. These often complain bitterly, but as a rule they learn to get on very well, and I should not rate the function as by any means an indispensable one.

Dr. Frederick H. Verhoeff, Boston: I agree with Dr. Blaauw that ophthalmologists in general have given very little attention to stereoscopic vision and know very little about it. It would take too long to discuss the general aspects of the subject, but I should like to call attention to a simple test for stereoscopic vision that I published about thirteen years ago, which seems to have been generally overlooked. Most tests for stereoscopic vision are based on the principle either of the bird in a cage or of stereoscopic pictures of a cone. The first, as Dr. Blaauw has pointed out, is no test of stereoscopic vision at all. The second is of uncertain value because a patient with one eye may guess correctly. The test that I have described cannot be passed unless the patient has stereoscopic vision. If we can explain the phenomenon exemplified by this test we have a thorough idea of the stereoscopic vision. The test in its simplest form may be represented by two figures, one a circle with two vertical parallel lines passing through it, and the other a circle with a single line passing through it which is bent in the middle. Put these two figures in the stereoscope and ask the patient what he sees. If he sees with one eye he will describe the corresponding figure, if he sees with both, both parallel lines appear bent in the middle. The only place the picture he sees exists is in the brain—in what I regard as a special center for stereoscopic vision.

Dr. George T. Stevens, New York: Dr. Blaauw tells us that "following the development along historic lines, it must be acknowledged that the mathematical treatment as well as the psychologic interpretation (of binocular single vision) has done more harm than good," and he declares that the subject of binocular single vision is "essentially a physiologic process." Yet he suggests for our consideration but one practical method for the determination of the presence or absence of binocular single vision; that is, by means of the

binocular parallax, which is a purely mathematical process. Then, returning to the question of a psychologic interpretation of the phenomena of binocular vision, which he characterizes as "filling the belly with east wind," how does he interpret the well known experiment of Dove, which he quotes approvingly, by any process except physiologic? There is, in that case, no movement of the eyes, conscious or unconscious; no physiologic contact of physical impressions. We have a combination of psychologic phenomena with a physical basis, on the same principle that every psychologic process must and invariably does have. Dr. Blaauw remarks, "Stereoscopic vision implies two distinct actions, namely, the relaxation of the convergence, accompanied by a synergetic relaxation of the accommodation, which is an adaptation chiefly of the muscular apparatus and the fusion of disparate retinal points, which produces the sense of relief, and is a sensory property." We might perhaps be permitted to regard the estimation of the force expended or demanded by a given "adaptation of the muscular apparatus" as to a large extent a psychologic process, but just here we are more interested in the radical difference in the teachings of Wheatstone (whose entire essay Dr. Blaauw says "should be printed as a classic," in which I quite agree with him), and the statement of Dr. Blaauw above referred to. Quoting, then, from Wheatstone, we read: "Suppose two stereoscopic pictures . . . are presented to the two eyes, it is possible, by an effort, so to converge the eyes as to throw the images on corresponding points, and when this is done the objects are seen in relief." Now, there would seem to be some disparity in the statement quoted from Dr. Blaauw and that just cited from Wheatstone. Perhaps we may in some measure bring the two into a show of harmony if we remember that persons with a certain degree of exophoria easily obtain binocular single vision of stereoscopic pictures by directing the eyes, not necessarily in divergence or even in parallelism, but with only a slight or moderate convergence, the psychologic effect of the effort of convergence actually taking the place of the physical act of convergence spoken of by Wheatstone, thus offering another illustration of the psychologic element in single binocular vision. The fact is that under all ordinary circumstances we call on both physiologic and psychologic activities to form our visual impressions of external objects, and to express in definite form the facts revealed to us by these two processes we must call to our aid the principles of mathematics. Dr. Blaauw tells us "The identity of the retinas remains the fundamental property of binocular single vision, but it must be separated from the conception of the horopter." This is true, just as a quart cup may be distinguished from its contents of beer, but unlike the quart cup, the horopter changes at every instant, while it is the contents that undergoes the changes within the cup. The assertion

that the appreciation of relief of stereoscopic pictures is an "abnormal binocular vision, obtained through virtual images with false projection," would apply equally to images of objects seen through different mediums or through the atmosphere in different states.

Dr. Francis Valk, New York: I think the time may come when it will be essential for us to have some ready means by which we can examine the true binocular vision and also the true sense of depth, or the third dimension of those who claim to be "one eye blind." I do not fully accept the theory of corresponding points of each retina. I am inclined to consider that we do not have corresponding points but that we do have corresponding regions, whose extent I am not prepared to say, and must leave that to science. But from my point of view, as I fuse the images, as shown in Dr. Blaauw's paper, A and B, and can then move my eyes to the right or left without the loss of this fusion, or stereoscopic vision, it seems to me that this stereoscopic image must be controlled by certain regions of my retina and not points. I am looking for Dr. Blaauw to explain this as well as the fact that I do it without the use of my accommodation. To assist in the study I would show to the section Pfalz' stereoscoptometer. The use of this seems to me a much more simple procedure than that of Hering's falling balls or that of the ingenious box that is illustrated by Dr. Blaauw. It is always ready for use and requires no special preparation. The person to be examined sits in front of the little screen, about 2 or 3 feet distant. He sees nothing but the screen and the two wires with the little balls on the ends, and is asked to state quickly the position of the balls. As soon as he answers he then looks away for a moment, and while doing so we quickly change the position of the balls, and he is again questioned. We try this several times and note the average of his correct answers. A scale indicates the distance the balls are apart, which can be quickly noted with his answers. I think this instrument may be useful in testing the binocular vision and the sense of depth, when we estimate the loss of working power in one who may claim a loss from an accident, and also, if that loss will be, or is, permanent. Perhaps this may answer Dr. Blaauw's seventh conclusion. Although one may claim a certain loss of earning power, yet that test will prove that the loss of the sense of depth may not be permanent. The loss of an eye may be quite serious and may for a time cause some inconvenience to workmen, yet can we not prove that in time, even with one eye, they seem to regain this sense of depth and will possibly give correct answers to these tests? I have verified this with the instrument of Pfalz.

Dr. Linn Emerson, Orange, N. J.: The dispute of Dr. Blaauw with some authorities on the presence or absence of

stereoscopic vision, is purely arbitrary and due to different interpretation. Worth arbitrarily gives three grades of fusion, but in reality there are "57 varieties" or grades, varying all the way from the faintest simultaneous macular perception, to those who have complete, instantaneous fusion, with perspective. In eliciting the lower grades of fusion, I have found nothing so satisfactory as the amblyoscope of Worth, varying the illumination of the test object before the amblyopic eye, either with the lighting device of Black, or varying the distance from an electric light set in the side wall of the consulting room.

Dr. Elmer G. Starr, Buffalo: I think Dr. Blaauw has done good work in calling attention to the distinction between binocular vision and binocular single vision. We are apt to overlook the fact that we may have binocular vision but not true stereoscopic vision. It is true this subject is vast and obscure in its nature. The work to which Dr. Blaauw calls attention, that of Wheatstone in 1835 or 1838, is still the basis of almost all our deductions. I think some of Wheatstone's conclusions have been questioned by Hering, and I know in my own case I do get stereoscopic vision, a sense of solidity, even where the two images fall on identical points and not on disparate points of the retina as Wheatstone laid down as the rule. In certain conditions of light, and with magnification, I get a true sense of solidity with a single eye. That merely shows how obscure the whole subject is. But when it comes to the practical point I think some of Dr. Blaauw's suggestions are very good indeed; and it seems to me the instrument shown by Dr. Valk should be of great value to us in our tests. I also make use of the fusion of two dissimilar charts to be used in the stereoscope as shown by Dr. Verhoeff; but I believe in many instances the patient may have imperfect distant fusion when he fuses two images at the near point.

Dr. Lucien Howe, Buffalo: I rise only for a question. Why do the abducting prisms not cause a certain amount of abduction, and why does not the convex glass, which is a part of the stereoscope, lessen the amount of effort for accommodation that we make?

Dr. Edmond E. Blaauw, Buffalo: I will answer the last question first. The use of the stereoscope with two pictures would be nothing different than without the stereoscope. Stereoscopic vision with the stereoscope should not be compared with binocular single vision. In the latter we must have normal accommodation and convergence, which is not present in stereoscopic vision. Therefore, the question of depth should not be called stereoscopic vision. I am especially gratified at the discussion of Dr. Stevens who has such great interest in the scientific aspect of the subject. I agree with him that this is psychologic and physiologic both.

James' apparatus has this advantage, that when we test for binocular single vision we do not see the top or bottom of the threads looked at, which cannot be said of Pfalz' stereoscoptometer.

I cannot agree with Dr. Lancaster, but if I would show everything in which I disagree with the textbooks it would be almost in every sentence. Every one who will study the question will in a short time agree with me. This chapter, as it is written, does no credit to ophthalmology.

THE TREATMENT OF HYPOPYON KERATITIS *

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Of the serious corneal ulcers the most frequent are those due to pneumococci, while next in frequency of occurrence are those due to diplobacilli. The characteristics of the pneumococcus ulcer, or ulcus serpens, are so well known that it is not necessary to discuss them here at length, but it may be well to emphasize certain features. Undoubtedly the most common cause for this type of ulcer is trauma, but there are two other causes that are often not recognized, namely, herpes corneae and glaucoma. Peters,[1] in fact, insists that the peculiar character of ulcus serpens is dependent on a neuropathic basis. This I think is doubtful, but it is probable that in many cases corneal herpes permits the pneumococcus infection to take place. After the infection has become well established the herpetic condition is obscured and so escapes recognition. The course of these cases, however, usually seems to be no different from the frankly traumatic cases. In cases due to glaucoma the failure to recognize the cause may prove disastrous. In many of the eyes that have been removed for perforating ulcer of the cornea and submitted to me for examination I have found deep cupping of the optic disk, although the glaucomatous condition was previously unsuspected. This mistake will less frequently be made if the tension of the affected eye is taken with the finger and the other eye is carefully examined for

* From the Massachusetts Charitable Eye and Ear Infirmary.

1. Peters: Ueber traumatische Hornhauterkrankungen und ihre Beziehungen zum Herpes Corneae, Arch. f. Ophth., 1904, **57**, 93.

evidences of glaucoma. The tonometer should not be used on an infected cornea, even after it has apparently healed, as the traumatism may have an unfavorable effect.

As a rule an ulcus serpens appears less serious than it really is. This is due to the fact that the leukocytic infiltration is often relatively slight and is most marked at the periphery of the ulcer, leaving the center comparatively clear. The central clear area, however, is really the most severely damaged part of the cornea, for it is almost completely necrotic. The pneumococci are usually most abundant in the infiltrated border of the ulcer because those in the center are cast off with the superficial layers as loss of substance occurs. In rapidly advancing cases, however, before much loss of substance has taken place, I have found the surface layers of the central portion loaded with pneumococci. The center of the ulcer may later appear more opaque owing to the deposition of fibrin and pus cells on Descemet's membrane behind it. This is analogous to thrombus formation on the injured wall of a blood vessel.

Although ulcus serpens is now known to be due to the pneumococcus, yet much bacteriologic work remains to be done in regard to it. The fact that ulcus serpens never occurs in cases of pneumococcus conjunctivitis, while it is not infrequent in cases of dacryocystitis, would seem to show that some special strain or strains of pneumococci are required to produce it. The fact, too, that ulcus serpens is rare in children, although the latter are especially susceptible to pneumonia and to pneumococcus conjunctivitis, also seems to point in the same direction. It is possible, on the other hand, that this may be due to the fact that children do not often sustain the kind of injuries most likely to cause ulcus serpens, and that they are seldom affected with corneal herpes or glaucoma. So far as I know, however, no careful bacteriologic investigation of this question has yet been made. In view of the beneficial

results of serum treatment obtained by Cole at the Rockefeller Hospital in cases of pneumonia due to certain strains of pneumococci, it is possible that such an investigation would be of value from the standpoint of treatment of corneal ulcers.

The diplobacillus ulcer differs considerably in its clinical appearance from ulcus serpens, but if not properly treated is fully as destructive to sight. There are several varieties of diplobacilli that may cause corneal ulcers, the most common being the Morax-Axenfeld. All such ulcers seem to have about the same clinical characteristics, so that for practical purposes the diplobacilli may be identified with sufficient accuracy by the examination of smears. It has been stated that it is impossible to differentiate by clinical appearances alone between pneumococcus and diplobacillus ulcers, but this is not in accord with my own experience. Perhaps the most striking feature of the diplobacillus ulcer is the relatively slight general reaction of the eye compared to the extent of the ulcer. This is manifested especially by the size of the hypopyon, which is relatively very small. The ulcer itself is more uniform in appearance than an ulcus serpens, the marginal infiltrate being less marked and the whole ulcer having a gelatinous appearance, due to plastic exudate.

The treatment of hypopyon keratitis has hitherto been highly unsatisfactory, judging by the fact that out of all the diverse methods of treatment no single one has been generally adopted. It would be impracticable here to discuss each of these methods in detail, and I have therefore attempted to classify them as follows:

Symptomatic Treatment.—This includes the use of an occlusive bandage, hot applications, and atropin to combat the iritis. The use of various nonirritating ointments may also be included under this head, since, judging by the ineffectiveness of still stronger antiseptics, their only value is to act as lubricants. Ethylmorphin hydrochlorid (dionin) is also frequently used,

and some surgeons employ miotics instead of atropin. This method of treatment undoubtedly yields a fair percentage of relatively good results, a fact that is apt to be lost sight of when estimating the value of other methods.

Serum Therapy.—The antipneumococcus serum of Roemer has not yielded the results anticipated, and has therefore never come into general use. As already pointed out, however, in view of the results obtained in cases of pneumonia at the Rockefeller Hospital, there is still a possibility that an immune serum may be developed that will prove effective in corneal ulcers due to certain strains of pneumococci.

Vaccine Therapy.—If vaccine therapy is under any condition effective against pneumococci, it should be especially valuable in cases of ulcus serpens, for here we have a relatively minute localized focus insufficient to cause any appreciable systemic reaction. Nevertheless, no conclusive evidence has been brought forth that vaccines are of the slightest benefit in cases of ulcus serpens. Recently interest has been shown in the nonspecific treatment of infectious diseases by injections of other foreign proteins, but I am not aware that it has yet been tried in cases of corneal ulcer.

Chemotherapy.—As regards the treatment of localized infections in general, the ideal germicide, of course, would be one which destroyed the micro-organisms without seriously injuring the tissues. In 1907, with E. K. Ellis,[2] I made experiments with a large number of antiseptics claimed to be noninjurious to the tissues, and found that in every case their germicidal activity was destroyed by serum. This explained at once their ineffectiveness against infections as well as their nonirritating qualities. It seems to me that it is altogether unlikely that any chemical substance of simple structure will be found to fulfil the ideal requirements.

2. Verhoeff, F. H., and Ellis, E. K.: The Bactericidal Values of Some Widely Advertised Antiseptics, THE JOURNAL A. M. A., June 29, 1907, p. 2175.

In any case, dilute solution of the germicide and prolonged exposure to its action would probably be necessary for differential effects as between the organisms and tissues. Thus far, no germicide fulfilling the foregoing requirements has been brought forward with the possible exception of optochin.

Ethyl-Hydrocuprein (*Optochin*).—This was introduced by Morgenroth as a specific germicide against the pneumococcus. It seems, however, to have proved a disappointment in the treatment of pneumonia, owing to the fact that it cannot safely be given in doses sufficiently large to be effective. It has also been used as a local application in the treatment of ulcus serpens, and almost all the reports of cases so treated have been highly favorable. When added to serum, the hydrochlorid is immediately and abundantly precipitated, so that it is evident that only a minute quantity could penetrate inflamed tissues. It is stated, however, that enough remains in solution to give a certain amount of germicidal effect. It seemed improbable to me that this could be sufficient to be of service in the treatment of ulcus serpens, and I have therefore waited for more conclusive evidence before personally giving optochin a trial. In the meantime I have noted its action in a few cases of my colleagues in which it has been highly ineffective. It is noteworthy that even those reporting favorable results admit failures in certain cases. These are explained by assuming that the organisms develop tolerance toward the drug. I suspect, however, that the real difficulty is that the germicide cannot reach the organisms when the cornea is deeply infected. This is suggested also by the fact that many treatments are necessary, whereas if the germicide actually reaches the organisms in effective concentration, as supposed to be the case, it should be possible to sterilize the ulcer in one or two vigorous treatments.

Organic Silver Salts.—The best known of these are argyrol and protargol, but many others have been

brought forward. Pitzman[3] has pointed out that their germicidal properties depend entirely on the amount of uncombined silver salt they contain. It is obvious that their irritating properties depend on this also. Thus argyrol contains a very small amount of uncombined silver salt and hence is almost nonirritating and nongermicidal, while protargol, which contains a relatively high proportion, is correspondingly more highly germicidal and also more irritating in solutions of the same concentration. So far as germicidal activity goes, the large amount of combined silver in argyrol is wasted. In other words, if solutions of each of the various organic silver compounds were made in such concentrations as to have the same irritating properties, they would each have practically the same germicidal powers. In 1906 I showed that the germicidal properties of argyrol and protargol are destroyed by serum, so that it is impossible for them to kill bacteria within inflamed tissues even if they reach the organisms.[4] Although it seems certain, therefore, that they are valueless in the treatment of corneal ulcers, their use for this purpose has not even yet been abandoned.

Tincture of Iodin.—This has been highly recommended for the treatment of corneal ulcers, but I fail to see how it can be of service except in the most superficial infections. While it is highly germicidal to bacteria immersed in it, as ordinarily applied to corneal ulcers, it cannot penetrate the tissues effectively. When it comes in contact with moist surfaces the alcohol is diluted and the iodin precipitated, or the alcohol precipitates the albuminous bodies encountered and is thus checked in its penetration. In this connection the following experiment seems to be of considerable significance: I applied tincture of iodin to a large colony of staphylococci growing on coagulated blood serum,

3. Pitzman: The Antiseptic and Germicidal Properties of the Silver Salts and Preparations, Am. Jour. Ophth., 1912, **29**, 1.

4. Verhoeff, F. H.: A Further Note on the Antiseptic Properties of Sodium Aurate, Correspondence, THE JOURNAL A. M. A., May 12, 1916, p. 1462.

allowing the solution to act for over two minutes. On then making a culture from the colony, I obtained an abundant growth. Unless, therefore, the solution is applied sufficiently long and in sufficient abundance as markedly to injure the tissues, no important germicidal action can be expected from it. This conclusion, I may say also, is in accord with my practical experience.

Zinc Sulphate.—Axenfeld[5] states that he has never failed to check the progress of a diplobacillus ulcer of the cornea by means of zinc sulphate. He uses strong solutions applied directly to the ulcer and weak solutions instilled into the conjunctival sac. A few observers report unsuccessful results with this treatment in severe cases. In laboratory tests zinc sulphate is said to have but slight germicidal action on diplobacilli, so that its therapeutic value is probably dependent on an inhibitory effect.

Anilin Dyes.—Pyoktanin (methyl violet) and methylene blue have been used in the treatment of corneal ulcers, but without marked success. Basic fuchsin and gentian violet have in recent years been advocated as effective nonirritating germicides, but experiments made by me go to show that their germicidal action is too feeble to promise any success in the treatment of corneal ulcers. Thus in 1 per cent. solutions I found that neither of these dyes would destroy the *Staphylococcus aureus* with exposure of over an hour. A saturated aqueous solution of methylene blue was not destructive to this organism after an exposure of four and one-half hours.

Ion Therapy.—The attempt to sterilize corneal ulcers by treating them with solutions ionized by an electric current seems to me irrational, for in this way the antiseptics are dissociated into simpler elements whose action on the bacteria and the tissues must be still less differential than the original solutions.

5. Axenfeld: Bacteriology of the Eye, translated by MacNab, New York, 1908, p. 301.

Thermotherapy.—The effectiveness of the thermocautery in the treatment of corneal ulcers has been well demonstrated by actual experience. The objection to its use is that so much tissue must be destroyed or injured in order to insure destruction of the organisms that excessive scarring results. Several attempts have been made to obviate this difficulty. Bourgeois[6] employed a blast of hot air, such as is used by dentists; Weekers[7] held the cautery point at a distance from the ulcer, while Wessely[8] employed a tube with a blunted point heated by steam. More recently Shahan[9] has made a careful experimental investigation along this line, and has devised an instrument by means of which he can accurately control the temperature to which the cornea is heated. He found, however, that with the moderate temperatures and relatively long exposures with which he worked, it was impossible to destroy bacteria within the cornea without severely injuring the corneal tissue.

Prince[10] has advocated what he terms the pasteurization of corneal ulcers. A conical shaped piece of copper is heated to a red heat and then held close to the ulcer until it cools. The treatment is given once or twice daily. Prince contends that the organisms are checked in their growth, thus allowing the natural defenses of the body better opportunity to cope with them. I have tried Prince's method both clinically and experimentally, and have reluctantly concluded that it is of little if any value. It was used in five clinical cases of ulcus serpens, all of a severe type. In two of these cases perforation occurred; in one, Guthrie-Saemisch section was required, and two came to enu-

6. Bourgeois: Nouvelles considérations sur le traitement des ulcères infectieux de la cornée, Ann. d'ocul., 1899, **122**, 55; Le chauffage des ulcères infectieux de la cornée, ibid., 1911, **145**, 273.

7. Weekers: Nouveau traitement des ulcères serpigineux de la cornée par le chauffage, Bull. de la Soc. belge d'ophth., 1910, No. 28, p. 70.

8. Wessely: Zur Behandlung des Ulcus serpens, Ophth. Cong. Heidelberg, 1912, p. 339.

9. Shahan: Effects of Heat on the Eye, THE JOURNAL A. M. A., Aug. 5, 1916, p. 414.

10. Prince: Pasteurization in the Treatment of Corneal Ulcers, Ophth. Rec., 1916, **25**, 177.

cleation. Experimentally I tested out the method by injecting staphylococci into each cornea of a rabbit and then "pasteurizing" one of them. I found that when care was taken to inject about the same number of organisms in each eye, the resulting abscesses were practically identical in size.

Working with Dr. C. E. Hill, I have also investigated the possibility, suggested previously by Shahan, that intense heat briefly applied might be more injurious to bacteria than to the cornea. It seemed to me that radiant heat offered the greatest possibility of success, since, owing to the transparency of the normal cornea, there would be a selective action on an ulcer owing to the absorption of the rays by the infiltrate, and since also, working with Bell,[11] I had found that the effect of radiant heat was greatest in the deepest layers of the cornea. We therefore made use of a carbon arc and lens system which delivered enough radiant heat at the focus to ignite a match almost instantly. The experiments were made by injecting staphylococci into one cornea of a rabbit and then exposing each eye in turn to the light for the same length of time, which was varied in different experiments. We found that even when the control cornea was severely injured by the heat, an abscess nevertheless developed in the infected cornea. These experiments seemed so conclusive that it appeared useless to proceed further. If they had resulted more favorably, it was our intention to standardize the apparatus and also to attempt to devise some method for protecting the iris and fundus of the eye from injury.

Phototherapy.—Hertel[12] in 1903, and again in 1907, brought forward what he regarded as conclusive evidence of the value of ultraviolet light in the treatment

11. Verhoeff, F. H., and Bell, Louis: The Pathological Effects of Radiant Energy on the Eye, Proc. Am. Acad. Arts and Sc., 1916, **51**, 692.

12. Hertel: Experimentelles über ultraviolettes Licht, Ber. u. d. 31 Vers. d. ophth. Ges., Heidelberg, 1903, p. 144; Experimentelles und Klinisches über die Anwendung lokaler Lichttherapie bei Erkrankungen des Bulbus, Arch. f. Ophth., 1907, p. 275.

of corneal ulcers. I have elsewhere discussed Hertel's experiments and have pointed out that none of them were conclusive. In addition I have recorded a series of experiments[13] which prove that it is impossible to destroy bacteria within the cornea by means of ultraviolet light without severely injuring the cornea. In most of these experiments the cornea was exposed to the light immediately after it had been injected with bacteria, that is, while it was still transparent. If under these conditions the bacteria could not be killed without undue injury to the cornea, it is obvious that it would be futile to attempt to kill them in a cornea infiltrated with pus cells and so made practically impassable to germicidal rays. In fact, by the use of suitable screens, I found that the relatively long ultraviolet waves, the only ones that are able to penetrate the cornea, are much less injurious to the bacteria than to the cornea.

Cauterization.—Various chemical caustics have been advocated in the treatment of corneal ulcers, and probably any of them will check the ulcerative process if properly used; but the thermocautery seems to be generally preferred. Of the chemical caustics, pure phenol (carbolic acid) is probably as good as any, if not better. It has been shown experimentally by Guillery[14] that mineral acids should be avoided, owing to the danger of injuring the lens. The chief objection to the use of caustics of any kind is the same as that which applies to the thermocautery, that is, the excessive scarring which results.

Subconjunctival injections of irritating solutions for the purpose of causing severe reactions and thus combating the infection have been highly recommended in the treatment of corneal ulcers. Fuchs[15] stated that he regarded this method of value in early cases but of

13. Verhoeff, F. H.: Ultraviolet Light as a Germicidal Agent, THE JOURNAL A. M. A., March 7, 1914, p. 762.
14. Guillery: Hornhautverätzung durch Säuren und ihre Behandlung, Arch. f. Augenh., 1909, **63**, 258.
15. Personal communication to the author.

no service in advanced cases. I myself have found other treatment so effective in early cases that it has seemed unnecessary for me to give this painful method a trial.

Operative Treatment.—Stephenson[16] has pointed out that priority for the operation of incising corneal ulcers belongs to Guthrie, who performed the operation twenty-seven years before Saemisch. The beneficial effect of this operation is generally so great as well as prompt that there is no question as to its effectiveness. The explanation of its effect is probably that it reduces the pressure on the cornea and allows drainage to take place from the cut surfaces, while at the same time it causes the ulcer to be continually irrigated by fluid from the anterior chamber. This fluid is no longer ordinary aqueous, but owing to the low intra-ocular pressure as well as the inflammatory reaction, it is almost pure serum and hence richly contains the bactericidal properties of the blood. In rare cases, however, this operation fails and the ulceration continues, or intra-ocular infection takes place through the incision. Probably in these cases either the organisms are unusually virulent or the patient's serum is lacking in immune substances.

In spite of the remarkable results usually following the Guthrie-Saemisch operation, it is generally employed only as a last resort, for although it may put a stop to the ulcerative process, the condition finally resulting is apt to be far from desirable. Almost always there is anterior synechia with its attendant dangers of secondary glaucoma and sympathetic uveitis, while the irregular astigmatism resulting from the scar is apt to be of an extreme degree.

Keratectomy.—Foroni[17] reports exceptionally good results from dissecting away the superficial layers of the cornea in the ulcerated area, curetting the wound

16. Stephenson: Priority; Guthrie or Saemisch Operation for Hypopyon Keratitis, Ophthalmoscope, 1910, p. 266.
17. Foroni: Keratektomie, Arch. f Augenh., 1915, **78**. 279.

and irrigating with a 1:1,000 solution of mercuric cyanid. That the results of this treatment are not all that could be desired, however, is shown by the fact that he finds it necessary in some cases to perforate the cornea in its periphery with the galvano-cautery. I have never tried this method, but it seems to me unnecessarily severe.

Conjunctival Flaps.—The placing of a conjunctival flap over a corneal ulcer has been strongly recommended by Kuhnt and others as an effective method of treatment. In the few cases in which I myself have tried this procedure, the flap has not remained in position long enough to be of any service. Possibly my technic has been at fault here.

Author's Method.—With the limited number of cases at my command, it has seemed to me better to continue a method that was giving good results, to find out its exact limitations, and to attempt to improve it, rather than to change continually from one method to another and thus reach no definite conclusions. In this way, during the past three years, I have worked out a method of treating corneal ulcers that has given results more satisfactory than has any other method I have employed or seen used. I am far from regarding the method, however, as the final solution of the problem.

The patient is placed in bed, the affected eye thoroughly cocainized, and a speculum inserted. He is then told to look at various points on the ceiling until a position is found at which the ulcer is directly upward. This position must be maintained throughout the treatment, but the patient seldom finds any difficulty in keeping his eye sufficiently steady. Occasionally it has been necessary for him to fix his other eye on the finger of an assistant. With a Beer's knife, incisions are now made in the ulcer thus: The back of the knife is held toward the cornea and the point entered slantingly into the margin of the ulcer. The

knife is then pushed through the middle of the ulcer to the opposite margin, care being taken to make the incision as deep as possible without entering the anterior chamber. It will be found that, owing to the slanting position of the knife, Descemet's membrane is pushed away from the point so that the incision may be carried down almost if not quite to this membrane without danger. The point of the knife is now entered in the center of the incision just made, and a radial incision perpendicular to the latter carried to the margin of the ulcer. A similar incision is then made in the opposite direction, so that the final result is a crucial incision. In the case of large ulcers, several additional incisions should be made, starting each time at the center. With the point of the knife the infiltrated border of the ulcer is now superficially curetted, and the material obtained used for the bacteriologic examination. After this, the entire surface of the cornea around the ulcer is dried by touching it with small dry swabs of sterile absorbent cotton.

The ulcer is now ready for treatment with the germicide, a highly concentrated Lugol's solution: iodin, 25; potassium iodid, 50; water, 100. This is applied by means of cotton-tipped tooth picks, and the application should be limited to the ulcer itself, as the solution will quickly injure the epithelium. Owing to the cornea's being dry, the solution has no tendency to spread beyond the ulcer. After the entire area of the ulcer has been moistened, additional solution is added until there is an actual puddle on the ulcer. This is allowed to remain five minutes, when it is quickly flushed out of the conjunctival sac by a jet of boric acid solution. A rubber bulb filled with boric acid solution should be in constant readiness for this purpose, for should the patient move his eye abruptly and cause the Lugol's solution to flow over the cornea, it is necessary to wash it away at once. If this should happen before the five minutes have elapsed, the cornea should

be dried again and the Lugol's solution reapplied. The treatment is absolutely painless.

In cases of very small ulcers, the Lugol's solution is applied without making the incisions described. In deciding whether or not to make the incisions, I do not depend entirely on the size of the ulcer, however, but also on the rapidity of its progress and the amount of general ocular reaction, as evidenced particularly by the size of the hypopyon. In active cases, even when the ulcer is small, it is probable that the organisms have become disseminated for a considerable distance from the ulcer proper and hence may not be reached by the germicide unless the incisions are first made. When there is doubt, the incisions should always be made, as they do not noticeably increase the opacity left by the ulcer. In fact, as the ulcer heals, all traces of the incisions disappear.

In cases of rapidly progressing ulcers of large size, with large hypopyon, after the foregoing treatment has been completed, I make a small puncture through the center of the ulcer with the Beer's knife, and evacuate the aqueous. This puncture is not large enough to evacuate the hypopyon, and I believe it advantageous for the latter to remain. It holds both the iris and lens away from the opening, and thus prevents anterior synechia and injury to the lens. I have never seen any harm result from this central puncture, and I now always make it unless I am absolutely certain it is unnecessary. In cases of suspected glaucoma, it should never be omitted. Its chief disadvantage is that it causes more or less pain.

The after-treatment of these cases consists in keeping the patient in bed with both eyes bandaged, the use of White's mercuric chlorid ointment as a lubricant, and in the instillation of atropin to keep the pupil open. The beneficial effects of the iodin treatment do not become apparent until after forty-eight hours, when the ulcer appears cleaner, the hypopyon smaller, and the eye becomes much more comfortable. In case

the central puncture was made, the longer the anterior chamber remains empty the better. If no improvement is noted at the end of seventy-two hours, and the anterior chamber has become reestablished, the treatment should be repeated.

It may be of interest to give the reasons that led me to adopt this method of treatment. Although it was evident that we possessed no germicide that would destroy bacteria in the cornea without injury to the cornea itself, it seemed to me that some of the germicides must be far less injurious than others. Moreover, since the cornea was already severely injured in the ulcerated area wherein the bacteria chiefly occurred, it seemed that a certain amount of additional injury would make relatively slight difference as regards the visual results. It occurred to me, therefore, that Lugol's solution, which was known to be a powerful germicide, might answer my purpose. I found that a freshly prepared solution even in as high dilution as 1:2,000 (iodin, 1; potassium iodid, 2; water, 2,000) would destroy the *Staphylococcus aureus* in less than one minute. I also found, however, that when mixed with an equal volume of ascitic fluid, a solution of 2:1,000 would not kill this organism in twenty-four hours. It was evident, therefore, that dilute solutions would be useless, since their germicidal activity would be destroyed as they permeated the tissue, and solutions sufficiently strong to overcome the neutralizing effect of the tissue fluid would be necessary. In this connection I found by experiments on rabbits that while strong solutions promptly killed the corneal corpuscles, they had remarkably little effect on the corneal stroma. For instance, a small amount of a 1 per cent. solution could be injected directly into the cornea without causing a permanent opacity. Evidently new corneal corpuscles made their way in from the periphery, and the original condition of the cornea was restored. Since in corneal ulcers the corneal corpuscles are already largely destroyed by the bacterial

toxins, it appeared likely that strong Lugol's solution would do no additional harm except so far as it destroyed a greater or less number of corpuscles at the periphery of the ulcer. On the other hand, it was found that if an area about 5 mm. in diameter was injected with the 1 per cent. solution, it caused, owing to its prolonged action, softening of the corneal stroma and a permanent opacity.

It is unfortunately not possible to produce in animals progressive corneal ulcers similar to those caused in man by pneumococci. I was compelled, therefore, to test the value of Lugol's solution by actual trial in human cases. At the outset I employed a 2 per cent. solution and applied it by placing a glass tube over the ulcer and filling the tube with the solution. This method seemed effective, but was difficult of application. I then increased the strength of the solution to 5 per cent. and used the method of making a puddle on the ulcer as mentioned above. In cases of small ulcers this was uniformly successful, but in cases of larger ulcers it sometimes happened that the process would start up again at one point or another, thus indicating that bacteria in the comparatively normal cornea had not been reached by the germicide. I then adopted the plan of making incisions in the ulcer for the twofold purpose of allowing the germicide to permeate the tissue more freely and of permitting drainage afterward. At the same time the strength of the Lugol's solution was increased to 25 per cent. Even with this powerful solution applied for five minutes, there was an occasional case in which evidently not all the bacteria were not killed. In fact, judging by the amount of discoloration of the tissues, it was clear that in no case did the solution penetrate far into the normal corneal tissue. For rapidly progressing cases, therefore, in which it was probable that the organisms had become disseminated far into the cornea away from the ulcer, the plan was adopted

of making a central puncture in the ulcer to obtain as far as possible the additional advantage of the Guthrie-Saemisch operation without its disadvantages.

Altogether I have treated forty-two cases of corneal ulcer with hypopyon by this method. These cases were unselected except to the extent that only such cases were treated in this way as were regarded as sufficiently serious to require admission to the hospital. Bacteriologic examinations, which were made in almost every case, showed the ulcers to be due either to pneumococci or diplobacilli.

In twenty-one cases the ulcer was small, from 1.5 to about 4 mm. in diameter, and the process was checked in every case. In these cases the visual acuity at the time of discharge from the hospital was $^{20}/_{70}$ or better in seven cases, $^{20}/_{100}$ in five cases, $^{20}/_{200}$ in six cases, and $^{12}/_{200}$ in two cases. In one case the acuity was equal only to the counting of fingers at 8 feet; but this was due to an older scar. In eight cases the ulcer was of moderate size, but was sufficient to reduce the visual acuity to the perception of light or the counting of fingers. Here the process was also checked in every case, but the resulting visual acuity was of course not great at the time of discharge, ranging from the counting of fingers at from 1½ to 8 feet. In thirteen cases the ulcer was very large, involving from one-half to two-thirds or more of the corneal surface. Here the process was checked in eight cases, but the visual acuity obtained was only equal to the perception of light or shadows. In five cases the process was not checked, and in three of these enucleation was performed. No doubt in some of the cases the visual acuity could later have been improved by optical iridectomy.

The average age of the patients with small ulcers was 39 years, while that of the patients with larger ulcers was 50 years. It would seem, therefore, that older persons allow the ulcerative process to gain

greater headway before applying for treatment. This, I think, is due in part at least to the lessened sensibility of the cornea and the weaker mentality of the older persons.

In one of these cases the ulcer was associated with dacryocystitis. In twenty-two cases there was a definite history of recent trauma. Glaucoma was noted in one case, but may have been overlooked in others. Diplobacilli were found in six cases. In each of these the process was checked, except in one in which three-fourths of the corneal surface was involved at the time of treatment. Owing to illness, this patient was not treated by me personally.

Judging by my results, as well as by those of some of my colleagues who have also tried this method of treatment, I feel safe in saying that it will stop the progress of any corneal ulcer of small size and leave a minimal scar. In cases of ulcers of moderate size the process will also almost always be checked by it, but in cases of very large ulcers failures will often occur, owing to the fact that the germicide cannot reach all the bacteria. In the latter cases, however, so much of the cornea has already been involved that little is to be gained by checking the ulcerative process. It is important, therefore, in the case of any ulcer to apply the treatment as soon as the patient presents himself. In hospital practice, to save unnecessary delay, the treatment should be carried out in the outpatient department before the patient has been admitted to the wards.

FURTHER STUDY OF THE EFFECTS OF HEAT ON THE EYE*

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In this work and in the work[1] reported at the 1916 session of the American Medical Association, the method of applying heat to the cornea has been one of direct conduction by means of hot metal placed in contact with the cornea. Methods of radiation by holding hot objects near the eye, by convection, through hot air or steam, etc., have been unsatisfactory because of the difficulties in exact control both as to intensity and as to quantity.

The difference between success and failure in applying heat in such grave conditions as serpiginous ulcer of the cornea is measured by only a few Fahrenheit degrees, as will be shown in the following experiments and case reports. Moreover, different materials have different degrees of heat conductivity, just as they do of electric conductivity. For an exact definition, therefore, it is necessary to state the intensity as indicated by the thermometer or thermocouple, and the material used for conducting the heat. In all these experiments and cases the material used was brass plated with nickel.

An experiment will make clear the need of stating the material used for conduction. A mass of nickel-plated brass (the thermophor applicator described at the 1916 session) was heated to 150 F. and applied to the cornea of a rabbit's eye for one minute. The epi-

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1. Shahan, W. E.: Effects of Heat on the Eye, THE JOURNAL A. M. A., Aug. 5, 1916, p. 414.

thelium was destroyed over an area somewhat greater than the surface placed in contact with the cornea. Bowman's membrane and the substantia propria were clouded so that the iris was scarcely visible through the heated area. It took five days for the epithelium to be completely replaced, and the cornea was somewhat clouded for a month or more. In contrast to this a test tube was filled with water, a thermometer placed in the water, and the whole heated until the thermometer indicated 160 F. The rounded end of the test tube was then placed in contact with a rabbit's anesthetized cornea for one minute. During this time the thermometer fell from 160 to 150 F. There was no visible effect on the epithelium, Bowman's membrane, or any part of the eye. The quantity of heat in the water and test tube was much greater (as measured in calories) than that in the brass applicator, but the heat in the brass applicator passed into the cornea with so much more rapidity that marked effects were produced. If glass should be used as an applicator, it would be necessary to institute a series of experiments to determine how high its temperature would have to be raised in order to produce desired effects. Glass is a poor heat conductor. The same condition applies to all other conductors. This probably accounts for the fact that the limit of heat tolerance for different eye tissues has been variously stated by different observers.

The most important application of heat is in serpiginous ulcers of the cornea. It was the routine loss of eyes afflicted with such ulcers that inspired this whole work. These ulcers are ordinarily pneumococcus infections, and are characterized by appearing nearly always singly as virulent ulcerative processes deep within the substantia propria. There is marked conjunctival injection, sometimes much chemosis, and after a few days, pus in the anterior chamber (hypopyon). The organisms advance in all directions from the primary focus, and are most virulently active beneath Bowman's membrane and where not exposed to the air. It

is owing to the fact that the organisms are deep within the substantia propria that practically all surface medication has proved a failure. It is also noteworthy that experimental inoculation with pneumococci in numerous cases was produced only by breaking through Bowman's membrane and inserting the bacteria beneath it, and also that when large areas of epithelium were removed around such ulcers, further progress was not made *on* Bowman's membrane, but *beneath* it, while the epithelium was rapidly replacing itself. This replacement of epithelium occurred so rapidly in some cases that the ulcers were roofed over and converted into abscesses. This is important because it permits us to use therapeutic agents which destroy the epithelium wherever applied. This does not hold, however, in streptococcus or pyocyaneus infections.

When the investigation was started it was hoped that pneumococci could be killed within the substantia propria by a degree of heat below the limit of tolerance of the substantia propria. It was found that 130 F. for ten minutes was the limit of tolerance of the substantia propria when the heat was applied through a nickel-plated brass conductor. This was tried on various pneumococcus ulcers and resulted in disappointment. It was found that after such applications positive cultures could be obtained from the site of application, and that the heat which passed through the cornea was sufficient to cause atrophy of iris tissues without permanent clouding of the cornea. By means of thermocouple measurements it was found that heat so applied passed rapidly through the cornea, and that when a constant temperature was applied to the anterior surface of the cornea, the posterior surface reached a constant level in about one minute.

Another series of investigations was then undertaken. Inoculations of pneumococci were made in a number of eyes. Each eye of each rabbit was inoculated. One was treated with heat and the other left

untreated, as a control. There is a tendency to spontaneous healing of pneumococcus ulcers in rabbits' eyes. Accordingly rigid controls are necessary for preventing misleading results. Gradually increasing degrees of heat were applied by means of nickel-plated brass thermophor points for one minute only. When applications of 152 F. for one minute were reached, the ulcerative process was stopped both bacteriologically and clinically. A detailed history of one of the rabbits will show the action of treated eye and control:

RABBIT 26.—Inoculated O. D. and O. S., July 13, 1916, with pneumococci.

July 14: O. D., vigorous infection. Area 7 mm. in diameter stains with fluorescein. Took culture. Applied 152 F. somewhat intermittently for one minute, after which ulcer appeared dry and clean. Washed with physiologic sodium chlorid solution. Took culture. O. S., vigorous infection, area 3 mm. in diameter stains with fluorescein. Washed with physiologic sodium chlorid solution.

July 15: O. D., ulcer quiet. No discharge, no hypopyon. O. S., ulcer very active, much discharge, no hypopyon.

July 17: O. D., profuse growth on culture, taken before heating. No growth on culture taken after heating. Eye quiet. Epithelium completely replaced. Thin gray spot on site of ulcer. O. S., much discharge, cornea clouded. Deep ulcer. Area 5 by 8 mm. stains with fluorescein.

July 21: O. D., entirely quiet. Gray central area becoming thinner. O. S., active ulceration. Pupil filled with a membrane. Whole cornea opalescent. Considerable discharge through the epithelium, which has covered ulcer so that fluorescein does not stain.

July 24: O. D., entirely quiet. O. S., severely inflamed. Vessels extend into cornea about 4 mm. from all parts of limbus. Center of ulcer stains with fluorescein.

July 27: O. D., entirely quiet. O. S., still severely inflamed. Vessels extend as a dense red zone 5 mm. into cornea from all parts of limbus.

July 31: O. D., entirely quiet. O. S., site of ulcer yellow, broken down and excavated. Whole cornea up to margins of ulcer filled with a dense network of blood vessels.

August 3: O. D., entirely quiet. O. S., site of ulcer organized. Still yellow deep within substantia propria. Hyphemia.

September 29: O. D., thin gray area at center of cornea. No iris changes. O. S., vessels in cornea all atrophied. Small gray area center of cornea. Yellowish deposit along lower part of cornea. Some clouding anterior capsule. Firm posterior synechiae.

This was repeated on rabbits with variations, sometimes with, sometimes without hypopyon, until it seemed reasonably certain that 152 F. applied for one minute was sufficient to stop the ulcerative process.

It now became desirable to know how much damage this amount of heat did when applied to a normal eye. This was applied to various eyes, and it was found in general that there was complete destruction of the epithelium over an area somewhat larger than the point used. With a 5 mm. point, for instance, there was an area 5 by 6 mm. of denudation. The surface of the cornea was clouded so that often the iris was not visible through it. The epithelium replaced itself in four or five days, and the clouding slowly diminished. A rabbit with O. S. heated so, July 27, 1916, now (Jan. 30, 1917) has a faint nebula at the site of application. The nebula is difficult to see without oblique illumination and the aid of a magnifying glass.

It would seem, therefore, that this method of thermotherapy offered much in the deep treatment of pneumococcus infections, and it was felt that the time was ripe for trial on human eyes when opportunity offered.

To date the following patients have been treated:

CASE 1.—Coal miner, aged 39, consulted Dr. E. C. Spitze, East St. Louis, Dec. 17, 1916. A foreign body was removed from near the inner limbus. There were two small foci of infection at the site of the foreign body. Ulcers were cauterized with saturated phenol iodin solution. The foci coalesced and the ulcer slowly increased in size despite active daily treatment. Dec. 28, 1916, Dr. Spitze brought the patient to me for trial of heat treatment. The ulcer then bordered the limbus and extended about 3 mm. out into the cornea. Trial of methylene blue and 1 per cent. silver nitrate solution to the walls of the ulcer was advised. Dr. Spitze did this and also made subconjunctival injection of 20 mm. of 1:3,000 mercuric chlorid solution. The patient was now in the hospital. The ulcer was steadily growing worse.

Jan. 8, 1917: Dr. Spitze again sent the patient to me. The ulcer now extended 5 mm. along the limbus and 4 mm. into the cornea. There was a dark border where the silver nitrate was applied, and beyond this a yellow zone of virulent infection. Hypopyon was 1 mm. high. The patient complained much of pain. Heat was applied at from 150 to 152 F. with a 5 mm. applicator for one and one-fourth minutes to the

ulcer under thorough anesthetization with cocain, 5 per cent. in suprarenal extract, 1:1,000. The application appeared to cause but little pain. After the application the yellowish color was changed to a grayish hue. The epithelium was destroyed over an area extending 1 mm. beyond the margin of the ulcer.

January 9: The patient spent a more comfortable night; the ulcer was quieter and grayish in appearance. The hypopyon was unchanged. Subsequently there was very little pain. The hypopyon disappeared and the ulcer became quieter until January 13, when there was a slight renewal of activity deep within the substantia propria in the border of the ulcer nearest the center of the cornea. There was a slight hypopyon with renewal of pain. Heat at from 150 to 152 F. was then applied for one minute with a 5 mm. applicator. The edge of the applicator was pressed with special firmness against the most active part of the ulcer. There was rather extensive coagulation of the new epithelium that had grown over the site of the ulcer since the first application of heat.

January 15: The ulcer was quiet. There was no discharge. The hypopyon was gone. There had been no pain since three or four hours after the application. Most of the heated area was still bare of epithelium.

January 20: The patient had been slightly uncomfortable for the last two days. There was no discharge. There was a faint diffuse staining with fluorescein along the edge of the ulcer nearest the center of the cornea. There was no hypopyon. A slight gray infiltration along the central and lower edge of the site of the ulcer may have indicated some pneumococcic activity deep within the substantia propria. Atropin and cocain (oil solution) was used. It was decided to wait another day before applying heat.

January 21: The patient was comfortable last night. The gray infiltration appeared to be breaking up into isolated foci. There was no hypopyon. There was no staining with fluorescein. Coddington lens (1 inch focus) revealed the site of the ulcer nearly surrounded by a narrow bulla. Atropin and cocain were used.

January 24: The eye was quiet. The site of the ulcer was filling in. There had been a severe iritis along with the ulcer. There were numerous posterior synechiae and some deposit of the anterior capsule. V. = 3/60. The ulcer did not reach the center of the cornea, and if the deposit on the anterior capsule clears up good vision should be obtained. This patient was seen daily either by Dr. Spitze or myself, and is now in Dr. Spitze's charge. Treatment was complicated by a suppurative postethmoidal and sphenoidal sinusitis. These conditions were cared for by Dr. Arbuckle.

February 10: Dr. Spitze telephones that there has been no renewal of the ulcerative process. The cornea has cleared up and the globe is slowly quieting down.

CASE 2.—Man, aged 56, appeared in Dr. William F. Hardy's service at Washington University Clinic, Jan. 16, 1917, with a corneal ulcer 6 by 8 mm. staining brightly with fluorescein. The eye was blind as the result of an old injury. It was treated with silver nitrate, 0.5 per cent., and atropin. It was bandaged.

January 17: All the symptoms had increased. The treatment was the same.

January 18: A very profuse mucopurulent discharge was soaking the bandage and gumming up the lids and lashes. The patient had had much pain last night. There was moderate hypopyon. The ulcer was larger. Cultures revealed probably pneumococci submerged in staphylococci. With the thermophor, heat was applied at from 150 to 152 F. for one minute to the upper half, and one minute to the lower half of the ulcer with a 5 mm. point. The whole surface of the ulcer was made somewhat gray by the heat.

January 19: The patient complained of pain in the head and the left jaw last night. There was much less discharge. The ulcer was cleaner. There was not much pain in the eye. The hypopyon was about the same as yesterday. Silver nitrate, 0.2 per cent., and atropin were used. A bandage was applied.

January 20: The patient passed a comfortable night. There was very little discharge. The ulcer was quiet. The patient had the notion that the treatment (heat) had affected the other eye. No physical evidence of this was found. He could not be rid of the notion. Treatment was the same as before.

January 22: The eye had not been dressed for two days. The dressing was dry. The "eye felt fine." The hypopyon was gone. There was a small active area at the upper margin of the ulcer. This should have had heat treatment with a small crescentic applicator. The patient refused to permit this. Treatment was the same.

January 23: The patient had much pain last night. There was a moderate discharge. The active area noted yesterday was extending rapidly downward over the former site of the ulcer. The patient refused heat. He was turned over to an assistant for routine treatment. He disappeared from the clinic. This eye might have been saved either by a higher or more extensive initial application, or by repeating the application over the small area of exacerbation as soon as it appeared.

CASE 3.—A coal miner, aged 46, was referred to me by Dr. Spitze for heat treatment, and has since been cared for by us jointly.

January 18: The patient was struck in the right eye five days ago by a piece of coal. There was an ulcer at the center of the cornea 5 mm. in diameter. Hypopyon was 1 mm. high. Cultures and smear revealed pneumococci. There was very

marked chemosis. The bulbar conjunctiva overhung and lay on the cornea for about 1 mm.

In this case the serious mistake of explaining the working of the thermophor to other physicians in the presence of the patient was made. The applicator was felt by these physicians and the audible comment on its hotness made. As a result the patient was nervous and apprehensive, expecting his eye to be burnt. Heat, from 150 to 152 F., was applied for one minute somewhat intermittently. The patient did not hold his eye still. This was done by artificial light, and slight changes in the hue of ulcer could not be made out.

January 19: Dr. Spitze reported that the patient had very little pain last night.

January 20: The ulcer was much worse and was spreading in all directions. The hypopyon was larger. The ulcer was too large to be covered by a 5 mm. thermophor point. Heat, from 150 to 152 F., was applied to the upper temporal surface of the ulcer for one minute and also to the lower nasal surface for one minute. Continuous applications were made. The patient exhibited some signs of pain.

January 21: Dr. Spitze reported the ulcer clean and much better.

January 22: The eye had not been dressed for about twenty hours. There was little complaint of pain. The ulcer was clean. The bandage was dry. There was no discharge. There was a small area of activity at the upper margin and another at the temporal margin of the ulcer. Heat, from 150 to 152 F., was applied to each of these for one minute with a 2 mm. applicator. They were washed with physiologic sodium chlorid solution. Atropin and cocain (oil) solution was used.

January 23: The patient had considerable pain, especially in the head, last night. The ulcer was generally clean. The active area at the upper margin of the ulcer was still somewhat active. The active area at the temporal side was quiet. The eye "felt fine" this morning. The hypopyon was smaller.

January 24: The conjunctiva at the upper limbus overhung the cornea and extended downward on it for 2 mm. Beneath this fold and sharply defined by it was a very active deep area of infection. All the rest of the cornea was quiet and translucent, allowing all parts of the iris and pupil to be seen. This focus emphasized the previous statement that pneumococci are most virulently active where not exposed to the air. By means of forceps the overlapping conjunctiva was held away from cornea and 154 F. was applied to the ulcer for one minute with a crescentic pointed applicator.

January 25: The patient had rather marked pain last night. The ulcerative process had stopped. There was a small quantity of fresh blood within the substantia propria at the center of the cornea. This was probably due to a small central perforation, although the anterior chamber was not collapsed and the iris did not touch the cornea. This perfora-

tion confused the effects of the heat somewhat, as such ulcers often improve rapidly after corneal perforation. The hypopyon had gone.

February 6: There had been no pain since the last note. Chemosis was gone. Epithelium was growing in over the surface of the cornea. Tension was subnormal. The anterior chamber was very shallow or absent. The iris and pupil were still visible, although the cornea was now somewhat turbid, as a result of reparative processes. There had been no foci of infection since the last heat application.

The ultimate outcome is still pending. The usual course of such a severe infection as this is total loss of the cornea with expulsion of the lens, etc.

CASE 4.—A laborer, aged 39, was referred to me for heat treatment by Dr. Meyer Wiener, and treatment has been conducted by us jointly. The man had been struck in the left eye three weeks before by a nail. There was a perforating wound of the cornea somewhat nasal to the center. A yellow abscess 2 mm. in diameter extended nasally from the wound toward the limbus. Earlier in the case this was an abscess with hypopyon, and the diagnosis of pneumococcus infection was established by Dr. Wiener. Under surface medication this improved until the epithelium covered the ulcer and converted it into an abscess. Improvement then ceased and extension began in the substantia propria. There was an exudate on the anterior capsule, and numerous posterior synechiae.

Jan. 29, 1917: Under thorough anesthesia with 5 per cent. cocain in suprarenal extract, 1:1,000, from 154 to 155 F. of heat was applied for one minute with a 2 mm. applicator. Continuous application was made. The patient had some pain. After the application an area 4 mm. in diameter stained with fluorescein. The center of the abscess stained more deeply than the rest of the area, which would indicate that the abscess had been unroofed.

February 2: The patient had had very little pain since the application of the heat. There was now a small moderately active ulcer at the deepest part of the former abscess. The epithelium was all replaced except over this small area.

February 5: There was an exacerbation, with more pain. The ulcerative process was active; 153 F. was applied for one minute with a 2 mm. applicator. The area about the ulcer turned slightly gray. The deepest part of the ulcer still appeared of a somewhat yellow hue. The application caused but little pain. Half an hour after application, the patient complained of the eye burning. This was quieted with an instillation of atropin and cocain oil solution.

February 8: There had been no pain since the last application. There was no discharge. There was no activity except possibly in the center of the site of the ulcer deep within the substantia propria.

February 10: There had been continued improvement, and no further pain. The site of the ulcer was somewhat gray. There was no staining with fluorescein. In this case it would have been better to use an applicator with a somewhat rounded, bullet pointed tip than one with a flat surface 2 mm. broad. The heat would have then been carried more effectively into the center of the ulcer. Such applicators have since been constructed.

These cases tend to confirm the experimental conclusion that heat, when properly used, is an effective therapeutic agent in pneumococcus infection of the cornea. It will be observed that the temperature is about what is stated as necessary for the immediate death of pneumococci in culture (from 65 to 70 C., 149 to 158 F.[2]). All temperature readings given in this article are those indicated by the thermometer. These readings are the ones most convenient to use in actual practice. In the previous article it was shown that the temperature of the surface in contact with the eye was less than that indicated by the thermometer, and that as the temperature increased this difference increased. When the thermometer indicates from 150 to 155 F., the temperature of the surface in contact with the cornea, while in contact with the cornea, is about 4 F. less than the thermometer shows. The temperature of 152 F., therefore, which was found sufficient for the death of pneumococci in the cornea of the rabbit's eye, was actually only about 148 F. This difference was measured by means of a junction of a thermocouple placed in a fine hole drilled through the tip of the applicator parallel to the surface in contact with the eye, and as close to it as possible.

In all the work up to the present, the thermophor used has been the compact but rather complicated one shown at the Detroit Session.[1] It consists of a resistance coil, sensitive regulating strip, platinum contacts, etc., and was primarily designed for maintaining constant temperatures in the applicators for long periods of time. For practical purposes for one minute applications, a much simpler apparatus can be used.

2. Besson: Technique microbiologique, Ed. 4.

The parts of such a simplified instrument are shown in Figure 1. In the left hand part of this illustration are four bullet-shaped objects (*a*). These are the applicators. Each one is hollowed out so as to receive the bulb of the thermometer. Just below these is shown the "core" (*b*) with an applicator and thermometer in position. This core is a heavy piece of brass (or copper) tubing 4 inches (about 10 cm.) long and 9/16 inch (about 14 mm.) in outside diameter. One end has a lock-nut in it for holding fast the thermometer, while the other end is reamed out properly for snugly receiving the applicators. The larger object (*c*) below is a metal case 1¼ inches (about 32 mm.) in diameter, and a little over 4 inches (about 10 cm.) long. This is heavily lined with felt, has an opening at each end and a hollow space through the center just large enough to receive the core. It is an insulating jacket for retaining the heat. Figure 2 shows the jacket with core, thermometer and applicator in position as when ready for use.

When it is desired to use the instrument, a properly shaped applicator is selected and slipped into the end of the core. The thermometer is then slid through the lock-nut and core until its bulb is in the applicator as far as it will go. The thermometer is then fastened in place by a few turns of the lock-nut. Then, with the thermometer used as a stem, the core is held over a flame, as that of a Bunsen burner, until the mercury in the thermometer reaches about 170 F. The core is then slipped into the insulating jacket, where it will be held by the friction of the felt. The mercury in the thermometer is then observed until it falls to 156 F. At this instant the tip of the applicator is placed in contact with the ulcer and held there for one minute. During this time the mercury will fall from 5 to 10 F., according to the size of the tip of the applicator. The method of use is illustrated by the following case:

A coal miner, aged 50, was referred to me for heat treatment, Feb. 13, 1917, by Dr. E. C. Spitze of East St. Louis.

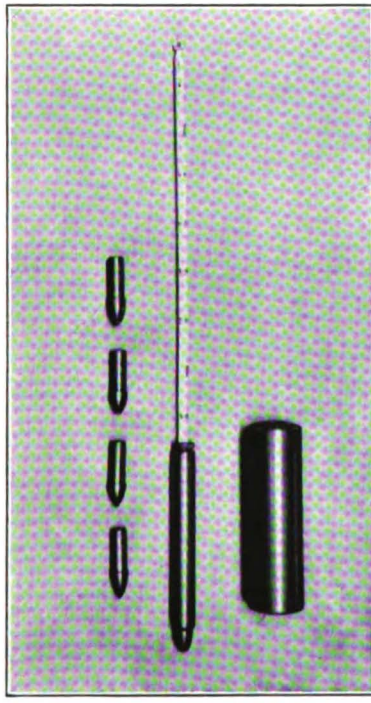

Fig. 1.—Thermophor parts: applicators, core with applicator and thermometer in position and felt lined insulating case.

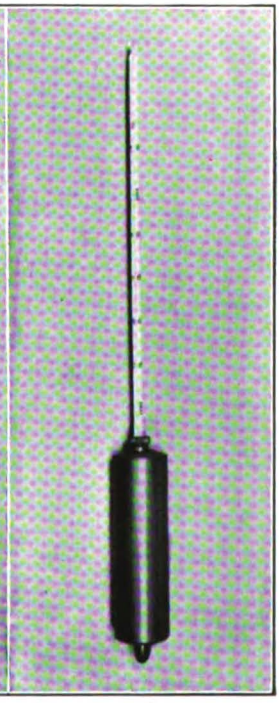

Fig. 2.—Assembled thermophor, as it appears when ready for use.

He had been struck in the left eye ten days previously by a piece of coal. A pneumococcus infection set in and was treated by various cauterizing agents by Dr. Spitze. Also a hot copper mass was held as near the ulcer as the patient could stand for five times. Despite these measures, the ulcer steadily increased in size and severity. When he came to me, February 13, there was an ulcer 3 by 2.5 mm. in the lower nasal quadrant of the cornea nearer to the center than the limbus. There was marked injection of the bulbar conjunctiva, beginning iritis and much pain. The patient had been unable to sleep the past two nights. There was moderate discharge. The stage of hypopyon had not yet been reached. A smear revealed pneumococci.

The eye was thoroughly anesthetized with 5 per cent. cocain in suprarenal extract, 1:1,000, for about fifteen minutes. During this period the applicator, in position in the thermophor, was held against the ulcer several times before it was heated so as to accustom the patient to it. Care was taken that the word heat was not used in his hearing. When the eye was ready the core was slipped out of its jacket and heated over a Bunsen burner out of sight of the patient. The core was then slipped back into its jacket and the instrument laid on a table in view of the patient while the mercury was falling to 156 F. During this time more anesthetic was instilled and the thermophor picked up. At the proper moment the patient's lids were held apart with the fingers of one hand while the applicator in the thermophor was held against the ulcer with the other. The patient did not notice any difference between this application and the former ones made before the core and applicator were heated. Consequently he was calm and held perfectly still. The application, therefore, was constant and thorough. A casual question afterward elicited the response that it did not hurt. A 3 mm. applicator was used for this. An area 4.5 mm. in diameter was denuded of epithelium. Shortly after the application considerable pain developed and was controlled by cocain, or oil solution of atropin and cocain, about every half hour for about three hours. The pain was such as follows a burn.

February 14: The patient "slept fine" last night. There was no discharge. No pain was felt. The ulcer was clean and everywhere free from yellow infiltration. Epithelium was replacing itself from all parts of the periphery. The area of denudation was now 3.5 mm. in diameter. Atropin and cocain (1 per cent. each) oil solution was used several times. Excellent mydriasis was obtained.

February 15: There were no pain and no discharge. The ulcer was replaced by gray surface. The area of denudation was 3 mm. in diameter. It was washed with physiologic sodium chlorid solution. Atropin and cocain in oil solution were used. A light bandage is kept on all such eyes until the epithelium is completely replaced.

February 16: There was no complaint. The area of denudation was 2 mm. in diameter. Treatment was as described above.

February 17: There was no complaint. The area of denudation was 1.5 mm. in diameter.

February 19: There was no complaint. The conjunctival injection was disappearing. An area of 0.75 mm. stained with fluorescein. There was steady filling in of the excavation in the substantia propria. There was no undermining or any symptom of pneumococcic activity. With + 1.50 sph. V. 20/20 +. The iritis had done no harm. The nebula was eccentric. The prognosis for complete recovery was absolutely good. Yet I have seen just such cases as this go to complete destruction of the cornea with extrusion of the lens, in my own hands as well as in the hands of my associates, despite most vigorous and conscientiously applied methods. The somewhat higher initial temperature of 156 F. was used because the temperature of 152 F. constantly maintained did not always stop the process in one application in the human cornea, and because in the simplified thermophor the temperature is steadily falling during the application.

In using the thermophor care must be taken to see that the thermometer is in order. One series of experiments was spoiled by a small volume of gas (some thermometers have a small amount of nitrogen in their stems) breaking the column of mercury and holding it so that the parts could not reunite. When this occurs the thermometer bulb is put into a freezing mixture and cooled until the mercury is all in the bulb. The gas then diffuses itself in the stem and the mercury column rises unbroken. Another series was spoiled by an appreciable quantity of the mercury becoming fixed in the small dilatation at the top of the thermometer stem. This gave false readings which caused the use of points hotter than was desired. In this case the bulb of the thermometer is cautiously heated over a flame until the mercury in the stem reaches and unites with that in the dilatation. On cooling, the mercury sinks as a continuous column and correct readings are obtained.

NOTE.—Since the foregoing article was written, nine additional cases of serpiginous (pneumococcus) ulcer have been treated by means of the simplified thermophor. The results have been most gratifying. The infective process was stopped

definitely in all but one case by one or two heat applications. It was found best, however, to use an initial temperature of 158 F. instead of 156 F. as being more certain to stop the process in one application of one minute's duration. Also a series of experiments on the normal corneae of rabbit's eyes showed that one minute applications up to 170 F. merely increased the depth and intensity of the resultant nebula without causing any necrotic changes leading to corneal perforation. A temperature of 190 F. (dropping to 180 F. during the one minute application) caused intense whitening of the substantia propria, followed in a few days by extensive vascularization of the treated area and some bulging of this area but no necrotic changes leading to perforation.

The effective temperature 158 F. for one minute is, therefore, far within the limit of safety for therapeutic uses on the cornea.

I am under much obligation to fellow ophthalmologists, especially Dr. E. C. Spitze of East St. Louis for collaboration in the cases reported.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. VERHOEFF AND SHAHAN

DR. WILLIAM C. POSEY, Philadelphia: At Wills Hospital we see many infected ulcers of the cornea. Our method of procedure is as follows: In mild cases if we find the Morax-Axenfeld bacillus we use zinc, often with excellent results. If the pneumococcus is found we use ethyl-hydrocuprein (optochin), and often in mild cases the results are good. During the past winter we have been using the pneumococcic serum. Before coming here, on questioning the house surgeon, who has been following up these cases, I found that he was not sure that he had seen any one case in which the serum had been of positive worth. We see many bad cases in which the patients have been injured in the mines. They do not come to us until some days afterward, so that we frequently find half or three-quarters of the cornea tissue broken down and extensive exudate in the anterior chamber. In these cases we usually perform a Saemisch section at once, or, as it might be called, a modified Guthrie-Schwenk-Saemisch section. We follow the method practiced by Dr. Schwenk. The patient is etherized and after the knife is carried into the anterior chamber, instead of drawing it out at once so that the aqueous flows off rapidly, favoring prolapse of the iris, the knife is rotated and the pus permitted to slowly flow away. Frequently such eyes heal without prolapse of the iris and very often without anterior synechia; so that often after iridectomy useful vision is obtained. I hope Dr. Schwenk will speak of the method. After the hypopyon has been drained off and the plugs of pus removed from the cornea, iodoform is dusted on the surface of the eye

and a compress bandage is applied. Dr. Verhoeff's deductions are scientific and logical, and his method of treatment certainly demands a fair trial. His results, however, in his bad cases I do not think are as good as those we have had at Wills with the methods I have just described. Of his cases, twenty-one bad cases, no vision was saved in any case; three eyes had to be enucleated; two others, he says, were lost, probably also demanding enucleation. I had planned to have an analysis made of our cases at Wills during the past winter, to compare the results with Dr. Verhoeff's, but unfortunately a recent illness prevented.

Dr. Edgar S. Thomson, New York: I am somewhat at a disadvantage in that I received one of Dr. Shahan's thermophors only a few days before the meeting and was unable to use it because we had no suitable cases at the time. However, I should like to say that my experience has been the general one with regard to these cases. The pneumococcic serum, it seems to me, has doubtful value. With optochin some improved and others were disappointing; and of course we know the value of cauterization. The principles on which Dr. Shahan is working are incontrovertible. A method to do the least possible damage to the cornea and at the same time destroy the pneumococci is what we want. We have had evidence of the clinical value attached to heat, so that I think his method deserves extensive trial. He has fairly well shown that there is not much damage to the cornea in this one-minute application. Whether it will destroy all the infection in this class of ulcers is another question. We all know that the virulence of bacterial strains varies, and a large series of observations is necessary to determine the status of the method. The outlook seems to me to be rather hopeful.

Dr. Peter N. K. Schwenk, Philadelphia: The modification of the Saemisch incision is a simple one and to my mind very important. Saemisch passed his knife across the anterior chamber, made a puncture and counter puncture, the cutting edge of the knife anteriorly, and cut directly forward, cautioning not to rotate the knife on its axis. That relieved the tension suddenly and naturally pushed forward the iris, lens and the contents of the ball. It occurred to me that after the counter puncture if the knife was made gently to press backward to equalize the tension, then rotated on its long axis, causing a gap or opening at both ends of the knife to allow the fluids in the anterior chamber to flow out and gradually release the pressure until tension was nil, then to replace the knife edge outward and cut out slowly, it would prevent the prolapse of the iris. The operation must be done under general anesthesia, and the incision must not be made rapidly but patiently and slowly. If you do that you will have no anterior synechia. At Wills Eye Hospital

we get the majority of the patients before the time for operation has arrived and cure them. Unfortunately, we get neglected cases from the country towns, and especially from the coal regions, and these are the patients on whom we are obliged to operate. Of late, I have paid much attention to the subject, and I believe there are several grades of the pneumococcus varying in strength, as described by Henderson and Lehnfels.

Depending on infection with malaria, a 3 per cent. solution of quinin has been used, supplemented by about 8 grains of quinin a day in 2 grain doses. This arrests the process without corneal destruction. But the pneumococci of the first type are the destructive pneumococci. Lately, after having cocainized the cornea, and applied phenol, making a thorough application, wiping off the white film and again reapplying the phenol, followed by a free application of glycerite of tannin, which will agglutinate the streptococci or pneumococci in the conjunctiva, I have found this to be one of the most satisfactory treatments. My last four patients have been treated by this method. Some say we should confine the patients to bed. Three patients coming from the country nearby I wanted to admit to the hospital. I believe they saved their eyes by declining. Confinement is deleterious and they should stay out doors. It is not only the local treatment, but also the supporting constitutional treatment that is helpful. If the home environment is better than we can offer them in the hospital by nutriment and support, they do better by going home. So that I believe in these cases the outdoor life, going to and from the hospital, is beneficial, the eye with the ulcer being always protected by a bandage. If the Saemisch incision has to be made you will find this modification a satisfactory one.

Dr. Verhoeff's principles remind me of the learned professor, William Pancoast, thirty-five or forty years ago, who treated leg ulcers by puncturing them a hundred times or more with the therapeutic knife. Dr. Verhoeff's operation is on that principle, endeavoring to get rid of the bacterial condition. His principle is scientific and worthy of a trial. I have used tincture of iodin, but it is too weak to produce the desired result, but Dr. Verhoeff's method of using it much stronger is quite promising.

Dr. John E. Weeks, New York: In regard to the diplobacillus ulcer, if our bacteriologic examination shows it to be such, the zinc salts are specific. But let us understand what the condition is in pneumococcic ulcer as described by Morax and Axenfeld and demonstrated by microscopic section. There is no overhanging lip and a shallow edge when the ulcer is not advancing, but when the ulcer is advancing there is an overhanging lip and the ulcer is deeper. We will find the pneumococci in the lamellae of the cornea which

have not broken down beneath the overhanging lip. We should reach these pneumococci with our treatment. It is my habit to cocainize the eye thoroughly, cut away the lip and gently curet the base of the ulcer. Then I have a surface to which I apply my antiseptic or germicide, whatever it may be. I have found, if I use a germicide other than the cautery or heat, that formaldehyd is one of the best we have. It in my hands has given better results than the tincture of iodin or any iodin preparation. Formaldehyd solution in the ordinary commercial strength reduced to 8 or 10 per cent. with sterile water, should be applied to the surface of the ulcer, particularly in the vicinity of the pneumococci infection. It should not be allowed to spread out over the epithelium. A surer way to destroy the pneumococci is to apply heat to the infected portion of the ulcer. It does not matter whether there is hypopyon or not. For the application of heat it is my plan to use a platinum bulb and a spirit lamp, bringing the bulb to a red heat, applying it carefully, touching every portion of the surface lightly, so that the resulting scar will not be very dense. Afterward I use an ointment of mercuric chlorid 1:3,000 or 5,000 in petrolatum and bandage the eye. If the tension of the eyeball is plus, I use a drop of pilocarpin. That, in cases in which the ulcer is not very far advanced, will bring about a cure in a short time. The eye should be treated and the bandage renewed about three times a day. If after a day or two there is a spot of inflammation, renew the cauterization at that point.

The Saemisch incision is a very valuable procedure. The gentlemen have spoken of the pus running out of the anterior chamber. Any of you with experience know that hypopyon, with few exceptions, is gelatinous and does not flow; you have to wash it out or pull it out. The anterior chamber should be evacuated slowly. A hypopyon which is not infected is composed of leukocytes in a stroma of fibrin. If you wash it out with a saline solution, cutting away the overhanging lip and treating the surface of the ulcer much as I have indicated, you will not get an adherent leukoma. The tendency to adherent leukoma is less with the Saemisch incision for ulcer of the cornea than after similar incisions for other purposes.

DR. ELMER G. STARR, Buffalo: I have great faith in covering the ulcerating cornea with conjunctiva. Dr. Verhoeff speaks of this, but is not impressed with its virtue. He speaks of the difficulty of keeping the conjunctival flap in place. It is often difficult to do this. Of course if the ulcerating area is not covered we cannot expect much benefit from the flap. Most authorities who mention covering the ulcer with a tongue or flap of conjunctiva speak of this difficulty, and it is true that where a small tongue of conjunctiva is stretched across the cornea under considerable

traction the retaining stitches are prone to cut out within one to three or four days. The difficulty which I experienced with that led me to adopt a different technic. Latterly now in extensive corneal ulcer, my plan is to incise the conjunctiva all around the cornea, undermine it well, put in a purse-string suture and cover the entire cornea in that way. There is no particular tension at any point and the suture holds very much better. I believe the ulcer must be covered and kept covered, not for a few days, but for a good many, preferably for a week or two in extensive, deep-seated ulcer. Where I have been able to cover the cornea and keep it covered I have yet to be disappointed in the result. It seems to me Dr. Shahan's results have been very promising indeed. I should like to ask what his procedure is in ulcers involving one-half or two-thirds of the corneal surface. Does he attempt to cover the entire surface with one treatment?

DR. JOHN A. DONOVAN, Butte, Mont.: A marked feature of these papers is the authors' anxiety to save tissue and to do no harm. It is my experience that so much tissue is already lost that to save a little extra is immaterial. Dr. Verhoeff has added another method to corneal ulcer treatment, and we are grateful for an additional method, because times come when we use them all. When Dr. Verhoeff gave his results, however, I was disappointed. The method of treatment by applying heat, which Dr. Shahan has given us, is another excellent method. There is no question about the efficacy of heat. His method of using it makes it a little more complicated. I still stick to the electric cautery. The point made by Dr. Weeks of undermining the edge of the ulcer is good. When using the electric cautery in making a puncture, I use the cautery point just red, sometimes a little more, and make it deep enough to get down into the corneal tissue between healthy and diseased tissue. This will always check the ulcer, at least for a while, but in some portion of the spreading edge it may not stay checked. Then I repeat the little punctures with the cautery, having the point heated before putting it on; then to dry the surface of ulcer I use the cautery at a black heat, testing the temperature on a bit of cotton, which will smoke. One can accomplish everything by that method, and do it more quickly and easily, and I find I lose as little tissue as with any other method. Hundreds of cases of simple ulcer can be touched with black heat and they will heal up with apparently no scar or loss of tissue. If they are bad, then you do not need to worry so much about loss of tissue; to save a useful eye you are fortunate. The results from cautery treatment I think are better than those Dr. Verhoeff has described for his treatment. In addition to the methods described, the injection of salt solution or mercuric cyanid solution, 1:1,000, is very beneficial. It is painful. But a few drops of a local anesthetic solution added

just before injection relieves this to a great extent. As to putting patients to bed, this is advisable in severe cases, and if I see it is not beneficial I let them up.

Dr. Melville Black, Denver: Dr. Verhoeff does not have much to say about subconjunctival injections. After a long experience with mercuric cyanid I insist that it is of very great value. If I had an eye going to the bad from keratitis with hypopyon, I should wish it used at once. I am sure that many have failed to get results from it because they have not used it right. A few drops injected at intervals of a few days is worse than useless and only brings the method into discredit. If results are to be obtained, from 20 to 30 minims must be injected, and the strength should be from 1:1,500 to 1:2,500 in accordance with the severity of the infection. I add ⅓ grain of novocain to the dose to be injected, and if the pupil is not well dilated 1/100 grain of atropin is also added. The eye is first well cocainized. The needle should be pushed through the conjunctiva by a quick thrust and the latter not picked up with forceps, as they tend to tear it and allow the fluid to escape. The average hypodermic syringe does not hold more than 15 minims; therefore an extra large size should be procured for these injections. The novocain prevents all pain at the time of the injection and for an hour or so following. The next six or eight hours there is a dull, deep ache, but it is not at all unbearable. The next day there is great chemosis and lid swelling, but like the singed cat, it looks worse than it feels.

Arthur G. Bennett, Buffalo: I want to bear testimony to the efficacy of the method spoken of by Dr. Starr. In Buffalo we have a great many ulcers to deal with, and covering the cornea with a conjunctival flap has in our hands acted most admirably. I have seen the most desperate cases, cases that I did not think could be saved, make the most wonderful recoveries under the flap. The point Dr. Starr makes is that you must keep it covered for a considerable time. The stitches will have a tendency to break away, but it is not difficult to put in a reenforcing stitch which will hold it for about seven days. It is not always necessary to keep it in position for seven days, for I have seen the conjunctiva adhere to the ulcer. That appears to be a complication which one would think would give an eye with a dense leukoma; but in a case treated about a year ago in which the conjunctiva became adherent to the ulcer, after the process had ceased I divided the conjunctiva from above the ulcer, and then, after the eye had quieted down, simply stripped the conjunctiva off, with a remarkably clear cornea. That was a most desperate case. The ulcer was advancing. From the size of 1 mm. one day it had almost entirely covered the cornea the next day. That case Dr. Starr saw afterward with almost a clear cornea. My success with

optochin has been greater than Dr. Verhoeff says his has been. I think it is good.

Dr. R. L. Randolph, Baltimore: My experience with this variety of corneal ulcer has not been very extensive, yet I see cases now and again. I do not regard the mere presence of exudate in the anterior chamber as necessarily serious, for, as Dr. Weeks has said, this exudate is usually sterile. Touching the area of the ulcer with phenol in the early stage of the ulcer has given me better results than anything else. Along with this measure, irrigations with normal salt solution are very valuable. The irrigation should be directed exactly to the spot, and this can be done best by erecting over the patient (on his back) a drop-bottle such as we use in the laboratory when making sections with the microtome. These instillations should be made every hour and should last five minutes at a time. Atropin of course is always demanded. The use of phenol, then, and frequent irrigations with salt solution at body temperature, constitute the treatment. I have never been at all satisfied with the use of heat in these cases, and have been disappointed with the galvanocautery. In my experience a pressure bandage acts admirably; exactly how, I am unable to say. It may help the nutrition and circulation of the eye and in this way promote the more rapid disappearance of the exudate in the anterior chamber.

Dr. S. Lewis Ziegler, Philadelphia: I am glad to learn from Dr. Verhoeff's investigations that a form of iodin can be applied to the eye without causing pain. I have used Lugol's solution internally in exophthalmic goiter with success, but I have never applied it locally. Tincture of iodin is extremely painful when applied to the cornea. A patient some years ago stated that tincture of iodin had been applied to his eye once, but that he would shoot the next man who would use it. Naturally, I did not apply it. The serum treatment has proved successful in certain cases in my hands, but not sufficiently so to depend on. For many years we relied on the Saemisch incision. Seeing a number of adhesions and a certain weakening of the cornea in large sloughs, led me to try peripheral paracentesis, which gave the same effect. I think it is really the flaccidity of the cornea which has a great deal to do with stopping the suppurative process. Following paracentesis we could obtain, if you choose, the removal of the hypopyon as a coagulum either with the Graefe dressing forceps or the Schmidt spoon forceps, and follow this with irrigation.

Speaking of Dr. Shahan's method, I have used heat only in the form of the heated cautery point, sometimes applied directly and sometimes applied as radiant heat; that is, not in contact. After cauterization I use the method Dr. Weeks spoke of, applying a solution of formaldehyd, not stronger, however, than 3 per cent., usually 1 per cent., with a small

pledget of cotton wound tightly on the end of an applicator, wetting it with the formaldehyd solution and removing the excess of moisture so that it will not spread to healthy tissue. The formaldehyd is then rubbed into the sloughing cornea until the ulcer is thoroughly macerated. The eye is then washed with boric acid solution. This method is just as applicable to corneal ulcer when you do not make a cauterization. I prefer to use it after cauterization, because the resulting scar is reduced and modified by the application of the formaldehyd; in fact, there is always less scarring when formaldehyd solution is used. Another point that might be mentioned is that a great many cases of sloughing keratitis, even after traumatism, are due to infection from the tear duct. The cause should, therefore, be removed. It has been my habit to stretch the tear duct by rapid dilatation in order to promote drainage and to prevent recurrence of the infection. This procedure has a very marked influence on all forms of corneal ulceration due to lacrimal lesions.

DR. F. PARK LEWIS, Buffalo: In the first place, the desideratum we all have in view is simplicity of apparatus, with efficiency. I believe that one of the added elements in the use of heat is the desiccation that comes from it. I have therefore used superheated air, by which one gets as nearly as possible the temperature Dr. Shahan thinks most desirable. After a good deal of experimentation to get an apparatus to get the air at the right temperature I found that such an instrument had been made for the rhinologists. It is simply a rheostat which can be attached to the air tank and a gradation of the heat obtained as you choose, and the superheated air directed on the infected area. In that way we not only sterilize the point covered, but it goes deeper into the tissues, as Dr. Weeks pointed out, under the overlapping edge and into the layers of the cornea; and the heat can penetrate without destruction of tissue. I have found its use in this way has in many cases produced results which no other method gives.

DR. L. WEBSTER FOX, Philadelphia: I have been expecting some of the essayists to name one of California's pioneers in ophthalmology, Dr. Barkan, as it was he who first applied heat to corneal ulcers. He introduced this procedure many years ago, and it has been followed by operators in all parts of the world. I have tried all the drugs that have been introduced and some of them have proved to be of value: there is, however, one additional drug not mentioned, and which I should like to add to the list, and that is trichloracetic acid. I have used it in 1, 2, 3 and 20 per cent. solutions, and have found it to be one of the most valuable drugs in our armamentarium. It is exceedingly penetrating, and will find its way into the spaces of von Recklinghausen where the cocci find lodgment and break down the tissue. I feel

sure that if it is given a fair trial it will be found most valuable. As a means of combating hypopyon it is one of the most efficacious drugs of which I know.

As to heat, I have tried Prince's method and found it not as satisfactory as I had hoped. Later, I devised a coil of platinum, which is held close to the corneal ulcer; this was applied 5 seconds on and off, alternating for one minute. The results were most satisfactory. As to the Saemisch operation, if any one will read Guthrie's method of puncturing the cornea he will admit that the Saemisch method was an old operation revived. Fortunately, I have not experienced the flowing out of pus and consequent prolapse of the iris. In every irritation of the cornea, as in ulcer, which is a terminal infection, we find the iris more or less contracted, and where there is evidence of pus there is inflammation and the iris will not prolapse unless a great deal of pressure has been used in executing the incision. Therefore, I feel that in performing a Saemisch incision, gentle pressure must be used.

DR. WILLIAM ZENTMAYER, Philadelphia: I have had experience with ethylhydrocuprein in about fifteen cases. I am quite sure my results have been much better than by any other single method or better than all the other combined methods. In fifteen cases I have not lost a single eye. Perforation occurred in about three cases. All the patients had good light perception and many had useful vision. I believe that this is a good result for any method of treating pneumococcic ulcer, especially the class of cases at Wills Hospital. You have been told that we often get them in a desperate condition. I believe optochin is worthy of further trial.

DR. FREDERICK H. VERHOEFF, Boston: I think those who have criticized the visual results I have reported have failed to note that the tests were made at the time the patients were discharged from the hospital. I attempted to keep track of the patients afterward, but it was so difficult I gave it up. I do not myself attach a great deal of importance to the visual results, but to the fact whether the ulcer was stopped in its progress. For instance, in some cases, although the ulcer was promptly healed, the iris was already tied down so that there was no clear space around the corneal scar. Since my paper was written, some of these cases have returned for optical iridectomy and obtained useful vision.

In regard to Dr. Shahan's method of applying heat, it seems to me it might be effective for moderate sized ulcers, but that the resulting scar would not be less in extent than that following other methods of cauterization. In large ulcers it seems to me that to kill the bacteria at the edge of the ulcer sufficient heat would have to be brought to bear to cause considerable opacity in the clear cornea. As he says, sufficient heat to kill bacteria will cause permanent

opacity in the rabbit's cornea. This means considerable injury, because the rabbit's cornea has a greater power to clear up than the human cornea.

DR. WILLIAM E. SHAHAN, St. Louis: Twenty-two cases have been treated by this method since December. Saemisch section was not required in any; none came to enucleation; the process was stopped in all. Final vision acuity depended on the size and situation of the original ulcer. The scar from a small central ulcer caused a greater visual loss than that from a larger peripheral one.

With ulcers larger than the largest point, the point at 158 F. was applied steadily for one minute to a part of the ulcer, then reheated and applied to another part, until all the ulcer had had a continuous application for one minute.

The advantage of this method of thermotherapy over all others is its exactness. We can reproduce the same condition every time and soon learn precisely what to expect. Moreover, heat applied in this way affects not only the surface of the ulcer, but passes directly through the cornea into the anterior chamber. This was proved by thermocouple measurements in the paper read at the Detroit session, and its practical value shown in a case of intracorneal abscess due to a perforating wound. There was a pneumococcus infection with hypopyon, etc. The wound closed leaving an abscess which would not heal and could not be reached by surface applications. Two or three one-minute applications of heat at intervals of several days stopped all symptoms and brought about permanent cure. This was a case of Dr. Wiener's.

The additional clouding caused by the heat can be limited accurately to the area of the ulcer and is of negligible importance as compared with an extension of the ulcer.

Several of the cases cured by this method had been fruitlessly treated with optochin. In fact, most of the cases had been vigorously treated by various methods before the heat treatment was applied. Three rodent (Mooren) ulcers were treated by this method without any benefit. This type of ulcer does not appear to be due to bacterial activity.

CRYSTALLINE DEPOSITS IN THE EYE

F. PARK LEWIS, M.D.
BUFFALO

A study of life processes, even though we are not able to arrive at final conclusions, is always a subject of great interest. For the ophthalmologist a condition in which side by side with the normal functioning of an organ or a tissue may be seen a return to the elemental substances of which it is composed is peculiarly important, because in the eye alone, of all of the tissues of the body, he may follow Nature at work in her laboratory. He may see the blood vessels swelling in congested areas. He may observe the serous transudation, becoming in time a plastic exudate. If he cannot actually discover the blood vessel in the act of breaking, he can at least determine its location and disposition when a hemorrhage occurs. He is able to note the process of absorption or of disintegration of the transuded fluid, following it finally to the point at which, through retrogressive changes, it is reduced to its ultimate constituents, and glistens in the retina or scintillates in the vitreous as scattered masses of bright light reflecting crystals of cholesterin or atrophic patches of pigmentary degeneration.

It is of interest to the ophthalmologist, moreover, as he recognizes these end-results, degenerative changes which he may trace back to their origins, for the reason that having observed their termination he may the more readily perceive the earliest tendencies which, if continued, may lead to like disastrous results. It is a condition, furthermore, that appeals to him as a pathologist, as the same forces that impede the progress of normal physiologic processes in the eye during

life may be producing similar changes in remote parts of the body where they cannot be seen, and where their existence may be only inferred from the presence of symptoms which are more or less misleading.

I have ventured, therefore, to choose a somewhat unusual subject, for though I may be unable to add any very new facts in relation to it, it may be worth while for us to consider in what tissues of the eye a reversion to the deposit of crystalline substances may occur, how these processes are brought about, and what they connote.

CRYSTALLINE CASTS IN THE CORNEA

It has been exceedingly rare in my experience to find in an eye, otherwise functioning normally, an extensive deposit of lime salts in the cornea. That they should occur is in itself an evidence that that particular part of the cornea has lost its vitality. One special case, coming under my observation some years ago, impressed me strongly:

The patient, a man then beyond middle life, and seemingly in perfectly normal health, had been under my care some twenty years before. At that time he suffered from an extensive corneal burn, involving both eyes and leaving deep opacities. Only the upper parts remained clear, and he was able to see through these by reason of iridectomies that I had made at the time, and had been thus enabled to carry on his work as a carpenter. After so many years he came again—with the ocular conjunctiva of the better eye on which he had chiefly depended highly injected and intensely painful. The sensation was as though it were filled with sand. The condition had been growing worse for weeks until it finally grew unbearable. The surface of the cornea presented the usual white nebulous appearance of a thick leukoma, with the vessels of the conjunctiva everywhere enlarged and extending to the limbus. On touching the surface with a probe I found that it gave a hard stony or glassy impression, and by running the point of the probe over the surface I found that a solid limy deposit had settled on the surface of the cornea, and while the surface seemed smooth, the epithelial covering had been wholly rubbed away by the lid and it was actually covered with fine elevations like pin points, in reality giving the effect of an extensive foreign body. With some little effort I succeeded in prying the mass from the eyeball, and it came away in the form of a perfect cast, the inner sur-

face especially being honeycombed where it had adapted itself to the roughened surface of the substantia propria of the cornea. The outer superficies were also roughened, giving the appearance of having at one time been smooth, but the more prominent parts seemed to have been worn off, either from the attrition of the lid or from the chemical action of the secretions of the eye. On the edges of the cornea several smaller flakes were scaled off in the same way. In some places it was necessary to take a sharp knife to scrape off the mineral deposits, so intimately had they fixed themselves in the interstices of the corneal fibers. The removal of the deposit ended the discomfort from which the patient suffered, and he has never had any recurrence so far as I know.

Similar deposits not infrequently develop in scar tissue, resulting from old corneal ulcers in which the epithelium has been destroyed. They are always slow in forming, and the pathology seems to be the same in both instances.

Occasionally lime deposits may be found in the substantia propria of the cornea in which there have been no previous inflammatory changes. These seem to be of much the same character as the structural degeneration which occurs in the elastic tissue of the vessels in arteriosclerosis.

CALCAREOUS DEGENERATION IN THE SUBSTANTIA PROPRIA CORNEAE

A spare, feeble old man, whose arteries had for many years been hard, had had for a long time nebulae scattered through the cornea. More recently these had grown dense. In the right eye a mass occupied the pupillary area and was perhaps 2 mm. in diameter, gradually thinning toward the edges. The arcus semilis had taken on such a degree of density that it could with difficulty be distinguished from the white scleral tissue. A relatively clear area formed a circular zone of sufficient breadth to show almost the entire iris and a small and not very mobile pupil. In course of time the lens became opaque, and as there existed a good projection field and blindness was already present, with no hope of improvement, I yielded to his importunities and extracted the lens. As the knife engaged the cornea the sensation conveyed to the hand was that of cutting a tissue filled with ashes, and even the sound of scraping could be heard as the blade encountered the hard limy deposit with which the tissue was strewn. This was undoubtedly a very rare condition and was allied to the "nodular opacity" in which Treacher Collins says that calcareous deposits are sometimes found.

The operation proceeded without any other unusual incident. Healing was followed by no inflammatory reaction, but so advanced were the degenerative changes that the small amount of improved vision obtained by the operation gradually disappeared.[1]

The concretions, which in the conjunctiva or on the tarsus have been considered of sufficient importance to have been carefully studied by Fuchs, are not of infrequent occurrence in the eyes of elderly people or when the eyes have been irritated or inflamed. Sometimes they are wholly encysted, occasionally they protrude through the epithelium, appearing as white or yellow bodies, and until they are removed cause much discomfort.

The concretions are the result of mucoid exudate in retention cysts. They are of the same character as the concretions found in degenerate chalazia.

Calcareous and osseous degeneration of the choroid have been so carefully studied, and so much has been written concerning these conditions, that I shall only briefly refer to them. The deposits, as was shown many years ago, are not in the choroid proper, but in the fibrous exudate between the choroid and the retina. Ossification is the last of the degenerative changes that occur in plastic choroiditis.

In eyes which have been subject to any long continued inflammatory process, calcareous concretions may be found in almost any of the tissues.

Bietti[2] found limy deposits in the optic nerve head. There was glaucomatous cupping in which new tissue was formed as a result of retinal inflammation, and in this the lime salts were deposited.

In nine cases reported by Rumschewitsch,[3] ossification and calcification were found in different structures in the eye. In six there was a deposit of bone in the choroid which was not confined to the choriocapillaris. In two there was ossification of cyclitic membranes.

1. A condition closely allied to this rare form of corneal degeneration is described by Collins and Mayou as "transverse calcareous film of the cornea."
2. Bietti: Ztschr. f. Augenh., January, 1908.
3. Rumschewitsch: Arch. f. Ophth., 1908.

In one there was a bony mass inside the vascular layer of the choroid with calcareous deposit in the retina.

Albertotti reports two cases of bony mass within the choroid.

Roy[4] records a calcifying fibroma of the orbit.

Arboleda, a South American ophthalmologist, describes a condition characterized by the presence of minute calcareous bodies in the palpebral conjunctiva, frequently seen in Bogota, usually at the lid margins and at the culdesac.

All of these conditions must be distinguished from conjunctivitis petrificans, to which attention has within the last few years been directed. Sidler-Huguenin records a remarkable case of the latter condition occurring in a hysterical girl, aged 16. It was induced by her putting lime in the conjunctiva sac and grinding it into the lids, by scarifying the conjunctiva and by contusing the lids. Chemically and anatomico-pathologically it differed scarcely at all from previously described cases of this remarkable form of conjunctival disease, and the author thinks that by a careful study of the patient, this disease as an entity may disappear from our literature.

The disease is so unusual that it may bear a brief description. The best description of it available is that found in the American Encyclopedia of Ophthalmology. It was first described by Leber in 1893, and is characterized by the presence in the conjunctiva of inflammatory swelling in which opaque white spots may be seen. The spots consist of lime in organic crystallizable combination. They increase in size as the disease spreads spasmodically, and finally coalesce, forming a hard, stony mass. New foci will appear while others are healing, and the process thus lasts for months or years. These smaller foci are said to vanish by absorption, while the larger ones coalesce into shriveled, thickened spots on the conjunctiva; but those who have seen cases of this kind do not

4. Roy: Montreal Med. Jour., 1908.

explain how it is possible for a limy concretion to be absorbed. Blindness is through corneal involvement, and the treatment consists of removing such a focus when it is possible to do so.

Del Monte, the Italian ophthalmologist, gives a detailed history of a case of this kind which came under his observation, in which the *Bacillus xerosis* was found to be present. The *Bacillus xerosis,* we know, is commonly found both in health and in disease. Indeed, it was the only micro-organism present in a large proportion of the cases of infantile ophthalmias reported to our local board of health during the past winter. There seemed to be a xerosis epidemic.

In Del Monte's case, in an eye previously sound, hyperemia of the tarsal conjunctiva and the fornices suddenly came on and remained there for about four days, accompanied by very copious secretion, and then on the middle portion of the tarsal conjunctiva below and in the inner sectors of the conjunctivae of the globe, sometimes relatively large and single, at other times minute and multiple, these being accompanied by sharp pain and profuse lacrimation. The epithelium over these hemorrhages was shed as grayish white detritus, the aspect of the parts resembling that of a burn of the membrane, the margins clean cut, but the floor irregular, covered by whitish or yellowish foci, not easily removed even with scraping, the whole aspect of the exudate tough, membraneless, smooth, not elevated, and surrounded by considerable hyperemia, as well as the resulting scars, strongly suggesting the appearance of a diphtheritic ulcer. Other little foci appeared also without being preceded by hemorrhages, similar in appearance, always small in size, and whitish. The healing of these superficial ulcerations, and then the return of pain, hemorrhages and the formation of fresh lesions went on for a time as narrated. Apart from an injection of serum no treatment seemed to produce any effect whatever.

The pathologic examination was exceedingly interesting. The process, it seemed, divided itself into two periods, as shown from the microscopic examination of portions of the tissue excised at an early stage in the morbid process. At a very early stage there was loss of epithelium, although the surface was smooth. In the substance the lymph spaces were very large, so much so that at parts the tissue was almost reduced to a large mesh network. In this tissue the vessels were dilated, with edema of the endothelium in all the coats. In the parts later removed there were signs of copious serous and cellular exudation which had parts in the interior of the fibrous tissue.

Del Monte's impressions from carefully made examinations are that the first step in the morbid process was an edema and cellular inflammation. Following this, by the absorption of the fluid, and in consequence of the pressure undergone which would, of course, be accentuated by the existence of highly resistant fibers, calcification might easily take place with partial necrosis of the tissues. Micro-organisms were obtained from the surface of the ulcers and from the broken down tissue, as well as from the surrounding zone of infiltration. Occasionally under the epithelium, at the edge of the ulcers, would be found large cells such as are seen in xerosis. Two forms of organisms were frequently present, one bacillary and the other coccal, but not, the author concludes, staphylococcal. He was unable to identify it with any measure of certainty. The xerosis bacillus was found deeply penetrating the tissues, sometimes even in leukocytes and in great numbers in the epithelial cells. Successful treatment followed the use of Behring's antitoxin serum.

Few cases of this rare disease have been reported. Among the most important is that of Leber before the Heidelberg Congress in 1895. Cases have also been observed by Reif and Mayweg and by Posey in this country.

Perhaps the most typical form of degeneration which may go through the various phases to the deposit of the lime salts is found in the lens as it develops into cataract.

When the lens begins to sclerose the fibers are drawn more closely together. With this is a contraction of the intrafibrillar cement, a narrowing of the fibers, and a firmer interlocking of their crenated edges. Between these shrunken fibers the interfibrillar fluid collects in drops. Gradually the opacity extends until the lens is ripe. Following the period of maturity comes that of hypermaturity, in which the cortex liquefies, forming an opaque milky fluid. The surface of the capsule loses its radial markings and presents a homogeneous surface on which little white specks may sometimes be seen. These are due, according to Mayou and Collins, to calcareous deposits in secondary cataracts which form as a result of the diminished intracapsular tension. Ultimately degenerative changes progress in morgagnian cataract, the fibers undergo fatty

degeneration, minute droplets form in them, giving rise to a granular appearance, these run together, and the fibers break down. In this way spaces are formed in the cortex of the lens containing a mixture of albuminous globules and drops of fat. As the cataract progresses, these spaces open up into one another, and more and more fibers undergo degeneration. Ultimately the whole cortex may become liquefied, and sometimes contains cholesterin crystals.

I have given this detailed description of the retrogressive changes which take place in the reduction of the lens from a vital organ into its primitive salts because it is exactly interpreted by our knowledge of cytology. In all life processes the structure proceeds from the simple to the more complex, from the elemental to the composite. When the vital bone ceases to be dominant the complex structures revert, going back again, step by step, through the same processes to their simple constituents.

The more important advances that have been made during the past decade have been along the line of physiologic chemistry. With the upbuilding in its simplest form of the substances found in air and water into the protoplasm which becomes organized into living structures, capable of performing the functions for which it is designed, there must be corresponding retrogressive changes. In order that that perfect equipoise may be secured, the balance of the increment and the excrement of the cell must be maintained. This balance being once destroyed, the initiation of structural degeneration will have begun.

In considering this reversion in the living body to primitive forms we are looking on the last phase of organic life. It is the end-product of a long series of changes, of which the beginning was in the cell. There would be little of value, or even of interest, in recounting such conditions, even as curiosities in pathology, if they carried with them no intimation of the reason why the vital energy had departed, and why with its

departure the structure, which it had held together, fell to pieces. This the study of dead tissues, no matter how carefully performed, fails to tell us.

The first altered pathologic condition which we see is macroscopic; the next is microscopic; the third — that which is most helpful — deals with physiologic chemistry.

BIOLOGY OF THE CELL

In every case of disease there must have been a wide range of gradations, from the initial disturbance of nutrition through the breaking down of structures, up to the final dissolution of the material into the elements of which it is composed. The end-results are visible to the naked eye. They are more fully detailed under the microscope, but they can be comprehended only through our study of physiologic chemistry. Permit me, therefore, in a word to recall a few of the important facts which have a bearing on the biology of the cell.

Every living cell, no matter what may be its function or ultimate destination, is a mass of protoplasm enclosed within a cell wall. It has in its substance proteins, lipoids and an enzyme. It is in fact a colloid in which crystalloid substances are readily soluble. Under the influence of chemical action, of heat, of electricity, of light or of radioactive forces, the cell is split up into the component carbohydrates, the molecules forming new groupings, through a long series, until the simpler forms are finally reached.

As Pauli has shown in his various lectures on "Physical Chemistry in Medicine," physiologic experience compels us to believe that chemical reactions of the most different kinds are simultaneously possible in the homogeneous ground substance of the cell; and in even the smallest particles of protoplasm, antagonistic chemical reactions, such as oxidation and reduction, hydration and loss of water, condensation, synthesis and their opposites, assimilation and dissimilation, are likely to occur through and beside each other.

Just as the chemist allows different chemical reactions to take place in different vessels, the cell is believed to utilize the different chambers of the honeycomb structure, and with the help of the colloidal ferments, the number and knowledge of which is daily growing, to allow the necessary reactions to go on independently of each other.

In order that we may visualize these cellular activities, which are constantly taking place, we must think in terms of cell structure continuously related. With such colloids as the cornea, the membrane of Descemet, the lens capsule, the lens cortex, the vitreous, the hyaline membrane, the nerve cells of the retina and the membrane of Bruch in contact with such colloids as the lymph and the blood plasma, with the aqueous filled with crystalloids in solution, with each cell penetrable by the toxins given off from bacteria or from chemical changes, we may begin to realize the enormous possibilities that are opened up to us, not only concerning the physiology and the pathology of the eye, but as to the therapy as well. The same laws, it will be remembered, govern the cell contents and its membrane as to osmosis and surface pressure as apply to other animal membranes, and while it is not yet proved, it is not at all improbable that the edema within the eye, whether it takes the form of an acute or a chronic, a simple or a fulminating glaucoma, may ultimately be found to be due to the swelling within the vitreous cells of soluble toxins. Equally the atrophic changes in the lens fibers, like the fibrous and subsequently fatty changes in xanthelasma, or in the arcus senilis, are evidences of the presence of soluble toxins which have disturbed the normal functioning of local cells.

Therapeutically it is clear that every drug substance introduced into the animal body must be ionized before its constituent electrons can activate those within the living cell. This is, of course, aside from the subject under consideration. It has been most

interestingly treated in a little volume by Leduc entitled "Electric Ions and Their Use in Medicine."

The acute or chronic inflammation of a tissue is only an intermediate stage, and is produced by a local irritation which may be due to a toxin introduced by some pathogenic bacterium.

Practically all forms of uveitis that are not traumatic, and many that are traumatic, are due to this cause. We no longer speak of rheumatic iritis. We look for the source of infection of both the rheumatism and the iritic inflammation. It may be found in an apical tooth abscess, it may arise from an infected sinus, or it may be as remote as the appendix. From such a locally infected point the toxin given off by the bacterium, the staphylolysin or the streptocolysin, is readily absorbed by the blood plasma and carried to the uveal tissues. Many illustrations come readily to mind, but one will suffice:

A lad of 18 had for five years been suffering from choroiditis. Large patches of exudation had been followed by atrophy, involving the macula of one eye, and coming dangerously near the visual center of the other. He had been under able, conscientious and expert care. When he was seen in midsummer in consultation it was evident that there must be somewhere a nidus, a focus of infection. He was seemingly perfectly well, free from both tuberculosis and syphilis. An investigation of his nose at first revealed nothing but a swollen middle turbinal. His rhinologist was urged to remove it for exploratory purposes. When this was done, immediately a mass of thick yellow pus appeared. The ethmoid was fully explored, opened and cleaned, and the active disease in the choroid became quiet. Two months later a new attack supervened, when a deeper hidden infected cavity was disclosed, and a roentgenogram revealed a blind abscess at the root of a molar tooth. Both were relieved when some weeks later a third attack developed. On this occasion the small, hard, sunken tonsils were removed, and both were found diseased. Since then there has been no recurrence of the active uveal disease; one cannot but feel, however, that had the source of the trouble been recognized much earlier the young man might have escaped the handicap of defective sight for the remainder of his life.

One hesitates to speculate, but the facts so rapidly accumulating all seem to point to the absorption of

different toxins as a possible, if not a probable, cause of both glaucoma and cataract.

In a recent case of glaucoma, in which excellent iridectomies had been made by an eminent Scotch surgeon, and in which only a remnant of sight remained, there were evidences of extensive choroiditis. The poor woman, finally in perfect health, had lost every tooth. She said that during the years in which she was steadily going blind she suffered constantly from toothache. It is well known that no sanitary precaution has been more neglected among the poorer classes, and even among the fairly well-to-do, than the care of the teeth.

One can readily understand from the cesspools that one finds in the mouths of those from whom more would be expected that these must be never ending sources of infection. I think that we will find, too, if we go outside of our special lines of work, that there is not a patient who comes to us, with commencing striae in the lens, who will not be found to have impaired metabolic functions. There will be a greater or lesser degree of intestinal indigestion, with the resultant absorption of toxic products. And if we would avoid the final degeneration changes, which so often are presented to us, we must seek for all possible sources of infection before the damage has been done.

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ABSTRACT OF DISCUSSION

DR. BURTON CHANCE, Philadelphia: Nothing should interest us more than to observe the physiologic action of the processes which maintain the health of the eye, and to seek for the causes of the degeneration of its tissues. Dr. Lewis does well to draw our attention to a consideration of certain aspects of the activities of Nature's laboratory. In spite of an earnest desire to get at the bottom of things, it is so difficult to obtain a satisfactory view of the chemical reactions which are constantly going on in the ocular tissues that anxious and careful observers are only too often disappointed by the results of their labors. We see general states, the depravity of which certainly justifies us in assuming that they must tend to the manufacture of compounds which act

deleteriously on the delicate and sensitive ocular tissues. And again, when consulted for the first time by patients whose eyes exhibit the well-marked signs of degeneration, we are nonplused by our inability to exactly link up the degenerate tissues with a bodily ailment, the signs of which might have been observable from its inception. The disease entities which we speak of as "degenerations," in conjunctiva and cornea, are prominent examples of this subject, for their origins are obscure, in so far as their presence has been unaccompanied by the classical signs of inflammation.

It has been my experience to have had a number of cases of nodular degeneration of the cornea in several instances in more than one member of a family, yet in none of them have my colleagues skilled in laboratory examinations been able to throw light on the origin of the affection. Neither have they conceived of any state of the system which might be taken as indicative of the earliest signs of disturbance within the general economy, which disturbance, if uncorrected, should inevitably lead to conditions similar to what we, as ophthalmologists, might take to be the end-results of a definitely deranged physicochemical process such as, for instance, diabetes. The whole subject, therefore, as the ophthalmologist views it, is all but metaphysical.

Nevertheless, the ophthalmologist is daily confronted by problems in which he finds present in diseased eyes calcareous, crystalloid or colloidal products of inflammation which greatly interfere with the function or, it may be, destroy the health of these organs. How frequently we find, do we not, on sectioning the quadrate globes of phthisis bulbi, plates of bony tissue in the midst of matted choroidal exudation?

In this connection, it may be interesting to show a wholly calcified lens taken from a shrunken globe which had been diseased for forty years, the choroid of which was bony. Such a specimen vividly shows the chemical resultants of inaction in a highly specialized organ; it remained as a truly dead inorganic structure. Is it not fascinating to imagine what were the physicochemical processes which destroyed the cellular elements in this crystalline body, as well as to endeavor to consider fully the processes concerned in the case of extreme hypermaturity, in which occurs complete dissolution and dispersion of the lens so that only the capsule remains?

Can it be that ordinary senile dissolution depends on the wearing out of the cell? There is not even here "perpetual motion" in destruction and repair, while the reactions of the cell to toxins excited or generated by bacteria yield as their results inflammation with consequent degeneration.

Dr. Lewis has touched on many obscure subjects, to any of which we at this time can give only a passing glance. When he cites cases of uveitis dependent on disease of the

dental structures, immediately we are reminded of similar cases which have occurred in our own practice. From the earliest years of my professional life I have regarded the dentist's function to be truly a part of the great body of medicine. And, from teachings received from my deceased brother, a dentist with keen powers of observation, I have insisted on the examination of the teeth and gums in all cases of uveal and protracted tarsal and conjunctival diseases, as well as before operations on the eyeball. It has not always been possible, however, to pursue such attention in public practice. As a comment on this state of things, it has been my experience to note in cases of delayed recovery after uncomplicated cataract operation that none occurred in subjects with good teeth, but in those whose gums were diseased and whose teeth were broken and decayed.

DR. MELVILLE BLACK, Denver: I have here a beautiful specimen of a bony choroid and calcareous lens. I had the chance to see this man when the eye was originally involved twenty-six years ago. It was a luetic case of uveitis running a protracted course, resulting in softening of the eyeball and blindness. The eye became divergent and the lens opaque, and it was evident that it had undergone calcareous degeneration. He submitted to enucleation after beginning sympathetic irritation of the other eye.

THE ANAPHYLACTIC BASIS OF SYMPATHETIC OPHTHALMIA

AN EXPERIMENTAL STUDY *

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The anaphylactic theory of the etiology of sympathetic ophthalmia is the most recent of the many theories brought forward in the effort to explain the true nature of the disease.

The theories offered in explanation of sympathetic ophthalmia fall into three general groups:

1. The theories which explain the disease through ciliary nerve reflexes. These theories may be dismissed without comment.

2. The theories which assume that the disease is bacterial in origin, through bacterial metastasis from the exciting to the sympathizing eye, either through the blood and lymph channels, or by actual migration of the bacteria along the optic tract. The objections to this theory are many. There is no proof of such metastasis or migration. Deutschmann's[1] assertions of having found an organism specific for the disease have not been substantiated. A selective affinity for the unknown exciting organism is assumed, which cannot be conceded, despite the work of Rosenow. Sympathetic ophthalmia resulting from noninfectious disorders of the exciting eye, such as subconjunctival scleral rupture and disintegrating intra-ocular sarcoma, has been reported. This is a type of sympathetic ophthalmia to which the bacterial theories cannot possibly be applied.

* From the John Herr Musser Department of Research Medicine, University of Pennsylvania.

1. Deutschmann, F.: Zur Pathogenese der sympathischen Ophthalmie, III, Arch. f. Ophth., 1912, **81**, 35.

3. The cytotoxic and the anaphylactic theory. This group of theories has arisen to meet the foregoing objections. The cytotoxic theory, advanced by Golowin,[2] assumes that the disintegrating uvea in the exciting eye liberates a cellular poison, a cytotoxin, which is specific for uveal tissue. This cytotoxin is carried by the blood stream to the second eye, where it attacks the uvea, and causes the clinical condition of sympathetic ophthalmia. Valuable as was this hypothesis, as it opened up channels for new thought and investigation, yet the theory itself must at present be disregarded as an explanation of the disease, for, in spite of much investigation, little or no experimental proof has been brought forward in its support.

The anaphylactic theory may be regarded as the outgrowth of the great number of observations of the local anaphylactic phenomena which could be produced in the eye by experimental means. Before Elschnig stated his belief in the anaphylactic theory and presented his experimental work in its support, a great amount of work had been done on experimental ocular anaphylaxis. This experimental work has already been reported in detail.[3] For our present purposes it may be summarized by saying that the following well controlled experiments have been clearly established: (1) A prompt inflammatory ocular reaction is produced by intra-ocular or intracorneal injection of foreign protein in animals previously sensitized by similar injection of the other eye; (2) intravenous injection of foreign protein in animals previously sensitized by intra-ocular injection produces inflammatory reactions in the injected eye, and (3) the intra-ocular injection of foreign protein in animals previously sensitized by intravenous, intraperitoneal or subcutaneous injection produces similar ocular inflammation. It had thus been established that local ocular phenomena of an anaphylactic nature could be produced.

2. Golowin: Hypothese der Autozytotoxischen Entstehung von Augenerkrankungen, Klin. Monatsbl. f. Augenh., 1909, **7**, 150.

3. Woods, A. C.: Ocular Anaphylaxis, I, The Reaction to Perfusion with Specific Antigen, Arch Ophth., 1916, **45**, 557.

With the possible exception of the well known experiments of Wessely on keratitis anaphylactica, these experiments seemed to have but little clinical significance. Yet these observations of ocular anaphylactic phenomena seem to be responsible for the conception of the anaphylactic theory.

The anaphylactic theory of the etiology of sympathetic ophthalmia assumes that the disease is an anaphylactic uveitis, brought about thus: The injury to the uvea in the exciting eye, by trauma, intra-ocular tumor, etc., leads to a destruction or disintegration of the uveal tissue. This uveal tissue is absorbed, and acts as a foreign protein, or antigen, to the organism, and produces a hypersensitiveness of the organism, and especially of the homologous organ, the second eye. A reaction now takes place between the sensitized uvea of the second eye and the antigens circulating in the blood or lymph. This anaphylactic reaction or intoxication is manifested clinically as a sympathetic ophthalmia.

Bold as this theory appears at first glance, it readily explains many of the clinical observations and symptoms of the disease. In the first place, it explains sympathetic ophthalmia, whether the disease in the exciting eye is bacterial or otherwise. Moreover, it explains most readily the incubation period, usually from two to six weeks, elapsing between the disease in the exciting eye and the outbreak in the sympathizing eye. This is the period after sensitization in which the organism develops the greatest sensitivity.

This theory, however, could not for a moment be tolerated in the absence of experimental proof. It assumes several fundamental points, which were in general distinctly opposed to the prevalent ideas concerning the properties of native body protein. It assumes that uveal tissue, or some constituent of the uvea, could act as an antigen, or foreign protein, to the same (homologous) animal. This means that it

must differ from practically all the other proteins of the body, for normal body protein cannot so act.[4]

This uveal tissue, after acting as antigen and producing a sensitization, must likewise be alone capable of producing intoxication. Were this not so, enucleation of the exciting eye (and the dangerous uvea) would be of no value, while the body contained other equally dangerous protein. To express it more technically, after sensitization from uveal tissue, intoxication must be from uveal tissue alone, and not for other body tissue. This is equivalent to assuming an organ specificity, and a lack of species specificity.[5]

The anaphylactic theory, as will be shown later, assumes many other points which need to be proved before the theory can be finally accepted. These cardinal points, however, (1) the ability of uveal tissue to act as foreign protein in the homologous animal, (2) organ specificity, and (3) lack of species specificity, are of such a fundamental and striking nature that it was necessary to establish these in order to command any consideration for the anaphylactic theory.

Elschnig fully realized this, and, in his presentation of the theory in its present form, presented experimental work to substantiate these assumed points.

HISTORICAL

Elschnig's[6] work, appearing in a series of papers, may be thus summarized: Using as his methods the intra-ocular injection of sheep erythrocytes and cholera vibrio extracts, and as his indexes the hemolytic titer and agglutination reactions of the blood serum, he established the fact that absorp-

4. This is a general rule. An exception, however, lens protein, has already been proved to exist.

5. Organ specificity implies that in an organism sensitized to a given organ or tissue, anaphylactic intoxication may be elicited only by reinjection of the same organ or tissue, with little regard to the species of animal from which obtained. Species specificity implies that, in animals sensitized to the ordinary protein of any given species, anaphylactic intoxication may be elicited by the reinjection of any protein from this species animal, with little regard to the organ. Lens, testicle and a few other proteins have been shown to be organ specific and not species specific.

6. Elschnig, A.: Studien zur sympathischen Ophthalmia, I. Wirkung von Antigenen vom Augeninnern aus, Arch. f. Ophth., 1910, **75**, 459; II, Die Antigene Wirkung des Augenpigments, ibid., 1910, **76**, 509; III, ibid., **78**, 549. Elschnig, A., and Salus, R.: IV, Wirkung des Augenpigments, Arch. f. Ophth., 1911, **79**, 428.

tion from the eye could lead to immune body production. He then sought to determine three points, to give scientific support to the anaphylactic theory. These points were: 1. Does uveal tissue possess antigenic properties? If so, in the homologous animal? 2. What constituent of the uveal tract is responsible for such properties? 3. What are the antigenic properties as regards organ and species specificity? Elschnig immunized rabbits against various heterologous (foreign) uveae, and against homologous (rabbit's) uvea, and finally against so-called "chemically pure" pigment from the uveal tract of various animals. Thus he obtained hetero-immune and iso-immune uvea serum,[7] and pigment-immune serums. He then used the complement fixation reaction for the detection of immune bodies in these serums and the study of their properties. He found that heterologous uvea produced immune bodies, as would be expected. Homologous uvea, also, acted as antigen and produced immune bodies. With the serums of animals immunized to uvea, complement was fixed by any uvea antigen, irrespective of species; the immune bodies were organ specific. As regards species specificity, the serums of animals immunized to emulsion of whole uvea were species specific in their reactions. The serums of animals immunized to pigment, however, were not species specific. The reason for this apparent anomaly is evident. Emulsion of whole uvea contains two elements, (1) pigment which is not species specific, and (2) such tissue as blood, smooth muscle and connective tissue, which is species specific. Immunization with heterologous uveal emulsion gives an immunity not only to nonspecies specific pigment, but also to the other species specific elements in the uveal tract. The species specificity of uvea immune serums is due to the last elements.

Elschnig therefore concluded that uveal tissue could act as antigen in the same animal, and that the pigment was the constituent responsible for this property. The pigment was organ specific and not species specific.

The remaining work on this subject may be quickly summarized. Weichardt and Kümmell,[8] using the doubtfully valuable epiphanin reaction, substantiated Elschnig's findings. Likewise, with the epiphanin reaction, and complement fixation, Kümmell[9] demonstrated uveal antibodies in a percentage of the serums of patients with sympathetic ophthalmia. Wissmann[10] supported the anaphylactic theory by experimental work, and showed uvea immune bodies in the serum

7. The term "hetero-immune serums" is used throughout this paper to designate the serums of animals immunized to foreign uvea or pigment. Likewise the term "iso-immune serums" designates the serums of animals immunized to their own species uvea or pigment.

8. Weichardt, W., and Kümmell, R.: Studien über Organ-Specifizität des Uvaeiweisses, München. med. Wchnschr., 1911, **58**, 1714.

9. Kümmell, R.: Versuche einer Serumreaktion der sympathischen Ophthalmie, Arch. f. Ophth., 1912, **81**, 486.

10. Wissmann, R.: Ueber Versuche mit Augen-Extrakten, Arch. f. Ophth., 1911, **80**, 399.

of two sympathetic ophthalmia patients by the precipitin reaction, but failed to substantiate Kümmell's work with complement fixation, on the serums of these patients. Rados[11] substantiated Elschnig's work only partially. Fuchs and Meller[12] were unable to demonstrate uveal antibodies in the serums of patients with sympathetic ophthalmia. Von Szily,[13] after lending much valuable support to the theory, criticizes the antigenic properties of pigment.

In this unsatisfactory state the anaphylactic theory remained. Elschnig asserted that he had shown that uveal tissue possessed the fundamental properties necessary to make the theory an actual possibility. These properties were: (1) the ability of uveal tissue to act as an antigen in the same species animal; (2) organ specificity, and (3) lack of species specificity, with the pigment being the constituent of the uvea responsible for these properties. The remaining work may be said to have enhanced the probability of the suggestion without detracting from it.

THE PURPOSE AND PLAN OF THIS WORK

The attractiveness of the anaphylactic theory and the importance of Elschnig's work cannot be disregarded or discounted, yet the theory, in the state it remained, could not be accepted. In a critical review of the subject, and of the experimental work on ocular anaphylaxis, several points are at once apparent. In the first place, Elschnig's work has never been repeated and substantiated in toto. Even granting that the antigenic properties of uveal tissue are established, other fundamental points remain to be proved. Although anaphylactic phenomena have been demonstrated in the eye by sensitization and intoxication, either the sensitization or the intoxicating dose, in every

11. Rados, A.: Ueber das Auftreten von Komplementbindenden Antikörpern nach Vorbehandlung mit arteigenen Gewebezellen, nebst Bemerkungen über die anaphylaktische Entstehung der sympathischen Ophthalmie, Ztschr. f. Immunitätsforsch., 1913, **20**, orig., 305.

12. Fuchs, A., and Meller, J.: Studien zur Frage einer anaphylaktischen Ophthalmie, Arch. f. Ophth., 1914, **88**, 280.

13. Von Szily, A.: Anaphylaxie versuche mit seg. chemisch reinem Augenpigment (von Rind, Schwein und Kaninchen) nebst pathologisch-anatomischen Untersuchungen, I, Klin. Monatsbl. f. Augenh., 1916, **56**, 79.

case, has been by ocular injection, with the resultant trauma obscuring the results. The anaphylactic theory assumes, however, that sensitization must be from general absorption, and intoxication from antigen circulating either in the vascular or lymphatic apparatus. Ocular anaphylactic phenomena have never been demonstrated by this means. It must be shown, therefore, that ocular anaphylactic phenomena can be produced by this vascular sensitization and intoxication, and that uveal tissue can exert its antigenic properties through the same mechanism. It must be shown that homologous uveal tissue absorbed from one eye can create a hypersensitiveness in the second. Finally, sympathetic ophthalmia should be reproduced experimentally by anaphylactic means.

In accordance with this conception of the problem, we have made an experimental study of the entire subject, attacking each phase separately. It is the purpose of this paper to present the work as a whole, bringing together the various points which establish a scientific basis for the anaphylactic theory of sympathetic ophthalmia.

Accordingly, the plan of this work was: 1. To repeat, and establish, if possible, Elschnig's work with complement fixation on the specific antigenic properties of uveal tissue. 2. To determine whether or not ocular anaphylactic phenomena could be elicited, after general sensitization, through antigen carried to the eyes by a vascular apparatus. 3. To determine the ability of uveal tissue to act through this mechanism, and at the same time to determine the specific constituent of uveal tissue responsible for this action, and its properties in regard to organ and species specificity. 4. To determine the ability of homologous uveal pigment free to be absorbed from one eye, to create a hypersensitiveness in the second eye. 5. To attempt the experimental reproduction of sympathetic ophthalmia by an anaphylactic means, through the use of uveal tissue as antigen.

I. THE ANTIGENIC PROPERTIES OF UVEAL TISSUE AS SHOWN BY COMPLEMENT FIXATION

This, as mentioned before, is substantially a repetition, in toto, of Elschnig's work.

Dogs were immunized by intraperitoneal injections repeated every six days, against cow's uveal emulsion, cow's uveal pigment and dog's uveal emulsion and uveal pigment.[14] In order to avoid nonspecific fixation, the serums of the dogs selected for this work were all examined in the complement fixation reaction with the various uveal emulsion and uveal pigment antigens, before immunization was begun. Eight dogs, giving completely negative reactions with all antigens in this preliminary work, were selected for immunization.

At suitable intervals after the last injection, the serums of these dogs were tested again against all the antigens. The results are shown in Tables 1, 2, 3 and 4. In order to quantitate the strength of the reaction, three different quantities of serums were used with every antigen. A + + + reaction indicates complete, or practically complete, fixation of complement with all quantities of serums; a + + reaction, similar fixation of complement in the tubes containing two largest amounts of serums, and a single + reaction indicates that such fixation was observed only in the tube containing the maximum amount of serum.

The first two complement fixation reactions were done with the same antigens used in the preliminary reaction. The last reaction was done with freshly prepared antigens. All antigens were used in one-third the anticomplementary dose.

All animals showed immune serums, differing, as might be expected, in the various dogs. Dogs 16-96 and 16-98 showed the lowest immunity.

Table 1 illustrates the complement binding phenomena shown by heterologous immune serums. As would be expected, these serums gave complement

14. The details of technic used to prepare the uveal emulsion and uveal pigment may be found in the papers already published and to appear shortly.

binding with their specific antigens. Moreover, the uvea-immune serums gave fixation of complement with pigment antigens. Pigment-immune serums fixed complement with pigment antigens, showing here that the pigment itself is capable of acting as antigen.

Table 2 illustrates the complement binding reactions of the serums of dogs immunized to dog's uveal emulsion and dog's pigment, iso-immune serums. The iso-immune serums gave fixation of complement with their specific antigens, showing the presence of iso-immune bodies. In other words, uveal tissue possesses the power to act as a foreign protein — as antigen — in animals of the same species. This is the first, and cardinal point, to be proved in order to establish the anaphylactic theory. Furthermore, the purified pigment of the uvea possesses this power of immune body production in the homologous animal, and uvea-immune serum gives fixation of complement with pigment antigens. This substantiates Elschnig's contention that it is the pigment which is the responsible factor for the peculiar antigenic properties of uveal tissue.

Table 3 illustrates the organ specificity of the immune serums. Iso-immune serums gave positive complement binding reactions with heterologous antigens, and the heterologous immune serums gave complement binding with the iso-antigens. In other words, the immune bodies were organ specific, showing an affinity for antigens of the specific tissue, without regard to the species from which obtained. This substantiates Elschnig's second point of the organ specificity of uveal tissue. The pigment-immune serums show this property also, and it is evidently to the pigment constituent that this organ specificity of uveal tissue is due.

Table 4 illustrates the question of species specificity. With the serums of animals immunized to cow's uveal emulsion and cow's uveal pigment, it was determined whether or not fixation of complement occurred with antigens of cow's protein, other than uveal tissue. It will

TABLE 1.—HETEROLOGOUS SENSITIZATION (COW'S UVEA AND PIGMENT)

Serum	Antigen	Dec. 14	Period of Immunization	Jan. 31	Feb. 14	Feb. 21 Fresh Antigens
16-89. Uvea-immune	Cow's uvea	Negative		+ +	+ +	+ + +
	Cow's pigment	Negative		+ +	+	+ + +
16-90. Uvea-immune	Cow's uvea	Negative		+ + +	+ + +	+ + +
	Cow's pigment	Negative		+	+ +	+ + +
16-91. Pigment-immune	Cow's uvea	Negative		+ + +	+ +	+ + +
	Cow's pigment	Negative		+ +	+ +	+ + +
16-92. Pigment-immune	Cow's uvea	Negative		+ + +	+ + +	+ + +
	Cow's pigment	Negative		+ + +	+ +	+ + +

TABLE 2.—HOMOLOGOUS IMMUNIZATION (DOG'S UVEA AND PIGMENT)

Serum	Antigen	Dec. 14	Period of Immunization	Feb. 6	Feb. 14	Feb. 21 Fresh Antigens
16-95. Uvea-immune	Dog's uvea	Negative		+ + +	+ +	+ + +
	Dog's pigment	Negative		+ +	+ +	+ +
16-96. Uvea-immune	Dog's uvea	Negative		+ +	+	+ + +
	Dog's pigment	Negative		+	Negative	+ +
16-98. Pigment-immune	Dog's uvea	Negative		+	+	+ +
	Dog's pigment	Negative		+	+	+ +
16-99. Pigment-immune	Dog's uvea	Negative		+ +	+	+ +
	Dog's pigment	Negative		+	+ +	+ +

be seen by a study of this table that the uvea-immune serums give complete fixation of complement with antigens of cow's liver and kidney extract. The reason for this is that already stated — that these dogs were immunized with the whole uvea, which contained not only the uveal pigment, but also some blood, smooth muscle and connective tissue of the cow. The fixation of complement with the liver and kidney extracts is dependent on the immunization with the last factors.

On the other hand, the serums of dogs immunized to cow's pigment alone gave either weak or negative reactions with cow's liver and kidney. The pigment was purified as much as possible, but it probably still contained traces of the other elements of the uveal tract —the blood, smooth muscle and connective tissue. The weak reactions occasionally observed are probably due to this impurity.

However, the difference in the degree of fixation of complement between the uvea-immune and the pigment-immune serums with the same antigens is striking. It seems reasonable to conclude from this, with Elschnig, that the pigment, acting as antigen, lacks, at least in a degree, species specificity.

These observations of the complement binding reactions of uvea and pigment-immune serums corroborate Elschnig's findings. Heterologous and homologous uveal tissues have the power of acting as antigens. In the case of homologous uveal tissue, the pigment is the factor responsible for its antigenic properties.

Whole uveal emulsion is both organ specific and species specific, the species specificity probably being due to the blood, smooth muscle, and connective tissue in the emulsion. The pigment, however, is organ specific and probably not species specific, and in this respect is analogous to lens protein, and differs from other body protein.

These properties of uveal pigment — ability to act as foreign protein to animals of the same species, organ specificity, and lack of species specificity — are, as

TABLE 3.—ORGAN SPECIFICITY

Serum	Antigen	Dec. 14	Period of Immunization	Jan 1, Feb. 6	Feb. 14	Feb. 21 Fresh Antigens
16-89. Cow's uvea-immune	Dog's uvea	Negative		++	+	++
	Dog's pigment	Negative		+	+	++
16-90. Cow's uvea-immune	Dog's uvea	Negative		++	+	+
	Dog's pigment	Negative		+	Negative	+
16-91. Cow's pigment-immune	Dog's uvea	Negative		++	Negative	++
	Dog's pigment	Negative		+	Negative	++
16-92. Cow's pigment-immune	Dog's uvea	Negative		++	+	+++
	Dog's pigment	Negative		++	+	++
16-95. Dog's uvea-immune	Cow's uvea	Negative		+++	++	+++
	Cow's pigment	Negative		++	+	+++
16-96. Dog's uvea-immune	Cow's uvea	Negative		+++	++	+++
	Cow's pigment	Negative		++	+	+++
16-98. Dog's pigment-immune	Cow's uvea	Negative		+++	++	++
	Cow's pigment	Negative		+++	++	+++
16-99. Dog's pigment-immune	Cow's uvea	Negative		+++	++	++
	Cow's pigment	Negative		+++	++	++

TABLE 4.—SPECIES SPECIFICITY

Serum	Antigen	Period of Immunization	Jan. 31 and Feb. 6	Feb. 14	Feb. 21 Fresh Antigens
16-89. Cow's uvea-immune	Cow's liver		+++	+	++
	Cow's kidney		+++	++	++
16-90. Cow's uvea-immune	Cow's liver		+++	+++	+++
	Cow's kidney		+++	+++	+++
16-91. Cow's pigment-immune	Cow's liver		Negative	Negative	++
	Cow's kidney		+	Negative	+
16-92. Cow's pigment-immune	Cow's liver		+	+	Negative
	Cow's kidney		+	++	++

before emphasized, the fundamental properties which uveal tissue needs to possess to make the anaphylactic theory a possibility.

II. OCULAR ANAPHYLAXIS THROUGH GENERAL SENSITIZATION AND VASCULAR INTOXICATION; THE PERFUSION OF THE EYE

The second phase of the problem is the determination of whether or not ocular anaphylactic phenomena can be elicited in a generally sensitized animal through antigen circulating in a vascular system, as must be the case if sympathetic ophthalmia is the result of intoxication of a sensitized eye. Efforts have been made to study this problem in the past, but have been unsuccessful for the reason that little is known of the actual mechanism of anaphylaxis, and the relatively crude methods commonly employed for the production of anaphylactic shock did not produce ocular reactions. In an animal generally sensitized and intoxicated, the stormy general reaction masks any ocular response; and the quick death or recovery of the animal makes protracted study impossible. If a smaller intoxicating dose is given, the action is evanescent, and finer anaphylactic reactions may not occur. In order to determine, therefore, whether or not anaphylactic ocular phenomena may occur in a generally sensitized animal through antigen carried to the eye by a vascular apparatus, we have resorted to the perfusion of the eye. The apparatus employed has already been described.[3] Figure 1 shows diagrammatically the operation and the position of the inflow and outflow cannulas.

With this technic we were dealing with what was to all purposes a living eye, maintained on an artificial circulation with defibrinated blood oxygenated by an artificial lung. Although the dog's heart always ceased to beat after the final ligature was placed above the heart, nevertheless the winking reflex persisted in the eyes often for an hour or more.

In this experiment the eyes were perfused for three hours, constant observations being made throughout that period.

It was found that when a sensitized dog was perfused with the defibrinated blood of a normal dog,

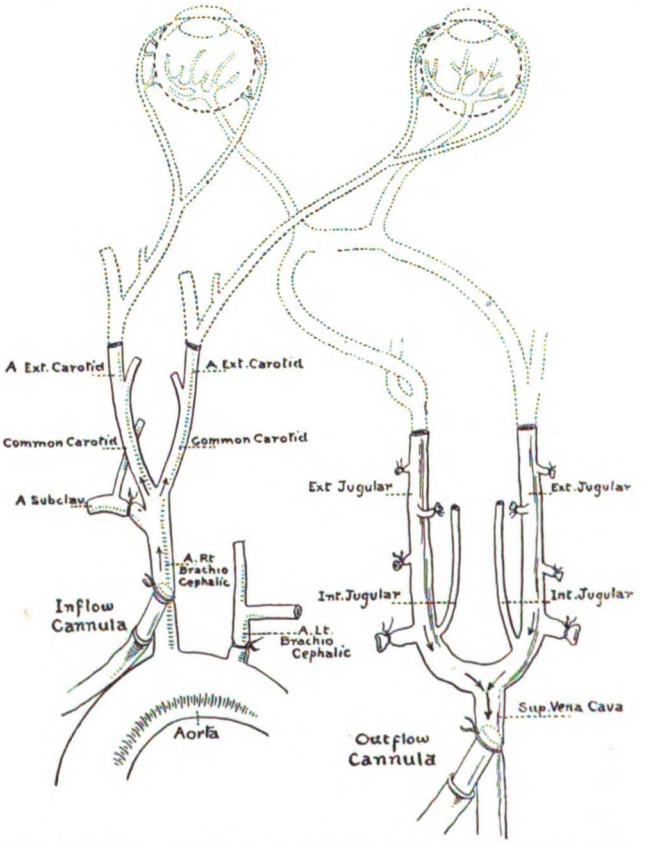

Fig. 1.—Diagram showing operation, position of inflow and outflow cannulas and scheme of perfusion.

there was no ocular reaction. When, however, the specific antigen (the antigen to which the dog was sensitized) was added to the perfusion fluid, a prompt contraction of the pupil occurred, and as the perfusion continued, small hemorrhages appeared throughout the

fundus. The contraction of the pupil was marked, usually from a dilated pupil from 10 to 12 mm. in diameter to a pupil from 2 to 4 mm. in diameter. This observation is in direct accord with those of Dale and Schultz, who observed the contraction of sensitized smooth muscle in the presence of specific antigen, and is another observation in support of the cellular theory of anaphylaxis. The hemorrhages observed cannot be so easily explained. Petechial hemorrhages have been observed over the peritoneum of animals recovering from anaphylactic shock, and it may be that these are analogous. From their method of formation it appeared that they were caused by an alteration of the endothelium of the capillaries, allowing a diapedesis of red cells.

In this experiment, the antigens used were horse serum and cow's uveal emulsion. Before we proceeded with the second phase of the perfusion work, a number of perfusions were done to control this observation. These controls are illustrated in Table 5. One of these controls requires a little explanation. Normal dogs perfused with horse serum showed a slight contraction of the pupil, never over 3 mm. Schultz[15] has shown that fresh horse serum possesses the power to produce a slight contraction of smooth muscle, and this is evidently what occurred with us. This contraction of the pupil in normal dogs, perfused with horse serum, was never over 3 mm., while the contraction in the sensitized dogs was from 8 to 10 mm. Normal dogs perfused with uveal tissue showed no contraction of the pupil.

It may be concluded, therefore, that the eyes may be sensitized as a part of general sensitization, and that anaphylactic phenomena may be elicited in the eyes by means of antigen carried by the blood stream. These anaphylactic phenomena consist in a marked contrac-

15. Schultz, W. H.: Physiological Studies in Anaphylaxis, I, The Reaction of Smooth Muscle of the Guinea-Pig Sensitized with Horse Serum, Jour. Pharmacol. and Exper. Therap., 1910, **1**, 549.

tion of the pupil, and in small extravasations of blood throughout the fundus.

With the artificial condition under which we were working, it is manifestly impossible to expect inflammatory phenomena of any kind, but the establishment of the fact that ocular anaphylaxis, no matter how manifested, may be demonstrated by a general sensitization and vascular intoxication gives us the second important point in the establishment of a scientific basis for the anaphylactic theory. Moreover, this observation affords us a means of studying the anaphylactic properties of uveal tissue, in vivo, by direct observation of the eye.

TABLE 5.—CONTROLS

Sensitization	Perfusion Fluid*	Result	
		Contraction of the Pupil	Hemorrhages in Fundi
None	D. B.	None	None
None	D. B. + uveal emulsion	None	None
None	D. B. + horse serum	Never over 3 mm.	None
Uveal emulsion	D. B.	None	None

* In this and the following tables, D. B. = defibrinated blood of normal dogs.

III. THE ANTIGENIC PROPERTIES OF UVEAL TISSUE IN THE PRODUCTION OF OCULAR ANAPHYLAXIS; PERFUSION OF THE EYE

In the section on complement fixation, it was shown that uveal tissue possessed the peculiar antigenic properties necesary for the anaphylactic theory of sympathetic ophthalmia to be possible. The next question is whether or not the same peculiar properties can be utilized in the actual production of ocular anaphylactic phenomena through such a mechanism as must be used in the production of an anaphylactic sympathetic ophthalmia, namely, general sensitization and intoxication through antigen carried to the eye by a vascular apparatus.

It has already been shown that an anaphylactic reaction can be obtained through the perfusion of the eye with specific antigen. This reaction has been used to determine the antigenic properties of uveal tissue. We first sought to determine whether or not whole uveal tissue possessed the antigenic properties necessary to make an anaphylactic uveitis a possibility. This point has already been established and reported.[16] We next sought to determine the constituent of uveal tissue responsible for its peculiar antigenic properties. Earlier work[16] showed that there was much reason to believe, with Elschnig, that the pigment was the responsible factor. A pigment solution has finally been prepared which was suitable for use in perfusion. Similar experiments to those in which uveal emulsion was used as an antigen have been performed to determine if the pigment was the responsible factor. The reactions given by the pigment were in every way similar to those given by whole uvea, except as regards species specificity. Moreover, dogs sensitized to homologous uvea reacted to perfusion with pigment, establishing more conclusively the fact that the pigment is responsible for the peculiar antigenic properties shown by uveal tissue.

The results of this work, both with uveal tissue and uveal pigment, are shown in Tables 6, 7, 8 and 9. The work is presented in the following form: 1. The ability of heterologous uveal emulsion and uveal pigment to act as antigen. 2. The ability of homologous uveal emulsion and pigment to act as antigen. 3. The properties as regards organ specificity. 4. The properties as regards species specificity.

Each individual observation was confirmed by at least two perfusions, and in those experiments in which for any reason (clots, thrombosis) the nature of the reaction was not clear after two perfusions had been performed, subsequent perfusions were done until the

16. Woods, A. C.: Ocular Anaphylaxis, II, A Contribution to the Anaphylactic Theory of Sympathetic Ophthalmia, Arch. Ophth., 1917, **46**, 8.

presence or absence of the anaphylactic phenomena was established beyond question.

Table 6 represents the results obtained by sensitization and perfusion with heterologous (cow's) uveal emulsion and uveal pigment. The anaphylactic reac-

TABLE 6.—HETEROLOGOUS SENSITIZATION (COW'S UVEA AND PIGMENT)

Sensitization	Perfusion Fluid	Result	
		Contraction of the Pupil	Hemorrhages in Fundi
Cow's uveal emulsion	D. B. + cow's uveal emulsion	Marked	Marked
Cow's uveal pigment	D. B. + cow's uveal pigment	Marked	Marked

tion was observed in all of these perfusions, establishing the power of both uveal emulsion and pigment to act as antigens.

Table 7 illustrates the fundamental fact necessary in the anaphylactic theory — the power of homologous uveal tissue to produce an ocular anaphylactic reaction

TABLE 7.—HOMOLOGOUS SENSITIZATION (DOG'S UVEA AND PIGMENT)

Sensitization	Perfusion Fluid	Result	
		Contraction of the Pupil	Hemorrhages in Fundi
Dog's uveal emulsion	D. B. + dog's uveal emulsion	Marked	Marked
Dog's uveal emulsion	D. B. + dog's pigment	Marked	Marked
Dog's uveal pigment	D. B. + dog's pigment	Marked	Marked

in a sensitized eye when carried there through vascular channels. It shows also that it is the pigment which is responsible for this remarkable antigenic property. Dogs sensitized to homologous uveal emulsion give an anaphylactic reaction when perfused with normal defibrinated blood to which pigment is added, and dogs

sensitized to pigment give the reaction when perfused with the pigment-containing blood.

Table 8 illustrates the organ specific property of uveal emulsion and uveal pigment. As shown before by the complement fixation reaction, uveal tissue and pigment are organ specific. The sensitization resulting from the introduction of uveal tissue into an animal is specific for uveal tissue, without regard to the species from which the tissue is taken. Dogs sensitized to cow's uveal emulsion react to perfusion with dog's uveal emulsion, and vice versa. Similarly,

TABLE 8.—ORGAN SPECIFICITY

Sensitization	Perfusion Fluid	Result	
		Contraction of the Pupil	Hemorrhages in Fundi
Cow's uveal emulsion	D. B. + dog's uveal emulsion	Marked	Marked
Cow's uveal pigment	D. B. + dog's pigment	Marked	Marked
Dog's uveal emulsion	D. B. + cow's uveal emulsion	Marked	Marked
Dog's uveal pigment	D. B. + cow's pigment	Marked	Marked

dogs sensitized to cow's pigment react to perfusion with dog's pigment, and vice versa. There evidently results from sensitization with uveal tissue a strong chemical affinity for similar tissue — organ specificity.

Table 9 shows the species specificity reaction of both uveal emulsion and pigment. The same properties are shown here as were shown by complement fixation; the uveal emulsion is species specific, while the pigment is not species specific. Dogs sensitized to cow's uveal emulsion give an anaphylactic reaction when perfused with other cow protein, for here the sensitization is not alone with the pigment, but also with the other species specific protein contained in the whole uveal emulsion. On the other hand, dogs sensitized to the pigment alone show no reaction when perfused with other cow protein.

The last perfusion illustrated in this table is largely a control perfusion. Dogs sensitized to uveal emulsion give no reaction when perfused with other dog protein. Perfusion with the other elements of the uveal tract — blood, smooth muscle and connective tissue — evokes no anaphylactic reaction.

To sum up the work thus far, both the complement fixation reactions of immune serums and the perfusion reactions have shown that uveal tissue possesses the power to act as antigen in animals of the same species, and that the pigment is the constituent of the uvea responsible for this property. In its antigenic action,

TABLE 9.—SPECIES SPECIFICITY

Sensitization	Perfusion Fluid	Result	
		Contraction of the Pupil	Hemorrhages in Fundi
Cow's uveal emulsion	D. B. + cow's serum, liver and kidney extracts	Marked	Marked
Cow's uveal pigment	D. B. + cow's serum, liver and kidney extracts	None	None
Dog's uveal emulsion	D. B. + dog's liver and kidney extracts	None	None

uveal pigment is organ specific and not species specific. The sensitization resulting from the absorption of uveal pigment is specific. Other body protein can produce no anaphylactic reactions in animals so sensitized. Pigment alone may produce intoxication.

In its immunologic reactions, uveal pigment stands apart, in a biologic isolation, from other protein of the body. It is of especial interest that a somewhat similar isolation in its chemical structure has also been shown.[17]

IV. OCULAR SENSITIZATION FROM ANTIGEN ABSORBED FROM THE OTHER EYE

The next point to be demonstrated in the establishment of a scientific basis for the anaphylactic theory is

17. Samuely, F.: Tierische Pigment und Faserstoffe, in Abderhalden, E.: Handbuch der biochemischen Arbeitsmethoden, Berlin, 1916, **2**, 762.

the fact that uveal tissue, absorbed in one eye, can create a hypersensitiveness to uveal tissue in the second eye. To demonstrate this point we have sensitized dogs by the injection of uveal tissue, both heterologous and homologous, into the vitreous of one eye. After a suitable period had elapsed for sensitization to occur, the injected eyes were enucleated, in order to remove any factor which could give a possible intoxication. One week after this, the remaining eye was perfused with the specific antigen. In every case, the perfused eye gave an anaphylactic reaction, indicating that ocular sensitization had taken place as a result of the absorption of antigen from the fellow eye. Table 10 illustrates this experiment.

V. EXPERIMENTAL SYMPATHETIC OPHTHALMIA BY ANAPHYLACTIC MEANS

The last step in the problem as outlined is the experimental production of sympathetic ophthalmia by anaphylactic means. This must be done with the use of homologous uveal tissue as antigen. Sensitization must be from absorption of uveal tissue from the exciting eye, and intoxication must be from antigen carried to the eye by a vascular apparatus. This phase of the work is still in course of progress, and a final report cannot as yet be made.

Sensitization of the eye by means of homologous uveal tissue absorbed from the fellow eye has already been demonstrated (Table 10). In the course of this experiment, the following phenomenon was twice noted: From three weeks to one month after the intraocular injection of homologous uveal tissue, the uninjected eye showed photophobia, ciliary injection and a sluggish pupil. The first eye was enucleated at once, with the prompt clearing up of the inflammation in the surviving eye, which was subsequently perfused with positive results.

Following this observation, we have injected a number of dogs in the vitreous of one eye with homologous uveal pigment. The eyes so injected all showed a

more or less severe iridocyclitis. From fifteen days to three weeks after the intra-ocular injection, the second (uninjected) eye of these dogs began to show a ciliary injection, a lusterless pupil, somewhat sluggish in its reaction, and a marked photophobia. This condition of sympathetic ciliary irritation continued steadily with little change.

After the signs of ciliary irritation had appeared in the second eye, two of these dogs were given intraperitoneal injections of pigment—an intoxicating dose. These dogs have not done well after receiving the

TABLE 10.—OCULAR SENSITIZATION

Sensitization	Enucleation	Perfusion Fluid	Result	
			Contraction of Pupil	Hemorrhages in Fundi
Vitreous injection left eye; cow's uveal emulsion	Left eye	D. B. + cow's uveal emulsion	Marked	Marked
Vitreous injection left eye of dog's uveal emulsion	Left eye	D. B. + dog's uveal emulsion	Marked	Marked

intoxicating dose, but we have as yet made no studies on such dogs for the detection of general anaphylactic symptoms. One dog so injected died ten days later from pneumonia, the second dog was dull and listless, and appeared ill for two weeks, with a gradual recovery to normal. The ocular reactions in these two dogs became much more intense after the intoxicating injection had been given. The dog which subsequently died showed a particularly violent ocular reaction, appearing clinically as an intense plastic iridocyclitis which progressed up to destruction of the eyes. This reaction appeared in both eyes, the injected or the exciting eye, and the uninjected or the sympathizing eye. Although it is impossible to say that the intercurrent pneumonia may not have had a bearing on the ocular

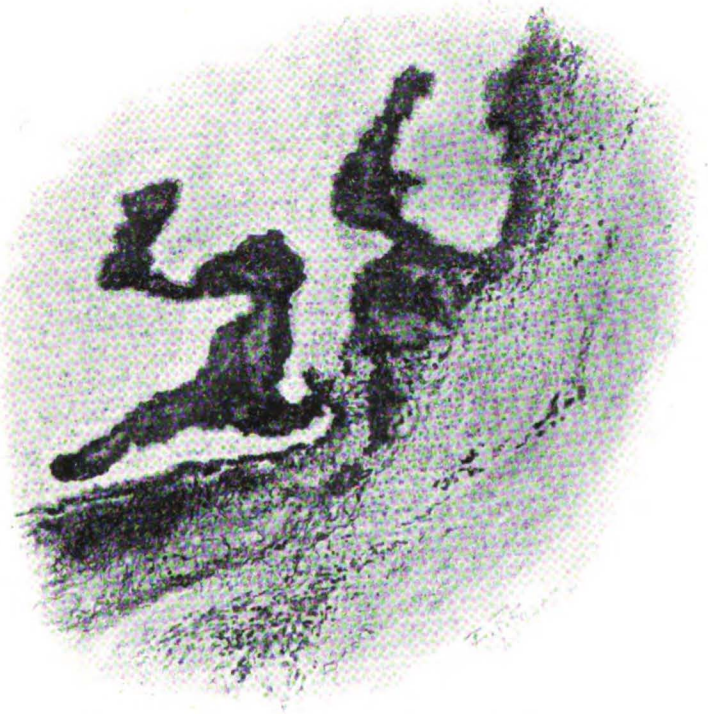

Fig. 2.—Ciliary region of eye of Dog 17-12, showing exudate around ciliary processes and round cell infiltration of choroid.

condition, this appeared to be a distinctly anaphylactic reaction, the ocular flare up beginning twenty-four hours after the intoxicating injection had been given, and progressing steadily. The histologic picture, moreover, was not that of an acute bacterial injection.

The clinical history and pathologic findings in this dog are as follows:

Dog 17-12. Normal dog.

January 17: Eyes were negative to external and ophthalmoscopic examination. Anterior chamber of the right eye was tapped and the aqueous allowed to flow out. At the same time 1 c.c. of dog's uveal pigment was injected into the vitreous.

January 22: Right eye showed a rather strong iridocyclitis, with a cloudy cornea. Tension was elevated. Left eye was entirely clear.

January 30: Right eye showed the same severe cyclitis. Left eye showed a fine pericorneal injection, a slight conjunctivitis, and a faint dimming of the upper edge of the cornea. Ophthalmoscopic examination was negative.

February 2: Right eye showed the same cyclitis, not so severe as formerly. Left eye showed a moderate pericorneal injection, and a faint dimming of the upper part of the cornea.

February 6: The eyes showed no further change.

Intraperitoneal injection of 5 c.c. of dog's uveal pigment.

February 7: The process in both eyes was of the same character, but the pericorneal injection was much more intense.

February 9: The right eye showed an opaque cornea, a violent ciliary injection, and a distinct diminution in the intra-ocular tension. The left eye showed a marked pericorneal injection, and clouding of the upper edges of the cornea. The iris was regular, but reacted sluggishly.

The dog showed a marked photophobia, lying with both eyes tight shut.

February 12: Right eye showed the same violent picture, and was very soft. *Left eye:* There was a moderate conjunctivitis, marked pericorneal injection, and a generally hazy cornea. The iris reacted slightly, and there was a slight exudate over the lower half of the iris. Ophthalmoscopic examination revealed many dark masses throughout the vitreous. The tension was distinctly below normal.

The photophobia in both eyes was marked.

February 15: Right eye showed violent ciliary injection, and a cloudy cornea. The iris could just be seen, and was covered with exudate. The fundus was not seen. The eye was mushy.

Left eye was soft and mushy. Conjunctivitis was marked. Ciliary injection was very intense. Cornea was cloudy, and

there was a small ulcer over the pupillary area. The iris was covered with exudate, and was irregular and immobile. The vitreous was filled with opacities.

The dog was listless, and lay with the eyes tightly closed. There was a slight nasal exudate today, and the dog appeared very ill.

February 16: The dog was dead in the cage. Necropsy revealed old petechial hemorrhages over the peritoneum, and a pneumonia of the upper lobes of both lungs in the stage of gray hepatization. The necropsy otherwise was negative.

The eyes were preserved in 10 per cent. dilution of liquor formaldehydi, washed, frozen and cut.

Gross appearance.—Right Eye: The cornea was thickened, and there was an exudate in the anterior chamber. The iris was adherent to the anterior surface of the lens. Both the iris and the ciliary body appeared larger than normal. The posterior chamber was practically filled with a large exudate.

Left Eye: The cornea was thickened except over the pupillary area, where on the outer surface there was a loss of tissue. The cornea bent in at this point and was here adherent to the iris, practically obliterating the anterior chamber. The iris was adherent to the anterior surface of the lens. Both the iris and the ciliary body appeared swollen, and there was a light fluffy exudate in the ciliary region, extending up to the lens, and filling the anterior half of the posterior chamber.

Microscopic appearance.—Right Eye: The picture was obscured by the large amount of hemorrhage that had taken place. The cornea was thickened. The iris was swollen and adherent to the anterior capsule of the lens. The vitreous was filled with a mass of fibrin and red blood cells, in which the ciliary processes were embedded. There was a very small amount of pigment scattered throughout this mass. Throughout the stroma and pigment of the choroid there was a large amount of hemorrhage, and in the stroma there was an infiltration of small round cells, with a few leukocytes and epithelioid cells. The sclera was slightly thicker than normal, and showed occasional small extravasations of red blood cells.

Left Eye: There was a small eroded area on the outer surface of the cornea, over the pupillary area. In the collapsed anterior chamber there was a serous exudate in which were enmeshed many small round cells and a few leukocytes. The stroma of the ciliary body and choroid was swollen, and throughout the choroidal stroma, especially in the ciliary region, there was a dense infiltration of small round cells, with occasional epithelioid cells and leukocytes. There were also occasional small extravasations of red blood cells. The ciliary processes were embedded in a serofibrinous exudate and around them were small extravasations of red cells, with round cells and occasional leukocytes.

There was a slight migration of pigment throughout the sclera, but neither the sclera nor the retina appeared to be involved to any extent. Figure 2 illustrates the appearance of the ciliary region.

COMMENT

Although the method of sensitization assumed by the anaphylactic theory — absorption of pigment from the exciting eye — is simple, and can readily be shown, the mechanism whereby intoxication results is not so clear. It can be explained, however, by the analogy of serum sickness. Serum sickness is universally believed to be an anaphylactic phenomenon, and may occur after a single injection of foreign serum. Von Pirquet explains this phenomenon thus: In the incubation period intervening between the introduction of the foreign serum and the breaking out of serum sickness, antibodies are developed by the organism. After these antibodies have been formed, if any foreign serum (antigen) still remains in the body cells or fluids, the antibodies react with this antigen, and this interaction causes the symptoms and lesions of serum sickness. It is only a short step to apply this reasoning to sympathetic ophthalmia. After sensitization occurs through the absorption of pigment from the exciting eye, antibodies are developed by the organism, and by the second eye as a part of this general process. The interaction of these antibodies and any pigment still present in the body cells or fluids causes a disturbance, manifested in the eye as a sympathetic ophthalmia. This supposition is strengthened by the fact that we have every reason to believe that anaphylaxis is a cellular phenomenon. It is of interest, further, in this connection, that the dangerous pigment is present in the eye and nowhere else in the body. After sensitization has occurred by absorption of pigment from the exciting eye, it is readily conceivable that the antibodies in the cells of the second eye might react with the native uveal pigment, causing a localized anaphylactic phenomenon — sympathetic ophthalmia. However, the

actual mechanism whereby intoxication occurs in this or in any anaphylactic condition must be hypothetic until we know more of the true nature of the anaphylactic reaction.

The ability of dog's uveal tissue to produce sensitization and intoxication in the dog has been shown. There is, however, one point further in this connection which for the present must needs be assumed. This is the ability of uveal tissue to create sensitization and intoxication in the dog from which the uvea is taken — the question of autosensitization. An anaphylactic reaction of extreme delicacy will probably be needed to show this. It is manifestly impossible to use the ocular anaphylactic reaction for this, for the eyes must be removed to obtain uveal tissue with which to work. We have attempted to show such autosensitization by removing the eye of a dog, excising and macerating the uvea, and injecting this to sensitize. Two weeks later the second eye was removed, the uvea excised and macerated, and this again injected. Under ether anesthesia this dog was observed for a fall in blood pressure, change in the coagulability of the blood, and drop in body temperature — the general signs of crude anaphylactic shock. No such signs were observed, and we have little hope of demonstrating autosensitization by this method. A more delicate method must be devised. For the present we must content ourselves with the demonstration that dog's uvea can produce ocular anaphylactic phenomena in the dog.

Fuchs[18] has asserted that the anatomic findings in sympathetic ophthalmia are characteristic. The picture is that of a round cell infiltration throughout the uvea, with occasional clumps of epithelioid cells, in which are larger cells, resembling giant cells. This observation, although not entirely confirmed by a few workers, is generally conceded to be true. There is a striking similarity between the lesions described by Fuchs as

18. Fuchs, E.: Ueber Ophthalmia sympathica, Arch. f. Ophth., 1909, **70**, 465.

characteristic, and those shown by the dog whose history we have reported. It seems more than probable that the ocular condition in the uninjected or sympathizing eye of this animal was a true sympathetic ophthalmia. To prove conclusively, however, that an anaphylactic sympathetic ophthalmia can be produced, a greater number and more clear cut demonstrations than can now be presented are needed. Experiments in progress at present indicate that this further demonstration is a probability.

In connection with the contraction of the pupil observed in the perfusion experiments, it is of more than historical interest that oscillation of the pupil is described by Nettleship to be one of the earliest signs of sympathetic ophthalmia. The contraction of the pupil noted here, and the well known fact that sensitized smooth muscle contracts in the presence of specific antigen, is in direct accord with this observation.

The final proof of the anaphylactic nature of sympathetic ophthalmia, and indeed of the true nature of any disease, must be in man. That this will be demonstrated by serologic studies is problematic. The researches of Kummel and Wissman indicate that it is probable; but a failure by this means will not necessarily invalidate the theory, for Gay and Southard[19] have shown that, in animals sensitized to horse serum, specific antibodies cannot be shown by complement fixation.

In this work we have endeavored to establish a scientific basis for the anaphylactic theory of sympathetic ophthalmia and to present what experimental proof we have of the anaphylactic nature of the disease. The theory assumes that uveal tissue must possess peculiar antigenic properties, and it has been shown here, and by Elschnig, that uveal tissue possesses these properties, and that the constituent of the uvea responsible is the pigment. It has also been

19. Gay and Southard: Further Studies in Anaphylaxis, Jour. Med. Research, 1908, **18**, 407.

shown here that local ocular anaphylactic phenomena can be elicited in sensitized animals by antigen carried to the eye by the blood stream, and that uveal tissue can exercise its antigenic properties through this mechanism. Uveal tissue absorbed from one eye can produce a hypersensitiveness in the second eye, and ocular anaphylactic phenomena can be produced there by uveal tissue carried by the blood stream. This is the scientific basis which can at present be advanced to support the theory.

SUMMARY

The anaphylactic theory assumes sympathetic ophthalmia to be an anaphylactic uveitis, caused by sensitization and intoxication of the sympathizing eye from disintegrating uveal tissue in the exciting eye. To fulfil the assumption of this theory, uveal tissue, or a constituent, must (1) be able to act as antigen in the homologous animal, (2) be organ specific, and (3) lack species specificity. Also (4) ocular anaphylaxis must be demonstrated in a generally sensitized animal by intoxication from antigen carried by the blood stream, and (5) uveal tissue absorbed from one eye must be capable of sensitizing the second eye.

Experimentally, these points have been demonstrated as follows: 1. By the study of the complement fixation reactions of the serums of animals immunized to foreign and homologous uvea and uveal pigment, it has been shown that uveal tissue may act as antigen in the same species animal and that the pigment is the constituent responsible. In its immunologic reactions, pigment is organ specific and not species specific. 2. The eyes of a generally sensitized animal, when perfused with normal defibrinated blood containing specific antigen, show local ocular anaphylactic phenomena. 3. Uveal tissue exercises its antigenic properties through this pathway. The pigment is the constituent responsible, and its antigenic properties are again demonstrated by this means. 4. Uveal pigment absorbed from

one eye produces a hypersensitiveness of the second eye, and a subsequent perfusion produces ocular anaphylactic phenomena.

These facts constitute the present scientific basis for the anaphylactic theory.

5. Experimentally, by anaphylactic means with homologous uveal tissue as antigen, sympathetic irritation, and in one case sympathetic ophthalmia, has been produced in dogs.

ABSTRACT OF DISCUSSION

DR. CHARLES E. DE SCHWEINITZ, Philadelphia: For the purpose of introducing the discussion which shall follow—because I do not for a moment assume to do aught else—the ciliary nerve theory and the theory of bacterial metastasis may be for the present largely disregarded, and reference may be restricted to the cytotoxic and anaphylactic theories. The credit of advancing the first-named of these hypotheses, namely, the cytotoxic theory, I would agree with Harold Gifford, should be given to Brown Pusey, who a year before Golowin's work was published suggested as an explanation of sympathetic ophthalmitis that the cells of the injured eye, probably the cells of the ciliary processes and the iris, give rise to a cytotoxin, which, having selective affinity for corresponding cells in the other eye, there sets up that inflammation which is known as sympathetic ophthalmia. For this hypothesis experimental proof is lacking, and thus far its value, as Woods points out, would seem to be that it "opened up new channels for thought and investigation."

Acting on the suggestion of Bail and Heim, given alike to himself and Kümmell, Elschnig developed the theory of anaphylactic uveitis in 1911, which not unnaturally appears to have developed in the course of his investigations of enterogenous autointoxication as an etiologic factor in certain types of nonspecific and nontraumatic iridocyclitis. According to him, in the development of sympathetic ophthalmia two factors are necessary, anaphylaxis of the uvea on account of tissue disintegration, the chief rôle being taken by the pigment, and an anomalous condition of the organism which is evident either as an organic lesion, like nephritis or diabetes, or which is called into action by autointoxication in the widest acceptation of that word.

It is historically interesting to point out that the late Dr. Alvin A. Hubbell of Buffalo, in 1900, also advocated a relationship of autoinfection and autointoxication to sympathetic ophthalmia. Hubbell says: "I believe with Professor Panas of Paris that sympathetic ophthalmia furnishes another illus-

tration of autoinfection or autointoxication, not in the sense that it is a migratory disease in which germs are transported from the injured to the sound eye, but in the sense that the uveal tract of the sound eye, by reason of a lowering of its vital tone, through the extreme reflex and vasomotor disturbances kept up by a foreign body in or injury to the other eye, becomes truly poisoned or infected by the materials in the general circulation to the extent of inducing a most obstinate and uncontrollable inflammation."

Elschnig's theory, thus briefly summarized, should be supplemented with his own statement, and for this purpose Harold Gifford's condensed translation may be utilized: "I assume," says Elschnig, "that the cause of sympathetic ophthalmia is not infectious, that is, it has not a bacterial origin, but, on the contrary, depends on some anomaly affecting the whole organism which can easily be detected, as a nephritis, diabetes, or something of that sort, or else is produced by autointoxication in the widest sense of the word; or by some anomaly of the constitution. . . . A trauma severely injures the first eye, at the same time, perhaps, by virtue of the anomaly of the body, develops from the trauma as such, a nonbacterial inflammation. The disintegration of the tissues which accompanies the inflammation leads to absorption of uveal tissue, and thus to a hypersensitiveness of that part of the uvea not immediately affected by the injury or the primary inflammation of the first eye, as well as the uvea of the second eye. As the result of this local hypersensitiveness of the uvea, the existing somatic anomaly finds a point of attack, and leads to that inflammation of the uvea of both eyes which is known as sympathetic inflammation."

At first there was little experimental proof of this theory, but now research work has accumulated and is especially noteworthy in Alan Woods' investigations. Whether Fuchs' objections to the results of experimental work, to wit: that dogs are unsuitable material because they never clinically are known to develop the lesions which we consider characteristic of sympathetic ophthalmia, or indeed, any other animal, so far as I know, should have weight, remains to be seen. Perhaps these animals never have the necessary somatic anomaly.

Naturally, a number of observers have advanced arguments in opposition to the theory of Elschnig, especially E. v. Hippel, Reis, Fuchs, Harold Gifford, F. Schiek, and others. While Alan Woods has demonstrated that uveal tissue may act as antigen in the same species of animal, and that the pigment is the constituent responsible, that the eyes of a generally sensitized animal when perfused with normal defibrinated blood containing specific antigen show local anaphylactic phenomena, that uveal pigment absorbed from one eye produces hypersensitiveness of the second eye, and a subsequent

perfusion produces ocular anaphylactic phenomena, and that these facts are the basis for the anaphylactic theory, we must none the less consider whether the clinical features of sympathetic ophthalmia can be brought into accord with what Schiek calls the essence of anaphylaxis. Here are summaries of some objections to the anaphylactic theory:

1. Anaphylactic manifestations, in general terms, are of an active character, attaining quickly their maximum intensity, differing thus in large measure from the usual course of sympathetic ophthalmia, a point made by Gifford.

2. Anaphylactic uveitis and the undoubted value of preventive enucleation are not compatible.

3. With rare exceptions sympathetic ophthalmia arises only after penetrating wounds, chiefly through the so-called danger-zone, but if the anaphylactic theory is correct the incidence of sympathetic ophthalmia should be equally great in nonpenetrating injuries and diseases which disorganize uveal pigment.

4. Sympathetic ophthalmia is undoubtedly more frequent in children and in robust young persons than in those who give evidence of a somatic anomaly, for example, "an autointoxication in the widest sense of that term."

5. Gifford finds it difficult to understand how an eye in which all uveal tissue must be sensitized at the same time could develop anaphylaxis in the form of isolated areas which are found affected in sympathetic ophthalmia, and cannot reconcile the theory with the usual exemption of retinal pigment epithelium, except in severest cases.

From the clinical standpoint the anaphylactic theory may better explain the incubation period, although Gifford disputes this, and the occurrence of sympathetic ophthalmia with intraocular sarcoma. The cytotoxic theory, however, would equally well explain this. Dr. Woods' work is doing its best to satisfy the exacting conscience of the laboratory, but the final proof of the theory is not at hand; indeed, as Dr. Woods himself says, this must be in man.

Dr. J. W. Jervey, Greenville, S. C.: It is of course true that very few of us have either the time, training, talents or equipment at our disposal to prosecute the kind of research that Dr. Woods has placed before us. The vast majority of us have to accept the facts adduced by the few, and then all that we can do is either to accept or reject their logic as they correlate their facts with their theories. I am sorry that Dr. Woods has so curtly dismissed Rosenow's demonstrations of selective affinity, for to my mind his own work is at least in some degree corroborative of Rosenow's, though I readily confess the weakness of my grasp of the subject. But surely it is not a very far cry from "selective affinity" to "sensitized localization." A micro-organism is protein, foreign when introduced, but tolerated and multiplied in the tissues, and may, with some license, be regarded

as autogenous protein. Is it fair to say sympathetic ophthalmia has occurred following scleral rupture without a solution of conjunctival continuity? Who can say there was not an invisibly minute wound? And besides, bacterial presence intra-ocularly in the absence of a perforation is a matter of common observation. Witness hypopyon attending a small superficial ulcer; witness intra-ocular tuberculoma, and the protozoal infections.

Dr. Woods has elaborated a beautiful theory. I hope it will prove to be correct. He has not touched on the occurrence of sympathetic ophthalmia twenty or thirty or forty years after the initial injury. That is a long time for an acquired anaphylaxis (I use the term as distinguishing it from a congenital sensitization) to persist, though I must admit, no longer than the same forty years would be in developing the selective affinity of Rosenow. May it not well be that there is virtue in both theories?

Dr. Frederick H. Verhoeff, Boston: Dr. Woods' attempt to solve the question of sympathetic ophthalmia is certainly most praiseworthy and I hope he will continue his work. It does not seem to me, however, that thus far his experiments afford convincing proof of the anaphylactic theory. His perfusion experiments seem to show that a dog's retina may be sensitized by uveal emulsion or pigment so that retinal hemorrhages are produced when the eye is perfused with the same antigen. The pupillary contraction may have been due to direct action on the smooth muscle of the iris as assumed, but, on the other hand, may have been due to irritation of the nerve centers. Only one control experiment seems to have been made in which the eye was perfused with uveal emulsion without previous sensitization, and none made with uveal pigment alone. It also is not clear that failure to produce similar results with other antigens was not due simply to difference in dosage. If the anaphylactic theory is correct, it is difficult to see why the choroid did not react in the sensitized animals even without perfusion, since in the dog the choroid is loaded with pigment. Granting, however, the validity of the perfusion experiments, they simply show that it is possible to sensitize the retina, not the choroid, to uveal protein. Now in sympathetic uveitis it is particularly noteworthy that the retina is not involved in the process and that retinal hemorrhages probably never occur. Moreover, the pigment epithelium from which Dr. Woods evidently assumes the sensitizing pigment is chiefly derived, is not markedly affected, in fact, the changes it shows seem to be secondary to lesions in the stroma of the choroid. Dr. Woods claims that in one case actual sympathetic ophthalmia was produced in a dog. The protocol of the experiment in question, however, does not seem to warrant this. The ocular inflammation in the experiment can

be explained by the poor general condition of the dog which led to his death from pneumonia. Certainly ulcerative keratitis is not a manifestation of sympathetic ophthalmia. The description of the microscopic appearance of the eye is too meager for an accurate opinion to be formed as to whether or not the changes in the uvea were similar to those in sympathetic uveitis, but it seems to me they may well have been due to the corneal ulcer. Personally, the germ theory of sympathetic uveitis still seems to me to be the most probable one. The cases reported as following nonperforating injuries, sarcoma of the choroid, etc., may be explained either as instances of erroneous diagnosis, the condition being due to syphilis or some other infection, or, less likely, to the specific germs having entered the blood from some other part of the body.

Dr. Steuart B. Muncaster, Washington, D. C.: A lady in Washington, 22 years of age, had an injury to the eye at the age of 3, and during the last two years the vision in the eye was very little. Two months ago all of it disappeared in a week after she arrived in Washington. I trephined. The tension was about 60 in that eye. The field of vision in the other eye was beginning to fail. After operation she had a great deal of edema; even the lids were puffed out and the conjunctival membranes were down below the lids and in very bad condition. I thought the eye would have to be removed, but after treatment the tension of the eye is now about normal and the sympathetic trouble in the other eye has improved very much. I think the whole matter was that the edema was due to the nephritis which the patient had. The physician in attendance said the temperature had been 100 F., or a little over; now the temperature is about normal and remains that way. I made five incisions in the conjunctival membrane around the eyeball to reduce the edema. The blood pressure has been reduced from 205 to 165.

Dr. Mark J. Schoenberg, New York: The paper has interested me, since for a few years I have been doing experimental work along the line of general anaphylaxis in relation to the eye. During the Franco-Prussian war of 1870, and in the Civil War, a large percentage of cases of sympathetic ophthalmia occurred in injuries of the eye, while now, if we read over the literature, in the experience of the French and English ophthalmologists there has not been one single case in this war in the last two years. If anaphylaxis has something to do with sympathetic ophthalmia, we ought to get anaphylaxis now as well as thirty or forty or fifty years ago. On that point I should like to call Dr. Woods' attention to his experimental work. The interest of his work will be to draw conclusions on human sympathetic ophthalmia. I believe he will be much nearer the solution of the problem if he will try his work on the higher ani-

mals—monkeys. I made quite a number of experiments on rabbits regarding ocular anaphylaxis and when I repeated the experiments on dogs I obtained entirely different results, which goes to show that we cannot draw conclusions from the findings on one series of animals and apply them to another type of animals, and much less could we apply conclusions from experiments on dogs to human beings. I think it makes a great difference. Otherwise the work of Dr. Woods is most admirable, well conceived and well gone over, and he deserves our thanks.

DR. ALAN C. WOODS, Philadelphia: Dr. de Schweinitz' objection is first that anaphylactic manifestations are usually active in character. These are objections advanced by Fuchs and Muller. They have emphasized the difference between experimental and actual sympathetic ophthalmia. Fuchs and Muller's cases were produced by vitreous injection, with the production of obscure, painful, subacute and chronic conditions. There are some diseases which we now believe to be anaphylactic in character—serum sickness, asthma and certain skin diseases. In general, however, the criticism is true that anaphylactic phenomena are found much more frequently in the acute conditions, as in anaphylactic shock. Chronic conditions such as serum sickness, asthma and the skin diseases run a rather chronic course.

The question of enucleation has been advanced by Seyd. Elschnig and Kümmell have answered the criticism, and it does seem almost as good a case can be made out on the other side. If the sensitization in sympathetic ophthalmia is due to an intoxication from the uvea of the exciting eye, the removal of the exciting eye should lead to the amelioration of the symptoms. Whether that can be demonstrated experimentally, or whether it has been done, I cannot say. Eighty-six per cent. of cases of sympathetic ophthalmia are supposed to be from traumatism; in fact, some observers have claimed 100 per cent. In regard to the influence of absorption on the uvea, we have done nothing at all. We hope subsequently to take up that field. It is a very interesting point, in support of the anaphylactic theory, that the condition occurs more frequently in the young, because they are more subject to anaphylactic conditions than the old.

In relation to the question of selective affinity, I did not mean to dismiss that summarily, but I believe that it is not generally accepted, and numerous experiments in other laboratories have not confirmed Dr. Rosenow's work.

Dr. Verhoeff's contraction of the pupil may be due to ciliary irritation. These dogs when perfused for forty-five minutes, later showed an apparently living eye, winking, etc., and continued respiratory movements. If at this time normal blood is perfused, the pupil will redilate, and in some dogs a second injection of pigment will produce the reaction following the specific contraction due to the specific antigen.

OXYCEPHALUS

REPORT OF THREE CASES WITH OPERATION IN ONE

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That oxycephaly has existed for ages is undoubted; that the ocular changes are among the most prominent symptoms is common knowledge; but that a cranial decompression may relieve the intracranial pressure, preserve vision and enable the patient to be a self-supporting member of society is too little recognized. It is to review the symptoms of three cases and to stimulate interest in this subject on the part of ophthalmologists that this communication is presented.

MacKenzie[1] in 1854 seems to have been the first to attract attention to cranial deformity as a cause of blindness. Friedenwald,[2] Fletcher,[3] Enslin,[4] Patry[5] and Hanotte[6] have recorded their experiences in communications of great value. Within the past few months, Sharpe[7] has presented a paper clearly describing his cases and recording the results of four operations. His paper should be read for the reason that only one other worker has published the report of an operation, Eiselsberg of Vienna, although Cushing has

1. MacKenzie: Practical Treatise on Diseases of the Eye, 1854, p. 77.
2. Friedenwald: Cranial Deformity and Optic Nerve Atrophy, Am. Jour. Med. Sc., 1893, p. 529; On Optic Nerve Atrophy Associated with Cranial Deformity, Arch. Ophth., 1901, p. 405.
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performed several operations and will soon make his report. A similar proceeding has been suggested by several, but their cases were not so treated. I am able to find the records of only seven operations, including the one here reported for the first time.

Symptoms may pass unnoticed, but usually the headache is so severe that it attracts attention, whereupon it is found that the head is deformed and vision failing. The three symptoms of this disease are such as usually to bring these cases to the ophthalmologist primarily: (1) exophthalmos; (2) impairment of vision, and (3) cranial deformity.

1. The exophthalmos is probably the result of the shallow orbits, and is usually accompanied by divergent strabismus and nystagmus.

2. The poor vision results from secondary optic atrophy following increased cranial pressure.

3. The cranial deformity is mainly shown by the high forehead and shallow orbits. Roentgenograms show sulcine impressions and bulging anterior fontanel.

Many other malformations, including webbed fingers and cardiac murmurs, have been noted.

My experience is unique. Three members of one family were afflicted; the youngest one showed optic atrophy, the middle one optic neuritis, and the oldest was free from optic nerve lesion.

The father of these children died at the age of 38, after having been in an insane asylum for two years. Necropsy proved that he had general paresis. The Wassermann reaction of his spinal fluid was positive. From Figure 1 it is seen that his head was without marked change.

The mother died at the age of 30, probably from acute alcoholism. She was said to have had scars on her corneae, and Figure 2 shows that she had marked divergent strabismus with facial expression suggesting acromegaly.

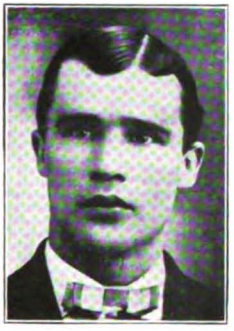

Fig. 1.—Father: Normal head; no divergence.

Fig. 2.—Mother; marked divergence; gross facial expression.

Fig. 3 (Case 1).—Patient at age of 3.

Fig. 4 (Case 1).—Full face view of patient, showing normal fingers.

Fig. 5 (Case 1).—Profile of patient.

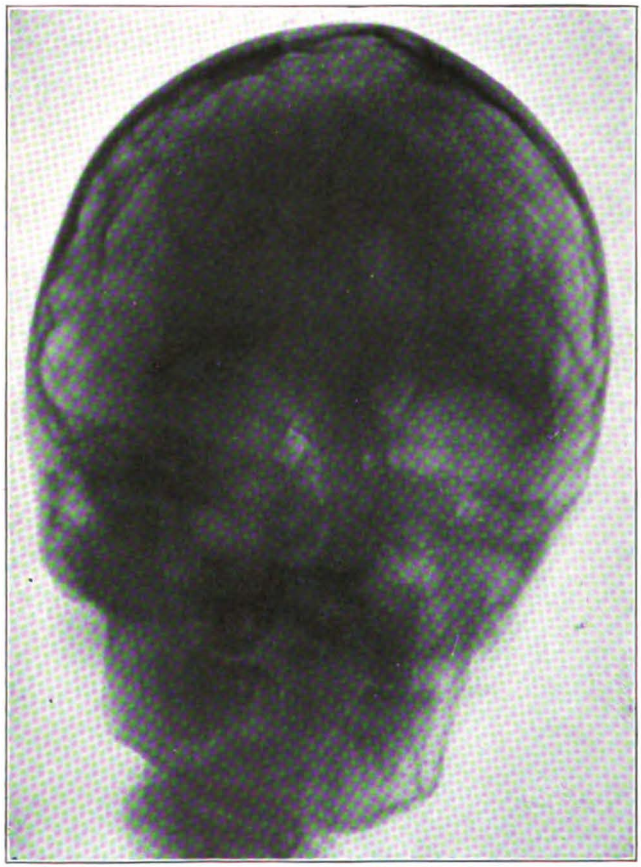

Fig. 6 (Case 1).—Roentgenogram of full face; well defined skull depressions.

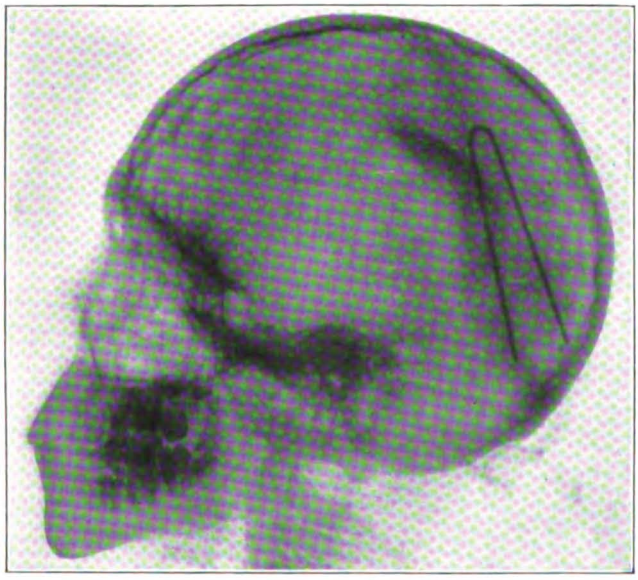

Fig. 7 (Case 1).—Roentgenogram of profile, showing depressions; deep sella turcica.

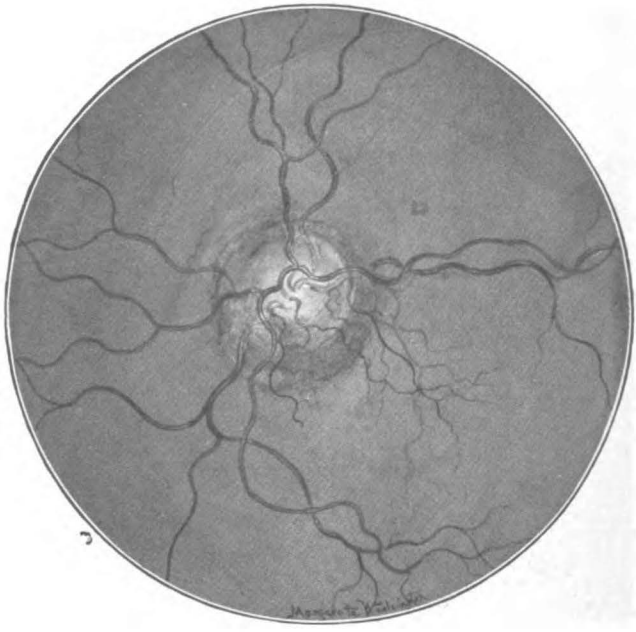

Fig. 8 (Case 1).—Practically normal fundus; slight indistinctness of the nasal margin of the nerve with slight increased vascularization.

Fig. 9 (Case 2).—Patient when 2 years old: prominent frontal region.

Fig. 10 (Case 2).—Full face view of patient: high forehead; tendency of left eye outward.

Fig. 11 (Case 2).—Profile of patient: bulging frontal region.

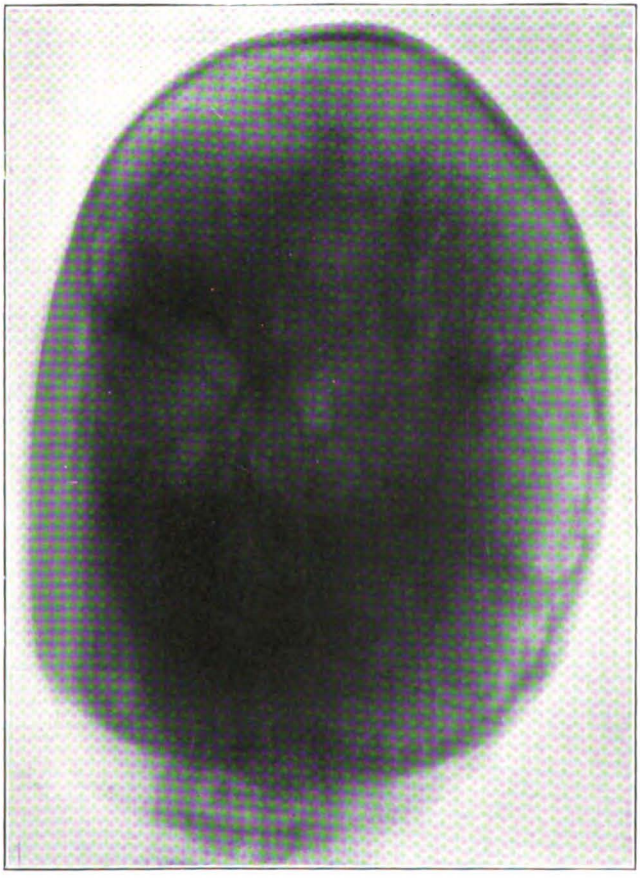

Fig. 12 (Case 2).—Roentgenogram of full face, revealing depressions on skull.

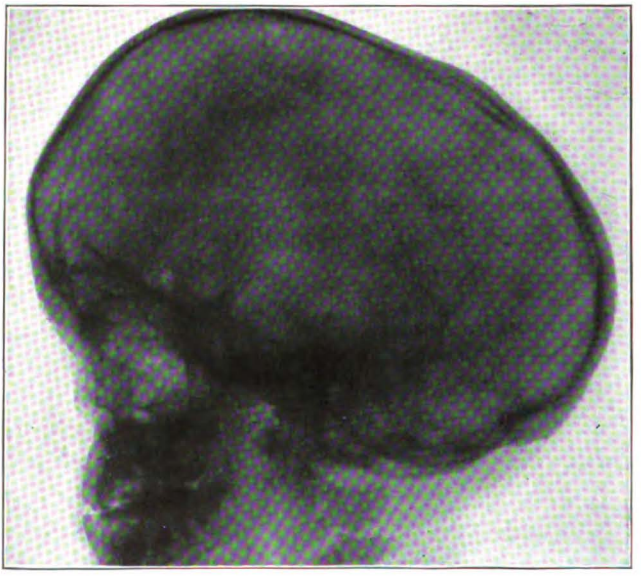

Fig. 13 (Case 2).—Roentgenogram of profile, revealing deep sella turcica.

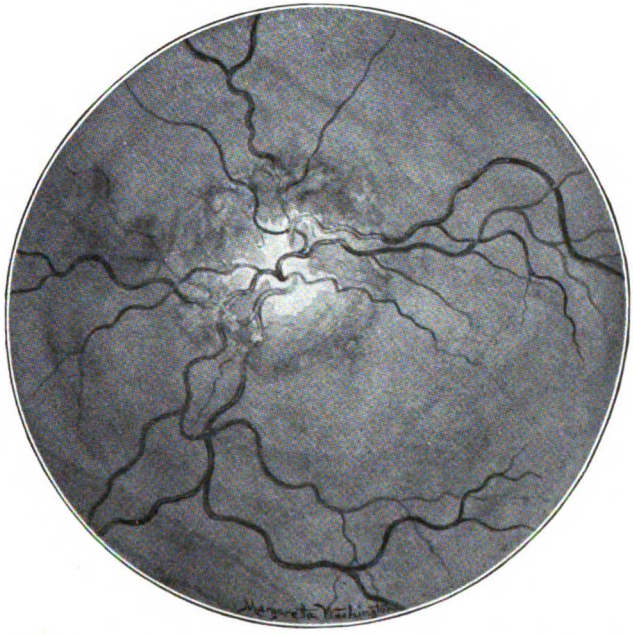

Fig. 14 (Case 2).—Fundus: blurred indistinct outline of the disk with partial obscuration of the inferior temporal vein; swelling = to 3 D.; papillitis.

Fig. 15 (Case 3).—Patient at age of about 10 months; normal shaped head.

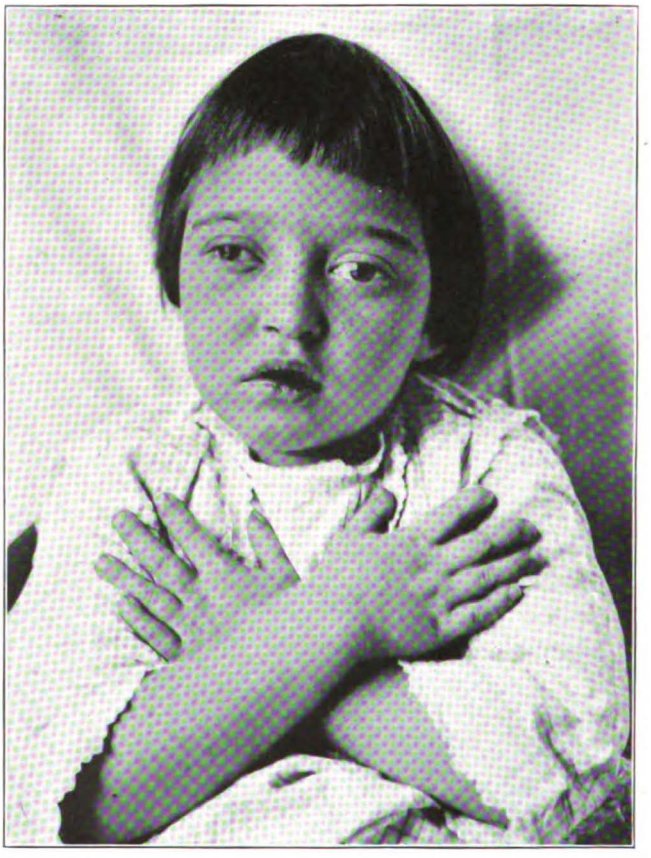

Fig. 16 (Case 3).—Full face view of patient: tower skull; extreme divergence; rapid nystagmus.

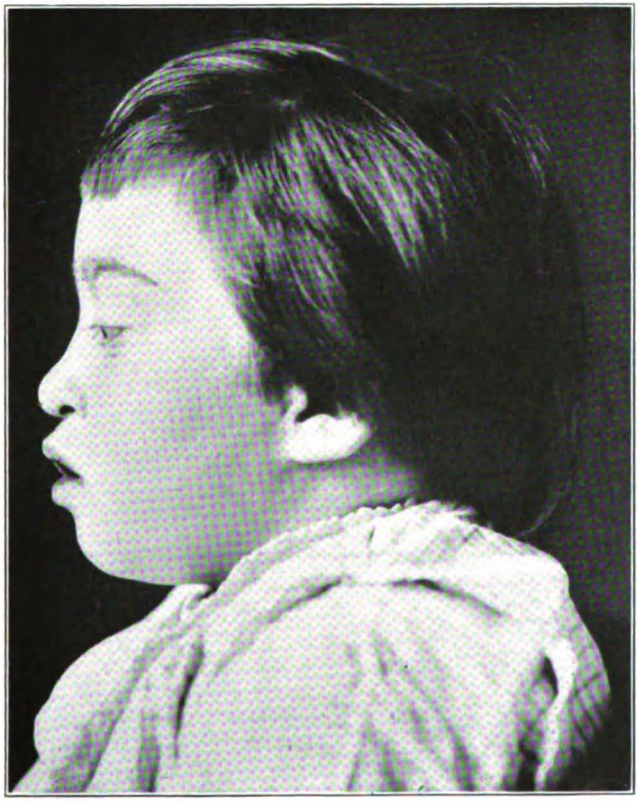

Fig. 17 (Case 3).—Profile of patient: adenoid expression.

Fig. 18 (Case 3).—Roentgenogram of full face: skull depressions, apex in anterior fontanel region.

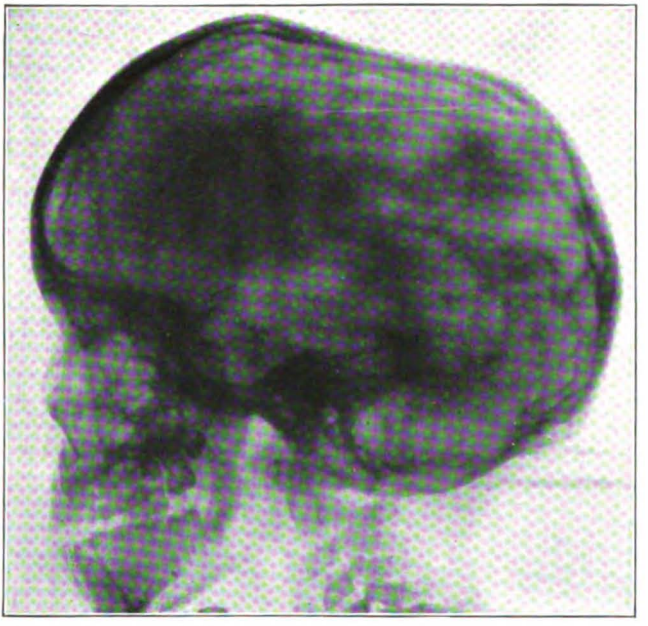

Fig. 19 (Case 3).—Roentgenogram of profile: very large sella; distinct ridge forward.

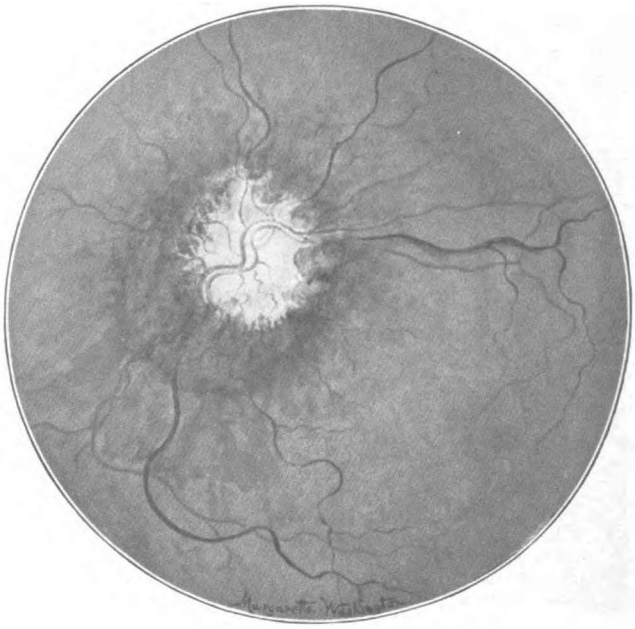

Fig. 20 (Case 3).—Fundus: disk decidedly pale, with marked obscuration and irregularity of its border, with considerable surrounding pigmentation; both arteries and veins more prominent than normal; post-papillitic atrophy.

The paternal grandmother died at the age of 49, from cancer of the uterus, and the paternal grandfather at the age of 78. One uncle and four aunts are living and well. The paternal great-grandparents died of old age, but it is especially noted that their heads tended to be small and the eyes closely set.

The maternal history was unobtainable.

The patients now recorded were all brought on the same day, Oct. 17, 1916.

CASE 1.—A well nourished, intelligent girl, aged 11 years, with a clear skin excepting a few freckles, had normal mucous membranes; the tongue was clean, and protruded mesially with a slight tremor. The teeth, of which there were twenty-five, were well kept, eleven in the upper and fourteen in the lower jaw. There was a high palatal arch; the tonsils were not enlarged; the patient had chronic pharyngitis. The shoulders were high, the right more so than the left; the chest was comparatively short, and the patient was round shouldered. The supraclavicular and infraclavicular fossae were not depressed. The chest was flattened anteroposteriorly over the upper part. The circumference at the sixth rib was 59 cm., on forced expiration 57 cm., and on forced inspiration 62 cm. Right side 31 cm., left side 30 cm., anteroposterior diameter 17 cm., lateral diameter 22 cm. There was free and equal movement on both sides. There was slight scoliosis in the upper dorsal region.

The heart was normal in position. The apex beat was in the fifth interspace within the nipple line. Palpation revealed nothing abnormal. There were no abnormal pulsations or thrill; pulmonary resonance over the right side was normal; over the left side of the chest below the fourth rib, resonance was slightly impaired. The respiratory murmur over both sides was normal; there were no rub and no râles. The spleen and the liver were not palpable. The abdomen was 56 cm. in circumference, and not prominent. There were no herniae. Examination of the nervous and osseous systems revealed nothing abnormal excepting that the left elbow joint resisted full extension, and when forcibly extended deflected outward with the inner condyle especially prominent.

The head was somewhat square shaped, with a definite ridge over the bregma; the forehead was low. The patient had abundant brown hair. There were dilated superficial veins over the left frontal and temporal region. The circumference was 48.5 cm. From the root of the nose to the occipital protuberance, the distance was 30 cm. Biaural, 33 cm. The transverse diameter was 16.5 cm.

Right eye: vision $^{20}/_{30}$ with $+1.25 = +.75$ ax $90 = {}^{20}/_{20}$. The pupil, about 4.5 mm., was irregularly quadrate, presenting no adhesions; it was active to light and accommodation; the media were clear; the disk, with a suggestion of yellowish tinge about its margin and a small shallow central excavation, was distinctly outlined. Several small vessels passed from the temporal side of the nerve, at which part pigmentation was greatest. There were no pathologic changes, exudate or hemorrhage.

Left eye: vision $^{20}/_{30} = +1.25 = +1.00$ ax $105 = {}^{20}/_{20}$-1? The same irregularly quadrate 4.5 mm. pupil, promptly reacting to light and accommodation, and the same fundus detail were observed. There were 8 degrees of exophoria. With the Hertel exophthalmometer, both eyes 16.5 degrees.

The only illness this patient had had was a mild attack of diphtheria when 9 years old. Figure 3 shows her condition when 3 years old. Her birth was said to have been easy, not instrumental.

CASE 2.—Boy, aged 8 years, born without difficulty, not instrumental, had never had any illness. His head was of odd shape practically from the time of his birth. The earliest photograph, taken when 2 years old, revealed a peculiar face and the suggestion of frontal swelling.

He was 114 cm. in height, with a dull, distant, vacant, stupid expression, and he was a decidedly noisy mouth breather. The skin and mucous membranes were normal. His nose was of the Roman type. The palate was decidedly arched and very high. The nasal septum deviated to the right; the turbinates were large. The ears were negative. The teeth were good, though irregular, fairly clean and not decayed, ten in the upper and twelve in the lower jaw. The lower jaw protruded, with prominent lower canines. The superficial veins of the neck were prominent. The posterior cervical glands were palpable. The chest was long, narrow and rather thin, with supraclavicular and infraclavicular fossae not remarkable; the interspaces were narrow, and the angle of the ribs more marked than normal. The epigastric angle was very acute. Breathing was of the abdominal type. The chest movement was good, and equal on both sides; there were no abnormal pulsations. The shoulders were rounded, the scapulae prominent and the spine straight. Palpation confirmed all signs of inspection. There was no evidence of early rachitis. The spleen and the liver were not palpable. There was normal pulmonary resonance on percussion. The heart outline was normal. The apex beat was in the fifth intercostal space one-half inch to the left of the nipple. Respiratory and heart sounds were normal; there were no murmurs. There were no herniae; the testicles had descended; the prepuce was adherent. The osseous system was apparently normal. Examination of the nervous system revealed nothing abnormal. The urine was negative. Blood examination

revealed: hemoglobin, 95 per cent.; red cells, 4,900,000; white cells, 6,300. The forehead was high, sloping upward to the vertex, with a bony ridge at the bregma. The circumference measured 51 cm. From the root of the nose to the occipital protuberance the distance was 34.5 cm.; from the root of the nose to the bregma, 15 cm.; bitemporal, 28 cm.; anteroposterior diameter, 21.5 cm.; from bregma to tip of mastoid, 17.5 cm.; transverse diameter, 16 cm. There was an abundant growth of dark brown hair.

Right eye: vision $^{20}/_{70}$. The pupil, 3.5 mm., was not perfectly round because of a straight nasal margin; it reacted to light and accommodation; the media were clear, the disk irregularly round with whitish nerve head, vessels overfull, in parts blurred because of the swelling of the nerve, which was + 3.00 D. There were no areas of hemorrhage, exudate or pigmentation.

Left eye: vision $^{20}/_{30}$. The pupil was irregularly oval, 4.5 by 5 mm., with straight nasal side, and reacted to light and accommodation. The same whitish swelling of the nerve head was present, with obstruction of veins and blurred outline of disk swelling = + 3.00 D.

Retinoscopic findings for each eye + 1.50. There was marked bilateral exophthalmos, by the Hertel instrument 20 degrees; nystagmus and tendency to divergence were present.

Dec. 20, 1916, the patient was operated on. A large subtemporal decompression was made on the right side. There was considerable intracranial pressure, with annoying dural hemorrhage. The patient died in a few hours. On necropsy the following was found:

The skull was irregularly elongated, measuring 21.5 cm. anteroposteriorly and 16 cm. transversely. The frontal region was unusually prominent. The inner surface of the calvarium and base of the skull showed unusually well marked depressions separated by sharp ridges. The bones of the skull were thicker than normal, measuring in the temporoparietal region 0.3 cm. in thickness and from 0.4 to 0.8 cm. in the frontal and occipital regions. The petrous portions of the temporal bones were negative. The middle ears and ear drums also were negative.

The brain weighed 1,380 gm. The general volume of the brain was rather considerable. It was elongated, but the two hemispheres were of equal length. The convexity appeared flattened. The left hemisphere was apparently smaller than the right. The base presented nothing unusual. There was very definite atrophy of both optic nerves.

CASE 3.—Girl, aged 7 years, well built and nourished, expression not intelligent, was delivered with forceps. The skin and mucous membranes were normal. The hair was light brown and abundant. The patient was a mouth breather. There were twenty-two teeth, which were clean and decayed. The palatal arch was high, with the nasal septum deviated to the

left. The tonsils were not enlarged or cryptic; there was moderate pharyngitis with adenoids. The posterior cervical glands on the right side were enlarged and palpable. The chest measured 56.5 cm.; on thorough physical examination, nothing abnormal was found in it. There was no sign of present or past rachitis. The circumference of the abdomen was 55 cm. Nothing abnormal was noted. Examination of the nervous system revealed the deep reflexes sluggish, the superficial reflexes active. The urine was negative. Blood examination revealed: hemoglobin, 95 per cent.; red cells, 5,280,000; white cells, 6,000. The forehead was high, sloping upward in conical form to a point at the bregma. The superficial veins were prominent in the frontal region. At the bregma, there was a distinct bony ridge. The circumference of the head measured 48 cm. From the root of the nose to the bregma the distance was 12.5 cm.; anteroposterior diameter, 20 cm.; from the bregma to the tip of the mastoid, 17 cm.; transverse diameter, 15.5 cm.

Right eye: pupil, 5 mm., slightly irregular, reacted to light and accommodation; the media were clear, the disk irregular in outline with marked pigmentation, decidedly pale, bluish white; the blood vessels were of irregular caliber. Hertel exophthalmometer, 10 degrees.

Left eye: pupil 5 mm. slightly irregular, reacted to light and accommodation; the media were clear; the disk irregular in outline with marked pigmentation, decidedly pale, bluish white; the blood vessels were of irregular caliber. Hertel exophthalmometer, 8.5 degrees.

Retinoscope + 1.25 in each eye. There was divergence of 30 degrees with short excursive rapid lateral nystagmus. It was impossible to test the vision although she was able to get around unassisted.

The patient had never had any illness, and it was positively known that the head was without change when the patient was 2 years old. It was impossible, however, to state when alterations began.

In all of these cases, the Wassermann reaction was negative.

Fletcher divides oxycephaly into three classes: those in whom cranial deformity is present at birth, those in whom the head is normal up to 2 years of age, and finally those in whom the change takes place between the second and third year of life. This classification, although to a certain extent arbitrary, offers a suggestion as to etiologic factors. Many writers consider rachitis a necessary preexistent element; but more numerous are the reporters who do not consider it an agent. Whether the syndrome be the result of menin-

gitis, of too early closure of the fontanel, of connective tissue or even bony compression of the optic foramina is as yet undetermined. Our cases which came to necropsy showed no closure of the optic foramina, no thickening of the meninges and no sign of rachitis.

In the cases here reported, it would seem that the disease in Case 2 probably was present at birth; the only apparent fact militating against this is that although the boy was 8 years old, both optic nerves were swollen, whereas Case 3 presented optic atrophy far advanced, although it is known that the child's head was normal until she was past 2 years of age. This is a point of considerable interest in the etiologic study of the condition; for if the head in Case 2 was deformed from the beginning and the patient was only in the nerve swelling stage and the head in Case 3 did not change until after the patient was 2 years of age and she was in the optic atrophy stage, we should be forced to look for a cause in something other than just the head deformity. Furthermore, at the time of necropsy in Case 2, there was no narrowing of the optic foramina, nor were there changes about the nerve or in the dura.

Oxycephaly should be considered a condition easy of diagnosis and amenable to surgical relief.

I am indebted to Dr. Frederic C. Conway for the general physical examinations, to Dr. Joseph A. Cox for operating in Case 2, and to Dr. Ellis Kellert for performing the necropsy.

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THE ENDONASAL OPERATION ON THE LACRIMAL SAC

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To many it may seem a presumption for a rhinologist to enter the sacred domain of this section and discuss a subject so intimately the field of the ophthalmologist. The *Grenzgebiet* is still a cause for strife between surgeons as between nations. My own hope is that in the domain of medicine, at least, it may soon be transcended by that higher aim, "the ultimate good to the patient," just as in national life it may ultimately be a question of "the greatest good to the greatest number." Then will humanity and benevolence rather than selfishness be the guiding principle in men's lives.

The demands of modern medicine become each day more extensive and exacting, and the specialties each day more circumscribed in their limitations. Whereas, in this country at least, it is but a short cry back to the times when the same individual encompassed, or attempted to encompass, within his domain, the diseases of the eye, ear, nose and throat; such a condition exists as a rule today only in the relatively smaller communities. Ophthalmology has come into its own as a distinct specialty, just as otology bids fair to do in the not too remote future. This assumption of authority and finality in our own limited field makes us in a way more than ever dependent on help from our fellows, according to the extent to which that field affects or is affected by the field of our fellow worker. So the ophthalmologist, who calls in the rhinologist to perform an endonasal operation on the lacrimal sac, is no more of an anomaly than the internist who

invites the technician of his field, the surgeon, to perform an operation for appendicitis; the surgeon who invites the otologist to perform an operation on the mastoid process; or the rhinologist who calls to his aid the dentist in his care of a diseased antrum, or to complete the result of his tonsil and adenoid operation by attending to the proper alinement of the child's teeth. Here is an instance in which team work and not selfishness, but mutual cooperation, spells success for both, and assures, or helps to assure, a successful outcome for the patient. My own cases are only those which have been referred to me by the ophthalmologists. In fact, I have no opportunity of seeing such cases in any other way.

Until a few years ago, according to West,[1] the treatment of dacryostenosis had made no real progress for a period of twenty-five years. In 1910 West published his paper entitled "The Window Resection of the Nasolacrimal Duct," and exhibited in all seven patients operated on by this method. This paper, as the title indicates, referred only to the operation on the duct, but laid no claim to being an endonasal operation on the sac. In three of the cases, however, the sac itself was really opened. West soon became convinced that the opening of the nasal duct alone could not be sufficient, and that any operation, to be successful, must concern itself rather with the opening of the lacrimal sac as well. He immediately set himself about the new task, and, through the generosity of Professor Silex of Berlin, gained access to an exceedingly rich clinical material. The results of his later endeavors he reported in 1913. At that time, he reported the investigation of over 300 cases of various diseases of the lacrimal apparatus, and the operation on the lacrimal sac in 130 cases by the endonasal method. He claimed a favorable result in over 90 per cent. of the patients so operated on.

1. West: Berl. klin. Wchnschr., 1914, **51**, 1633; Arch. f. Laryngol. u. Rhinol., 1913, **27**, 224, 504; Berl. klin. Wchnschr., 1913, **50**, 926.

West asserts that Caldwell was the first to open the lacrimal canal, in 1893, when he reported one case. Still later, Killian and Passow reported similar cases. The idea of reestablishing the connection between eye and nose, as a passageway for the tears, was known to the ancients, who were for the most part unsuccessful in their attempts. In 1863, Berlin had revived the extirpation of the sac from without, and this had remained the operation of choice almost to the present. In 1904, Toti published the results of his attempts to reestablish the natural pathway from without by means of a skin incision at the inner angle of the eye. In 1910, von Eiken published a paper in which the approach to the sac by way of the antrum was advocated. West performed his first operation in 1908.

The various operations, previously described, were successful only in a relatively small proportion of cases. The external operations often gave rise to fistulas, and, if successful, as far as the curing of phlegmon was concerned, necessitated later the excision of the lacrimal gland to get rid of the epiphora. The resulting scar, too, was often unsightly. West's earlier operations on the nasal duct were also unsuccessful, because the stenosis occurred at the inferior border of the sac, where it joins the duct. The operation was, accordingly, inferior to and outside the field of the real difficulty in a large proportion of cases.

West's modified operation concerns itself with the lacrimal sac alone, the duct being disregarded. The free opening of the sac itself he regards as essential, if a cure is to be expected. That the sac is really opened in all cases is shown by the fact that a probe introduced into the lower punctum passes horizontally into the nose. A minute knowledge of the anatomy of the parts is absolutely essential. The technic is as follows: A quadrangular flap, covering the end of the lacrimal sac, and extending well forward over the ascending process of the superior maxilla, is resected submucously, its base of attachment being inferior.

After its resection it is turned downward out of the field of operation, this inferior attachment acting as a hinge. The denuded area extends vertically through a space roughly limited by an anterior extension of two lines, the upper marking the attachment, and the lower the inferior border of the middle turbinal. Neither turbinal is encroached on, unless an obstructing hypertrophy is present. By means of appropriate chisels, a portion of the posterior border of the nasal process of the superior maxilla is chipped away, and the thin bone, covering the sac, resected. The sac itself is then grasped with forceps and a large portion of its nasal aspect resected with a thin scalpel. The submucous flap is now replaced, the posterior part, over the area of the resected sac, being first removed. The flap is held in position for twenty-four hours by packing of iodoform gauze. The after-treatment is exceedingly simple, and consists in keeping the nose free of crusts and granulations, and irrigating the sac by way of the canaliculus with a 3 per cent. solution of boric acid. As stated before, West claims favorable results in 90 per cent. of his cases.

The advantages of West's, over the previous methods, for the treatment of the various diseases of the lacrimal apparatus, he enumerates thus:

"1. The physiologic function of the path for the tears is again restored, so that not only a suppuration of the sac, a lacrimal fistula or a phlegmon is healed, but also the tears flow normally through the nose. A later epiphora is accordingly avoided.

"2. A so-called cure by probing is rendered unnecessary.

"3. The lacrimal gland is spared.

"4. A skin incision or a curetting from without, with eventual scar formation, is avoided."

This operation he has performed in every possible sort of disease affecting the lacrimal apparatus.

After the operation, certain persons, by sharply blowing the nose, can force air out through the canaliculus. West does not consider this a disadvantage, as the patients do not complain of it. Halle's suggestion for forming a valve of the mucosa, in order to prevent this, he regards as impossible of accomplishment.

A more recent endonasal operation on the lacrimal sac is that of Yankauer.[2] The latter considers the West operation unsatisfactory because of the tendency of the opening of the middle meatus to close, such objection being avoided by his improved operation.

In the operation of Yankauer, the horizontal incision is begun at the attachment of the anterior end of the middle turbinal and carried forward for a distance of 5 mm. It is then carried downward to the anterior border of the inferior turbinal and backward along its inferior, free border, for about 2 cm., or from one-third to one-half the length of the turbinal. The incision is carried well down to the bone and the roughly rectangular flap, thus outlined, resected submucously, its posterior attachment acting as a hinge. On this hinge it is folded backward and held in place by tucking it under the anterior free end of the middle turbinal. A part of this submucous resection consists of the mucous membrane and periosteum on both sides of the anterior end of the inferior turbinal to a point well back of the opening of the nasal duct. The bony portion of the inferior turbinal, so uncovered, is then resected with punch forceps. The bony covering of the canal and sac is now removed with chisel and punch forceps, and the canal at its extreme posterior aspect slit from the opening in the inferior meatus to a point well above the junction of the inferior portion of the sac with the duct. When the sac is found to contain pus, a portion of its inner wall is resected to allow for free drainage into the middle meatus. This

2. Yankauer: Laryngoscope, 1912, p. 1331, Vol. XXII, Tr. Am. Laryngol., Rhinol. and Otol. Soc., 1913, 294.

opening into the middle meatus closes subsequently. The internal walls of the duct and sac are now folded forward and held in position by folding the previous submucous flap down on it. The latter flap is held in position by a single stitch as well as by packing. The subsequent treatment consists in removing the packing after twenty-four hours, and irrigations through the lower punctum, the nose, of course, being kept free of crusts while healing is taking place.

Yankauer reports in all nine patients operated on by this method, during a period of three years: "Two were cases of mucocele of the sac; the other seven were suppurative. The suppuration ceased in all cases after the operation and has not recurred in any of them. The epiphora was relieved in all but one of the cases."

The most recent operation devised is that of Mosher.[3] This method of opening the sac and duct was come on, as it were, by accident from an observation of the specimens on the cadaver in the development of Mosher's operation on the ethmoid labyrinth and frontal sinus. After removal of the anterior end of the middle turbinal and free exposure of the processus uncinatus, Mosher's stiff probe is introduced through the duct into the inferior meatus, the canaliculus having previously been slit. A roughly rectangular flap, limited anteriorly by the posterior lip of the ascending process of the superior maxilla and a portion of the superior border of the inferior turbinal, superiorly by the extreme limit of the middle meatus, and posteriorly by the extreme inner tip of the uncinate process, is now resected submucously and deflected downward and backward.

The inner wall of the lacrimal cell and bony covering of the duct are now broken through with an appropriate curet, the nasal process of the superior maxilla acting as a guide anteriorly. The fragments are removed with a conchotone. The inner walls of the duct and sac are now broken through by slowly with-

3. Mosher: Laryngoscope, November, 1915, p. 739.

drawing the probe, at the same time turning its tip sharply inward toward the septum. The probe is then reintroduced, and serves as a guide to the curetting away of the posterior tip of the ascending process of the maxilla, as well as the upper part of the processus uncinatus. The canal is subsequently widened by biting forceps after the probe is withdrawn, practically to double its previous width. A ligature is then passed through the nose upward and out through the dilated punctum, a piece of gauze being attached, kite-tail fashion, to its middle. This gauze is then drawn upward into the lacrimal sac, and the ends of the ligature fastened to the face with adhesive tape. The flap is now replaced.

After-treatment consists in removing the plug after two or three days, keeping the nose free from granulations and crusts, and keeping the passage free by passing the probe wherever it is indicated. Mosher asserts that the probe can be passed either from the nose or from the inner canthus. The operation "has been abundantly tried on the cadaver and three times on the living." Mosher admits that "it is too soon to say much about the results on the living, except that, so far, they are good."

From a review of the foregoing methods and operations it would seem that in the development and perfection of the endonasal method a possible solution of a difficulty, which has long been a perplexing one, may be reasonably expected. Certainly the external operation and its after-results have left much to be desired. The endonasal route obviates many of the disadvantages of the external operation. Whether or not the physiologic pathway for the tears into the nose can be maintained permanently in a sufficient percentage of cases at the hands of the average rhinologist is a question which time alone can answer.

The results in my own series of eight cases have been fairly good. The West operation was that of choice in all but one. In this the Yankauer operation

was done. In only one case was there a complete failure. In this case the antrum was accidentally entered. Whether or not this had anything to do with the unsuccessful outcome it is impossible to say. In three cases a secondary operation was performed, that is, the stenosed opening at the beginning of the middle meatus was enlarged. All were cases of dacryostenosis and cystitis, except one. This was a case of stenosis and epiphora following a permanent opening into the antrum for empyema.

My own technic differs slightly from that of West. After the submucous flap has been outlined and elevated, a probe is inserted into the canaliculus, sac and duct. If this is difficult, the assistance of the ophthalmologist is summoned. This probe is held in place by an assistant. Its pressure renders the uncovering of the duct and sac easier. When the duct is freely uncovered, the point of the probe is directed inward toward the septum, thus bulging in its septal wall. A thin scalpel is now inserted between the probe and the lateral nasal wall, and the incision carried well up beyond the isthmus, so that the probe ultimately passes horizontally into the nose, as suggested by West. In this way a considerable portion of the duct and sac is completely removed. The infiltration of a few drops of 0.25 to 0.5 per cent. novocain solution, to the dram of which from 1 to 2 drops of epinephrin have been added, following the preliminary cocainization renders the operation bloodless, as well as absolutely painless. The only pain complained of in any of my cases was that from the pressure of the lacrimal probe.

To many this operation from its description may seem difficult or impossible of accomplishment. It *is* difficult, and this difficulty I shall not attempt to minimize. The difficulty, however, is no greater than that attendant on the submucous resection of the nasal septum, and I can assert with firm conviction that any one who can perform a submucous resection can successfully perform the endonasal operation on the lacrimal sac.

In my hands the difficulties have been considerably lessened by the use of the modification of West's instruments, which I herewith present.[4]

ABSTRACT OF DISCUSSION

DR. HARRIS P. MOSHER, Boston: As Dr. Chamberlain has stated in his paper, there are a number of methods of approaching the lacrimal sac and the nasal duct and short circuiting the drainage of the tears into the nose. The duct may be approached from the antrum. All intranasal methods must face the danger of reclosure of the canal. What is needed at the present time is to know the results of the various operations. It goes without saying that the procedure which is the simplest and gives the most permanent drainage will be the operation of the future. I have operated on nine patient. The results after a year of four of these cases are known. Three patients were operated on two years ago. When last seen the results were good. An attempt to trace these cases this year failed. If one were charitable one could say that no news is good news. In one recent case the patient is still under treatment and the result is not yet apparent. One patient was not gracious enough either to come to the hospital or to answer a letter.

The results in four cases only are known. Of these four, one was a case of mucocele of the sac. This patient after nine months has had no return of swelling of the sac, and there is a patent opening into the nose. The second patient, who had a suppurative sac of long standing with a skin fistula, to quote her words, reports "the eye is fine and the tears run over only when I get a cold." A more definite report is desirable in this case, but the woman had a good excuse for not coming in. She lives in another state and is trying to run a boarding house in a small New England town.

The third patient had a suppurative sac for seventeen years, complicated by an infected mucocele of the ethmoid labyrinth. He has now a patent opening into the nose and the tears run over only on a cold day or in a strong wind. The fourth and last patient who reported was given to me as "a lemon," to use slang. He had a bony occlusion of the nasal duct which the ophthalmologists, after a number of trials, could not relieve. This man is still wearing a style (at the end of ten months). With the style in place there is no running over of tears except in a strong wind. Such are the results in my small series of cases. I asked one of the

4. In addition to the references already given, the following will be found of interest:
Onodi: Monatschr. f. Ohrenh. u. Laryngo-Rhinol., **46**, No. 4.
Mithoefer: Ohio State Med. Jour., September, 1916.
Horgan, J. B.: The Operation of Dachryocystorhinostomy: Its Indications and After-Treatment, Jour. Laryng., Rhinol. and Otol., London, June, 1916, 225.

ophthalmic house officers to look up the results after excision of the lacrimal sac. His report was that a third of the results were satisfactory.

Having given the results as I have found them, I wish to speak on two more points, namely, the technic of the operation and the use of the style. If there is a deviation of the septum to the side of the diseased sac, the lacrimal operation should be preceded by a submucous resection of the septum. I feel that the easiest approach to the sac is through the thin bone of the anterior ethmoidal region. The steps of the operation as I now do it are as follows: The anterior end of the middle turbinate is removed and the slitting probe is introduced through the cut canaliculus into the sac and on through the nasal duct to the floor of the inferior meatus. Next the anterior ethmoidal cells are exenterated. Then a cut is made in the mucous membrane of the nose along the posterior edge of the ascending process of the superior maxilla. The slitting probe is withdrawn to the upper margin of the inferior turbinate, and when clear of the turbinate the point is pressed upward and inward and made to break through the nasal duct into the nose. The tip of the probe is then slowly forced upward, all the while slitting the inner wall of the nasal duct. The upward excursion of the probe is finally stopped by the increased resistance of the superior part of the lacrimal bone. Experiments on the cadaver have shown that this manipulation opens not only the nasal duct, but the lower two-thirds of the inner wall of the lacrimal sac. The slitting probe is now withdrawn and the posterior edge of the ascending process of the superior maxilla is bitten away. Special attention is given to the thick upper part of the process, the portion which is chiseled away in the West operation. With the finger tip in the inner canthus of the eye, the amount of bone which is removed can be easily followed. It is possible in this way to uncover the whole inner wall of a dilated sac. In every operation enough of the lacrimal bone should be broken by moving the slitting probe up and down and forward and back, followed by the removal of the bone fragments with the curet, to allow the finger tip placed in the inner canthus of the eye to feel the head of the curet, separated only by soft tissue. The area of the bone removed is a quarter inch in each diameter. Unless it is the purpose of the operator to exenterate the lacrimal sac intranasally the operation is finished by inserting a silver style. Every two or three days the style is removed and cleaned, but the style is not removed for any length of time or for good for at least six weeks or two months. This does away with the after probing which the patients dread. The end of the style lies in the inferior meatus in plain view. Upward pressure on the lower end of the style causes the head of the style present in the inner canthus.

The removal and reinsertion are simple and painless and the patient has no fear of the manipulation.

After the style has been removed permanently, signs of reclosure of the duct are watched for and if they appear the style is reinserted for a few weeks or a few months. The great advantage of the style is, as I have said, that its use eliminates the probing, plus the fact that all the while the style is in place it is helping to make a lasting fistula from the lacrimal sac into the middle meatus. This operation, therefore, as I now do it, is a combination of old and new methods, and the old is as important as the new. The head of the style can be made invisible, and after the operative field has become reepidermatized it is not necessary to remove the style for cleaning for weeks at a time. Judging from my limited experience, the style completes the operation. Every form of intranasal operation on the lacrimal sac and the nasal duct is only a temporary success unless supplemented with frequent probing or the prolonged wearing of the style. Without the style the intranasal operation carries with it a feeling of helplessness; with the style you are master of the situation. To me, West's high percentage of successes without resort to frequent probing or the use of the style is a mystery.

Intranasal extirpation of the sac is not only possible but feasible. The whole inner wall even of a much distended sac can be laid bare if the lacrimal bone is freely broken down and the upper posterior part of the ascending process of the superior maxilla chiseled away or bitten off with suitable bone forceps. In one of my cases there was a skin fistula half an inch below the inner canthus of the eye. An area of bone three-quarters inch by one-half inch was removed and then the sac and neighboring soft tissues were bitten away with a small punch until but little more than skin covered the bone defect. This was the patient who answered my letter by saying that her eye was fine and that the tears ran over only when she got a cold.

Some one will ask, What is the use of going to all this fussy intranasal operating when an external removal of the sac is simpler and just as efficient? My answer, supported by one case, is that the external operation is not just as efficient. In the case which I am now citing there was a mucocele of the sac and an infected mucocele of the ethmoid labyrinth and the frontal sinus. The sac was extirpated intranasally and a style worn for a short time afterward. This patient now has no tearing except on a cold day or in a high wind and the color test as well as blowing the nose show that he has a patent fistula into the nose. If there is any advantage in having a patent fistula into the nose, and ophthalmologists tell me that there is, the intranasal operation alone will accomplish this.

In attempting the intranasal removal of the sac it is a great help, after the inner wall of the sac has been extensively exposed by a free removal of bone, to put the finger tip in the inner canthus of the eye and to press the sac into the end of a short, self-illuminated bronchoscope and to work through the bronchoscope with small biting forceps such as are used in direct work on the larynx.

Before operating on a sac intranasally one should make up his mind how much he is going to do. In cases of recent mucocele without infection, removal of the inner wall of the sac, access to the sac being gained by removing the anterior ethmoidal cells, combined with slitting of the nasal duct above the inferior turbinate and the use of the style for a moderate period, is sufficient. When the sac is suppurating, or there is a skin fistula, the sac should be extirpated and the style used for a longer period.

DR. WILLIAM B. CHAMBERLIN, Cleveland: One point I did not have time to bring out in the paper is the subsequent closure. This is a difficulty in any endonasal operation. Three of my patients were operated on the second time; but the secondary operation is comparatively a simple thing. The probe is introduced through the sac again and at the point of stenosis you can see a slight bulging. It is a comparatively easy thing to resect this with the punch forceps. In the after-treatment, if one is careful in keeping down granulations, as in any other part, epidermization will not be retarded. In many cases a preliminary submucous resection, especially a resection as high up as we dare go, is absolutely essential. It is a *sine qua non* of the operation.

Another point Dr. Mosher brings out in the discussion is the resection of the posterior part of the ascending process of the superior maxilla, which makes a groove for the duct. This is dense, firm bone. It is for the resection of this ascending process that West has devised his chisels, and it is for the resection of this process that I have modified his chisels, making them very much thinner, and also putting on the handle, thus enabling one to see every step of the operation. The view is less obstructed than with the West chisels, and you can insert it under the bone, and if it does not make a clean cut the bone may be fractured by a twisting motion. The forceps are a modification of the familiar Gruenwald forceps for the ethmoid, and are quite as effective as the instrument which West has especially devised for the purpose.

NEUROLOGIC PERIMETRY AND A METHOD OF IMITATING DAYLIGHT WITH ELECTRIC ILLUMINATION *

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In a previous paper[1] I have described much of the technic of modern perimetry in connection with the study of color interlacing in neurologic cases, from the surgical clinic of Prof. Harvey Cushing. It was stated there that I believed it possible to imitate the daylight illumination of the perimeter and perhaps improve it in many respects by use of an arc of electric lights properly controlled and arranged. Accordingly I wish to submit in this paper an apparatus for the satisfactory perimetric examination day or night of difficult neurologic patients.

THE PERIMETRIC PROBLEM IN A NEUROLOGIC CLINIC

Probably the greatest difficulty to contend with in this group is the matter of comfort. If the patient can be kept comfortable throughout the examination, half the battle in taking a complete field at one sitting may be considered won. This is true not only because the time is greatly shortened, since the patient can make a greater number of observations without rest, but also because the answers are more accurate and consistent, thereby greatly reducing the number of readings that have to be discarded.

After the physical capacity of about 1,500 neurologic patients had been observed, it was noted that practically all of them could be moved into a reclining

* The sequel to this paper will appear in the Trans. of the Am. Ophth. Soc. for 1917 under the title of "Quantitative Perimetry; Practical Devices."

1. Walker, C. B.: Tr. Am. Ophth. Soc., 1916, **14**, 684.

wheel chair and kept comfortable for a period of two hours or so. In the worst cases the patients could at least be moved so that the head would be at the foot of the bed, slightly elevated with a pillow or two (Fig. 1). In these cases, however, perimetry is apt to be prevented by the mental state of the patient, except in the postoperative group, in which a field may be desired the first day or two after operation. In the latter group the presence of a cast or head dressing may interfere, though almost always it may be trimmed away sufficiently about the eyes (Fig. 2).

Having determined what positions of comfort must be used, we may desire an apparatus for holding the perimeter in the proper corresponding positions; but still other conditions should be fulfilled. In the first place it is desirable to be able to take a field at any time of day or night, not only because there may be an urgent demand for an immediate report, but also because there may be only certain intervals at odd times when the patient is mentally lucid enough to be examined. Again, because of dark cloudy days, and short winter days, artificial illumination becomes necessary to meet the time requirement. But when the patient is placed in the recumbent position, even good daylight becomes unsatisfactory because most of the distribution comes from above, whereas it should come from below. Artificial illumination must therefore be used, not only because it is available at any time, but also because it can be adjusted to project in any direction.

Thus we find that the solution of our problem requires the fulfilment of two conditions; first, an apparatus to hold the perimeter in proper relation to the eyes of the patient while in a comfortable reclining or sitting position, and, second, an adjustable illuminating apparatus.

THE SUSPENSION APPARATUS

It was decided that the conditions cited with reference to the proper support of the perimeter could best

be fulfilled by suspension from above. Accordingly the apparatus shown in Figure 3 was designed. A glance at this shows the moving parts all supported by a right angle crane of 2 inch iron pipe held in a vertical position by a clamp to the window casing above, and below by a pivot on the floor. The projecting arm of the crane can swing through 180 degrees by this arrangement, so that the apparatus can be pushed slightly to one side or clear over against the wall, when not in use.

The perimeter is suspended from a carriage which is underslung from two rollers (*a*, *a*) traveling along the upper surface of the projecting arm of the crane. The carriage and the perimeter are connected by two telescoping pipes (*b*) which can be clamped together. This telescoping tube provides movement along and rotation about the vertical axis of the perimeter. At the lower end of the outside telescope pipe is a ratchet joint (*c*) to which the perimeter is clamped below. By means of this joint the perimeter can be tilted through an angle of 90 degrees from the vertical corresponding to the angle at which the patient reclines (Figs. 1, 2, 3 and 4).

In order that the horizontal motion of the carriage on the crane and the vertical motion of the telescoping pipes can be smoothly controlled, counterweights are used thus: Two cords inside the telescoping pipe are attached to the lower segment just above the ratchet joint. These cords, emerging at the top of the upper segment, pass over a pulley (*d*) which projects into the pipe through a slot. One of the cords then passes directly downward again to attach to the larger counterweight (*e*), while the other passes along the horizontal arm of the crane and over another pulley (*f*) on the upright pipe, and thence down to the smaller counterweight (*g*). The sum of these weights is equal to the combined weight of the perimeter and the lower segment of the telescoping tube. By this arrangement the smaller weight (6 pounds) not only acts as a

counterweight for the perimeter but also pulls the carriage slowly along the crane toward the patient, if the trigger (*h*) is depressed. This motion can be checked at any point by releasing the trigger (*h*), which allows a dog to escape in the notched strip of channel iron fastened to the under surface of the crane. The strip of channel iron also serves to prevent lateral swinging of the telescoping tube, the latter being grooved at its upper end to receive the projecting margins of the channel iron.

By use of these devices the perimeter may be easily moved forward or back, up or down, swung to one side, rotated or tilted to meet all of our requirements with respect to position. Any perimeter may be suspended in this way. The one we use is a very wide faced perimeter of special design which has been previously described.[2]

The illuminating device is also attached to the vertical portion of the crane, as shown in Figure 3. To make this apparatus a piece of 1 inch iron pipe 8½ feet long is bent to form an incomplete circle 3½ feet in diameter. The opening in the circle is wide enough (20 inches) to admit the back of a wheel chair. Holes are then drilled and tapped at an angle of 60 degrees with the axis of the circle, to receive sixteen sockets for electric light bulbs at intervals of about 6 inches. This brings the source of light to lie in a circle of about 32 inches diameter, the distance of each light from the center of the perimeter during working conditions being 30 inches. The electric lights are protected simply by two parallel circular wire guards running around on each side and a little in front.

This circular bank of lights is hinged at its upper midportion directly to a sliding sleeve (*k*) on the upright portion of the crane. The two lower ends of the bank are also hinged to a lower sleeve (*l*) but with the intermediation of two rods 2¼ feet in length so that as the lower sleeve is made to slide down while

2. Walker, C. B.: Arch. Ophth., 1915, **44**, 369.

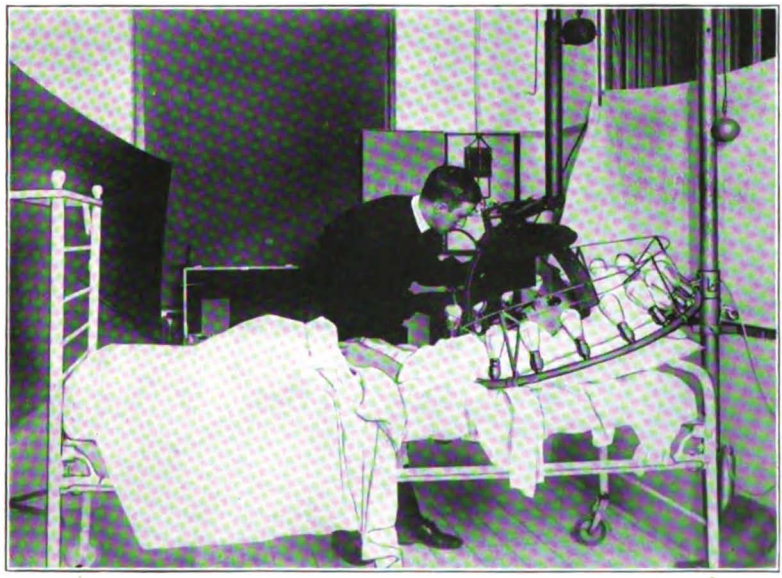

Fig. 1.—Adjustment of suspension perimeter for patient in bed.

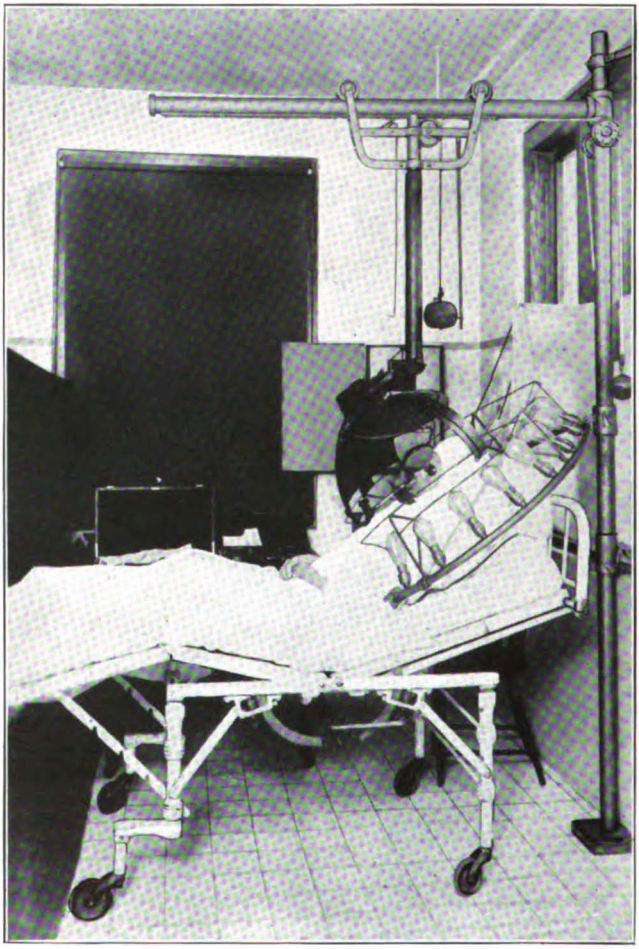

Fig. 2.—Adjustment of suspension perimeter for patient in Way bed.

Fig. 3.—Adjustment of suspension perimeter for patient in sitting position: *a*, *a*, suspension carriage rollers; *b*, telescoping pipes and clamp; *c*, ratchet joint; *d*, *f*, *m*, counterweight pulleys; *e*, *g*, counterweights; *h*, trigger controlling horizontal motion of carriage; *k*, *l*, sliding sleeves for light carriage adjustment.

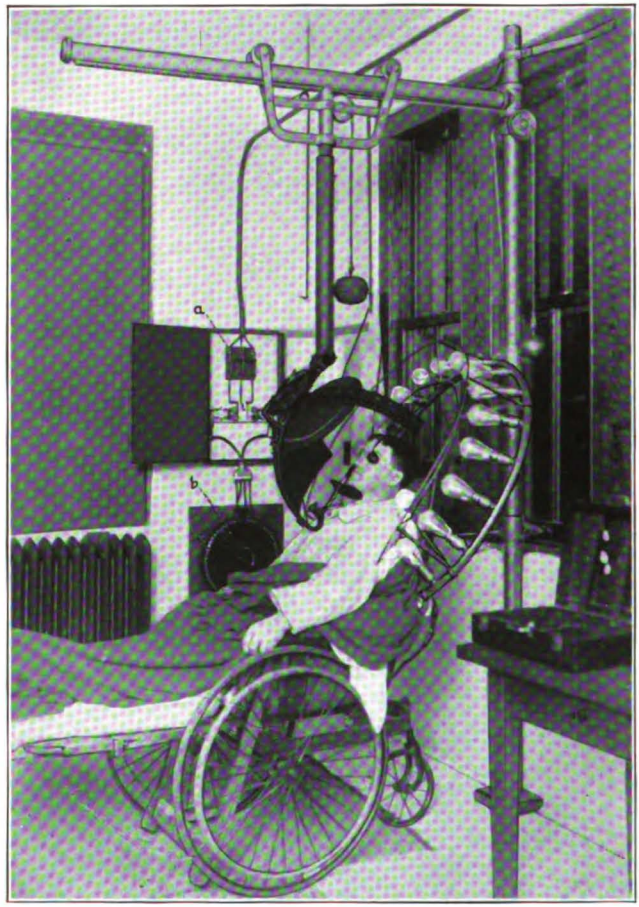

Fig. 4.—Adjustment of suspension perimeter for patient in wheel chair: *a*, switch board; *b*, rheostat.

Fig. 5.—Large black screen serving for Bjerrum test and as a dark background when the perimeter is in use, black gown and gloves are worn by the examiner.

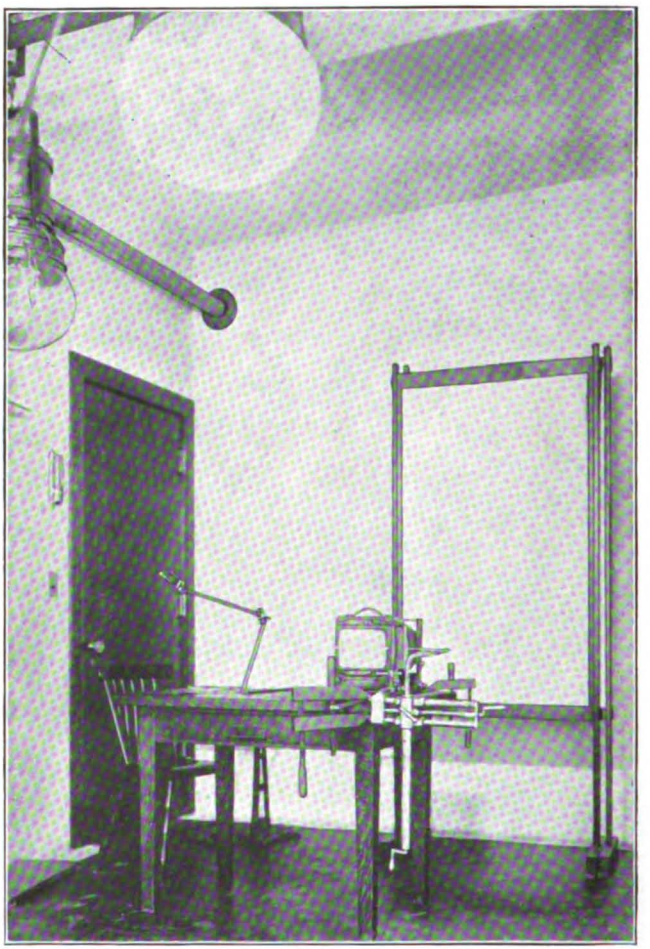

Fig. 6.—Arrangement used to make exposures for photographic wedge plates.

the upper sleeve is clamped, the bank of lights can be tilted forward to the desired angle and fixed in position by clamping the lower sleeve (Figs. 1, 2, 3 and 4). However, when both sleeves are free, the bank hangs vertically and can be easily raised or lowered, being counterweighted over the pulley (*m*) on the upright portion of the crane.

In connection with this apparatus, I have also designed a Bjerrum screen (Fig. 5), which is placed directly across the room at a distance of 3 meters or less if desired. The screen can be used with the greatest ease after the visual field has been taken by simply pushing the perimeter to one side, and propping up the patient's head somewhat, if in the reclining position. Artificial illumination from the perimetric lights is a great help, since a large screen such as we use is difficult uniformly to illuminate in an ordinary room. The screen is made of light weight black canvas stretched over a frame measuring 9 by 11 feet. The frame is made of ¾ inch pipe having at the bottom short (18 inch) extensions at right angles to the screen, giving sufficient base to maintain a firm upright position. The canvas overlaps the frame and is laced taut behind by stout cording. The screen may be moved about the room and is readily demountable if it is necessary to move it to another room.

The examiner wears a black gown and gloves as shown in Figure 5, and, standing close to the screen, is able to see the lines of circles drawn on the screen representing the field at 2 and 3 meters distance. The patient is not confused by these lines, since they are drawn with a soft pencil, and can be well seen only by the examiner. The lines for 3 meters are solid, those for 2 meters are broken. Three meters is the distance used for the most part, since only the very smallest visual angles (below 3 minutes) are tested on the screen, the larger angles (3 minutes and more) being readily tested on the perimeter by use of fine disks previously described.[2] The fixation point is slightly

above the level of the patient's eye in order to get a sufficient field of screen below. The error on this account is negligible, since most of the measurements are under 30 degrees from the center.

ILLUMINATION

In a previous paper I have already stated my objections to the use of transilluminated (if I may use that term) test objects as a method of accurate perimetry. Briefly they may be stated as follows: It is necessary to work in the dark in order to control retinal dark adaptation phenomena which are not constant for half an hour or more. During the entire field examination this dark adaptation must be maintained constantly. Diffusion and dispersion of light rays in transit of the vitreous lead to confusion in determining the boundaries of the field. The size of the pupil may vary with the distance of the transilluminated test object from the center. The central fixation light and the test light may produce after-images. But, worst of all, the patient's eye cannot be observed in the dark as to fixation, and a mechanical perimeter is necessary to make the record. With the mechanical perimeter we cannot be sure of our records in the dark, nor can we use the range of disk-sizes necessary for a complete examination, as stated in the paper already mentioned.

With transilluminated test objects and mechanical perimetry precluded, we have left the possibility of closely imitating good daylight on the plain perimeter. Daylight, that is, skylight or cloudlight, may be closely imitated by strongly transilluminated milk-glass. And our original idea was to use the new nitrogen bulbs (75 watts) for this purpose; but it was soon found that, although they could be used, the heat developed by them, when used in sufficient number, was considerable. However, certain helpful data were obtained before they were changed.

From the switchboard (Fig. 4 *a*) the lights can be controlled in intensity through a wide range by use of a theater dimmer (*b*). The lamps are connected through this dimmer in two circuits, alternate lights being on separate circuits. Either circuit, or both together, can be separately controlled through the dimmer. It was found that sixteen of these lights are considerably more than are needed when not dimmed, but when they are dimmed to about the strength of skylight the "white" character of the light is lost, and reddish tints are more noticeable.

Accordingly it was decided that the rheostat is to be used only to determine, in the first place, how much light is required to approximate daylight, and later only for small adjustments. When the desired amount of light is thus determined, as will be explained later, it is duplicated by trying light bulbs of various wattages in the circular bank, with the rheostat out of circuit. Further adjustment cannot be done until daylight illumination of the perimeter had been carefully studied as a standard of comparison. This work may be outlined as follows:

CALIBRATION OF DAYLIGHT AND ARTIFICIAL ILLUMINATION OF PERIMETRIC TEST OBJECT

It was necessary first to provide a fairly accurate photometer of small size so that it would not itself block off illumination from the test objects. Also it should be possible to use the photometer when the test disks are at any point on the perimeter. Further, the observer's eye should be covered as little as possible so that the compensating devices of light adaptation and pupillary reaction shall be practically the same as in the patient's eye during field examination.

After considering several kinds of photometers, it was decided that a narrow wedge which could be moved vertically before the eye would best meet the requirements.

The light transmitting properties of a true wedge can be well imitated by proper exposure of photographic plates. Such wedges have been used by astronomers and Dr. Williams for measuring small or weak lights. Figure 6 shows an extemporized arrangement with which plates of sufficient uniformity for our purposes have been made. A 5 by 7 camera is fixed to a table in such a position that the slide of the plate holder can be drawn by the horizontal screw of a Killiam suspension apparatus. Any small windlass device will be just as satisfactory, since with a little practice one can develop a very uniform winding motion.

The exposure starts as soon as the slide leaves the end of the plate holder, since the shutter is left open, and ends when the shutter is closed at the moment the slide is out, the camera being pointed somewhat out of focus toward a white uniformly illuminated wall or screen. It was soon found, however, that uniform motion of the screw did not give a sufficiently short scale, no matter what the speed. If the speed, however, was increased from a very slow winding motion, at first, to a very rapid motion at the finish, a wider range was obtained. But if in addition to the increasing speed the diaphragm of the camera is gradually closed during the withdrawal of the slide by an assistant, a scale which is short enough, or even too short a scale, may result.

A variety of plates were made with these different exposures and then cut into strips 1 cm. wide. While no single strip gave us exactly the desired scale, a combination of two or more strips selected by trial gave a very satisfactory photometric wedge, since small variations in density were thereby averaged. With the glass side turned out and a marginal binding of adhesive or gummed paper, a handy little instrument was obtained (Fig. 7, reduced one-half size).

A satisfactory wedge for our purposes having been selected and mounted, there remained the problem of

calibration. Here again selection of a suitable method was necessary. The use of a point source of standard light and the square of the distance ratio was unsatisfactory, as was also the use of the stellar magnitudes used by astronomers. The reason for the failure of these methods, I think, is really a retinal phenomenon well shown in Tables 1 and 2. Here it is notable how

TABLE 1.—CENTRAL ILLUMINATION OF PERIMETER BY BRIGHT MIDDAY LIGHT AND BY CLOUDY DAYLIGHT*

Disk Diameter	Averages of Bright Days, Best Light		Light Failure on Afternoons of Ordinary Cloudy Days (Averages)						
	Noon	10 a.m. and 2 p.m.	2 p.m.	3 p.m.	4 p.m.	5 p.m.	5:30 p.m.	6 p.m.	6:30 p.m.
40 mm.	0.75	0.73	0.67	0.63	0.50	0.41	0.35	0.30	0.20
20 mm.	0.74	0.72	0.65	0.60	0.49	0.40	0.35	0.27	0.18
10 mm.	0.73	0.71	0.63	0.58	0.48	0.38	0.33	0.25	0.15
5 mm.	0.71	0.69	0.61	0.56	0.47	0.37	0.30	0.22	0.13
2.5 mm.	0.68	0.66	0.57	0.51	0.45	0.35	0.28	0.17	0.10
1.2 mm.	0.52	0.50	0.48	0.45	0.40	0.30	0.20	0.11	
0.6 mm.	0.43	0.41	0.39	0.36	0.31	0.21	0.10		
0.3 mm.	0.31	0.30	0.28	0.28	0.22	0.10			
0.15 mm.	0.20	0.19	0.17	0.16	0.12				

* Tables 1 and 2 present a comparison of central perimetric illumination by daylight (general average) and by artificial illumination of different strengths.

TABLE 2.—ILLUMINATION POSSIBILITIES (CENTRAL) WITH VARIOUS LAMPS AND CIRCUITS

Disk Diameter	1 Circuit 60 Watt Lamps	2 Circuits 60 Watt Lamps	1 Circuit 40 Watt Lamps	2 Circuits 60 & 40 Watt Lamps
40 mm.	0.66	0.75	0.64	0.70
20 mm.	0.65	0.74	0.63	0.69
10 mm.	0.64	0.73	0.62	0.68
5 mm.	0.63	0.70	0.60	0.67
2.5 mm.	0.55	0.63	0.53	0.60
1.2 mm.	0.48	0.52	0.44	0.50
0.6 mm.	0.38	0.40	0.33	0.39
0.3 mm.	0.27	0.30	0.24	0.28
0.15 mm.	0.18	0.21	0.16	0.19

fast the reading decreases as the size of the disk decreases, although the illumination per unit of area is constant. This was noted roughly before the calibration was done. As the object becomes punctuate in size the readings become more uncertain until finally the point is reached at which the normal central sco-

toma begins to interfere. These factors make it more difficult to build up a scale from a point source as a standard, than to build down from an illuminated disk on which it is much easier to make a reading.

A consideration of our difficulties fairly suggests that the full moon in a cloudless sky would make an excellent standard for many reasons. It is diskiform, fairly uniformly illuminated, and of size comparable in visual angle to some of the disks to be examined. It is more strongly illuminated than any of our disks as used, so that the "lunar unit," as I shall call the strength of full moonlight illumination, must be divided preferably decimally to give the desired scale for use on perimetric disks.

The division of the "lunar unit" was accomplished in the simplest manner by combining ten strips from a plate of such strength that the full moon observed through them could be blanked out at some point as they were drawn past the eye. This point was determined in the same manner on the wedge which we wished to calibrate.

Since all the ten strips in the combination first described were of exactly the same density at the point at which the moon was blanked, we assumed that each strip represented one-tenth of a "lunar unit" at that point. If one of these strips is taken and the wedge to be calibrated is moved at right angles to the strips at the point mentioned, a place is found at which the moon is just blanked through the combination of strip and wedge. This point will obviously represent 0.90 lunar unit on the wedge. Next, two strips being taken, the point on the wedge representing 0.80 lunar unit was found; and by repeating the process, the wedge was divided into tenths of a lunar unit. The distance between each tenth was then arbitrarily divided into tenths so that our photometric wedge was finally calibrated in hundredths of the lunar unit, as indicated in Figure 7, which is drawn to scale.

Although the photometer described is not the most accurate, it has all the accuracy necessary and possible to apply under the conditions of observations by the patient. The results obtained with it are satisfactory and easily obtained. In taking the readings the observer seats himself at the perimeter with the eye at the center of the perimeter and observes the disk to be tested through the wedge held vertically before the eye. The wedge is slowly moved until the disk is just blanked out. A slight motion of the disk may be made at this point to assist the observation as the disk is made to appear and disappear several times until checking readings are obtained.

Fig. 7.—Drawing to scale of the completed photographic wedge, showing calibration in terms of "lunar unit." Reduced ½ size.

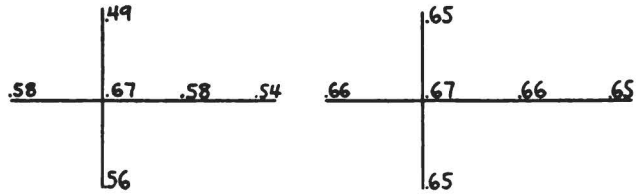

Fig. 8.—Comparison of illumination on principal points of perimeter by average daylight and by equivalent artificial illumination. Compare Tables 3 and 4.

With this simple device a great number of observations were easily and rapidly made on the various sizes of test objects, both centrally and peripherally placed on the perimeter. Observations were made in all kinds of daylight, from morning until night, and in all sorts of weather conditions. Likewise observations were made with artificial illumination alone and with combinations of various conditions of daylight and various grades of artificial light.

To reduce the mass of data obtained to the smallest possible space, we find that we can refer to the light of

any type of dark day, rainy, snowy, etc., as being equal to the light at some particular time between afternoon and dusk of any average day during which we have studied the light every half hour or so until dark.

Thus Table 1 shows the failure of light on an average bright, although short winter day. At noon the light is best, but for two or three hours before and after, the light is almost equally as good. On our scale when the readings are above 0.60 for the large disk and 0.50 for the 5 mm. disk, light adaptation seems to compensate to such a degree that the fields are satisfactory. The difficulty at this point is that it will not be maintained long enough to do much in case it is late afternoon of an ordinary day.

As failure of light continues beyond this point, the smallest disks begin to be thrown out of commission; and although the larger ones can be used, their fields are contracted. Thus the effect of light failure on the size of the field varies inversely with the size of the disk.

The weakened daylight of bad weather is apt to measure close to or less than the lower limit of usefulness mentioned above. While the larger disks can still be used under these conditions without great error, owing to the compensation of dark adaptation of the eye, the fields for the smaller disks both on the perimeter and on the Bjerrum screen are usually considerably reduced. One circuit if light of 40 or even 25 watt tungsten lamps will bring such light up to a satisfactory working strength. Since the testing with objects of small visual angles is most valuable in modern perimetry, the use of weakened daylight should not be tolerated when it is so easily brought up to a satisfactory working strength by addition of the artificial illumination of a few electric light bulbs.

The average central reading obtained by average daylight on the normal or 5 mm. disk has been found to be short 0.67 on our scale. Other disks will give readings according to their size, as indicated in the

tables. This degree of illumination is, of course, readily obtained artificially by use of the rheostat and strong light. But by trying lamps of various wattages, it was found that when eight 60 watt frosted tipped tungsten lamps were used in one circuit while seven 40 watt and one 60 watt lamps were used on the other circuit, the full strength illumination met our requirements. Table 2 shows the illuminating possibilities of 40 and 60 watt combinations. Since each circuit has a separate switch, three degrees of illumination are available without the rheostat. One circuit or the other alone may be used to strengthen weakened daylight, either late in the afternoon or in bad weather. In worse light, both circuits will be used. It is notable in the table that doubling the illumination does not double the readings due to light adaptation phenomena.

TABLE 3.—ILLUMINATION OF PRINCIPAL POINTS ON PERIMETER BY AVERAGE DAYLIGHT*

Position	Average Daylight						General Average
Centrally.....	0.67	0.65	0.68	0.68	0.69	0.70	0.67
60° laterally.....	0.58	0.57	0.57	0.54	0.50	0.62	0.58
90° laterally.....	0.54	0.48	0.56	0.49	0.57	0.58	0.54
60° above.....	0.48	0.46	0.49	0.45	0.50	0.54	0.49
60° below.....	0.58	0.56	0.55	0.50	0.58	0.61	0.56

* Tables 3 and 4 present a comparison of illumination on principal points of perimeter by average daylight and by equivalent artificial illumination. Compare Figure 8.

Thus far I have considered the illumination of the central portion of the perimeter. Daylight illumination falls off considerably in the region from 60 to 90 degrees from the center because of the disadvantageous angle at which the light falls on the disk in this region. Table 3 shows several observations, at different times, on the 5 mm. disk at characteristic points on the perimeter in average daylight.

In this respect artificial illumination has a decided advantage, as shown in Table 4. The peripheral readings under these conditions fall off a negligible amount. It must be noted, however, that these readings are all taken without the eccentric adjustment possible with

our perimeter. If we use 20 or 30 degrees of eccentric fixation, we have almost perfectly uniform illumination with artificial light in spite of the incompleteness of the circular bank, where it accommodates the patient's chest. The readings in the latter table serve also to show what degree of accuracy may be obtained after some practice with the photometer used, repeating readings checking usually within two hundredths.

Under artificial illumination of this type, the colors seem to give practically the same readings as in daylight, though it may be added that we find examination with the minute disks and smallest visual angles will usually give all the evidences obtainable with colors, especially now that we have given up color interlacing as a valuable aid to the diagnosis of increased cerebral

TABLE 4.—ILLUMINATION OF PRINCIPAL POINTS ON PERIMETER BY ARTIFICIAL LIGHT

Position	Artificial Illumination						General Average
Centrally.....	0.66	0.67	0.68	0.67	0.69	0.66	0.67
60° laterally.....	0.66	0.65	0.67	0.66	0.66	0.65	0.66
90° laterally.....	0.65	0.65	0.66	0.64	0.64	0.66	0.65
60° above.....	0.64	0.67	0.65	0.64	0.65	0.64	0.65
60° below.....	0.66	0.65	0.65	0.66	0.65	0.65	0.65

tension. Spectroscopically the difference between the colors illuminated by daylight and by this method is practically negligible, being much less than variation from week to week due to soiling and fading, and quite within the limits of variation in colors one may always find in different offices and clinics.

CONCLUSIONS

1. An overhead perimetric suspension apparatus such as described has the greatest possible range of service.

2. Artificial illumination arranged to imitate daylight has proved to be a perfectly satisfactory, complete or partial, substitute for daylight.

3. Artificial illumination is superior to daylight in uniformity, in constancy, in availability and in control.

ABSTRACT OF DISCUSSION

DR. LUTHER C. PETER, Philadelphia: The question of illumination in perimetry is of very great interest and exceedingly important. It is of decided value, I am sure, to follow Dr. Walker's mental processes and experiments in arriving at his conclusions because it impresses one with the intricacies of the problems involved in this apparently simple question.

The difficulties in the way of obtaining good perimetric studies are: (1) improper illumination in the average office; (2) the absence of uniformity of daylight due to weather conditions; (3) bedside perimetry, which involves problems of its own; (4) a total absence of standards either as to the amount of light used or as to color values.

To discuss the last question first, it must be apparent to all that we can only approximate uniformity in light, much as accuracy is desired for scientific purposes. The nearest approach to uniformity is to be able to say that the examination was conducted before a window, preferably of northern exposure, on a bright day. In our effort to imitate daylight we naturally concede that daylight is our standard, and artificial illumination is an imitation. In any given case of great importance the work should be done under these recognized standard conditions whenever it is possible. Qualitative color changes are of more value in recognizing early pathologic processes than are alterations in the form field, and very slight differences in the amount and character of the light will produce perceptible color alterations. The character and amount of light, therefore, are of paramount importance, and only absolute necessity should lead us to adopt a standard less constant than good daylight.

As to bedside perimetry, the conditions here demand special study. The apparatus which Dr. Walker has devised for this purpose seems to be admirably adapted to fairly accurate work with a minimum amount of tax on the patient's strength. If the equipment is not too complicated, its general adoption by the larger hospitals would add much to our equipment for doing efficient work, in the neurologic cases in particular. I believe Dr. Walker does not recommend this as well adapted to general use in the private office.

How are we to overcome problems one and two, namely, the poor illumination found in the average office, and the variation in the degree of light on clear and cloudy days? This naturally resolves itself into the question of the effect of any form of artificial illumination on the field study. There is now general agreement that the transillumination, as Dr. Walker calls it, of the test object should be entirely eliminated from the perimeter because light as well as form and color are tested at the same time, the results of the examination being therefore inaccurate and misleading. In the illumination of the campimetric surface I have personally

made careful studies with fields taken in good daylight and under suitable artificial illumination, and have not been able to find much, if any great difference in the size of the field, although I find it easier to determine the color limitations of my own field in good daylight. On cloudy days, therefore, I have not hesitated to use artificial illumination for the campimetric surface—Mazda lamps covered with ground glass being employed. The character of the illumination is noted on the chart.

The ideal artificial illumination is by the indirect lighting system—the light springing from one source in sufficient amounts to duplicate the even diffusion of daylight. When this is not possible, or for economic reasons, Mazda lamps covered by frosted globes may be substituted to illuminate the campimetric surface, care being observed that the entire surface shall be evenly illuminated. In collaboration with others, I am at present working on a lamp, both for campimetric and for test type illumination, which possesses the properties of normal daylight. The original cost, however, of such a lamp and the cost of operating the same is too much at present to admit of its general use in offices.

I should like to modify Dr. Walker's conclusion somewhat as follows: First, illumination such as can be obtained before a northern window on a bright day is the ideal and standard for perimetric work, and when this can be obtained, offers greatest uniformity in perimetric studies, a uniformity which will apply the world over. Second, when good daylight cannot be routinely employed, because of dark offices or when the sky is overcast, approximately uniform results can be obtained by the illumination of the campimetric surface, preferably by means of indirect lighting, or by direct illumination furnished by Mazda lamps with frosted globes. Third, the test object should never be transilluminated. Fourth, bedside perimetry may be conducted advantageously with artificial illumination, as suggested by Dr. Walker.

CONICAL CORNEA, OR ANTERIOR MYOPIA

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This subject, last brought before the section in the valuable symposium by Burnett, Sattler and Hermann Knapp, seventeen years ago, is of great scientific and practical interest, and our knowledge of it is still fragmentary. It has broad relations with other pathologic conditions of the eye and with more general problems of nutrition. The phrase "anterior myopia" is suggestive of relations to the larger class of posterior myopia and the process through which the great majority of cases of myopia develop. Both keratoconus and posterior or axial myopia arise from the pathologic distention of the sclerocorneal coat. In their production, states of general nutrition and local conditions both share, but to quite different extents. Axial myopia has been the subject of extended and thoughtful study, especially by our colleague, Dr. S. D. Risley. The succession of events, eye-strain, uveal congestion, scleral softening and distention, and passage of refraction from hyperopia to myopia have been often watched. Especially has astigmia been noted, as an important factor in the production of eye-strain; the eyes passing, as Risley has phrased it, "from hyperopia to myopia through the turnstile of astigmatism."

The use of the eyes for near work is also recognized as a very general condition of the causation and progress of myopia. General diathetic diseases have also been considered to cause myopia. Batten[1] made a strong argument for the connection of myopia with

1. Batten: Ophth. Rev., 1892, p. 1.

general disease, and reported seventeen cases in support of this view, mostly of myopia arising in connection with cardiovascular disease. The share of general impaired nutrition in causing myopia has not been so well demonstrated as the share of near eye work and strain from astigmia, but it seems necessary to assume such a factor to account for the fact that in many patients astigmia, eye-strain and retinochoroidal hyperemia leave undiminished hyperopia, while others become less hyperopic or myopic.

In the production of keratoconus the same factors may be concerned, but with far different relative importance. In anterior myopia, conditions of lowered general nutrition seem to be of first significance; anemia, acute general disease, chronic diathetic diseases, and impaired nutrition from many causes seem clearly associated with the beginnings of conical cornea. These may from the first cooperate with eye-strain, and later certain mechanical factors perpetuate and increase the trouble; but conical cornea rarely, if ever, begins when the patient is in robust general health. It is easy to see why the nonvascular cornea should especially suffer from lowered nutrition; while the most vascular portion of the sclera at the posterior pole of the eye should be most involved in a process, marked by active hyperemia. In axial myopia the brunt of the pathologic process falls near the posterior pole of the eye and near to the temporal side of the optic disk. The influences that produce keratoconus show little or no effect except toward the center of the cornea well removed from the vascular limbus. The beginnings of keratoconus are not closely associated with near work for the eyes, although after the defect has come into existence, and the eye is crippled for distant vision, the same vicious circle is established as in axial myopia; and the deterioration is likely to be more rapid and to go much farther because of the presence in keratoconus of high and variable astigmia.

The conditions under which keratoconus begins are often difficult to determine. But in all cases that I have investigated carefully in this respect there has been a clear history of some antecedent severe impairment of nutrition.

One patient had suffered from extreme epistaxis; another from many attacks of epistaxis resulting in anemia; another had suffered from marked anemia from other causes, and three others first noticed defective vision while recovering from typhoid fever. One began after a severe attack of pleurisy. Others traced the beginning of the trouble clearly to scarlet fever and measles, although no history of special ocular inflammation at the time could be obtained, and there was no evidence of keratitis. A woman of 40 came out of two years of invalidism with astigmia of 5 and 6 D. incapable of perfect correction, although there was no evidence that her sight had been defective before that. A poorly nourished girl rapidly developed conical cornea at puberty.

This importance of generally depressed nutrition in causing keratoconus is also illustrated in the fact that most cases are bilateral, thirty-one out of forty-eight. Of the remaining seventeen, one patient had lost one eye probably from conical cornea, and the operations done for it; and in six others there was evidence that the eye, which was not counted as having conical cornea, had at one time been disturbed by the same process, but had soon regained a nutritive equilibrium retaining good vision with moderate astigmia. That is, of forty-eight cases, thirty-eight were really in the beginning instances of binocular disturbance. The importance of impairment of general nutrition in causing keratoconus seems certain.

THE MECHANISM OF KERATOCONUS

Plaut[2] reported a case of conical cornea in which the affected part of the cornea was greatly thickened, and experimentally produced in rabbits a condition resem-

2. Plaut: Klin. Monatsbl. f. Augenh., February, 1900, p. 65.

bling conical cornea in which the membrane was greatly thickened. But the great mass of evidence indicates that thinning as well as softening of the tissue occurs so that it yields before normal or even diminished intra-ocular pressure.

Salzmann[3] reports examination of a keratoconic eye in which the thinnest part of the cornea, the apex of the cone, was 0.17 mm. in thickness, one-fourth or one-fifth the normal. He cites seven other reports of similar observations in which the apex of the cornea was found thinned to one-half or one-third the normal thickness. The ease with which the highly conical cornea can be indented by the lid margin, or altered in shape by lid pressure, makes it certain that the essential change in keratoconus includes thinning of the cornea with distention.

The actual protrusion in a case of conical cornea is generally much less than commonly occurs in posterior myopia. A protrusion of 1 mm. makes a very marked conicity. My notes show no case of protrusion of the cornea of over 2 mm. Yet that lengthening of the anteroposterior axis of the eyeball would cause only 6 D. of myopia. Anterior myopia is to no important extent axial. It is preeminently myopia of curvature (Fig. 1). The protrusion rarely involves the whole cornea. It often affects little more than one-half the area. In a few chronic cases of gradual development the whole cornea is altered in shape, and in some others the protrusion blends so gradually into the conicity of the cornea that the appearance suggests involvement of the whole cornea in the pathologic distention. Often the pathologic bulging is distinctly marked off from the normal corneal curve. Generally the apex of the cone is situated below and to the nasal side of the center of the cornea, often as much as 2 or 3 mm. from the center. I have never noted it exactly at the center. This eccentric bulging gives the high astigmia that marks all cases of keratoconus.

3. Salzmann: Arch. f. Ophth. (Graefe's), **67**, 1.

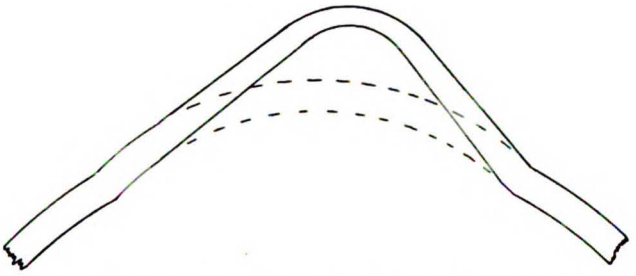

Fig. 1.—Diagram representing bulging of conical cornea of 2 mm. with thinning of apex.

Fig. 2.—Corneal curvature modified by lid pressure produced by traction with tip of finger near the outer canthus.

Fig. 3.—Face of patient at rest. Note wrinkles about the eye of a man of 27.

Fig. 4.—Face distorted by strong contraction of orbicularis to improve vision by lid pressure on the cornea.

The center of the protrusion may present a fairly regular spherical curve, although usually it does not. But this center is so small that the light admitted through it is a very small proportion of that entering the pupil; and it may be so enormously myopic as to be far beyond the limits of correcting glasses or any possible usefulness in vision.

ASTIGMIA

Generally the apex of the cone does not come in front of the contracted pupil. The patient is compelled to see through one side of the cone and so has to contend with high astigmia. In the direction of a line radiating from the apex of the cone, the cornea is relatively or actually flattened. In a direction at right angles to this, the tangent of a circle having the apex as a center, the curvature is progressively increased as the apex is approached. Thus arises the high regular astigmia which with irregular astigmia characterizes the refraction of conical cornea. Of forty-five cases of keratoconus given optical correction, all had marked astigmia. The amounts of regular astigmia noted in these seventy-nine keratoconic eyes are given in the accompanying table.

AMOUNTS OF REGULAR ASTIGMIA IN SEVENTY-NINE KERATOCONIC EYES

Diopters of Astigmia	Number of Eyes
From 1 to 2	1
From 2 to 3	1
From 3 to 4	11
From 4 to 5	13
From 5 to 6	24
From 6 to 7	10
From 7 to 8	7
From 8 to 9	7
9	1
10	2
14	1
21	1
Total	79

Almost as characteristic as the high degree of regular astigmia is its variability from day to day, or test to test, under variations of light; and its tendency to change from year to year, in amount, and to a less

degree in direction. The astigmia as indicated by the glass preferred also varies greatly from the curvatures of the corneal meridians as shown by the ophthalmometer. Take the following case under observation thirteen years, while passing through college, medical school and hospital internship:

CASE 1.—L. G. W., aged 19 when first seen.

Nov. 6, 1903:

Right, Ophthalmometer + 4. cy. axis 120°
Lens — 3.50 Sph. ⌒ + 2.50 cy. axis 153°
Left, Ophthalmometer + 3.50 cy. axis 60°
Lens — 5. ⌒ + 3. cy. axis 10°

Dec. 26, 1908:

Right, Ophthalmometer + 4. cy. axis 140°
Lens — 3.25 ⌒ + 0.75 cy. axis 160°
Left, Ophthalmometer + 4. cy. axis 65°
Lens — 4.50 + 2. cy. axis 5°

Aug. 29, 1910:

Right, Ophthalmometer — 4.50 cy. axis 130°
Lens — 3.75 ⌒ + 3. cy. axis 170°
Left, Ophthalmometer + 4.45 cy. axis 60°
Lens — 4.50 ⌒ + 2. cy axis 5°

Feb. 28, 1914: This patient's refraction changed with the use of pilocarpin.

Right, without: — 3.75 cy. axis 86° = 5/10 partly
With pilocarpin: — 0.50 ⌒ — 3.50 cy. axis 110° = 5/5 mostly
Left without: — 5.50 ⌒ + 3.75 axis 5° = 5/5 partly
With pilocarpin: —5.50 ⌒ + 3.75 axis 5° = 5/5 mostly

March 2, 1914: Without pilocarpin:

Right — 0.50 ⌒ — 3. cy. axis 95° = 5/12 partly
Left — 5.50 ⌒ + 3.50 cy. axis 5° = 5/5 mostly

March 6, 1914:

Right — 2.75 cy. axis 95° = 5/5 mostly
Left — 5. ⌒ + 3.25 cy. axis 180° = 5/5 mostly.

July 3, 1916: The eyes now became less variable and still required:

Right — 3. cy. axis 87°
Left — 5. cy. ⌒ + 3.25 cy. axis 180°

For reasons given below, the close correction of such astigmia, at least to the extent of securing the best vision, and the keeping of the correcting glasses closely adapted to the eye, is of great practical importance.

EFFECTS OF LID PRESSURE

The effort to get the best vision is instinctive and persistent. It prompts the patient to try all sorts of experiments. The presbyope goes through the "trombone performance." The patient with uncorrected astigmia twists his head until he looks through his glasses obliquely in a way that makes them give the greatest assistance; the myope with too weak glasses looks through them obliquely to increase their strength, getting all the theoretical disadvantages of a full correction with some very important practical ones added. The myope without glasses partly closes his eyes, to lessen the circles of diffusion, thereby getting from the Greeks the name for his defect, which it retains today.

The patient with keratoconus, especially if it be largely myopic, is likely to resort to similar pressure to overcome, so far as possible, the disadvantageous curvature of the cornea. The long recognized "nipping" of the lids not only narrows circles of diffusion; it can also alter to a marked degree the corneal curvature, especially when the cornea is thinned and softened.

CASE 2.—Miss G. E. F., aged 34, could change the curvature of her cornea equivalent to 1 D. change of refraction without closing the lids enough to prevent watching the ophthalmometric images. The effect was always to diminish the curvature in the horizontal meridian and increase it in the vertical meridian. The latter, however, was neutralized by the stenopaic effect of narrowing the palpebral fissure. Her cylinders, which were

Right — 6.50 axis 120°
Left —11. axis 120°

varied as much as 1 D. either way in frequently repeated tests, and as much as 15 degrees in the directions of the meridians chosen. The ophthalmometer reading indicated

Right —5. cy. axis 80°
Left —5.50 cy. axis 155°

at repeated observations.

Observations on other patients show that the usual effect of "nipping" the lids is that given above: to lessen the curvature in the direction of the palpebral fissure and increase it at right angles thereto, dimin-

ishing the myopia in the former and lessening the effect in the latter meridian. An unusual way of making such changes in the corneal curvatures is illustrated in the following case:

CASE 3.—A. M. H., a schoolgirl, aged 12, had the following vision and refraction:

Right $\frac{5}{27}$ partly $-4.$ sph. $\supset +1.$ cy axis $95° = \frac{4}{4}$
Left $\frac{1}{100}$ and with $-9.$ sph. $= \frac{1}{20}$

While not wearing glasses, she found she could bring up her vision so that she could see the blackboard by dragging on the outer canthus and upper lid of her right eye with the tip of a finger, as shown in Figure 2. In this way she brought up vision to 4/7.5 partly.

This patient was seen regularly at intervals of from twelve to thirty months, and by the time she was 19 her myopia had increased 2 diopters.

CASE 4.—K. F., man, aged 27, in 1903, had scarlet fever when 14, and was sick for a year afterward, from which period he dated his poor sight, which had gradually increased. He had high conical cornea. His corneas were almost clear, showing a very slight general haze, and the ocular fundus was normal. The ophthalmometer showed:

Right $+7.$ cy. axis $130°$
Left $+10.$ cy. axis $80°$

The following gave him the best vision:

Right $+5.$ sph. $\supset -8.$ cy. axis $75° = \frac{5}{5}$
Left $-6.$ cy. axis $80° = \frac{5}{40}$

He was not wearing glasses, having been unable to get any that helped him. Vision with either eye without lid tension was probably about $\frac{1}{100}$, but the instant his attention was directed to the letters, a curious distortion of the whole face occurred with lid tension that brought his vision up to $\frac{5}{20}$. The change is shown in Figures 3 and 4.

The apex of the cone was below the edge of the 4 mm. pupil in each eye, and the lower part of each pupil (half for the right and two-thirds for the left) was myopic. The upper part of each pupil was hyperopic. In his attempts to see (Fig. 4), the lower lid was pressed hard against the eyeball and stretched so that the edge of the lid went straight across the middle of the pupil. He was given correcting lenses, and warned of the importance of getting entirely out of the habit of compressing the eyeball with the lids.

At the end of eleven months he returned with corrected vision right $\frac{5}{5}$ and left $\frac{5}{40}$, and his corneas entirely clear. He had given up the habit of lid tension so completely that he could reproduce the effect very imperfectly and with special effort. It was eight years before he could come again wearing

the same glasses, which now required change, and the left eye had developed high myopia with choroidal absorption.

Right + 4. sph. ⌒ — 8. cy. axis 72° = ⅙ partly
Left — 4. sph. ⌒ — 8. cy. axis 140° = 1/18

Since than I have not seen him, but have heard from him that the sight of his left eye "is almost gone, and the right eye is in bad shape."

The following case shows how even the posterior distention of axial myopia may not save the cornea from protrusion, or how the malnutrition and softening of the coats may involve both the posterior and the anterior pole.

CASE 5.—B. G. M., girl, aged 15, a bright student devoted to music, and strenuous in all she undertook, but anemic and "nervous," was brought to me for her eyes, Sept. 21, 1916. She had been wearing glasses since 8 years old, and now required them strengthened to:

Right + 0.75 sph. ⌒ — 1.25 cy. axis 175° = ¼ plus
Left + 0.62 sph. ⌒ — 1.25 cy. axis 5° = ¼ plus

There was little choroidal disturbance, but the optic disks were red and slightly hazy. The ophthalmoscopic readings were:

Right + 1.50 cy. axis 90° added to 43.5 D.
Left + 2. cy. axis 90° added to 44. D.

There was some conjunctival hyperemia, burning and itching, in addition to the headache for which she came. She was under my care for four months and left with all symptoms relieved. June 19, 1912, she returned, having been under good professional care in Chicago and elsewhere. The strenuous educational process had been supplemented by social engagements. The ophthalmometer now showed:

Right + 1.25 cy. axis 90° added to 44. D.
Left + 10. cy. axis 100° added to 49. D.

The left eye was clearly suffering from conical cornea. A — 10. cy. axis 10° gave it vision of 1/50.

She next came, Sept. 14, 1912. The ophthalmometer showed:

Left + 9.50 cy. added to 52. D.
Lens, Left + 3. sph. ⌒ — 16. cy. axis 145° = 1/15 partly.

No marked change in the right eye, but

Right + 0.62 sph. ⌒ — 1.12 cy. axis 168° = ⅛ partly.

Oct. 4, 1916, she came once more broken down in health, highly anemic, listless, hopeless, and almost blind. She had been under treatment by various oculists in other cities. There was a history of operations, corneal ulcers, and finally enucleation of the left eye. Vision in the right was less than 1/100; she was wearing a compress on it much of the time, and

using eserin. It had suffered from an "ulcer" last winter. The ophthalmometer showed but little regular astigmia with a corneal curve of over 70 D., corneal radius about 5 mm. A —30 D. sph. lens gave vision of 9/100. There was a nebula near the center of the cornea. The use of a bandage was almost stopped, being occasionally used for pain, the eserin was continued, and everything done to build up the general health of the patient.

Jan. 11, 1917, the eye felt better, and the iris was of good color and structure. The ophthalmometer showed:

+6 to +8 cy. axis 80°, added to 60 or 65 D.

Hot bathing of the eye has been substituted entirely for the bandage. The eye feels worst in the morning. The patient is to keep the head elevated.

CASES 6 and 7.—F. B., aged 18, and his sister, aged 17, both came for conical cornea, with the history that every member of the family wore glasses. They were both students, the brother then in college. In all three eyes the area of protrusion was comparatively small and situated below the center. The brother's correction and vision was:

Right +4.50 sph. ⌒ —7. cy. axis 90° = 1/4 partly
Left +4. sph. ⌒ —7. cy. axis 100° = 1/15 partly

After eight years, the patient living on a ranch, these are:

Right +3.50 sph. ⌒ —7.50 cy. axis 90° = 1/8
Left —5. sph. —7.50 cy. axis 85° = 1/12

The sister had only the right eye affected. She had vision:

Right +1. ⌒ —4. cy. axis 38° = 1/8
Left +1. sph. ⌒ —0.25 cy. axis 90°

After four years she showed:

Right +2. sph. ⌒ —5. cy. axis 55° = 1/4 partly
Left +1.75 sph. ⌒ —0.37 cy. axis 65° = 1/4 plus

These patients have taken proper care of their eyes, although living about 200 miles away so that they could be seen only at long intervals. The same is true of the following case:

CASE 8.—Jan. 29, 1910, S. J. G., aged 19, high school student, dated his poor sight to typhoid fever five years before. The eyes had not grown worse since. Both corneas were conical, the apex of each cone being fairly central, and the reflections of the rings of Placido's disk fairly circular. The centers of the pupils showed with the ophthalmoscope a myopia of 20 to 30 D., and the margins of the dilated pupils in the direction of the radii hyperopia of 15 to 20 D. The ocular fundus was normal in each eye, so far as could be determined. The patient was put on the regular use of pilocarpin. Lenses given:

Right —15. sph. ⌒ —8. cy. axis 20° = 1/30
Left —10. sph. ⌒ —5. cy. axis 20° = 1/30

He was seen six months later with vision: Right 1/50, left 1/12.

Sept. 12, 1914:

Right $+3.\frown -6.$ cy. axis $25° = 5/15$ partly
Left $+6.\frown -1.$ cy. axis $180° = 5/15$

Jan. 26, 1917: He comes several hundred miles to be seen once.

Right $+4.\frown -6.$ cy. axis $25° = 5/15$

Left shows keratitis, hazy with facet at apex of cornea 1 mm. in diameter, no staining with fluorescein. He has completed school and college, and is now teaching school. He has used the pilocarpin continuously, but has not used his glasses sometimes during summer vacations.

In this paper no attempt has been made to give any complete account of conical cornea or complete histories of any of the forty-eight cases on which it is based. All of these cases have shown marked conicity of the cornea in some projection, asymmetrical except in Case 8. None of them has shown any marked choroidal disease or atrophic crescent except Cases 1 and 2. In the series of cases from which the forty-eight were taken, there were thirty-nine cases of myopia with 3 D. or over of astigmia, and seventeen cases of hyperopia, with astigmia of 3 D. or over. None of the forty-eight patients, except Case 5, was subjected to operation, and all the worst cases have been included above.

The points to which attention is directed are:

1. Keratoconus arises from yielding of the cornea to intra-ocular pressure during a period of impaired nutrition, commonly due to general disease.

2. It gives rise to curvature ametropia, about which the ophthalmometer and the shadow test give little information of value for the selection of glasses.

3. The subjective tests with lenses give widely variable results; and a decision as to the best lens is reached only after many trials, under varied conditions influencing the pupil.

4. It is extremely important that glasses should give the best vision under the conditions under which they will be used without lid pressure, which is to be carefully avoided.

5. The treatment should include:

Every effort to build up and sustain nutrition.

The continuous use of a miotic in the worst cases, usually pilocarpin.

The avoidance of softening the cornea by any form of bandages.

Under proper care operative treatment will rarely be needed.

A NEW OPERATIVE METHOD FOR THE RELIEF OF ADVANCED CASES OF KERATOCONUS

WITH REPORT OF TWO CASES

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ST. LOUIS

Conical cornea was observed by Demours as early as 1747, and has been described by many authors since that time. There has never been any satisfactory explanation as to its production or cause. It is known that it is more frequent in females than in males, and is generally first noticed between the twelfth and fourteenth years of life. The probability is that some disturbance of internal secretion is the most potent causative factor.

Many operations have been devised for the relief of this condition, none of which has given entirely satisfactory results. The earlier surgical attempts to relieve conical cornea were bent toward changing the refraction of the eye behind the cornea rather than to alter the cornea itself. In 1817, Sir William Adams advised breaking up of the crystalline lens by needling. Tyrrell suggested a still different procedure, that of performing a peripheral iridectomy, on the theory of there being less deformity toward the margin of the cornea than in the center, and consequently a peripheral pupil would be of advantage. Crichett modified this a little later by permitting the iris to prolapse, and tying a ligature around it, thus distorting the pupil. Frequent puncturing of the periphery of the cornea has also been resorted to for many years. Von Graefe suggested the greatest advance for the relief of this condition by proposing that the epithelial coat of the

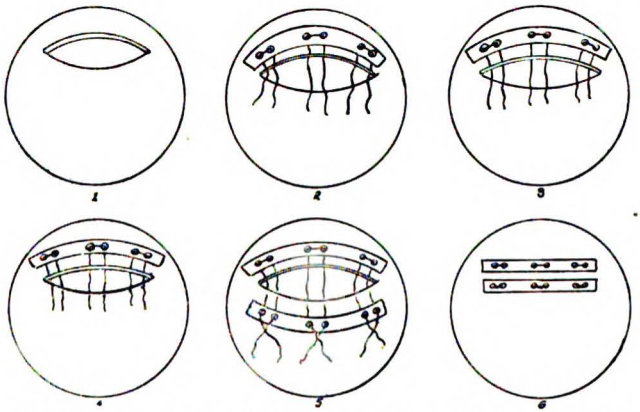
apex be shaved off and then cauterized with silver nitrate; finally the floor of the resulting ulcer was punctured, the object being to try to bring about scar contraction with resulting flattening. Bowman had previously excised a disk of corneal tissue from the apex with a trephine, but without any great success. A number of cases in which he excised an elliptical piece were reported by Bader. To prevent prolapse of the iris he passed horse-hair sutures through the cornea, previous to making the excision, afterward tying them over the edges of the wound. This operation has probably been the one most frequently advocated of those procedures which we might term purely surgical. The use of the electric cautery, or frequent punctures of the apex with a needle, being more simple and less dangerous, are the methods most often used today, when anything at all is advised. Recently a case of marked flattening has been reported from the use of the high frequency current applied to the apex of the cone.

The great objection to excision of the apex of the cone, or of a peripheral part of the cornea, has been the almost invariable prolapse of the iris into the wound with the sudden gush of aqueous, with its resulting infection, or irritation from an extensive leukoma adherens. The method which I am proposing is an excision of the corneal tissue in such a manner as to avoid iris prolapse. This I have succeeded in doing, and with fairly good visual results.

As the cornea near the apex of the cone is so thin as to preclude the probability of a successful dissection, I have elected to excise a segment near the periphery, as best suited to the method. The idea is to excise a segment of the cornea, including as nearly as possible its entire thickness, without puncturing Descemet's membrane, and then suture the wound. Necessarily there as an unusual tension on the stitches which would cause them to tear through almost immediately unless some extraordinary means were employed. This difficulty is met by tying the sutures

over thin gold plates, on the same principle as that employed in bringing together the gap in cleft palate. This method of suturing was described by me at the 1915 meeting of the Academy of Ophthalmology and Oto-Laryngology.

Gold strips 0.005 mm. in thickness and 1 mm. wide are used, with holes 1 mm. apart, and just large enough to permit the passage of the smallest curved eye needles. The length is cut according to the size of the wound to be sutured. An elliptic incision is made in the cornea near the limbus, with a small, sharp



Details of operation: 1. Elliptic incision of cornea near margin. 2. Gold strip with double-armed sutures already in place ready to be passed through peripheral margin of cut. 3. Sutures passed through peripheral margin of wound. 4. Sutures passed through central margin of wound. 5. Sutures passed through second gold strip. 6. Gold strips in place after completion of operation.

scalpel. The object is to go as deeply as possible without penetrating Descemet's membrane. The length is about 8 mm. and the width 3 mm.

The apex of the part to be excised is then picked up with a tiny sharp hook, and the resection carefully begun. The most important part of the dissection is to get a good start, as the apex of the ellipse can then be grasped with fixation forceps, and by putting the flap on the stretch, the layers of the cornea can easily be separated. This separation can be readily accom-

plished by following, with the point of the knife, the white line at the point of attachment of the flap to the cornea. By keeping the blade of the knife flat against the corneal surface, using only the tip, and alternately stretching and then separating, there is not much danger of entering the anterior chamber. Double armed sutures are then passed around the first gold strip through the small openings, then in turn through the central flap of the wound, through the limbus flap, finally through the openings of the second gold strip placed on the opposite side, and then tied over this second strip with one tie. The stitches are then alternately tied more firmly together until the wound is approximated. Linen threads are an advantage on account of their strength. The amount of tension which this form of suturing will withstand seems to be almost unlimited. If there is no infection they will not pull through, provided the technic is good. The technic is delicate and difficult, but can be acquired by any experienced operator with much practice and patience. It is essential that the bored holes shall be polished in order that the stitches may slide easily. I believe it to be an advantage purposely to puncture the anterior chamber at the periphery, away from the wound, after the sutures are all in place, and just before tightening and closing the wound, in order to permit easier approximation of the wound and facilitate healing with lessened tension. The plates are left in position, with comparatively little irritation, for eight or nine days. They are easily removed by simply cutting the loop over each gold plate, when the thread readily pulls through.

The following is a brief history of the patient on whose eyes this operation was performed:

M. S., girl, aged 15, first came to the Jewish Hospital dispensary for treatment, April 30, 1915. She had a very pronounced case of conical cornea in both eyes with ability to count figures at 7 feet with the right eye and $^{18}/_{100}$ with the left. Her refraction was carefully determined, but no improve-

ment of any value could be secured, minus lenses making letters clearer, but not bringing up the visual acuity by measurement. She was put on epinephrin chlorid, 1:1,000, three times a day, as we had seemingly had some good results in mild cases of keratoconus and keratectasia with this treatment, as recommended by Pontius. It did not help, however, and, Feb. 23, 1916, she was admitted to the Jewish Hospital, and the right eye operated on two days later. March 6, the stitches and plates were removed, and, March 12, patient discharged from the hospital. There was considerable photophobia and conjunctival redness for many weeks after the operation, but these slowly subsided.

Sept. 12, 1916, visual acuity had reached $^{18}/_{100}$ in the right and fallen to fingers at 7 feet in the left. As the operated eye was considerably better than the left, or unoperated one, the patient begged to have the same thing done for the left.

She was admitted to the hospital, Oct. 9, 1916, and operated on, October 11. A segment was excised from the lower quadrant, as in the right eye. March 7, the stitches and plates were removed, and the patient discharged from the hospital, Oct. 29, 1916. November 28, the visual acuity was $^{18}/_{100}$ in the right and $^{18}/_{200}$ in the left. The visual acuity in the right has remained stationary, and in the left, March 8, 1917, had improved to $^{18}/_{75}$.

A peculiar phenomenon about this case is that while the patient asserts that a —4.0 D. S. to a —7.0 D. S. lens will improve each eye very much, she cannot be made to read farther. There is a linear scar on each cornea in the lower quadrant which can readily be demonstrated but which does not disfigure the patient and would not be noticed by a casual observer. I believe that there will be still further improvement with time, but if this should not develop, I shall advise excision of another segment. The patient is willing.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. JACKSON AND WIENER

DR. SAMUEL D. RISLEY, Philadelphia: I have been deeply interested in Dr. Jackson's paper, and am entirely in accord with his conclusions. Conical cornea, produced, as it is, by a distention or stretching of that membrane, is closely allied, when viewed in its broader phases, to the same etiologic factors which are potent in the genesis of the myopic eye, and to the intricate questions underlying uveal disease and its sequelae; for example, the impaired nutrition of the eye leading to the affections of the avascular tissues—the

vitreous body, crystalline lens and the cornea; and also to the important and illy understood problems of increased tension of the globe. In the usually entertained concept of axial myopia I believe we have been too prone to fix our attention solely on the pathologic changes at the posterior pole. The posterior staphylomas, the macular atrophies, etc., because of their grave significance, have claimed our attention to the exclusion of other features frequently manifested in increasing or changing refraction of the dioptric system. In a relatively large percentage of young people, especially young children, these changes in refraction occur without notable evidence of disease at the posterior pole. Given a case of congenital anomaly of refraction, with or without, but usually with, some associated abnormality of binocular balance, the act of vision cannot be exercised without abnormal effort or strain. Under this strain the vascular uveal tract becomes unduly congested, the intra-ocular tension rises, the circulation of the nutritive fluids is impeded and the nutrition of the globe impaired. The relatively soft or tender sclera of childhood yields to the increased intra-ocular pressure and stretches or distends, whereas the tough and therefore resisting sclerotic of middle life fails to give way and we have the phenomena of so-called glaucoma.

The distention of the globe, however, in youth is by no means always at the posterior pole or along the antero-posterior axis of the globe, but in many instances in all of its diameters. The eyeball is enlarged. This condition manifests itself not only by impaired distant vision due to increasing refraction, but by a significant group of objective signs. The ophthalmoscope reveals a fluffy eyeground throughout the ophthalmoscopic field; the veins are full, the normal stippling is lost, there may or may not be a beginning absorption crescent embracing the temporal margin of the nerve, or there may be fine granular changes in the macular region. Externally the anterior sclera is bluish white, obviously from thinning or stretching of that membrane; the anterior ciliary vessels are engorged. In addition to these, another very significant symptom will often be found if sought for. The plane skiascopic mirror will discover in the illuminated, dilated pupil a faint granular ring, usually concentric with the pupillary margin, or a faint granular cloud, circular or oval, apparently covering the corneal pole. Occasionally I have been able with oblique light and the loupe to discover in these cases a faint gray area corresponding to the skiascopic findings situated apparently in the membrane of Descemet. I have seen this so frequently associated with cases of increasing refraction with severe asthenopia and weak eyes that I have come to look for it as an important factor in diagnosis of anterior myopia. Reasoning *a priori*, changes in the membrane of Descemet are to be anticipated in the presence of the existing uveal disease, which has been

described as the underlying factor in these cases of increasing refraction, since that membrane is, from the point of view of the embryologist, an extension forward of the uveal tract.

By way of illustration, in March, 1913, B. P., a boy aged 7, came for a phlyctenular chain along the limbus of both corneas. He had frequent styes, stuffed nostrils and enlarged tonsils. These conditions rapidly improved under treatment, but recurrences were frequent for two years or more. He received in April, 1913, + 2.00 sph. for each eye, V = 6/V. This glass was worn with comfort and normal vision until March, 1917, when he returned complaining of headache, and that he could not see clearly with his glasses. There were photophobia, red lid margins, marked thinning of the anterior sclera, and the anterior ciliary vessels markedly engorged. The fundus was fluffy throughout, and all details blurred. The skiascope showed the corneal ring. With his glass V = 6/XII. He had become interested in a series of boy's books, which fascinated him, and had been permitted for many weeks to read almost continuously while out of school to the neglect of exercise in the open, even far into the night; indeed, until the pain in eyes and head forced him to desist. A strong cycloplegic was prescribed, to be used three times daily in each eye, smoked protecting glasses to be worn in addition to his correcting glasses. The pain disappeared, the fundus conditions improved, the dilated ciliaries resumed their normal size and the Descemet ring grew faint and finally disappeared, but instead of + 2.00 sph. which he had worn with comfort and normal vision for two years, he now required + 2.00 ⊃ + 1.00 cyl. axis 90 degrees, with which his vision rose after three weeks' use of the cycloplegic to 6/V. At present there has been no return of the syndrome, but he will not return to his school work or be allowed to read continuously until the September semester. This case is reported simply as an illustration of a large group of young persons presenting similar conditions compelling the interruption of their work at school.

If these deductions are to be accepted, we need find no difficulty in regarding the resulting impaired nutrition of the cornea as an important factor in the changed curvature of the cornea, the shifting meridians, etc., which we so often are compelled to observe, or that in extreme cases the result may be conical cornea, or what I have frequently seen—a wavelike curve of that membrane instead of a demonstrable conelike distention forward. I quite agree with Dr. Jackson that these conditions are prone to occur in people in impaired health, or in fat, flabby patients without resisting power, or in the underfed or sick.

Dr. E. C. Ellett, Memphis, Tenn.: I regret that I have had no experience with Dr. Wiener's treatment of conical

cornea, and must therefore limit what I have to say to the technic of this method of corneal suture, based on animal experiment exclusively. This experience has impressed me with the difficulties of the procedure, to which I well refer under a number of heads.

1. Illumination: It is essential to have perfect illumination, preferably by a photophore or some other form of portable electric light.

2. It was not possible for me to do this work without the magnification secured by some form of binocular loupe.

3. The knife, preferably a scalpel, must be perfect.

4. It is necessary that the two cuts outlining the portion to be removed should penetrate the cornea to the same depth. This requires care and experience.

5. The removal of the portion included between the incisions is not especially difficult if the above precautions are observed.

6. The difficulty of handling several sutures and two rather long plates without tangling things up is very great, and if one suture breaks in tying, it will greatly try the patience of all concerned.

7. The last objection can be to some extent overcome by using a short plate, 3 mm. in length, drilled with two holes to accommodate a single double armed suture.

8. It would be still simpler to dispense with the plates, and pass each needle in and out along one edge of the incision, having the loop cross the incision at one end and the two free ends tied across the other end. For this purpose, as well as for use with the suture plates, the needles and thread suggested by Kalt for corneal suture are most satisfactory. The needles are round, not cutting, and make a wound which does not so readily cut out, although more difficult to push through the tissues than a cutting point.

9. By slightly undermining the edges approximation is made easier; but I am not sure that it would not make infection more probable.

One is surprised, in doing this and other work involving dissection or suturing of the cornea, to note the toughness of the membrane. It seems quite as capable of holding sutures as is the sclera.

Dr. William H. Wilder, Chicago: The question of the causation of keratoconus is an interesting one. It seems to me that the idea of a lack in general nutrition is not a sufficient explanation, for this condition of the eye is so infrequent, even in persons whose general nutrition is impaired, and is seen in persons apparently in good health.

I have always entertained the thought that there must be some anatomic peculiarity, possibly congenital, to account

for the progressive thinning and subsequent bulging of the cornea. That there is an actual anatomic change even at an early stage of the process is evidenced by the fact that the nerves seem to have suffered and there is a distinct lack of sensitiveness in the most prominent part of the cone. Even before there is much bulging, this may be demonstrated by touching the tip of the conus with a probe, when it will be found to be quite insensitive. Later when the cornea becomes thinner, one may observe a pulsation of the tip, shown by a slight movement of the reflected images of the mires of the ophthalmometer as pointed out by Axenfeld and others.

In advanced cases, with vision greatly impaired, it seems to me we are justified in resorting to operative procedures in an attempt to check the bulging or to improve the sight.

A method I have used in several cases with benefit is the following: With a galvanocautery tip heated to a cherry red, four small incisions are made in the cornea from the limbus to within 2 or 3 mm. of the tip of the conus, at points on the cornea corresponding to 12, 3, 6, and 9 on a clock dial. These burns should extend through Bowman's membrane and widen out slightly as they approach the limbus. The resulting contracting scars have a tendency to flatten out the central portion of the cornea which remains clear. A somewhat similar method has been proposed by Dr. Posey, who makes only one such radiating incision. Care must be taken not to penetrate the cornea. In the patients treated there was distinct improvement of vision, and the method does not interfere with any subsequent procedure that one may have to practice.

Dr. Edmond E. Blaauw, Buffalo: How is it that Dr. Jackson in a short time sees forty-five cases, and in other regions, at least in Buffalo, I have not been able to see forty-five cases in twenty years? There may be something in the region in which we live. In Germany, too. How can you make a diagnosis with this slight amount of astigmatism? I would like to ask Dr. Jackson how often he found the hemosiderin ring? We should always keep in mind that we have in many instances a self-limiting disease. I have seen many cases stop without apparent reason and nothing done in the way of operation. In the cases of keratoconus we have most probably a form of hyaline infiltration of the substantia propria. It occurs to me that we have to deal with a condition similar to nodular opacity of the cornea, often overlooked because it needs a great deal of research. I have seen fluorescein sometimes bring out opacities in the cornea before the binocular loupe. Uhthoff has recorded the microscopic findings in conical cornea with hyaline infiltration.

Now, if it is so important that lid pressure should be eliminated, are we not warranted in doing a canthoplasty? We can always resort to stitching up the defect. I have seen the pulsation. That is exceptional. Dr. Park Lewis showed me such a case. In my opinion it is rare. When must we operate? That is the great thing for us to know.

Dr. Hunter H. Turner, Pittsburgh: Drs. Jackson and Risley have both mentioned features leading up to a certain element which in my experience has been of paramount importance in the production of this condition. Skillern, who is recognized as an authority on diseases of the accessory nasal sinuses, says that infection of these cavities following the diseases mentioned by Dr. Jackson is very common, much more so than is ordinarily believed, and that the primary infection is followed by a secondary infection of chronic character which persists indefinitely. He claims that the resulting tissue hyperplasia constitutes a point of lessened resistance which is affected by constitutional toxemias, and that the local effect is manifested by a turgescence of the nasal tissues. I wish to go on record before this section as stating that this nasal turgescence is always associated with a turgescence of the vessels of the retina and conjunctiva, with a tendency to increase in intra-ocular tension. I have had quite a number of cases of conical cornea in the early stages under treatment for years, which have remained stationary, the principal treatment being directed to the controlling of the sinus condition by competent rhinologists, and care in the matter of diet. This tendency to congestion and hypertension, in my experience, is present in every case of progressive change in the ocular dioptrics toward the myopic side.

Dr. L. Webster Fox, Philadelphia: I consider conical cornea one of the most mysterious of all eye diseases. You who have read the classical work of Nottingham on conical cornea—a book of 200 pages—know that he has thoroughly covered the ground of operations devised and abandoned. Many of these operations were autoplasty, or various cuts and incisions made in the cornea to try to reduce the nipple-shaped projection, if possible. All of these experiments have been carried out by me—varied incisions and sutures. A suture through the cornea is not half as disastrous as it appears, for the cornea has great resisting forces. You will remember the old Critchett operation for staphyloma—incision and suture—from which no deleterious results followed.

I am greatly interested in this operation of Dr. Wiener's, but I have come back to the English method—the application by the cautery. At present I have two patients in the hospital who have been operated on within the last ten days, and in both cases I used this method. One operation may

not be sufficient; in that case a second one may be carried out. As I have said above, Morton's method and also that of Critchett have been my favorites. In following the cautery method I have obtained a deep saucer depression, and the final act is to pierce the center so that the aqueous will escape; in this way I have obtained good results. This mode of procedure makes a central scar. After performing the operation as outlined, an iridectomy is the next step, and after all inflammation has been allayed, a tattooing of the scar. A workable eye is thus obtained. Under the best conditions the chances are that one operation will not bring about the desired results. I have repeated the operation three times in one case, and finally, from about $\frac{5}{200}$ vision, got $\frac{20}{50}$.

Regarding the pathology as expressed by Drs. Jackson and Risley, we may agree in general with their ideas, but there are some cases in which it is difficult to believe that the pathology as outlined by my confrères has any bearing on some of these cases. One case in particular is that of a young man who was formerly a brakeman on the Pennsylvania railroad, in the passenger service, who had passed his visual examinations. He is a young man of perfect physique, 5 feet 11 inches tall. Conical cornea developed in one eye, and in three months the cornea projected 3 mm. The same defect is becoming apparent in the fellow eye, which requires a high cylinder glass to bring it up to the required vision. He is still undergoing treatment for his right eye.

Fario, an Italian surgeon, cut out a small portion of the cornea three different times, leaving a hole through which the aqueous humor gradually drained. The first excision was practiced at the upper part of the cornea, the second at the lower, and the third at the upper and inner. Each piece of the cornea removed was triangular in shape, the apex being near the scleral margin.

Dr. Wiener's method is worthy of our consideration, but I am afraid his difficult surgical technic will not take the place of the actual cautery. However, I shall give it a trial.

Dr. Edgar S. Thomson, New York: I should like to say a word in connection with Dr. Wiener's operation. I have done six or seven conical cornea operations, excising the summit of the cone. It seems to me the results were better than cauterization because of less scarring. In one instance I did not excise a large piece of the cone, but the sutures pulled through on the third day. They were deep. There seemed to be an unusual amount of traction on the cornea. I had difficulty in keeping the lips of the wound together. The eye was finally practically lost. I do not know that that is a very common accident. It seems to me Dr. Wiener's operation would keep the lips of the wound in close apposition and guard against an accident of that sort.

Dr. Edward Jackson, Denver: With reference to clouding of the cornea, I want to express my agreement with Dr. Risley's observation that a certain clouding of the cornea may be checked temporarily, and is rather frequent at an early stage. We know that the worst cases ultimately show some permanent clouding; but this early clouding may be entirely recovered from. I have not convinced myself that it is always located in, or confined to, the membrane of Descemet.

With reference to an anatomic peculiarity of the individual's cornea, I note that two of the cases I have given here were in brother and sister. They are the only two cases that I can recall occurring in the same family; but as far as this instance would go, it might point to a family peculiarity of the cornea. We certainly have myopia running in families to a striking extent; but it is quite unusual for conical cornea to affect more than one member of a family.

As to the number of cases, my forty-eight cases have not been seen in twenty-one years, but in thirty-five years. They include practically all the cases that I have had under observation for any length of time in private practice. The cases are not very common, and one case may be seen by a number of men—is apt to be so—and under observation for long periods of time, so that they seem to have an importance which the actual numbers do not support.

The pulsation of the conical cornea seen with the ophthalmometer I have occasionally noted; and I had one patient who spontaneously called attention to the pulsation of his vision. When the vision was about as well corrected as it could be, any active exertion gave him the pulsation of distinctness of vision. He could count his pulse by the changes in the distinctness of vision.

Infection of the nasal sinuses was not looked for in a good many of the earlier cases of the series. It has been present in some. My figures would not indicate how often. I am sure it is a condition worth careful looking for.

The lid pressure to which I called attention is not due to the deformity of the lids, or to the weight of the lids on the eyeball or from a narrow palpebral fissure. It is the pressure of a voluntary effort to change the refraction by lid pressure. The girl with myopia who could bring her vision right up did not do it by movements of the lids, but by placing the finger on the lower lids and making traction. In the case of the man, he made similar pressure on the cornea, but by a peculiar grimace; the lid was tensely drawn. The illustration was not as striking as his movements when first seen. As soon as he had put on glasses the pressure on the cornea stopped. The pressure then made his vision worse and he stopped it. That is the object of giving glasses, so that they will see best without additional pressure on the eyeball.

DR. MEYER WIENER, St. Louis: I believe illumination is very important. I use ordinary north window daylight and no modifications, but I still retain good accommodation. There is no question about the difficulty of handling the sutures. They are complex and are apt to get twisted. That is merely a detail which can be overcome by care and patience. These plates do not cause any irritation. They lie flat against the cornea and the lids can bat over them after the bandage has been removed. In the animals on which I experimented, there was never anything put over the eye. I had no infection that I can recollect in any of the animals, and there were two or three dozen. The operation developed first by using a loop suture before the plates were used. The loop suture pulled through after a few days, never lasting long enough for the wound to heal, so that the plates were suggested.

I think emptying the anterior chamber purposely by a small puncture near the periphery is good. It gives a chance for the wound to heal and with, eventually, a better result. The advantage over cauterization is that if the operation is successful it leaves only a linear scar which can only be noticed by careful examination. I believe the operation should be performed only in extreme cases when all other palliative measures have been tried and have failed.

I have used also in a couple of cases of mild form epinephrin 1 to 1,000, as advised by Pontius, and seemingly the cases were improved greatly by its use.

I should like to see Dr. Fox, of all men, perform this operation. I think men who have highly developed technical skill—and I have never seen one with greater skill—should perform this operation. He is the type of man who can perform it and get a successful result.

INTERSTITIAL KERATITIS

WITH SPECIAL REFERENCE TO THE END-RESULT

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The cases on which this study is based have been selected from the large series of persons with hereditary syphilis and interstitial keratitis who have been so fortunate as to come under the skilful care of Drs. Abner Post and C. Morton Smith[1] during the past five years.

Interstitial keratitis is one of the most studied of eye diseases. It can boast of a most voluminous literature, in which almost every phase of the process is exhaustively discussed. From the standpoint of history, symptomatology, pathology, experimental research and therapy, it has claimed the interest of many of the best ophthalmologic minds of the present and past. That not all of its problems are yet solved is due rather to their complexity than to a failure of competent men to attack them. One most important question has, however, received but scant attention, namely, an exact knowledge of the end-result. With that feature, the study reported here is principally concerned.

A large majority of the patients were personally examined, and most of the remainder were seen by Dr. Alexander Quackenboss, chief of service of the Massachusetts Charitable Eye and Ear Infirmary. In a few instances, when neither Dr. Quackenboss nor I was on duty, other members of the staff kindly took the task on their own shoulders. To them, and especially to Dr. Quackenboss, I wish to make my grateful acknowledgment.

1. Formerly presiding over the syphilis clinic at the Boston Dispensary; more recently in charge of the newly established syphilis department of the Massachusetts General Hospital.

In the whole series, except in a very few instances, only cases were accepted in which the inflammation of the eyes had come to an end at least two years previously. Thus, the visual results obtained may be regarded as reasonably permanent, although we cannot bar out the possibility of some slight further clearing of the cornea in some cases, and in others deterioration of sight due to recurrence of the disease or to the development of myopia.

The whole number of cases observed here is ninety-six, comprising ninety-four in which the disease had occurred in both eyes, and two in which up to the time of the examination only one eye had been affected; thus, a total of 190 eyes is available.

Corneal Opacity.—By careful examination with oblique illumination, it was possible to note a corneal opacity of greater or less density in 168 eyes, while in fourteen none could be seen. This opacity varied from thick leukomas in a few cases to the finest possible haze in others.

Vascularization. — The examination for vessels in the cornea was made with the ophthalmoscope and a strong convex lens, after the pupil had been dilated as much as the condition of the eye would allow. In 186 eyes, vessels were found in 171 and were absent in fifteen. In some cases I have observed them after the lapse of many years, as has been noted by others. Whether or not avascular cases occur is a matter of some discussion. Von Graefe, Vossius, Gutmann, von Michel, Knies and Elschnig are in favor of it; while Fuchs, Saemisch, Galezowski and others believe that this is seldom or never the case. As a rule, the vessels persist for years. Hirschberg has observed them ten, fifteen and even twenty years after the disease, and believes, as does Silex, that they never disappear.

Bearing on this question, the following case is of interest:

A doctor, aged 69, who had been in very delicate health as a child, at the age of 14 had inflammation of both eyes

lasting one year, with recurrence five years later. When 38 years old he was seen by Dr. O. F. Wadsworth of Boston. At this time corneal scarring was marked and there were many patches of choroiditis in the periphery of the fundi. When I saw the patient, Feb. 19, 1917, there were diffuse corneal scars of each eye. There were marked signs of old vascularization in the corneas. There were many patches of chorioretinitis in the periphery of the fundi.

V. R. —10. sph. —2.0 cyl. axis 90 = 1/20
V. L. + 7. sph. +1.0 cyl. axis 90 = 1/10

This case, therefore, shows unmistakable evidence of vessels in the cornea fifty-five years after the original inflammation.

Baas has reported a case in which no evidence of vessels could be detected clinically, and yet the histologic examination later showed their undoubted presence.

Iris.—As to the involvement of the deeper portions of the eye, we note the presence of posterior synechiae in sixty-two eyes. My experience, however, bears out that of Hoor,[2] namely, that in frequent instances the involvement of the iris and ciliary body bears but little relationship to the severity of the corneal process. Where the keratitis itself is of but average intensity, it is sometimes only with the greatest difficulty that dilation of the pupil can be obtained, while at times with a severe keratitis dilation is accomplished by very moderate instillations of atropin.

Lens.—In four eyes, slight opacification of the lens was left behind. Axenfeld has observed peculiar circumscribed opacities occupying the situation of previous posterior synechiae, and appearing as faint, star-shaped thickenings of the capsule, which show fine pigment deposits under high magnification.

Vitreous.—In eleven eyes, vitreous opacities were noted. In thirty-eight eyes, by reason of the corneal scars, or of the permanently contracted pupil, it was impossible to make a satisfactory examination of the deeper structures.

2. Hoor: Die parenchym. Hornhautentzündung, Samml. Zwangl. Abhandl., **7**, 788.

Choroid and Retina.—We have notes on the fundus examination of 148 eyes, and of these, eighty-one showed lesions of the choroid and retina (55 per cent.) mostly in the form of disseminated rounded lesions, usually situated in the equatorial region. That in some negative cases lesions existed forward of the equator is not to be doubted. The percentage of chorioretinitis recorded here is much higher than any I have come across in the literature. It should be said that the examination was made with the greatest care, with dilated pupil, and that special attention was paid to the periphery of the fundus.

As it happened, the eyes were examined in two series. In the first, comprising eighty-eight eyes, forty-eight were positive, 54.3 per cent. In the second, of sixty eyes, thirty-three were positive, exactly 55 per cent.

In regard to the relationship of the fundus changes to the corneal disease, I feel that while in a certain number of cases of interstitial keratitis in which the involvement of the uveal tract is beyond the ordinary, fundus lesions may develop, yet in the majority of cases they appear independently of the corneal process and usually antedate it. It has been possible for us to observe in several instances the presence of such lesions in the choroid and retina in the uninvolved eye of patients with a beginning interstitial keratitis.

Vision. — With the exception of Igersheimer's article,[3] I have been able to find little or no definite information as to the visual results among the sufferers from this disease, a feature of the greatest importance. Our results are recorded in the two tables presented. All visions were taken under a mydriatic and with the appropriate correction.

Table 1, which gives the vision of 161 eyes examined, does not truly represent the exact degree of disability in each case, as the two eyes must be reckoned together, for many children possessing one good eye

3. Igersheimer: Das Schicksal von Patienten mit Keratitis parenchym, Samml. zwangl. Abhandl., 1913, **9**, No. 4.

get on practically as well in school as do those with two. In later life their disability consists in being barred from certain occupations and having a lesser factor of safety, when we take the normal chances of injury to the one good eye into account.

TABLE 1.—VISION OF 161 EYES EXAMINED

Number of Eyes	Vision
32	10/10
17	7/10
18	5/10
13	4/10
25	3/10
14	2/10
17	1/10
25	less than 1/10

In Table 2 the vision of the better eye in eighty cases is given. Taking the figures from the standpoint of education, Table 2 shows that twenty-four children would have the same chance as the normal child. Twenty-five more could study in the same classes with normal children, but would be handicapped, especially in choosing an occupation. Twenty, or possibly twenty-six, could be educated in defective eyesight classes, if any were available, or failing that would have to be referred to institutions for the blind, as would the remaining five. It should be noted, however, that as some of these children grow older and more intelligent, it may be possible to improve their

TABLE 2.—VISION OF BETTER EYE IN EIGHTY CASES

Number of Eyes	Vision
24	10/10
9	7/10
11	5/10
5	4/10
11	3/10
9	2/10
6	1/10
5	less than 1/10

vision somewhat more with glasses. On the other hand, it may be diminished by recurrence of the disease, or by the development of myopia, especially prone to occur in these cases. Myopia was noted in nineteen of our eighty cases, about 25 per cent. Igersheimer noted it forty-four times in 152 eyes, or 29 per cent.

For the purpose of comparison, Igersheimer's statistics of vision in 152 eyes are given (Table 3). A glance shows a considerably graver situation among these cases than do our figures. Of our 161 eyes, 42 per cent. have vision of $\frac{5}{10}$ or better; of Igersheimer's only 22 per cent. Forty per cent. of his cases had vision of less than $\frac{3}{10}$, and only 26 per cent. of ours; in seventy-seven cases determined, a loss of earning power in forty-three. In any event, whether his figures or ours are nearer right, no one can deny that the disease causes a visual economic loss that deserves consideration.

TABLE 3.—IGERSHEIMER'S STATISTICS OF VISION IN ONE HUNDRED AND FIFTY-TWO EYES

Number of Eyes	Vision
7	10/10
11	7/10
16	5/10
32	3/10 to 4/10
24	2/10 to 3/10
34	1/10 to 2/10
28*	less than 1/10

* Five of these twenty-eight were blind.

Etiology.—By the diagnosis "interstitial keratitis" I mean the typical cases of this disease. To quote the words of Jonathan Hutchinson, "Only typical cases of chronic diffuse keratitis must be chosen, for although the affection is unmistakable to the practiced observer in a great majority of instances, yet it has, like all other diseases, a border ground on which mistakes may be made." In my opinion, it is usually the border line cases that have caused the endless controversy as to the etiology.

In a certain small number of cases, another factor probably enters in. I refer to the coexistence of another corneal disease, usually phlyctenular keratitis. In a small number of the cases which I have observed, naturally excluded from the series on which this eye report is based, we find on the hospital records a diagnosis of the first disease, and at a later period the second diagnosis was made. We know that tuberculosis not infrequently occurs in hereditary syphilitic children, and it may not be too much to say that these

children, especially the poorly nourished ones, are particularly liable to develop tuberculous lesions. I believe, therefore, that phlyctenular disease and interstitial keratitis can occur in the same person at different periods, and in making the ocular diagnosis in a doubtful case this possibility should be borne in mind.

The relation of interstitial keratitis to tuberculosis has never been definitely settled; the views of various authors differ markedly. In 107 cases Diez gave 42 per cent. as tuberculous. Von Michel once stated that 40 per cent. were of this nature, von Hippel[4] twenty-eight cases out of eighty, Enslin eight out of nineteen. In thirteen cases with a positive Wassermann reaction, Kümmell obtained a positive general reaction to tuberculin. Elschlepp found seven similar cases. A. Leber, in eighty cases, found six surely tuberculous and four probably so. Hess is of the opinion, and to his observation we must lay great weight, that tuberculosis is a cause of interstitial keratitis. In a series of cases examined with tuberculin injections for diagnosis, he found several which gave a focal reaction in the eye. Clausen[5] has observed a similar case. The last named writer, after considering carefully the evidence, concludes that 90 per cent. of interstitial keratitis is due to syphilis, and that tuberculosis is the next most important cause, and that in a number of these cases both diseases are present, but that generally in such cases syphilis plays the important part in the production of the eye disease.

To quote Jonathan Hutchinson again:

> I will not make so sweeping an assertion as that interstitial keratitis never occurs except in people of inherited taint, yet I cannot conceal from myself, and have no wish to do so from my reader, that such is my present belief.

Igersheimer, who perhaps has devoted as much attention to this disease as has any other ophthalmologist, came in 1913 to the same conclusion to which Hutchinson subscribed in 1858. And on the basis of the large series of cases which forms the groundwork

4. Von Hippel: Arch. f. Ophth., **42**, 194.
5. Clausen: Arch. f. Ophth., **83**, 399.

of this communication, I am inclined to the same view, with the reservation that, in the light of our present knowledge, we must admit a small number of cases of the disease may be of other than a syphilitic origin.

Recurrences.—It is my belief that recurrences are considerably more frequent than existing figures show. Some ophthalmologists have held that recurrences seldom occur (Silex, Nimier-Despagnet), others that they occur sometimes (Fuchs, Vossius, Hosch), and Arlt and Gutmann that they are frequent. In a carefully followed series of sixteen cases, von Hippel found five with recurrence, 31 per cent. In eighty-seven cases, many of which were observed only a short period of time, he found but 17 per cent., as did Jakolewa, Brejski in 18 per cent. and Stephenson in 22 per cent.

In thirty-seven cases in which I have carefully gone into the question of recurrences, I have found positive evidence in fourteen (38 per cent.), and in three more recurrence was probable.

Will the treatment of hereditary syphilitic children in their early years prevent the development later of an interstitial keratitis?

Hochsinger's Experience. — Sixty-three cases of undoubtedly congenital syphilitic children were followed by Hochsinger and Kassowitz from four to twenty years; of these, twenty-two were followed carefully for eight years, after they had been treated energetically with mercury in very early life. Among this group there was not a single case of interstitial keratitis, deafness or hutchinsonian teeth.

The following cases occurred in my practice:

CASE 1.—D. M., a boy with undoubted signs of hereditary syphilis, developed a paralysis of the third nerve of the right eye in the early months of life. As far as could be ascertained, his treatment up to the age of 4 consisted largely in the use of the iodids combined with inunctions. At 4 years of age he developed in this eye an interstitial keratitis of moderate intensity, which lasted for about five months. At this time he came under the care of practitioners thoroughly

qualified in the handling of syphilitics, and was given thorough treatment with mercury over the succeeding four years. In 1908, then at the age of 7, and three years after the involvement of the right eye, he developed a mild interstitial keratitis in the left eye, which ran its course in about ten weeks and cleared up almost completely. Seven years later, in 1915, the vision in the left eye with a minus cylinder was 10/10—, the cornea showed a slight opacity, and a number of vessels in the cornea were to be seen.

CASE 2.—G. V., boy, developed unmistakable active signs of hereditary syphilis soon after birth, and was given continued treatment with inunctions twice a day for two years and with mercury and chalk for a year more by his doctor. At the age of 9 months, he developed in one eye a uveitis with a small hypopyon. The ocular disease cleared up within a year, but general treatment was continued. Four years later he developed an interstitial keratitis of both eyes, of moderate intensity.

While cases like these are discouraging, I believe that they should in no way deter us from the energetic and long continued treatment of hereditary syphilitic children. Although we cannot, in the light of our present knowledge, influence markedly in many cases the course of the ocular inflammation, nor can we prevent the involvement of the second eye, yet we can and do produce improvement in the general condition and often in the mentality of our patients. It is well known that a positive Wassermann reaction is unusually persistent in these cases, but by adequate treatment of sufficiently long duration it can be made to disappear. Recurrence of the eye disease and other complications are certainly much less likely to occur, and if they occur at all, without the presence of active disease as evidenced by the Wassermann reaction.[6]

In conclusion, a quotation from Igersheimer may not be out of place:

> In the interest of the later career of patients with interstitial keratitis it is important to administer antisyphilitic treatment with the greatest energy, not only during the course of the ocular disease, but afterward from time to time as long as the Wassermann reaction is positive.

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6. The literature up to 1911 will be found in the exhaustive articles of von Hippel, Hoor and Clausen, which have been freely used. Other communications referred to are:
Axenfeld: Ber. d. ophth. Gesellsch., 1913, p. 261.
Wickerkiewitz: Ber. d. ophth. Gesellsch., 1913, p. 262.

ABSTRACT OF DISCUSSION

DR. ARNOLD KNAPP, New York: The better visual results which Dr. Derby obtained at the Massachusetts Charitable Eye and Ear Infirmary than those given in other statistics, may be due to the fact that the patients in the infirmary were all carefully examined, with dilated pupil, and with refractive correction. It is well known that the vision in this class of cases is often very much improved by a correction with glasses.

An important complication, and one which is not unusual, is that of glaucoma. When this occurs in the course of the keratitis, it necessitates a change of treatment, and if the tension still remains increased, an iridectomy should be performed. The results of an iridectomy under these conditions are very much better than one would be led to suppose.

An important association in the etiology is that of tuberculosis and syphilis in these cases; and in the treatment, the patients are much more benefited by the tuberculin treatment than by antisyphilitic remedies. A case in point is that of a young patient, aged 20, who, while at Saranac undergoing treatment for general tuberculosis, developed an interstitial keratitis. The Wassermann reaction was 4 + and a tuberculin complement fixation test was 3 +. Treatment for four months with injections of salicilate of mercury had no influence. The patient was then given tuberculin, with distinct benefit after a number of months. In this case the keratitis was characterized by lack of vascularization. Some years ago I published an article advocating the use of salvarsan in the treatment of interstitial keratitis, and I am still of the opinion that this is a very valuable adjunct. Though the effect on the corneal condition is not striking in the cases with but little inflammatory reaction, it surely tends to improve the patient's general health, which is a very important object in the treatment.

I have attempted to clear up the corneal opacities after interstitial keratitis with the use of radium. The results up to date have not been definite enough to say whether this treatment has been of any benefit.

Dr. Derby raises a very important question about the advisability of early treatment and the possible prevention of interstitial keratitis. This is a question which naturally will require many years of observation to be answered. At the same time, by means of a general use of the Wassermann reaction, cases which should be subjected to this treatment can be easily determined. I was interested to see in Dr. Derby's paper that in a certain number of cases the Wassermann reaction was made negative. In the cases of interstitial keratitis which I have been able to follow this has never been accomplished.

The section has the honor of having present Dr. John A. Fordyce, and I hope that Dr. Fordyce will be good enough to express his opinion on the prognostic value of a persistent positive Wassermann reaction in these cases.

DR. JOHN A. FORDYCE, New York: In experimental syphilis in the rabbit, involvement of the eye is interpreted by Nichols and Reasoner as indicative of nervous syphilis, being the equivalent of an intensive invasion which in man is expressed by some form of cerebrospinal lues. In other words, the organisms recovered from the spinal fluid in syphilis of the central nervous system and inoculated in rabbits produce eye lesions. In human syphilis, however, such changes as keratitis, retinitis and choroiditis, with a few exceptions, have in my experience been accompanied by a negative cerebrospinal fluid, and objective findings referable to the central nervous disease have not been elicited. It is possible that in ocular syphilis the lesion is such a localized one that the Wassermann-producing bodies are not in sufficient number to give a reaction in the spinal fluid, at least with our available methods. Although the blood in these cases may be strongly positive, there is no evidence that the complement binding powers pass into the fluid unless there is a simultaneous involvement of the meninges or nerve tissue.

In eye syphilis, positive clinical manifestations are frequently present with a negative blood Wassermann, as in a patient referred to me by Dr. Knapp, who developed in his early secondary period, one month after treatment with four full doses of salvarsan, a perivasculitis of the deep eye vessels. Under mercury the eye lesions regressed and with its discontinuance a relapse from a new focus took place, which again improved under further therapy. Three months after the cessation of all treatment his Wassermann was 4+. The blood in this case was evidently reinfected from the lesions in the eye.

Less than 1 per cent. of all eye patients are said to suffer from interstitial keratitis. It has been estimated that keratitis affects the female sex more often than the male (52 per cent. as against 48 per cent.), and that more than half the cases occur between the ages of 8 and 14. In a series of twenty-five cases under my observation, interstitial keratitis was met with in four patients with acquired syphilis, in one thirteen years after infection, in another seventeen years, and in the other two the interval could not be determined. Of the congenital cases, the youngest patient was 1 year old and the oldest was 33 when the first attack appeared. Another patient had her first involvement at 21; the remaining patients between the fifth and fifteenth years. In twelve cases the parents were investigated with the following results:

In five, both parents were found to be syphilitic. In four, the mother gave a negative history and negative Wassermann. The father was said in two of the cases to have had lues. In a third case two brothers had positive Wassermanns, one showing also corneal opacities. In two cases the father only gave a positive history. In one case two brothers also had interstitial keratitis—no other history obtainable. In four cases the fathers had suffered from syphilis of the nervous system; one paresis, one tabes, two cerebral endarteritis. In two of the twenty-five cases the Wassermann was negative; the patients were 28 and 54, respectively, with recurring attacks. The remaining twenty-three were 4+. Six cases were punctured. Two showed a slight increase in cells and globulin, otherwise negative. The other four were normal.

Interstitial keratitis is said to be associated especially with bone and joint affections. In this series, synovitis was met with in two cases and bone involvement seen but twice. The concomitant or previous symptoms of syphilis were as follows:

Girl, aged 21, had migrainelike attacks of headache which disappeared under treatment; girl, aged 15, had ulcer of the septum; girl, aged 15, had hydrocephalus; girl, aged 29, had scaling lesions of palms when 18; girl, aged 24, had Hutchinsonian teeth and is quite deaf; when 10 years old the patient had gummas of leg and arm; girl, aged 11, when 2 years old had a right-sided hemiplegia, with a second attack ten months later; when 10 years old, keratitis and deafness; at the same time, periostitis of both wrists and dactylitis of several fingers. She had Hutchinsonian teeth. Her pupils were irregular and fixed to light; she was undeveloped mentally.

The observation of interstitial keratitis supports the contention that spirochetes may remain localized in one tissue or one organ for a period of years without exciting an inflammatory reaction. This is illustrated in a number of cases which I have observed in which the first manifestations occurred after the patient reached adult life. It is possible, however, that if these cases had been suspected to be syphilitic when young, their blood reaction would have been positive; or there might have been discovered evidences of involvement of the deeper parts of the eye, as choroiditis. This was, in fact, observed in one of my patients, a physician, who developed an interstitial keratitis after he was 25. This was followed by a uveitis and later by a glaucoma for which he was operated on by Dr. Lambert of this city. In my notes of the case it is stated that old scars in the choroid were discovered by the ophthalmoscope. Keratitis may occur as the only evidence of the disease, but this is not infrequently preceded or accompanied by other manifestations of syphilis, as dactylitis, or joint involvement. An important

point to be emphasized is the necessity for the persistence of treatment in patients with interstitial keratitis until the Wassermann reaction becomes negative. This was well illustrated in a patient, who had several recurrences of keratitis after a definite quantity of salvarsan and mercury had been given. Her Wassermann reaction, however, remained persistently positive until the treatment was continued over a long period of time. The reaction finally became negative and since that time she has had no recurrence whatever of her trouble. This may be accomplished by a combination of mercury and salvarsan, in this particular case by salvarsan alone, the patient being intolerant to mercury and the iodids in any form.

Salvarsan, in my experience, produces a more rapid effect in acute attacks of keratitis than mercury or the iodids. Several cases previously treated by mercury and iodids without result rapidly subsided after administration of salvarsan. Patients treated over long periods by mercury and iodids may develop mercury-fast organisms which render them immune to the drug.

In the majority of cases of congenital syphilis the Wassermann reaction is very resistant to treatment, and in some cases cannot be changed.

Dr. F. Park Lewis, Buffalo: Every bit of cornea that we can preserve in an interstitial keratitis is of great importance. The measure which I have found useful, supplemental to the usual measures constitutionally employed, is cataphoresis. The manner in which I have used it leads me to believe that it may ultimately be of increasing value in other corneal diseases. I use it through an eye cup in which there is a normal salt or other solution, holding this so that the eye is immersed in the eye cup, the positive pole of the battery being attached to the cup holding the solution, and the negative pole being applied indifferently or the reverse, according to the effect desired. I use that for two or three minutes two or three times a week, and the rapidity with which the cornea has cleared has been in many cases exceedingly gratifying. Within the last two or three months in a case of interstitial keratitis in which there was a 3+ Wassermann, and in which the cornea was quite opacified, the clearing has been, I may say, absolute; there are no scars left at all. Within two weeks the brother of this young man presented himself with precisely the same condition in the same eye, and he is now under the same treatment; both receiving a course of constitutional treatment. I supplement this with 1 to 3,000 mercuric chlorid ointment, used at night. That, however, I have used in other cases without getting the results I have in these cases. I believe the effect of the ionized medicament in controlling the toxins in the substance of the cornea is the essential element producing resolution in these cases.

DR. R. L. RANDOLPH, Baltimore: I have been very much struck by the difference in effect obtained by the administration of salvarsan in cases of interstitial keratitis in acquired and in congenital syphilis. It has made a remarkable difference in the former class of cases. Cases of interstitial keratitis due to acquired syphilis get well with wonderful rapidity as compared with results obtained in the presalvarsan days, for once it took months or years to clear up the cornea. I have never found a case of interstitial keratitis due to congenital syphilis in which salvarsan seemed to help.

DR. HIRAM WOODS, Baltimore: I do not know of anything more interesting than the gradual change that has come in our own section in reference to studying the constitutional courses of eye diseases; in other words, the greater thoroughness with which we are going into these things. In this discussion a number of interesting things have been brought out. First is the diagnosis of the cause of interstitial keratitis. We used to put it down to hereditary syphilis without hesitancy. I was rudely jarred some years ago by a child with a typical syphilitic infiltration which on antisyphilitic treatment "got no better rapidly." The boy developed a pain in the knee joint, with suppuration—a tuberculous joint. It was drained and in a few weeks the cornea began to clear and went on to a total clearing. All other treatment was stopped. In other words, here was a family history of syphilis, in which mercury failed, with the eye rapidly clearing after removal of antiluetic treatment and drainage of the joint. It was a clear and definite evidence to my mind of a possible tuberculous cause.

Now as to salvarsan: I saw recently a 16-year-old girl with interstitial keratitis in one eye. There was a 4+ Wassermann. Salvarsan was given. The immediate result was a violent circumcorneal injection with total clouding of the affected cornea and rapid involvement of the other. It reminded me of a severe focal tuberculin reaction. Improvement began only after a prolonged course of mercury. She now has $^{20}/_{40}$ vision, from nothing; her Wassermann is still positive, even after salvarsan and several months of mercurial treatment.

By way of contrast, another girl, 15 years old, had a positive Wassermann. Salvarsan was not used. She lost one eye from organized interstitial opacities. The other eye became myopic; vision $^{20}/_{30}$ with, recently, a negative Wassermann, after two years of mercurial treatment.

In a fourth case, a boy was given salvarsan and got absolutely well with clear corneas. I thought that was a triumph for salvarsan; but I am not at all sure that Dr. Knapp's statement is not correct: that when salvarsan does good in interstitial keratitis, it does more by getting rid of the general infection and improving the general health, than by a

special localized effect on the cornea. I should like to hear that point discussed; there is evidence on both sides.

One other point with reference to all these cases, and that is the importance of a follow-up system. I have seen cases of recurrence from neglect after the eye got well. I think we should view our duty in interstitial keratitis cases along this line—that we have a *local manifestation* of syphilis, if the Wassermann is positive, and that when we get rid of this particular manifestation in the eye it is up to us to do the best we can to keep the patient under constitutional treatment till the disease is cured.

DR. ERASTUS E. HOLT, Portland, Maine: Dr. Lewis' reference to electricity called to memory a discussion of this subject thirty years ago when Dr. Derby, Sr., read a paper before the New England Ophthalmological Society. He was applying electricity in the treatment of this disease. I have used all the remedies usually employed in this affection, but even then we have cases that stay with us. I have always used anaphoresis. I take sodium iodid, perhaps 10 or 20 per cent. solution, and paint it over the eye lids of the patient and apply the negative pole. I am sure that I have seen the eyes clear up under that treatment. After we do get them to clearing up I practice cataphoresis, by applying a 1 per cent. solution of strychnin over the lids and then the positive pole. I am sure if any one will practice this treatment in these cases he will find it a help in curing them.

DR. F. H. VERHOEFF, Boston: In discussing the question of the relative frequence of tuberculosis and syphilis as causes of interstitial keratitis, the assumption seems to be generally made that either condition produces the same type of corneal changes. As a matter of fact, however, I have found that tuberculous keratitis and the keratitis of hereditary syphilis each has a characteristic clinical picture, and that cases are extremely rare in which the diagnosis can not be made from the appearance of the cornea alone. In general, tuberculosis is far less important than syphilis as a cause of interstitial keratitis. Up to the age of about 16 years interstitial keratitis due to tuberculosis is so rare that we can almost say that all cases are due to syphilis. Beyond this age tuberculosis gradually increases while hereditary syphilis decreases as a cause of interstitial keratitis. Hereditary syphilis is undoubtedly often associated with systemic tuberculosis, but I have never been able to determine that the two infections ever produce interstitial lesions in the cornea at the same time.

The general opinion seems to be now that interstitial keratitis is a manifestation of anaphylaxis, but some observations that I have made with Dr. H. Hartwell on rabbits infected with spirochetes go to show that it is due to the actual presence of these organisms in the cornea.

I employed specific treatment in all cases of interstitial keratitis for over ten years before it dawned on me that it was useless. Since then I have used only nonspecific measures and my results have been certainly as good if not better than formerly. As I have suggested elsewhere, in cases of interstitial keratitis the spirochetes have been present in the body so long that it is probable that they have acquired the same immunities as the body cells, so that the drugs at present available are no longer able to act differentially on them.

DR. H. F. STOLL, Hartford, Conn.: I am interested in the question of hereditary syphilis, as I have treated a number of such patients. In regard to the diagnosis, we have in addition to the Wassermann test a means of diagnosis that was not mentioned, though a very important one, taking more time than the Wassermann and requiring an equal amount of skill. I refer to the diagnosis by intensive family study. The patient often has brothers, sisters, a mother and father. In one you may get a high palate, in another Hutchinson's teeth. The father may have an eye palsy, the mother severe headaches, and perhaps aortitis. This means syphilis—*familial syphilis*—whether the Wassermann be positive or negative. I have seen people go two or three years without treatment because they had a negative Wassermann, until the disease became so manifest that treatment was finally started. In two of my cases interstitial keratitis was a manifestation of syphilis of the third generation. In one case the mother had Hutchinson's teeth. Both patients had positive Wassermanns. We must bear in mind that the treatment of symptoms is not the treatment of syphilis, and the man who treats his cases of interstitial keratitis with fresh air and no medication, though the local lesion may subside, is losing sight of the fact that the patient probably has spirochetes in his liver and testicles, his aorta and brain, and should have treatment for this general infection even though the local manifestation in the eye will get well without it.

In regard to the presence of syphilis and tuberculosis in the same individual, Fournier said they were peculiarly apt to coexist, and that the tuberculosis was generally severe. As to focal reactions following treatment—I think Dr. Woods spoke of it—they are really common, and yet, so far as I know, never described. I frequently tell patients, whatever their symptoms are, that when treatment is begun they will possibly feel worse for a time and perhaps dormant lesions will become manifest. I have seen a tertiary skin lesion and a typical secondary rash develop at the same time in a heredosyphilitic when treatment was started. Again, precordial and joint pains and various other manifestations may appear; these focal reactions indicate more, not less, treatment.

The last point I want to make is that the ophthalmologist, the gynecologist, the nerve specialist and the internist each has an obligation to the other members of the family when treating a patient with syphilis. The mother of the patient with interstitial keratitis may be pregnant, and it is the duty of the ophthalmologist to prevent, if he can, the unborn baby from the fate of a heredosyphilitic. By all means follow up the cases of interstitial keratitis, but as a family group, not as an individual.

DR. WILLIAM H. WILDER, Chicago: I wish heartily to endorse the remarks of the preceding speaker in criticizing the statement of Dr. Verhoeff that it is not necessary to treat cases of syphilitic interstitial keratitis with antisyphilitic measures.

The recent experimental work of Dr. Alan C. Woods, reported to the American Ophthalmological Society, in which he was able to demonstrate the presence of a variety of trypanosome in the cornea, suggests that in syphilitic keratitis the trypanosome of syphilis is probably present in the corneal tissue. In our present state of knowledge it is wise to act on the assumption that such is the case and to pursue active treatment for syphilis if the clinical history and Wassermann tests agree as to syphilitic infection.

I have been repeatedly convinced of the great value of salvarsan combined with active mercurial treatment by inunctions, or better, by deep injections in these cases, especially if the treatment is begun early enough, before the exudate in the cornea has organized. I am sure that in a number of cases in which I adopted this treatment in the early stage of syphilitic interstitial keratitis, I have had results that I could not have obtained with the old procedures. It is true that fresh air and the general constitutional measures of the follow-up treatment should not be neglected. The older syphilographers recognized the value of such measures and often advised the suspension of mercurials and the administration of tonics and general building up of the patient, and then a return to the specific treatment. This is all important, and it would not be well to have the idea go abroad that as ophthalmologist we need not pay attention specifically to the general infection that underlies such a local condition.

PRESENT STATUS OF CORNEAL TRANSPLANTATION

SOME EXPERIMENTAL DATA

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While this operative procedure has intermittently held the attention of many workers in the field of ophthalmology for over a century, to the present it has proved a failure, except in rare instances. A review of the literature on the subject will show to a fair degree the causes of failure and the main elements which might lead to success.

To Pollier de Quengsy, a French oculist, is given the credit of being the pioneer in this work. In 1789 he attempted to transplant a piece of glass into the center of the cornea, and from this time on other men, Nussbaum in 1856, Huszar in 1859 and von Hippel[1] in 1877, attempted the same thing, while Drummer in 1889 and Salzer[2] in 1899 tried celluloid, all with no success to speak of. Salzer reported a case in which the glass was retained for two and one-half years, but it ultimately sloughed out, and this at least showed the great vitality and endurance of the cornea.

F. Reisinger, in 1824, was the first to attempt the transplantation of the cornea from one animal to another, by means of a transcorneal keratoplasty. Half of the cornea cleared up in two and a half days, but later the eye sloughed out. Schon, in 1827, denied the practicability of this work, but did no experiments to prove his assertion. Both Dralshagen and Diffenbach,[3] in 1831, were unsuccessful and decided that if

1. Von Hippel: Arch. f. Ophth., 1878, **24**, 235-256.
2. Salzer: Arch. f. Augenh., 1909 **115**, 226; Ber. ü. d. Versamml. d. Ophth. Gesellsch.., 1911, **36**, 244.
3. Diffenbach: Ztschr. f. Ophth. (Ammon's), 1831, **1**, 172.

the foreign cornea did adhere it would soon become turbid and unserviceable.

Bigger,[4] in 1835, took up the problem with more vim than success. He first operated on the leukomatous eye of a gazelle by means of a complete section of the cornea, and reported a good result. Later he operated in the same manner on a timber wolf and thirteen rabbits. His chief difficulties were movements of the animal at the time of operation, and sepsis. His original account itself is worth the reading, from the standpoint of subtle humor, and an appreciation of the difficulties under which the earlier experimenters worked.

H. Power,[5] in 1872, took up the work along a somewhat different idea, and his experiments with a cork borer and sharp steel punches were the forerunners of the later trephine operations. His results were bad, and due, he said, to the lack of precaution and poor technic. Among many other investigators on the subject, von Hippel led a school that used the trephine. His was a mechanically operated instrument and there was great difficulty in controlling the depth of the cut and the sterility of the instrument. His results were not good, mainly because he attempted heteroplasty. Others followed von Hippel, using his trephine, among them Zirm,[6] who grafted the cornea of a child, aged 11 years, to that of a patient with total opacity, having vision of shadows. The result was that six months later the patient had vision $\frac{3}{20}$ with plus 5.00. One year later the patient died, and as the eye was not examined the depth of the leukoma was not known. Fuchs, in 1894, operated in eleven cases and, on account of the opacity of the graft and the penetration of new vessels, he gave up the project.

Magitot[7] lately took up the trephine method, but he used an instrument much different from von Hippel's.

4. Bigger: Dublin Jour. Med. Sc., 1837, **11**, 408-417.
5. Power, H.: Rep. Int. Ophth. Cong., 1872, **4**, 172-176.
6. Zirm: Arch. f. Ophth., 1880.
7. Magitot: Compt. rend. Soc. de biol., 1911, **36**, 369; Ann. d'ocul. Paris, 1916, **153**, 369, 417.

His was a modification of Eliot's trephine which would cut a graft approximately 5 mm. in diameter to the required depth, and after repeating the operation on the eye to be transplanted, he transferred the grafts. They were held in place by a nasal and temporal corneal stitch over the graft. Atropin was instilled and the eye bandaged for forty-eight hours. He used not only fresh tissue, but also infants' eyes which had been submerged in aseptic serum for some time, washing them in Ringer-Locke solution after taking out, and grafting as before. He reports that the grafts grew, and that the opacity was not great. This most certainly is an example of the great vitality of corneal tissue.

Probably the most spectacular operation of this type of work was one in which Chimanowski attempted a total corneociliaryplasty, if the term may be used. He operated on a patient, aged 20, who had perception of light, and removed the whole cornea and the ciliary body without touching the muscles. The graft was placed in physiologic sodium chlorid solution for three hours, and then sutured to the prepared host by ten suture points. Healing took place and the sutures fell out. On the twenty-fifth day one part of the cornea was clear and the other grayish, vision being light perception. On the fiftieth day no suture line was visible; the anterior chamber was 2 mm. deep, the iris was visible, and an astigmatism of 37 D. was reported. On the seventh month there was some organization, and the vision was fingers at 18 inches, tension being 18 mm. One year later there was a sensitive cornea tension of 23 mm. and there was opacity of the globe; but no atrophy was found. Iridectomy was tried, but it resulted in phthisis bulbi.

Morax followed the plan of F. Reisinger, except that he attempted to transplant the leukomatous portion to the periphery, and the clear periphery to the center. Strauch, in 1840, preferred to dissect from one limbus

to the other, using parallel blades, and Wolfe,[8] in 1873, finding the tissue opaque from lack of nutrition, made conjunctival flaps. Schaeler, in 1877, advised large grafts after complete resection of the leukoma, while Dürr and Rasenni,[9] in 1877, reported that only part need to be covered. Probably the thought of large flaps and better nutrition led to the transcorneal keratoplasty, which many men tried in modified forms. Among the most recent to devise and try this form of technic was Löhlein,[10] in 1910. His was essentially the idea of Wolfe in 1873, for his operation consists of a transcorneal section of required width across the cornea from limbus to limbus, making a generous conjunctival flap at each end. The thickness was controlled somewhat by the depth of the cut of the parallel blades. The flap was sutured in place at both sides by its conjunctival attachments, and the eye bandaged. His conclusions are that he has done forty of such grafts and is most satisfied with results, which are best, he thinks, following leukoma from burns, and most unfavorable after ulcer. He thinks that thin grafts are the best, but says that Thiersch grafts are hard to get.

Over a long period, therefore, many types and forms of operation were tried; and from them the larger questions arose, such as the type of tissue to be used, causes and prevention of opacity following operation, nutrition of the transplanted flap, and lastly the operative technic. We can therefore make these general deductions from the voluminous literature on this subject on the foregoing main points.

As is known, there are four different types of operation: autoplasty, homoplasty, heteroplasty and foreign body transplants. The latter can be at once dismissed, because of universal failure, and heteroplasty, which is at present a biologic problem. Bonnefon and Lacoste[11] stated that most failures were due to

8. Wolfe: Brit. Med. Jour., 1880, **2**, 780.
9. Dürr and Rasenni: Klin. Monatsbl. f. Augenh., 1877, **15**, 305-311.
10. Löhlein: Arch. f. Augenh., 1910, **154**, 179; Arch. Ophth. N. Y., 1912, **41**, 266.
11. Bonnefon and Lacoste: Compt. rend. Soc. de biol., 1912. Bonnefon: Arch. d'Ophth., Paris, 1913.

heteroplasty. Hence autoplasty and homoplasty are the two types to which most of the more recent experimenters have clung.

Autoplasty, as the name implies, is the transplantation of the cornea from one part of the eye to another part of the same eye, or from one eye to the other eye in the same animal. Homoplasty is the transplantation of corneal tissue from an individual of one species to another of the same species, or to a species not far removed. As the two types are similar, they will be dealt with together.

Among the causes of failures, other than those due to heteroplasty and foreign body transplants, has been the selection of cases. Healthy tissue should be the first prerequisite, although Fuchs asserted that he had had his best results in interstitial keratitis. Magitot doubted this. The depth of the leukoma should be taken into account. It should not extend to Descemet's membrane, for it has been shown by several men that it is necessary to get all of the leukoma and yet not puncture the anterior chamber. Magitot has stated that unless all the leukomatous tissue is obtained, the results will not be good, and no hope can be held out for a complete growth of the tissue. "The thing to know," he says, "is how deep the leukoma extends, but only at time of operation can this be determined." Leukomas as the result of ulcers, especially with posterior corneal synechiae, are not good operative risks. It is, of course, assumed that the conjunctival sac is free from infection, as this was the cause of most failures in the work of earlier operators.

Of all the operative accidents, puncture of the anterior chamber appears as the leading cause of failure, although union has been obtained under these circumstances. Trauma of the graft while handling, loosening of a graft after transplantation by lid movements, and local necrosis from stitches were given by others as causes of failure.

As regards nutrition, there is a wide variance of opinion; for some authors hold that too much nutrition causes vascularization of the graft, and others take the opposite view. Salzer stated that the more vessels present, the more cloudiness and connective tissue. Zirm, on the other hand, tried to cover the periphery of the cornea with conjunctiva, to promote nutrition of the graft. Löhlein, in his later paper, stated that the best nutrition followed vascularization.

Hence, in summary of the foregoing notes of many different authors, it can be said that foreign body transplants and heteroplasty are bound to lead to failure, and that autoplasty and homoplasty offer the greatest chance of success. Still even in the latter two, the technic of operation and the selection of cases have a great bearing on the final result.

EXPERIMENTAL WORK

The technic of this operation was developed on the eyes of dead dogs, of which I had a plentiful supply from the physiologic laboratory of the University of Chicago. It was first found that it was impracticable to obtain the corneal grafts with conjunctival flaps attached, similar to those of Löhlein, from a dog, on account of the anatomic form of the socket and the third lid. The curvature of the cornea, even in man, makes it difficult to follow a surface of this kind, especially when a cataract knife is used. Hence some other method was necessary to obtain a large graft with sufficient nutrition. For this reason, the modified Thiersch graft was decided on, and the technic was developed to a fair degree after a great many trials on dead animals.

The live specimens were then prepared as follows: A mercuric chlorid bath was given, and the animals were isolated in fumigated cages. The eyes of the animals were treated for a period of three weeks previous to operation by 2 per cent. protargol instillation each evening, and washing out of the sac each morning

with 1:4,000 mercuric chlorid solution. After smears showed the absence of the streptococcus and other virulent bacteria, the animal was ready for operation. The work was all done under ether, and with as much asepsis as a physiologic laboratory will permit.

Description of Operation.—The conjunctival flap is prepared thus: The bulbar conjunctiva is loosened up well over the whole area extending from 7 to 4 o'clock on the dial, care being taken to have it loose enough so that it can readily be pulled over the entire cornea without much tension. Some hemorrhage results, and the corneal graft is not removed until this has entirely stopped, as will be explained later on.

The eye is now prepared as in operation for cataract extraction, and after the conjunctiva has been grasped below with fixation forceps, a Graefe cataract knife is passed into the cornea at the required depth, and the point emerges on the other side as far as is possible or desired. Then by a sawing motion the flap is cut down as far as necessary, and the knife emerges, leaving the flap attached to the cornea at the upper pole. The flap is now grasped with the fixation forceps, and is gently lifted up and cut away at the upper pole. This yields a flap of corneal tissue about 3 by 5 or 4 by 6 mm. in size, which is somewhat irregular in outline, and of slight difference in thickness throughout.

The graft is now made ready for the host, by passing three or four fine silk ligatures through it at its corners and close to the periphery. On account of the difficulty encountered in this step, special forceps were designed which resemble standard fixation forceps with a notch cut in the middle third so as to allow the needle to be passed through the tissue in question.

The corneal cavity of the host is made in the same manner as described above and is larger in diameter than the flap. It is moistened with physiologic sodium chlorid solution, and the flap, immersed in the same solution, is laid on the eye. The sutures are inserted at the points at which the flap limits extend and just

deep enough to hold, namely, about one-fourth to one-third the depth of the cornea. When all sutures have been inserted they are pulled taut, so as to approximate but not to strangulate the tissue. The thread is now cut off close, and the eye washed gently with physiologic sodium chlorid solution.

The flap is now carefully brought down over the entire cornea and sutured fast below with silk sutures. In dogs, the third lid is sutured over the flap and the flap and lids bandaged.

Forty-eight hours after the operation, the bandage is removed and the conjunctival flap loosened. One finds at this time some of the corneal sutures gone, and those that are not are taken away. Ethylmorphin hydrochlorid (dionin), 5 per cent. solution, along with weak mercuric chlorid irrigations are now used. On direct examination of the eye at this stage, one finds the corneal graft clouded and there is some injection. In fifteen days the process tends to clear, leaving some small blood vessels which after three to four weeks gradually disappear. The final result is a leukoma discernible under strong light. The outlines of the graft gradually disappear in from two to three months.

It has always been the aim to obtain a flap thinner than the tissue removed from the host, and one can see, after healing has taken place, the new tissue filling in the slight peripheral depressions in the cornea. After from three to four months there is little left to show how extensive the flap was, so smoothly is the corneal surface epithelialized.

Experimental Data.—The laboratory experiments on live animals consisted of operations on twelve dogs, part of which had both eyes operated on and part only one.

The operations on the first three dogs were entirely unsuccessful, for the anterior chamber was punctured and the eye sloughed out. The next three were partially successful in that in one dog the anterior chamber was punctured but the graft held. Organization and exudate filled in about the graft, forming a dense leukoma, and the animal was discarded. The

other two were successful from the standpoint of technic, as the anterior chamber was not punctured and the flaps were held in place. After the first dressing one of the dogs managed to scratch his eye, and as a result the corneal flap sloughed out. The other dog retained his grafts, but a dense leukoma formed. In the foregoing six dogs, both autoplasty and homoplasty were tried.

Dogs 7 and 8 were operated on in the manner described and dressed on the third day. The grafts held, but there were slight depressions with some grayish material at the borders of the graft. On the sixth day both grafts were relatively clear, and injection was not so marked as before. The twelfth day showed beginning organization in both grafts, and on the twenty-fifth day there was marked organization and cloudiness. Subconjunctival injections of 8 mm. of sodium chlorid solution and two of 5 per cent. ethylmorphin hydrochlorid were given with no result. In six weeks the same condition was present, so the animals were discarded.

In Dog 9, autoplasty was attempted. Operation was performed as described. The final result was organization of the graft especially from the nasal side which foiled all attempts to correct. The dog had fair vision, as was evidenced by a good red reflex with the ophthalmoscope, only slight cloudiness, and ability to get about without much disturbance due to vision.

In Dogs 10, 11 and 12, profiting from previous work, we allowed the minimum amount of blood when the conjunctival flap was brought over the cornea. In all three dogs the grafts held well; some cloudiness was present on the fifth day which increased to the twelfth day and then gradually disappeared to the naked eye. The organization was less than in any previous group, and although it persisted for six weeks, under ethylmorphin hydrochlorid both it and the cloudiness gradually faded away. Ophthalmoscopic tests showed a normal red reflex, and focusing on the cornea revealed the graft, which could be discerned especially about its edges by its faint wavelike appearance. After four months the eyes in all three animals appeared normal to the naked eye, and the corneal surface was absolutely smooth and regular.

CASE 1.—J. H., white man, aged 26, had dense leukomas covering the central third of the cornea in both eyes as the result of smallpox ulcers. Autoplasty was attempted on one eye only. The operation was performed successfully with exception that the leukoma was so deep that it was impossible to remove it all. When the conjunctival flap was loosened on the second day it appeared grayish and was attached only at its upper border. The attachment was so small that the flap was later brushed off by the upper lid. The final result has left the eye in a better condition as regards the leukoma than before operation. This demonstrates that corneal grafts heal

poorly on diseased tissue, as has been shown by previous experimenters.

CASE 2.—T. J. S., white man, aged 23, had a slight opacity of central fourth of the corneal surface. Homoplasty was tried. A graft was taken from the eye about to be enucleated. The operation was performed successfully and at present the graft is in place with only slight cloudiness and organization, and appears to be gradually clearing up under atropin and ethylmorphin hydrochlorid.

CASE 3.—B. W., white woman, aged 19, had a slight opacity of one fourth of the cornea, the result of phlyctenular ulcer. Autoplasty was tried. The graft at present is in place, and cloudiness and organization are no more than would be expected at this time.

Data on the final result of the last two cases will be given later.

In my earlier experiments I allowed plenty of blood to remain on the surface of the cornea when the conjunctival flap was brought over, desiring to give the flap plenty of nutrition by this means. Invariably I found intensive cloudiness or organization, although healing had taken place readily enough. Later I reversed that procedure, and stopped all bleeding from the conjunctival flaps before bringing them down. This I find a much better method, for beside giving the corneal flap plenty of nutrition by means of the serum present, much less cloudiness and organization resulted.

Most of my earlier failures were due to sepsis and undeveloped technic, as punctures of the anterior chamber. Later, opacities and organization were due mostly to too much blood in contact with the flap, and the result therefore was not good.

CONCLUSIONS

1. Heteroplasty and foreign body transplants offer very small chance of success and should be discarded, according to the literature.

2. Autoplasty and homoplasty at the present time will terminate with fair results, depending on the technic of operation.

3. The modified Thiersch graft is a simple, satisfactory and expedient manner of obtaining graft.

4. The conjunctival flap, as described, offers sufficient nutrition with the minimum cloudiness and organization, besides serving to hold the graft in place while union is taking place.

5. With properly selected cases, this operation will give good results.

[Live specimens will be shown.]

25 East Washington Street.

ABSTRACT OF DISCUSSION

DR. JOHN E. WEEKS, New York: From my studies and observations on this subject, heterogeneous and foreign body transplantations are entirely useless. Heterogeneous transplants of the cornea either become opaque or disappear entirely. The majority of flaps disappear entirely, although there may be primary union, as the experiments of von Hippel, Zirm and others have shown. The experiments of von Hippel were very exhaustive, extending over many years. In regard to foreign body transplants, they were failures in every respect. In homoplasty and autoplasty the measure of success that will be obtained will not be great at the best. The most suitable cases must be selected to obtain any degree of success. The opacification should not be extremely dense and there should be no involvement of the underlying tissues. The anterior chamber should be deep and the lens, of course, clear. All kinds of flaps have been employed. The flap suggested by Dr. Walker is certainly promising. That suggested by Löhlein is something similar. His flap consisted in the removal of a zone of corneal tissue including the opacification and a portion of the conjunctiva extending horizontally to either side, and the transplantation of a transparent portion of corneal tissue over the defect obtained by the removal of such a zone of tissue. The operation by von Hippel in one case and by Zirm in another are the only cases that have been successful, so far as I can find recorded in the literature, and these cases were only observed about one year. I believe Zirm's patient died of some intercurrent disease after the cases were observed ten or twelve months.

My own experience in operations for keratoplasty is confined to one case, in which I attempted to follow the technic of Zirm, which consisted in the removal of a disk of opaque tissue 5 mm. in diameter and the transplantation of a similar disk. The patient was one whose cornea had become opaque as the result of trauma following an explosion of acetylene gas. In this case the tissue employed to furnish the flap was from a boy of about 14 years who had lost the eye from the entrance of a piece of steel. The cornea was in good condition so far as nutrition was concerned. Both patients

were put under ether at the same time. The disk was removed by the von Hippel trephine, and subsequently the injured eye enucleated from the boy and the flap of corneal tissue removed by the same trephine and transferred. The disk was not touched by the hands of the operator. It was transferred on sterile gauze moistened in normal saline solution; it fitted perfectly in the opening. The healing was perfect and by first intention. The flap remained transparent for ten days, when vascularization began at the margin, and we finally had quite complete but not very dense opacification of the transplanted disk. In this case the vision was improved from nothing to ability to see the color of a garment, an improvement which was encouraging but was not at all brilliant.

In regard to the conclusions Dr. Walker has reached, I think they are a little too sanguine. They will, of course, be modified as his experience grows. I might repeat again, only well selected cases in which the transplantation can take place from an individual whose cornea is vigorous, present any possibility of a favorable outcome.

Dr. L. Webster Fox, Philadelphia: Transplantation of the cornea is not a new operation with me. Long ago I passed through the experimental stages, just as Dr. Walker has, and I congratulate him on his energy and perseverance. After trying out many experiments I concluded that corneal transplantation from animal to animal is a failure. These grafts unite with the corneal tissue and become opaque. I found that the greatest resistance is in the cornea of the chicken when transplanted to the human eye. It grew, but became opaque in time. After these experiments I concluded that the only way to succeed, if success be possible, is to transplant from human to human eye. My further conclusion is that this operation should only be carried out where an iridectomy cannot be performed, or where the cornea is not clear. One of my patients, a young boy in Philadelphia, has a cornea sufficiently clear to find his way along the streets. Many of my colleagues have seen the patient. The operation was performed sixteen months ago. The eye was lost from gonorrheal ophthalmia. It is in these cases, where the true cornea has been destroyed, that we are justified in operating. Seven operations were performed before we succeeded, and let me say that it is necessary to have a normal cornea as a base to nourish the transplanted cornea. If the graft is surrounded by scar tissue, it is absolutely impossible to keep the graft clear. I conceived the idea of taking one graft after another and transplanting them side by side until sufficient space was covered to make practically a nearly normal base. The last graft transplanted in the patient's eye was directly in the center of this new formed area of corneal tissue derived from these various grafts. He can count fingers at about 6 to 10

inches, and he can find his way about the streets. Later on I shall plant another graft at the side of this, apparently, normal one, and I believe the nutrition of the transplanted graft will absorb substance from its fellow and by this source of nutrition remain clear. Then and only then will the crowning event of ophthalmic surgery be achieved.

DR. SYDNEY WALKER, Chicago: Perhaps it is true that I am too sanguine, but from the results that I have had, at least on dogs, I think I have had reason to be sanguine. Of course it has been normal tissue grafted on normal tissue in these cases. My article will show that I have followed up instructions as regards a clean base, because that is quite necessary. The greater majority of the tissues present at least are clean and normal so that the graft may grow. In human cases I did not hope, as indicated in the literature, that heteroplasty would be successful. Dr. Fox has confirmed that. Our only hope seems to be by autoplasty or homoplasty, taking a part of the cornea from the other eye or the same eye and bringing it over to the center, if necessary, or by using cornea from an eye that is going to be removed for sarcoma of the choroid, dead fetus, etc., if the cornea is clear. I think that further work on this subject is really a necessity, not only from my own standpoint, but from the standpoint that it will become of value in the future, for I think there will be a great many more leukomatous cases, especially from trauma, than we have ever had before.

TENDON SHORTENING

AUTHOR'S METHOD

H. H. BRIGGS, A.M., M.D.
ASHEVILLE, N. C.

Convergence is the most interesting function of the human eyes save the visual act itself. In the process of evolution it is the last oculomotor function to develop, reaching its highest degree in monkeys, apes and man. But convergence is a means to an end. It is a motor function necessary to the higher and more intricate sensory function of binocular vision, which in man is a still higher instance of evolution. A wonderful function it is when we realize that before fusion is possible, normally constituted eyes must receive from every object two sets of sensations which must be blended into one through the fusion center by carefully coordinated ocular movements so fixing the eyes that images may fall on corresponding retinal areas. In order to obtain binocular vision, certain mechanical conditions are necessary, the fulfilment of which requires a function of wonderful accuracy. With eyes of normal acuteness of vision, a deviation of the visual axis of $\frac{1}{50}$ degree means an error in the relative length of opposing muscles of only $\frac{1}{230}$ mm., being $\frac{1}{6000}$ inch, or one-half the diameter of a red blood corpuscle.[1] How futile it is, then, to expect, from mechanical readjustment alone, the fulfilment of conditions rendering binocular vision possible. Let us not forget the importance of fusion and its stimulus in this question of muscle readjustment and binocular vision. The treatment of strabismus is a matter of

1. Jackson, Edward: Principles Controlling Operative Interference in Strabismus, THE JOURNAL A. M. A., Nov. 1, 1902, p. 1105.

neuromuscular dynamics, and the question should always be, How will the proposed readjustment influence the innervation of the muscle? An operation should aim at as exact adjustment of the visual axes as possible, considering the age of the patient, the muscle and tissues to be operated on, the refraction and accommodative effort, the probable amount of cicatricial changes with healing and with increasing age, and in a certain class of strabismus, the fact that the eyes have not hitherto participated in binocular function.

PRECISION

No operation requires greater precision than that for readjustment of ocular muscles. We appreciate this the more if we remember that for each millimeter a muscle is shortened or lengthened, from 3 to 5 degrees of rotation is affected, and that 1 degree of deviation, if uncorrected, means diplopia for small objects. To illustrate: A letter in the $^{20}/_{200}$ line of the test card subtends an angle of 50 minutes of an arc, the width of the bar composing the letter subtending an angle of 10 minutes or $1/6$ degree, so that if there should be an uncorrected deviation of one eye upward, say of 1 degree, which could not be corrected by the stimulus of fusion, the person would see two letters, one above the other, with a space between them equal to the width of one of the bars making the letter. Such diplopia would be disturbing indeed, and an error of 0.2 mm. in readjusting the eye would produce this diplopia, were it not fortunately that physiologic stimulus of fusion comes to our rescue and corrects such small errors.

In certain methods of advancement, especially those involving section of the tendon, it is difficult to determine within 3 mm. the exact point of final reattachment of the tendon, and this possible error of 3 mm. means from three to five times this amount, or from 9 to 15 degrees of uncorrected phoria or tropia. Is it surprising, then, that our ultimate effect is often far

from what was intended? It is consoling, however, to know that for every 5 degrees of phoria or tropia which we have failed to correct, it has meant a failure of perhaps only 1 mm. of adjustment. The readjustment, therefore, not only at the time of operation, but also after final healing, should be within 1 mm. of the calculations in keeping with muscle balance.

VARIOUS METHODS

More than sixty methods of increasing the power of an ocular muscle may be found in the literature. These may be divided into three classes: (1) those in which the tendon is severed and a new insertion made nearer the cornea; (2) those in which the insertion is not severed but the muscle shortened by tucking or resection, and (3) those in which the tendon is tucked or folded and the overlapped portion attached anterior to the insertion.

AUTHOR'S METHOD

Since 1909 I have used almost exclusively the following method of tendon shortening without severing the tendon and without the use of needle and suture:[2]

The method is applicable to any case in which shortening of an ocular muscle is indicated. The principle involved is the looping of the tendon and maintaining the shortening thus attained by means of a silver link (Fig. 1 *A*) clamped over the loop and retained there until inflammatory adhesion takes place between the advanced portion of tendon and the sclera. The link, which is elliptic, is made of silver wire (No. 20, Brown and Sharpe Standard). It is long enough to extend across the tendon, and wide enough to allow a small tendon hook with the looped tendon and overlying conjunctiva to pass through its lumen. The major axis of the link should be about 5 mm., the minor about 2 mm. Pure silver wire gives the proper amount of resiliency, retains its compressed shape, is easily removed, and seems to serve better than an

2. Briggs, H. H.: Tr. Sect. on Ophth., A. M. A., 1909.

alloy. To insure against the tendon retracting through the link, burrs may be punched on the inner surface of the link to act as barbs against the pulling action of the tendon.

A clamp forceps (Fig. 1 *B*) is used to grasp the silver link in its jaws and hold it in position over the tendon until the loop of tendon and conjunctiva is drawn up through the link, when by compressing the forceps the link is compressed sufficiently firm over the loop to maintain the loop the required time after the forceps have released the link and left it in situ. Passing entirely across the approximating surface of the jaws near the tips are transverse grooves deep enough to hold the long parallel sides of the silver link. The length of jaw to the angle is sufficient to accommodate the longest loop of tendon that may be desired. A ratchet maintains the jaws in any compressed position over the link.

A special tendon hook (Fig. 1 *C*) is used, differing from the ordinary small tenotomy hook in that its shank, 6 mm. above its angle, is bent backward in the plane of the hook at an angle of about 15 degrees. This bend allows the handle of the tendon hook to be held out of the operator's line of vision, and at the same time the hook may remain parallel to the tangent plane of the eye at the site of operation. The hook should be short enough to pass through the major axis of the link, and yet sufficiently long and strong to pull the tendon through the link, overcoming the pull of the opponent muscle, which may or may not have been previously tenotomized.

For removal of the link, scissors forceps with wedge-shaped blade above and flat blade below (Fig. 1 *D*) are used to divide each end, when the severed halves of the link may be removed with small tissue forceps.

After the usual preparation of sterilization and anesthetization, the operator, standing on the side of the eye nearest the muscle to be shortened, makes a small

opening in the conjunctiva on one side (the operator's right side) of the tendon near its insertion into the sclera, the distance from the insertion varying with the degree of advancement intended. An ordinary tenotomy hook passed beneath the tendon is held by an assistant, the clamp forceps holding the silver link is held over the tendon with one hand, and the special tendon hook passed down through the silver link is substituted for the hook held by the assistant. The tendon with its overlying conjunctiva is drawn up through the link to the desired degree, and the jaws of the clamp forceps forcibly approximated. If the degree of deviation is great, and the tendon loop to be made long, the conjunctiva may be incised, allowing the tendon only to be advanced to any desired degree. It may be necessary in excessive advancements to excise the greater part of tendon and tissues protruding through the link, leaving only enough to insure non-retraction of the tendon ends through the link.

The moderate edema of the tissues about the link immediately covers it so that no irritation is felt from the presence of the foreign body. Within twenty-four hours the strangulated tissues become white and begin to atrophy, and on removal of the loop, in from ten to fourteen days, they should be excised.

The advantages of this operation over the usual methods of advancement are:

1. Simplicity.
2. Elimination of needles and sutures.
3. Avoidance of tenotomy of the advanced muscle.
4. Absolute security from slipping or retraction of tendon.
5. Short duration of time of operation.
6. Less pain.
7. Less amount of plastic inflammation during convalescence.
8. Short period of convalescence.
9. Less scar and deformity.

Since the operation was devised in 1909, I have operated in 131 cases of phorias and tropias with satisfactory success generally, the only exceptions being

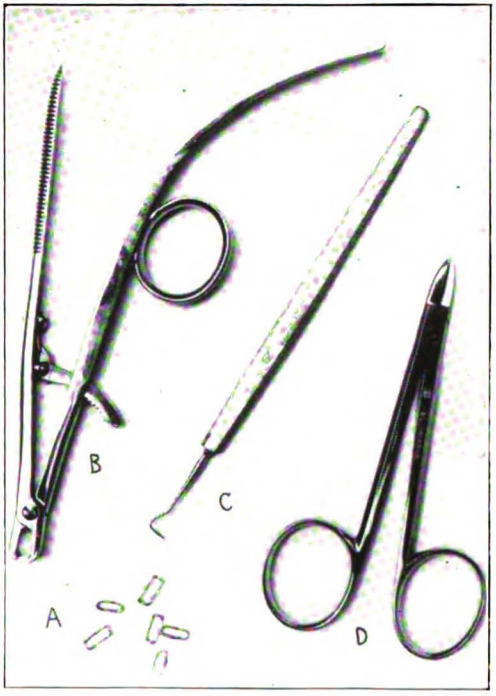


Fig. 1.—Instruments employed: *A*, silver link; *B*, clamp forceps; *C*, tendon hook; *D*, scissors forceps.

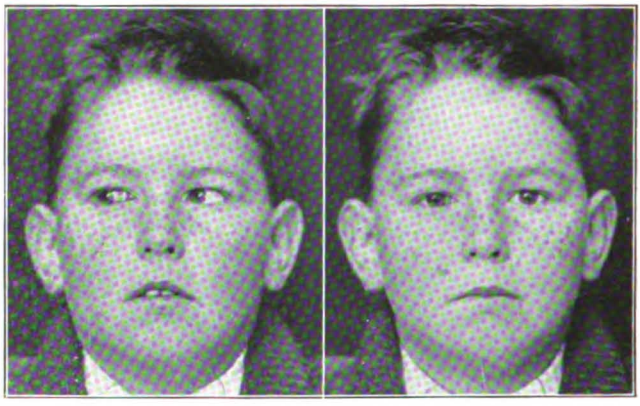
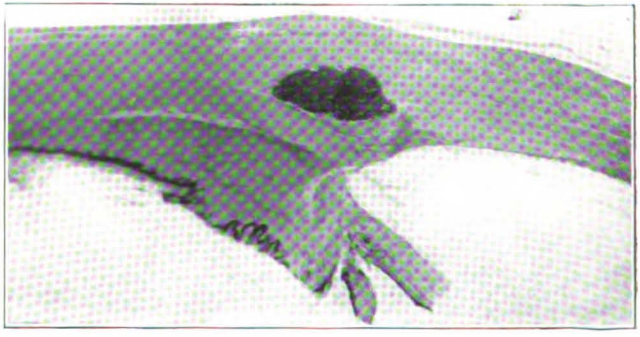


Fig. 2.—Condition of the eyes in patient one day after operation.



Wells' modification of Worth's advancement. Horizontal antero-posterior section. This double and twisted silk suture was inserted in an eye about to be enucleated in a direction at right angles to the long diameter of the muscle, and shows the depth of bite essential to prevent cutting out. (Illustrating Dr. Wells' discussion.)

in three cases, in which the link failed to hold. In the first case, the patient returned to the office on the day following operation, when the link could not be found either in the conjunctival sac or in the dressings. Whether it was not sufficiently compressed and slipped off the loop of tendon could not be determined. The muscle was immediately again operated on, and the usual good result followed.

Failure in the second case was due to imperfection of the link; it had been broken at one end, and, becoming loosened, was found in the conjunctival sac the following day.

Failure in the third case was due to the grooves in the jaws of the clamp forceps being made too shallow, allowing the link to slip out of their grasp. This operation was done in a demonstration clinic at the Southern Medical Association meeting in Atlanta last November, the imperfect instrument being borrowed from an exhibit for the occasion. The link came off a few minutes after the operation was concluded. In no other case has there been any slipping of the loop of tendon representing the shortening.

In no case has the advanced portion of tendon failed to unite with the sclera, at and immediately back of the original insertion of the tendon. This was found to be true in cases in which second advancements on the same muscle were found necessary—the tendon hook encountered no adhesion far back of the original insertion, as is usually the case after other methods of advancement. In no case has a vertical deviation resulted from operation on a lateral muscle, as sometimes happens when sutures are used and the anchorage on one side gives way.

Many patients prefer to have the eye unbandaged, and most patients complain of no more pain or discomfort than after tenotomy. One patient, a boy of 12, with intermittent exotropia, played baseball with eyes unbandaged the same afternoon and on each following day without seeming inconvenience.

Removing the link is the most difficult part of the operation — difficult principally because the link-cutting forceps are often not made accurately. The upper cutting jaw should be so shaped that it meets the lower jaw most firmly at the extreme tip, and it must be kept sharp; otherwise it will not cut completely through and sever the link.

Often failure in advancement operations is due to the character of tissues dealt with. The conjunctiva and subconjunctival tissues are extremely elastic and easily dragged away from their normal position. They may be cut through with sutures at the time of the operation, or later, by softening or sloughing, they become an unreliable anchorage, and militate against an accurate result.

The presence of sutures may induce infection, which in turn may add to the softening and insecurity.

The placing of sutures in itself is a somewhat dangerous procedure.

Howe[3] says, "One of the greatest difficulties in all these operations is that they require the anchorage to be made in what is usually described as the 'episcleral tissue'; but in reality the sutures are seldom or never in the episcleral tissue only. They are either in the conjunctiva, which is elastic and easily torn, or the needle enters the sclerotic itself," which, being but 1 mm. thick, is often punctured and the ciliary region invaded. He mentions a colleague who broke a small needle, the point remaining in the ciliary region and being extracted only after a careful and long search.

Worth[4] says:

> Sutures must have a firm hold in the sclerotic; no other mode of attachment can be depended on. My sutures traverse two-thirds of the thickness of the sclerotic. This exact depth which I have used for many years without accident is certainly safe, but if a suture were inserted through the whole thickness of the sclerotic it would probably lead to loss of the eye, and I have heard of such catastrophe.

3. Howe: Muscles of the Eye, **2**, 350.
4. Worth: Claud: The Operation of Advancement of a Rectus Muscle, Ann. Ophth., 1902, **11**, 377.

Apropos of this statement is the following from Howe:[5]

"The layer of scleral tissue near the cornea is only about 1 mm. thick. The smallest needles measure from 0.6 to 0.8 mm. A larger needle either does not penetrate the sclerotic, or if it does it is at the risk of passing through into the ciliary region." How difficult, then, it must be to pass a needle "exactly two-thirds"[4] through a membrane 1 mm. thick.

Woodruff[6] has seen two cases of perforation of the sclera, one of sloughing of the sclerotic and two in which the caruncle was drawn toward the cornea.

Wirts[7] reports a case of phthisis bulbi, following an advancement operation.

Cowgill[8] mentions an ulcer of the cornea in which there was no injury of the cornea at operation. The eye was preserved, but a leukoma extending to the center of the cornea resulted. He attributed the ulcer to the reduced nutrition from the severing of the conjunctiva and the pressure of the anchoring sutures.

Webster[9] stated that he had seen one eye lost of panophthalmitis, three or more cases of ulcer of the cornea, and in more than one case the operator's needle had penetrated the anterior chamber.

Noyes[10] saw needle points enter the anterior chamber.

Cogan, in operating on pigs' eyes, found he had perforated the sclera in half of them.

Zentmayer[11] has seen the needle enter the anterior chamber on two occasions, and he once had a perforation of the sclera and loss of vitreous from sloughing.

Woodruff[6] states that the reason serious accidents do not follow advancement operations oftener is that

5. Howe: Muscles of the Eye, **2**, 351.
6. Woodruff, H. W.: Shortening of an Ocular Muscle by Tucking, THE JOURNAL A. M. A., Aug. 5, 1911, p. 461.
7. Wirts: Ztschr. f. Augenh., January, 1910.
8. Cowgill: Ophth. Rec., **10**, 3.
9. Webster, David: Tr. Am. Ophth. Soc., 1908, **2**, 3.
10. Noyes, H. D.: Textbook, Diseases of the Eye, p. 172.
11. Zentmayer, William: Changes Occurring in the Refraction of Corrected Ametropic Eyes, THE JOURNAL A. M. A., Aug. 5, 1911, p. 470.

the operator, erring on the side of safety, prefers insufficient anchorage to perforating the sclera, and leaves us to infer that in operations of true advancement in which attempts are made of anchoring in the so-called episcleral tissue, the eye is endangered, on the one hand, or an insecure anchorage results, on the other.

Landolt,[12] who said that "the dosage of tenotomy is uncertain and from the dynamic standpoint its effects are unfavorable," might have added that the effect of advancements, as usually performed, is also, though to a less degree, uncertain.

While the conjunctival sac and the lacrimal secretions have certain bactericidal properties, wounds of the eye are prone to infection, and especially is this true when any foreign substance, such as silk or catgut, remains in the eye. The substance best tolerated in any part of the body, and especially by mucous membrane, is silver, which in the form of a small link is the only substance used in the author's method, and it is so placed that it does not even penetrate the tissues.

Some operators believe that by advancing a muscle above or below the original attachment a coexisting hyperphoria may, with the lateral deviation, be simultaneously corrected. It may be argued that the author's method (the same would apply to all tucking methods), leaving the insertion intact, precludes the possibility of effecting this dual result. While this is true, the operator is more than compensated for this lack by the assurance that by not severing the tendon and keeping the advanced edges in absolute juxtaposition, assured by the shape and length of the silver link, he not only cannot correct a deviation in the vertical meridian, in case of hyperphoria, but furthermore, he *cannot* produce a hyperphoria by miscalculation or by accidentally misplacing the tendon vertically in cases in which no vertical deviation exists, as often happens

12. Landolt in deSchweinitz: Diseases of the Eye, p. 552.

to the best operators after severing a tendon and attempting to advance it along the horizontal meridian.

The author's method also assures against the accidental production of cyclophoria. In an editorial, "Muscle Shortening versus Muscle Advancement," Savage[13] warns against the danger in advancements of accidentally turning the eyeball on its anteroposterior axis so as to throw unbearable strain on the obliques.

Leaving the tendon intact, aside from lessening trauma, insures, therefore, against induced hyperphoria, induced cyclophoria, and against the severed muscle retracting and producing a much worse squint than was first attempted to correct.

It may be said that the author's method does not allow the operator to watch the effect produced during the operation, and modify the tension of the sutures to meet the indication, as is done in the Prince and other methods of the pulley type. Concerning this, Jackson[14] says:

> This ought not be depended on to the neglect of careful calculation beforehand of the exact effect to be aimed at. Only when some unfortunate factor intervenes and the effect produced proves quite different from that expected is it wise to modify a carefully proposed plan of operation. Under the stress of operation with local anesthesia, the patient's answers as to the subjective tests become especially unreliable.

In the author's method the only trauma to the eye consists in making the smallest possible incision through the conjunctiva and subconjunctival tissues, barely large enough to admit a tendon hook to be passed underneath the tendon. The only manipulation otherwise is the traction of the hook on the tendon and conjunctiva in bringing them through the link. The entire traumatized area, including the incision, is drawn through the link and ultimately excised. Thus there not only is no suture in the tissues, but also the only foreign body present is one of silver, which clamps the tissues together rather than penetrates

13. Savage: Ophthalmologic Myology, Ophth. Rec., 1893, **2**, No. 9.
14. Jackson, Edward, in Wood: System of Ophthalmic Operations, **6**, 749.

them. There is less trauma than in any other operation done on the ocular muscle.

The time required will average about two minutes, and the amount of pain and discomfort is practically nil; far less than in any method of tenotomy.

ABSTRACT OF DISCUSSION

DR. WILL WALTER, Evanston: Any operation for shortening which tends to do away with the unsurgical procedure of sutures under tension, is worthy of study and trial. Those of us who have performed the O'Connor operation are, I believe, in agreement that the advantage of the heavy gut loop is to hold off tension while the advanced central tongue of tendon becomes firmly adherent, and if a gut would hold the loop long enough for adhesion to itself, no tongue of tendon and no sutures would be needed.

Advocates of the tendon-tucking types of operation also must agree that the friction resulting from the overlapping relieves the pull on the sutures while adhesions are forming. Then, too, the effect of prolonged bandaging, as employed in the usual advancement operation, is immobilization, and hence freedom from tension on sutures.

There is, therefore, underlying the ingenious operation of the essayist a fundamental principle of surgical technic, and a study of the successful operations for advancement and shortening throughout the development of the specialty will disclose the universal employment, consciously or unconsciously, of methods which tend to modify or to avoid this tension.

Distinct advantage would theoretically rest with those methods which allow freedom of motion and the employment of the eye in the new found relations of the globes to each other, and therefore operations of this type should excite our interest.

At the last meeting I showed some of you, and made preliminary announcement of, an operation without sutures, and this I had hoped, after further experiences, to demonstrate at this meeting; but I have been unable to do so.

I might say, briefly, that my operation employs two hollow gold tubes for the shortening. The tendon is split longitudinally and each strand is twisted on one of these tubes. The tubes are twisted in opposite directions so as to oppose forces to each other, and are twisted until the desired shortening is made. A smaller tube is then passed through both and they are locked together and left buried beneath the conjunctival and subconjunctival tissues.

Now the use of the silver links of the essayist may be a better plan than the gold tubes and my technic, but it occurs to me that the combination of that feature with the

distinctive features of my operation might prove ideal. Working together, we have established one thing, namely, the tolerance of the eye for such foreign bodies. Taking my experience with what the author has described, I shall at the first opportunity try out my operation, using the silver links instead of gold tubes, and I would like to cooperate with Dr. Briggs in working out a combined operation.

I have gone far enough to find that the gold tube may be left a very long time and cause no distress, and that finally it becomes free under the conjunctiva and may easily be removed.

The suggestions for the combination of features of my operation with that of Dr. Briggs would be these: (1) splitting the tendon and using two smaller silver loops; this enables one to correct tilting of the vertical meridians; (2) not cutting off the tendon loops, since they might slip and complications follow, and they do no harm; (3) burying the loops subconjunctivally; (4) leaving them a longer time, until they are free and may be released without cutting.

However, 131 cases is a goodly number, and three failures is a small per cent., and without experience it is presumptive to offer any criticism of the operation as described.

One cannot so easily subscribe to the preamble of the essayist in which it is said that for every millimeter of shortening there is a definite change in the degree of correction. This is certainly not the general experience of ophthalmologists.

Dr. J. W. Jervey, Greenville, S. C.: Dr. Briggs has perfected a procedure which in safety, simplicity, ease and celerity of accomplishment and attainment of good results, appears to me to leave little to be desired. It is an elaboration of the doctrine so freely expounded by Todd of Minneapolis and Savage of Nashville that muscle slack should be taken up and tendons should be left uncut. It is strange indeed that in spite of the definite detail and scientific accuracy which has of late years been introduced into ocular surgery, there yet are to be found men who will tenotomize a rectus, turn it adrift, with a "good-by everybody, *sauve qui peut,* anchor where you can" and let it go at that. I believe the day is coming when no man can look the world in the face and say, "I am going to sever a tendon." With this procedure in use I believe it will never be necessary to tenotomize to remedy a strabismus. By operating on the recti of both eyes, repeated, if necessary, any deviation, no matter how great, can be corrected without injuring a tendon. Dr. Briggs has enumerated the advantages of his procedure, but he has slighted an important one, to my mind, and that is the absence of conjunctival adhesions to the sclera after his operation, so that the operation can readily

be repeated. I have performed this operation fifteen to twenty times. Dr. Briggs has made an important contribution to ocular surgery, and I, for one, sincerely congratulate him and thank him for putting these possibilities within my reach.

Dr. David W. Wells, Boston: The surgical correction of heterotropia and heterophoria by a method which allows the patient to be up and about with both eyes open, is certainly ideal from the patient's point of view. In operating on 131 cases of phorias and tropias Dr. Briggs has certainly had sufficient experience to determine the value of his very interesting technic. This report, however, is so lacking in details that it is impossible for one to decide its value as compared with other well recognized procedures. Reports of this sort should state the number of cases of the different kinds of phorias and tropias, with the degree of each before operation and after, so, that one might judge in what class of cases it is especially indicated. In the tropias I am still quite well satisfied with the Worth advancement, with my modification of the scleral anchor, but have used it on only a few cases of phorias of high degree. But this necessitates the bandaging of both eyes for a week, and I should be glad to adopt something less arduous for the patient; but I must confess that the indications for this new procedure are not sufficiently clear. Does the essayist intend us to infer that it is a panacea? Is there no limit to its applicability. He states that from 3 to 5 degrees of rotation is effected for each millimeter of shortening or lengthening, but he fails to tell us how he measures the number of millimeters of the tuck, and how accurately his results substantiate this statement. My plea is that such reports be presented on a comprehensive schedule, giving results before and after operation by different measurements. Three years ago I presented such a report of sixty-five advancements, and I am extremely anxious that others should do the same, so that I may determine if there is a better way.

The author has collected a series of unfortunate results in advancement operations, quoting Worth's statement that he heard of the loss of an eye by the scleral suture perforating the sclera. I corresponded with Worth about this, and he said the case happened in the practice of a colleague, but that he was not sure the eye might not have been infected.

The scleral anchor is the difficult part of the Worth advancement operation, and doubtless the needle may at times penetrate; but that this necessarily causes trouble I doubt. In my own experience and that of the other ophthalmic surgeons of the Massachusetts Homeopathic Hospital, all of whom have adopted my modification of the Worth advancement, there has been no exception to first intention healing.

Three years ago I showed the section a so-called fixation fork which I insert through the conjunctiva into the sclera,

in order to get the necessary counterpressure as the needle travels through the sclera. In an eye about to be enucleated a double and twisted silk suture was introduced, as is done in advancement, and the photomicrograph shows the ideal depth.

Dr. Frank C. Todd, Minneapolis: This method of operation appealed to me as very simple and very useful in certain classes of cases, and accordingly I secured the instruments and tried it out. Now Dr. Briggs' results demonstrate to me that my technic was poor, and for your benefit I am going to ask Dr. Briggs to tell us wherein we may avoid the failures that I had. I want to try the operation again, but my percentage of failures so far has been 100, and joking aside, it seems evident to me that I failed somewhere in the technic. I was pleased to note that Dr. Briggs had three failures. The operation is exceedingly simple and is done very quickly. You draw the tendon and conjunctiva through in one mass and pinch them together. I pinched the link and the mass as hard as I could. I dressed the eye four days later in one patient and found the little clamp loose and just hanging by one edge. In the other case the clamp came out entirely and was lost. These were clamps sent by Dr. Briggs, so that I am sure they were all right. I am almost certain it is a matter of technic, and I hope we may have the benefit of Dr. Briggs' suggestions in that respect.

Dr. Archibald L. McLeish, Los Angeles: I have done Dr. Briggs' operation a number of times with gratifying results. In the earlier cases I found a decided difficulty in manipulating the little tendon hook underneath the clamp forceps. There is little room for the tendon hook to be pulled up through the link. The pull is oblique; you have to put the hook in at the side of the clamp and when you pull up along the hook you pull the tendon unequally through the loop. I had a modification of Dr. Brigg's instrument made by Hardy in Chicago, which helps to facilitate the passing of the hook. The action of these forceps is parallel. On the ends of the jaws there are little clips which embrace the ring, and by that means you can tilt the forceps aside as far as you please, and get free access to the link and hook. I have found that the application of the link is thus made much more certain. One can draw the tissues up equally above and below the clamp, and with absolute certainty.

Dr. George A. Moore, Palmer, Mass.: This operation of Dr. Briggs seems almost too good to be true. I have found to my sorrow that some eyes do not tolerate sutures, and have had the unpleasant experience of watching the process of extrusion. This operation would of course obviate such an occurrence.

I venture to mention a way of translating the degree of strabismic deviation into its estimated millimetric equivalent in tendon, making use of no new instrument except a special hook.

When the tendon is freed and raised on a strabismus hook, a hook of nontapering wire is substituted for the ordinary. This hook is made by bending a piece of steel or silver wire so that about three-eighths inch at the end is at a right angle. Make four or five ranging from 1 mm. to 4 mm. in diameter of wire. The only essential difference is that these must not taper; the reason will be obvious in a moment. If we wish to shorten 3 mm., a hook of 1-mm. wire is used; it is 3 mm. (good measure) around it. This hook is placed under the tendon; in the other hand a Prince forceps is held, with one jaw on either side of the hook but over the tendon. Downward pressure is made on the jaws of the forceps which are separated the diameter of the hook wire, and traction is made on the hook to bring the hook and tendon upward between the jaws of the forceps, and the tendon is thus wrapped around the hook like a tape around one's finger; the forceps jaws meet below the hook, holding the tendon; they are locked; the hook is slipped out of the engagement, leaving in the forceps a fold of tendon that you know is 3 mm., or three times the diameter of the hook wire. Before placing sutures the forceps may be balanced in the hand and the position of eye noted although it is assumed the operator knows before the operation how much he wishes to shorten the tendon. In extreme cases two loops are useful, so placed in relation to each other that the free ends of the two folds may be sutured without tension. If, later, a loop suture cuts out, the interloop sutures will have had time to produce union. My main point, however, is that in this simple way, loops of accurately known length may be made.

DR. H. H. BRIGGS, Asheville, N. C.: You will notice on the jaws of the forceps passed around that there is a millimeter scale for measuring the amount of tendon drawn up, remembering of course, that the actual amount of shortening is twice the distance the loop of tendon is drawn through the link. As to the indications for the operation, the scope of the paper did not allow me to go into this, but the operation is indicated in any case in which shortening of an ocular muscle is indicated. It is not applicable to any particular class of prophorias or tropias but may be successfully used for shortening a muscle, little or much, in any case where shortening is indicated. I am surprised that Dr. Todd did not succeed in securing the link on the tendon. On the inner aspect of the parallel sides of the link, little burrs are punched to prevent the tendon from slipping. If the links and the link forceps are properly made and the latter holds the former in its jaws securely and compresses it firmly over the loop of tendon and

overlying conjunctiva, there is no chance for the tendon to slip. Occasionally an imperfect forceps will allow the link to escape its grip during or prior to compressing the forceps, but if the instruments are properly made there should be no trouble from this cause.

The smallest possible opening is made in the conjunctiva and subconjunctival tissues alongside the tendon, the distance from its insertion being half that of the proposed shortening. An ordinary tendon hook passed through this incision under the tendon is held by an assistant. The special tendon hook passed down through the link previously placed in the jaws of the forceps is substituted for the assistant's tendon hook and gently pulls the tendon and overlying conjunctiva up through the link a distance half as many millimeters as the proposed shortening, which should be predetermined. The tendon is held rigidly without slipping at the point where it is clamped, and will remain there until the link is removed, in ten to fourteen days. Moreover, the attachment is very near the insertion and I have found no case where inflammation has caused the tendon and muscle to unite to the sclera more than a short distance from the insertion. I first learned of this in doing a second, and sometimes even a third, operation on the same muscle where a partial paralysis necessitated greater shortening than had been previously estimated.

Immediately after operation the slight edema of the conjunctiva covers the link so that it is scarcely seen. Consequently there is little irritation to the eyelids from the foreign body. Frequently the conjunctiva will grow over one or both ends of the link and slight dissection may be necessary when the link is removed, in ten to fourteen days.

TENDON TRANSPLANTATION OF THE EYE MUSCLES

H. W. WOODRUFF, M.D.
JOLIET, ILL.

By tendon transplantation is understood the transference of all or a part of a tendon from its normal insertion to a new one, in such a way that its physiologic function will be changed. The object to be gained by such an operation is not so much the restoration of function in a paralyzed muscle as the substitution of a normal one in its place.

Little in this particular field has been done in ophthalmology. Most cases of incurable muscle paralysis are dismissed as beyond surgical relief, or the operation of advancement of the paralyzed muscle with tenotomy of the contracting opponent is advised. In this way, in incomplete paralysis, good results may be obtained without doubt; but in complete paralysis of a single eye muscle I have never secured a satisfactory result by advancement with tenotomy. I have been a consistent follower of such writers as DeWecker,[1] Landolt[2] and Prince,[3] and I have no doubt that at present their operations, or similar ones, are followed by the majority of eye surgeons when time and treatment have determined that a paralysis is incurable. Dr. Jackson of Denver is the only American writer I know of who has suggested any radical departure from these methods, and it is due to his various references, notably one before this section in 1908, that I was induced to try tendon transplantation in two cases of abducens paralysis which recently came under my care.

1. DeWecker: Arch. d'ophth., 1904, **24**, 421.
2. Landolt: In Norris and Oliver's System, **4**, 66.
3. Prince: Am. Jour. Ophth., **19**, 259.

Tendon transplantation has yielded results in general surgery. May it not have a future in ophthalmic surgery? As early as 1770, Missa[4] restored function to a finger by substituting other tendons to an extensor which had been severed and could not be sutured. Velpeau[5] advocated this method in 1839.

In 1881, Nicoladoni,[6] at the annual meeting of the Society of German Naturalists and Physicians, showed before the surgical section a 16-year-old boy whom he had recently operated on for paralytic talipes calcaneus. As all the calf muscles were paralyzed, the tendons of the two peroneal muscles were divided and the central ends were implanted into a longitudinal slit in the Achilles tendon. He showed the members of the society that the contraction of the peroneal muscles produced plantar flexion of the foot, while the patient's gait was considerably improved. This brilliant result was unfortunately not permanent. The suture pulled out and the final result was nil. At about this time four or five others tried the operation with negative results, and it was given up by German and Austrian surgeons as early as 1886.

The first American surgeon to do the operation was Parrish[7] of New York, who in 1892 transplanted tendons in a case of clubfoot.

At present, tendon transplantation seems to occupy a place in the surgery of paralysis, although writers differ as to methods and results. Ryerson[8] of Chicago says:

> The two principal methods to be considered are those advocated, respectively, by Vulpius of Heidelberg and by Lange of Munich.
>
> In the Vulpius method, the entire healthy tendon, or a portion of it which has been partially split off from it, is sutured to the tendon or muscle which it is intended to strengthen. This method and its modifications have been so

4. Missa: Gaz. Salut., 1770, No. 21.
5. Velpeau: Operative Surgery, 1839.
6. Nicoladoni, cited by Waterman, J. H.: Med. News, July 12, 1902.
7. Parrish, in Da Costa: Surgery, 1908.
8. Ryerson, E. W.: The Surgery of Infantile Paralysis, THE JOURNAL A. M. A., Nov. 1, 1913, p. 1614.

extensively used by Vulpius and his followers that there would seem to be little doubt of its efficacy. In my experience, however, it has been found of value in only a limited field. It is useful, particularly in the arm and forearm, and in supplying power to the flexors and extensors of the toes; but when used where a considerable strain is to be thrown on it, as in the tibial or peroneal muscles, it shows a tendency to lose its efficiency. This may be due to the stretching of the paralyzed tendon, but in many cases cannot be referred to any definite cause. The operation is technically easier than the method of Lange, but it is not so certain in its results.

After a considerable experience with the method of Vulpius, I began, some years ago, to use the periosteal implantation of Lange. This gave satisfaction in many of the cases in which tendons were transplanted, but proved not strong enough for the silk suspension cords or ligaments used in cases of dropfoot.

The principal argument, then, seems to be over the method of implantation or attachment, and I will here quote from Mayer[9] of New York, to whose writings any one interested is referred. Mayer says:

A rational system of tendon transplantation must be based on an accurate knowledge of the anatomy and physiology of tendons and muscles. That our present systems are empiric rather than rational is shown by the marked discrepancy in the operative methods of prominent orthopedic surgeons. Thus Lange, on a basis of over 2,000 tendon operations, is radically opposed to Vulpius, whose experience is almost as great. Lange insists on the periosteal implantations of the tendon, and lengthens the tendon artificially by silk strands. Vulpius maintains the advantages of sewing the transplanted tendon to the paralyzed tendon as advocated by Nicoladoni in 1882.

In the same article Mayer says the principal cause of failure in addition to inefficiency of fixation is postoperative adhesions on account of disturbed relationship between the tendon and its sheath.

In the eye muscles, Parinaud[10] and Motais[11] were the first to utilize a part of one muscle to perform the work of another. The Motais operation has now become a rather popular method for the cure of ptosis.

9. Mayer: Surg., Gynec. and Obst., February, 1916, p. 182.
10. Parinaud: Ann. d'ocul., 1897, **117**, 12.
11. Motais: Bull. et mém. Soc. d'ophth. de Paris, 1898.

Edward Jackson[12] has operated for paralysis of the superior oblique by transplanting the superior rectus to a new insertion farther backward and outward from its normal insertion, thus relieving the tendency of the eye to turn upward, and at the same time increasing the power of intorsion and abduction.

Hummelsheim,[13] in 1907, performed several experimental operations on two monkeys by transplanting the nasal halves of the tendons of the superior and inferior recti to the attachment of the internal rectus after destroying its action by an extensive resection of its tendon. In the first animal there was no noticeable disturbance of motility after six weeks. The second animal died from some unknown cause, before any observation could be made.

Hummelsheim refers to a work of Marina in which he discusses the transplantation of the superior oblique for the removed internus or externus. Marina referred to Dr. Cofler as the operator, and said that he would report concerning the technic and the practical results in man. Hummelsheim, however, was not able to find any such report. His own experimental operation and the statements of Marina were sufficient to cause him to attempt the operation on the human eye.

A 12-year-old girl came to him with presumable congenital paralysis of the right abducens. The eye could not be turned outward beyond the median line. He transplanted the temporal halves of the superior and inferior recti and obtained an abduction of 30 degrees without any loss of vertical rotations. Binocular vision, however, was not obtained.

Hummelsheim[14] reports a second case one year later. This patient was a woman, aged 47, with an incurable acquired paralysis of the abducens, of eight months' duration. He performed the same operation with somewhat different technic and without tenotomy of

12. Jackson, Edward: Ophth. Rev., 1903, p. 61.
13. Hummelsheim: Ber. d. ophth. Geselsch., Heidelberg, 1907, p. 248.
14. Hummelsheim: Arch. f. Augenh., 1908, 62.

the internus, stating that he did not wish to alter the scope of the operation experiment. The result of the operation was parallelism when the eyes were looking straight forward. Double vision was abolished except on extreme abduction. In this case there was some limitation of the upward rotation, but fortunately not in rotation downward. As this paralysis did not appear until five months after the operation, he argues that it was not traumatic, that is, caused by his operation.

Stuelp,[15] Jan. 6, 1912, operated after the same method in a case of marked convergent paralytic strabismus. On the third day he noted that there was a marked improvement in the strabismus but no further active outward movement of the eye. This later was effected after three weeks more following a tenotomy of the rectus internus. Stuelp states that his result was functionally not brilliant but that the condition was improved.

REPORT OF CASES

CASE 1.—*History.*—Nov. 14, 1916, Dr. Charles Burkholder of Chicago referred to me a case of paralytic convergence in a girl, aged 20 years. She attributed her paralysis to a fall, when 6 years of age, in which she struck on the head behind the left ear. When 15 years of age she had had an advancement of the paralyzed muscle with tenotomy of the internus. This, she thinks, had improved her condition to some degree. About a month before Dr. Burkholder saw her she had had another tenotomy of the internus.

At the time of her admission to the hospital, she had 20 degrees of convergent strabismus when looking straight forward, but only the slightest perceptible movement of outward rotation (Fig. 1).

Realizing the hopelessness of the ordinary procedures and the repeated failures, I considered the advisability of transplanting the whole of the tendons of the superior and inferior rectus muscles to two points on the globe midway between the superior rectus and the externus, and between the inferior rectus and the externus, respectively. After reading of Hummelsheim's experiments and results in his two cases and the fact that the function of the superior and inferior rectus were not disturbed, I followed his method.

Operation.—Anesthesia was secured by subconjunctival injections of novocain and epinephrin over the sites of the four rectus muscles. The tendons of all four muscles were

15. Stuelp: Klin. Monatschr. f. Augenh., 1912, **50**, 467.

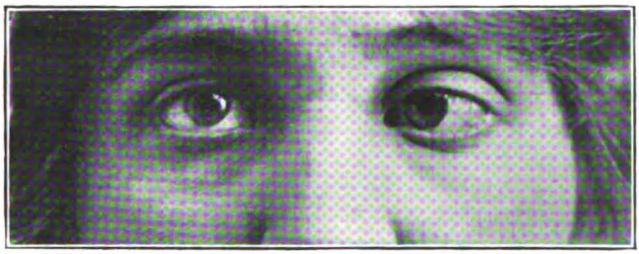


Fig. 1.—Case 1: Paralysis of the left external rectus; fourteen years' duration.

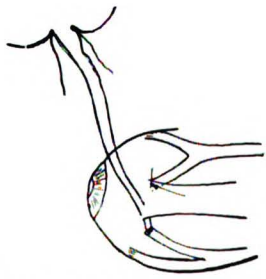


Fig. 2.—Method used by the author in performing partial tendon transplantation.

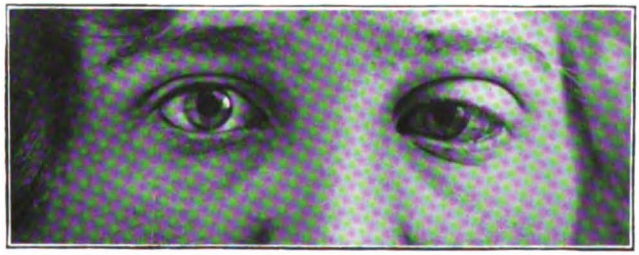


Fig. 3.—Case 1: Result after partial tendon transplantation of the superior and inferior recti muscles to the external rectus.

Fig. 4.—Case 2: Paralysis of the left external rectus, six and one-half years' duration.

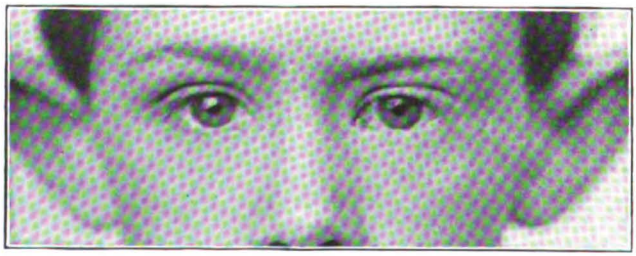


Fig. 5.—Case 2: Result after partial tendon transplantation of the superior and inferior recti muscles to the external rectus.

exposed by incisions through the conjunctiva and capsule. The outer halves of the tendons of the superior and inferior recti were separated for about 12 mm., and each tendon flap was sutured to the tendon insertion of the paralyzed muscle with 00 catgut treated with formaldehyd (Fig. 2). The internal rectus was freely tenotomized and the conjunctival wounds closed with silk. Both eyes were bandaged for three days. The operated eye was bandaged for one week. Figure 3 shows the results two weeks afterward. There was distinct improvement but still a slight convergence. This I attempted to relieve by shifting the remaining normally attached portion of the superior and inferior recti to positions slightly outward so that they would no longer act as adductors. This secondary operation was of doubtful success, so that at present the condition is substantially as in Figure 3, two weeks after the first operation.

CASE 2.—Dec. 26, 1916, I performed the same operation on a boy, aged 7 years. He had been brought to me the previous May with a marked internal paralytic squint of the left eye (Fig. 4). When he was 7 months old, the left side of his face was paralyzed, and the eye turned in at the time. He had had an operation of advancement and tenotomy four years before without result. It was suggested to the parents of the patient that there was a possibility of straightening the eye, which was finally attempted, Dec. 26, 1916, with the result shown in Figure 5.

The operation was the same as that in the first case. The result, however, cosmetically was better, but functionally the same, as there was no movement outward beyond the median line in either case. As both these patients were amblyopic in the paralyzed eye, the question of diplopia did not arise.

It will be noticed that in Hummelsheim's experiment on the monkey, he reports no loss of motility after six weeks.

Partial paralysis produced by operative interference is not exactly comparable to complete paralysis from central causes.

In his case of the 12-year-old girl there was ability to turn the eye outward to the median plane; therefore the paralysis was not complete. The securing of 30 degrees of abduction was a brilliant result, yet the patient undoubtedly possessed horizontal rotation of that amount before the operation.

In the case of the woman aged 47, he does not state the degree of rotation; but the result was good, as he

secured parallelism when the eyes were directed straight forward, and double vision abolished except on extreme abduction.

Stuelp himself claims an improvement only in the strabismus in his case; and my two cases would also tend to show that while restoration of function is not possible to any degree by partial transplantation, improvement in the strabismus is possible. The failure to secure altered function that is abduction in these two cases is probably due, as Mayer states, to adhesions, so that the superior and inferior recti do not act from the new point of insertion, but at the point at which the tendons were split, the transplanted tendon flaps acting more as ligaments to hold the eye outward, especial after the tenotomy of the internus.

Nevertheless, it would seem that the results in these five cases (omitting the animal experimentation of Hummelsheim) should place incurable paralysis in the operative class.

Whether or not work along these lines will lead to improvement in function, the static result is certainly worth while.

ABSTRACT OF DISCUSSION

Dr. F. C. Todd, Minneapolis (with moving picture demonstration): I wish to show a picture or two illustrating a case in which an operation of the kind described by Dr. Woodruff was performed. My experience has been much the same as Dr. Woodruff's, but you will see by the moving picture that some motion will result from the transplantation of the external half of the superior and inferior recti muscles.

The same principle would seem to hold true in respect to this case as in the cases described in the paper. It is a case of the congenital absence of the external recti. This patient gives a history in the family for some generations back of strabismus, and he is a man 60 years of age, with a strabismus of 95 degrees. His history showed that eight years previously his strabismus had been very much less, and I have a picture showing what it was seventeen years previous to the time I saw him. I did not know what the cause of the strabismus was until the operation. I operated on one eye, cutting the internal rectus completely. I could do

nothing else. I then attempted to do the zig-zag operation. Finding the absence of the external rectus, I attached the capsule of Tenon to the globe farther forward, as was described in the paper last year at Detroit. We had a limited improvement but it was not very satisfactory, and did not hold, and gave no motion. I then operated on the other eye later by completely tenotomizing the internal rectus, and did this operation described by Dr. Woodruff, separating the superior rectus and the inferior rectus and splitting it and attaching it to the globe on the outside. I then later did the same operation on the other eye. Now this man has not completely straightened eyes, but as nearly as I can get them; but he is able to see as he was not able to do before, and he is able to read and has some motion, which is improving. You will see two sets of pictures, in which the second shows some improvement of motion after some training.

Then I want to show a picture following that (because it fits in with this case), of a woman who had an operation for convergent strabismus in childhood, with complete tenotomy of both interni, and the strabismus which results from that sort of operation, and in which we could not find the internal recti anywhere. In the paper read last year I called attention to the fact that these muscles are really existing and in the capsule, and if you will advance the capsule you will find that you will get the use of these muscles as suggested in the paper of Dr. Woodruff. This picture shows that she has considerable movement in the direction of the cut muscles, so that there is some action in spite of the fact that they are not attached to the sclera, and I show it to compare it with the other case.

Dr. Arthur S. Tenner, New York: My experience with tendon transplantation is limited to one case. I presented that case two weeks ago before the eye section of the New York Academy of Medicine. The patient in that case was 19 years of age, with a congenital abducens paralysis of the left eye, which produced a convergent strabismus of about 30 degrees. The eye, as in Hummelsheim's case, could be rotated outward almost but not quite to the median line.

The operation that I performed was in almost every respect similar to the one described by Dr. Woodruff, with these exceptions. I performed no tenotomy of the internal rectus, thinking if that were necessary I could do it afterward; and secondly, the tendon flaps of the superior and inferior recti were sutured to the sclera somewhat in advance of the external rectus—about 3 mm. I got a very severe reaction in this case and there was some sloughing of the flaps, especially the superior. I attributed that, not to infection, but to the tension on the sutures. As in the cases presented, however, there was some improvement in the strabismus, quite decided, and I did get some increased motility outward,

and, as in Dr. Todd's case, that abduction increased as time went on. I cannot say that it is very great, and it is mainly due, in my case, to the action of the transplanted tongue of the inferior rectus, which probably got a better hold than the superior and probably less of it sloughed away. At any rate, the action is downward as well as outward.

While the results of these operations leave very much to be desired, I think they are sufficiently satisfactory, especially as in the case shown by Dr. Todd, to encourage further efforts in this direction.

As the result of my experience I would venture to make the following suggestions: To lessen the reaction after the operation it might be sufficient, it seems to me, to transplant only one muscle, or only one tongue of muscle; that is, the external half either of the inferior or superior rectus, depending on which seemed to have the stronger action. Another suggestion that I would venture to make is that instead of suturing the tongue of muscle to the external rectus, the paralyzed muscle, I would suture the tongue of muscle farther forward, nearer the limbus, because, on theoretical grounds, we should get better abduction if this were done.

Dr. Meyer Wiener, St. Louis: A few years ago I conceived the idea of solving this problem for the relief of paralysis of the external rectus, and I performed a number of experiments on dogs in the surgical laboratory of Washington University. I operated on five dogs, excising the externi in both eyes, leaving one as a control. and transplanting the outer half of the tendons of the superior and inferior recti to the insertion of the externus on the other. The results seemed very good. After I got ready to report my cases I found Hummelsheim's report. He had already reported his case. Afterward, however, I had occasion to operate on a patient with paralysis of the externus in which I used this operation with a modified technic. Instead of doing it subconjunctivally I exposed the three tendons. It is very much easier in the human eye than on dogs. This patient had complete paralysis of the externus, and when I last saw him he had an excursion of 20 degrees outward past the median line. The patient was followed for some time by Dr. Barnes of Enid, Okla. He had 25 degrees then, and the last time he saw him, he had 30 degrees excursion past the median line.

Dr. H. W. Woodruff, Joliet, Ill.: I congratulate Dr. Todd. His demonstration shows that he secured a magnificent result. The necessity of doing a tenotomy of the opposing muscle, it seems to me, is very great, because of the tension under which the transplanted flaps are held. Dr. Tenner advises placing the new insertion of the flaps farther forward than the insertion of the paralyzed muscle. Of course this adds

to the effect, but also increases the tension, which is considerable, while by fixing these flaps to the thick portion of the sclera where the external rectus is attached, and by doing a tenotomy, you will have a minimum amount of tension. As far as the reaction following the operation is concerned, of course it is considerable, but not at all alarming in any way. The operation is extensive, really four operations in one, the tendons of all four recti muscles being exposed—you might call it a double advancement operation and a tenotomy—so that it makes a very extensive and somewhat tedious operation; but the result justifies it.

LYMPHATIC-NODULAR KERATOCONJUNCTIVITIS (PHLYCTENULES)

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Phlyctenular keratoconjunctivitis has always impressed me as a distinct ocular evidence of a systemic dyscrasia; in other words, these nodular exudates indicate a weakened, anemic constitution. The impoverished patient requires more than general tonics, something more definite and specific in the line of treatment. As an increased heartbeat would be suggestive of some abnormality, so I take this eye condition to be a symptom of some abnormal constitutional condition. As interstitial keratitis is a manifestation of inherited syphilis, these nodular lymphoid infiltrates are evidences of a strumous diathesis or tuberculosis — if not tuberculosis, then something very similar, bearing in its symptomatology a striking resemblance to tuberculosis. These nodular exudates are warnings, and not outspoken evidences of tuberculosis. The absolute certainty, as clinical evidence of it, must remain unanswered for the present. The proof is not sufficient to warrant such an assertion, yet in the large percentage of these cases it distinctly gives evidence of an intimate association with tuberculosis. We all know of tuberculous cases undergoing spontaneous arrest, and these may be some of the cases that give symptoms accompanying tuberculosis but not made manifest by our present methods of examination. Of course further evidence must be forthcoming, but I believe a proper interpretation of this local sign will be a great aid to us in saving and restoring vision.

Since the vision which has been lost depends so much on the location of the resulting corneal opacity, it is not possible to generalize in regard to it.

It is by a thorough and complete physical examination of all cases showing these nodular lymphoid deposits on the corneoscleral border, especially when ulcerating, that we can get some evidence of tuberculosis in one of its many manifestations. In some of the thirty-nine cases which form the basis of this study we have distinct and clear evidence of tuberculosis.

I would not style the nodules tuberculous simply because giant cells are seen in some of their sections. If organisms are found they are a secondary infection, due to the lymphoid cellular exudates breaking down. This nodular cellular infiltrate is a manifestation of a disease and not the cause of disease. There is an underphysical tone, a latent tuberculosis, that causes nodular infiltrates to appear on the corneoscleral border. To me it is an exhibition of a tuberculous condition or one very closely allied to tuberculosis — call it scrofula.

In seven of the thirty-nine cases, pulmonary tuberculosis was found. I hold that when tuberculosis is fully established we shall not find this eye condition. These nodular exudates are seen only in a beginning tuberculous infection and not when tuberculosis is fully established. It is more of a manifestation of tuberculosis, especially in childhood, though we saw these cellular infiltrates in four cases in adult life.

In sixteen cases we found some form of tuberculosis. Fourteen of the patients had tuberculous adenitis. One of the sixteen had tuberculous wrist joint; one had a tuberculous hip.

Eight of the patients, while not having positive pulmonary signs, had suggestive lung involvement, as apical moist râles, impaired notes, dull percussion, and spotty infiltrates. One had an indurative mediastinitis. Three, from roentgenographic reports, showed upper apical infiltration. One had tuberculous arthritis.

Thirty-two of the patients had positive von Pirquet reactions; fifteen of them +, ten + +, four + + +, and two had a slight positive reaction.

Dr. J. H. Elliott[1] of Toronto says, "It has been our experience that every child that has phlyctenular conjunctivitis has given a tubercular reaction."

In four of the cases the tubercle bacillus was found in the sputum. Three of the patients were sent to the state sanatorium for tuberculosis.

Eighteen of the patients had adenoids and diseased tonsils. The interesting point about these cases was the fact that in microscopic sections the tubercle bacillus was found in six cases.

The eczema seen in these cases is caused by the profuse lacrimation, and the constant epiphora is the irritant to the skin and nares. Nasal obstruction aids the eczema and the excoriation of the skin surfaces. Improper nasal respiration in the upper air passages, caused by congestion and intumescent turbinals, prevents the proper flow of tears to the nasal cavity. The mucous membrane of the tear duct and its sac becomes swollen and the lumen is narrowed, so that the tears do not have proper drainage to the nose. The eczema is a secondary factor, an association and not a cause of the eye condition.

Thirteen of the patients had a negative Wassermann reaction. Two had positive reactions; one of the latter patients, aged 20, had a nodular exudate on the conjunctiva with slight photophobia, lacrimation and a negative von Pirquet reaction. Roentgenoscopy revealed infiltration beneath both clavicles. Because of the negative signs of a tuberculous involvement, and of a positive Wassermann reaction, the patient was placed on antisyphilitic treatment. The condition of the second patient, aged 25, was similar to that of the first, and the same treatment was given.

1. Elliott, J. H.: Tr. Natl. Assn., Study and Prev. Tuberc., 1916, p. 45.

In seven of the cases some member of the family had tuberculosis, four in parents (three paternal and one maternal), two in sisters, and one in a paternal uncle.

The age of these thirty-nine cases varied from 1 year to 30. There were thirty females and nine males. Eighteen patients were white and twenty-one colored. The cornea and conjunctiva were involved in thirty cases. Corneal ulceration was present in eighteen. The conjunctiva was involved in nine cases. Both eyes were involved in twenty-one cases, the right eye twenty-nine times and the left eye eleven times.

When local treatment was required for corneal ulceration we used atropin in 1 per cent. solution and protective glasses. When there was no corneal ulceration, sodium borate drops were ordered.

I have carefully gone over the cases and itemized each particular case for study: After the eye was seen and diagnosis made, the patient was referred to the Phipps Clinic for Tuberculosis for a physical examination and a skin test. It is needless for me to say that the work in that department is so thorough that the diagnosis will not be questioned. In fact, when one is working on a special subject, sometimes the enthusiasm gets the better of discretion, and one is liable to err. The reference of these cases to another department is bound to result in more accurate reports, and the results in the long run will be more valuable.

With their varied experience, the physicians in this clinic are unusually competent to make physical examinations and are especially skilled in giving the tuberculin injections. The dosage, temperature, pulse and respiration are followed more accurately. It is important to know how to administer tuberculin. It is a dangerous drug; and unless used skilfully, more harm than good will come from its administration. The minimum dose was 0.000001 gm.; to the small dose often repeated I think is due the success in giving

tuberculin. The tuberculin used was the bouillon filtrate and was made in the Phipps Clinic for Tuberculosis. I feel confident that had it not been for the skilled and expert administrations of tuberculin in the clinic, the results would not have been what they are. Unless the ophthalmologist has had experience in this line of work, it is better to have the work done by men who are doing that kind of work every day; they are more capable of judging the proper dosage, and the interval in time.

At least forty injections are necessary in an average case, and prolonged tuberculin injections a necessity for good results. Any rise in temperature is an indication of too large an amount of tuberculin, and with any redness or soreness at the point of injection, the dose should be diminished. Small doses often repeated constitute the treatment; this is so important that I wish to lay special emphasis on it.

In some of the thirty-nine cases we had a positive von Pirquet reaction, but the physical examination showed "no active pulmonary signs"; nevertheless we tried tuberculin to see if it improved the ocular condition. We were surprised to see the nodular exudate disappear. To satisfy the patient, sodium borate drops were ordered. This improvement in the patient I attribute to the tuberculin. These cases show no manifest tuberculosis, but probably have some latent form. If tuberculin improves the condition, there must be some association between the nodular exudates and tuberculosis.

When the patient was under tuberculin treatment, relapses were very few; in fact, there was only one distinct case, and that was very slight. When other methods were employed, recurrent attacks were often seen.

Some of the patients returned for observation; the eyes appeared in good condition, there were no active inflammatory signs, and the general remark of the patients was that they were much improved.

It is necessary in some cases and at certain intervals to give another course of tuberculin injections. By this means we are more than able to hold the corneal ulceration in check and prevent opaque areas through the cornea. I feel confident that with the proper administration of tuberculin, accurate taking of pulse, temperature and respiration, and the general condition kept under surveillance, we can get good results from this way of handling the cases. It is far from perfect, but better than giving general tonics, with no special medicine given for a special ailment. The word "tonic" would not carry far if one were questioned why and for what reason the tonic was given.

Norman[2] says:

> Under tuberculin treatment the process of ulceration was arrested very much more quickly than by ordinary methods alone, and the resulting area of corneal opacity was correspondingly diminished. Another advantage is a remarkable freedom from relapse, but to insure this we find it necessary to give the full course of fourteen injections in all cases.

The eyes were carefully observed and notes made after each injection, stating progress, or retrogression, or whether the eye was quiet.

The Department of Laryngology handled the cases of adenoids and tonsils. This removal was a great aid in the treatment. The examination of the adenoids and tonsils for microscopic diagnosis was thorough. Finding six cases with tubercle bacillus I think is exceptionally interesting. Their interest in the cases made the examination thorough and complete. Six cases were found positive for tubercle bacilli in the sections.

Miss Grace Pearson, our social service worker in the Department of Ophthalmology, did painstaking work in steering patients to the various departments for examinations and treatments. Had she not displayed so much interest in the work, our study would not have

2. Norman, A. C.: Tuberculin in Ophthalmic Practice, Hospital, London, 1915, **58**, 9.

been complete. Her arguments with the patients about the importance of the treatment brought them back. Her observations and reports of housing conditions of the patients, and her instructions to the patients about the best way to live hygienically, the best way to get fresh air, the proper food and the like made her aid invaluable. I am indebted to her for her interest displayed in following these cases.

SUMMARY

I am not claiming this manifestation as a certainty of tuberculosis, but that in a majority of cases, if we use all methods of diagnosis we will find some evidence of symptoms that go with tuberculosis. In the seven cases showing active pulmonary signs (four of the patients having the tubercle bacillus in their sputum) it is satisfying to know that we aided the patients in getting the proper treatment.

The eye manifestation will at least make us think of a tuberculous involvement and will aid us in trying to check a tuberculous tendency. Tuberculin injections when properly given are a great aid in our treatment. I have outlined eleven distinct points:

1. The nodular cellular lymphoid deposits, ulcerative and nonulcerative (phlyctenules) of the corneoscleral border are local eye manifestations of a constitutional dyscrasia, strongly suggestive of tuberculosis—if not tuberculosis, then something very closely associated, bearing a striking resemblance in its symptomatology to tuberculosis.

2. While the microscopic sections of these nodules show giant cells, it would not follow that the nodules are tuberculous, but rather indicate that the cellular infiltrate is a manifestation of the disease and not the cause.

3. The treatment has been hygienic and sanitary: sodium borate drops in nonulcerative case, and in corneal ulceration 1 per cent. atropin and protective

glasses; tuberculin bouillon filtrate given in doses varying from 0.000001 gm. to 15 mg. At least thirty-five to forty treatments are demanded, and as high as fifty or more. Prolonged tuberculin treatment is important, and in doses averaging 0.0001 mg. I think at stated intervals it is advisable to renew tuberculin treatments. During tuberculin treatment eyes rapidly improve, and there were no recurrent attacks with the exception of one case, which was slight. Temperature, pulse and respiration were carefully watched. Any rise or tenderness and redness at the point of injection would indicate too strong a dose. The patient should *sustain toxins given* with *no reaction;* this is important.

4. Of the thirty-nine patients seven had pulmonary phthisis.

5. Sixteen had some form of tuberculosis as cervical adenitis or tuberculous bone.

6. Thirty-two had a positive von Pirquet reaction.

7. Eighteen had adenoids and diseased tonsils; the tubercle bacillus was found six times in microscopic sections.

8. Four showed tubercle bacilli in their sputum.

9. Eight physical examinations were suggestive of some form of lung involvement.

10. Thirteen had a Wassermann test with eleven negative.

11. Seven had a member of their family with tuberculosis.

Through the kindness of Dr. Robert L. Randolph, I was privileged to use these cases in his clinic at the Johns Hopkins Hospital.

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THE PATHOGENESIS OF OPHTHALMIA ECZEMATOSA

A PRELIMINARY REPORT

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The acute interest of the profession in phlyctenular disease has been maintained for some time past, for two principal reasons: first, because the disease itself offers so much calculated to attract continued attention — its prevalence, complications, sequelae, and frequent recurrences — and second, because the problem of its pathogenesis is still *sub judice,* no one claim having succeeded in gaining a firm foothold in science.

As long as the etiology of ophthalmia eczematosa remains obscure, treatment will be empiric. Had we succeeded in accidentally finding a specific for the cure of phlyctenular disease such, for example, as we have in quinin for malaria and in mercury for syphilis, the fact that we have no absolute knowledge of the real causal factor would be only of academic interest; but, as will be seen, we have not been fortunate in discovering a specific as yet, and hence any investigation aimed at the finding of a cure of this troublesome malady must be in the form of a search for the causal factor of this form of ophthalmia.

Ophthalmologists throughout the world became much interested when a few years ago the teaching was promulgated that ophthalmia eczematosa, or phlyctenulosis, is due to either the tubercle bacillus or its toxins. Naturally the recognition of tuberculosis in some form as the cause of phlyctenular disease led to the application of a supposedly rational therapy. The

entire antituberculous therapy, as tuberculin, ferruginous tonics, fresh air and overfeeding, was utilized against this disease, but unfortunately with no special result. The fact that the von Pirquet and other cutaneous tubercle tests yielded a surprisingly large number of positive reactions justified that therapy, but the comparatively insignificant clinical results certainly prove practically conclusively that the etiology of ophthalmia eczematosa is not tuberculosis.

The only other important theory which has been advocated by competent observers is that the disease is due to intestinal fermentation. As will be seen later on, therapeutic experiments to confirm this hypothesis have proved negative.

It seemed to me that in this era of advanced pathology the inability to demonstrate the underlying cause of any form of ophthalmia must stand out as a reproach and that some light must be reached if a systematic search for it be made. That such a road is not devoid of difficulties is known to every one who has tried to secure diagnostic laboratory data of this disease. Might it not be that perhaps in this case the microscope and serologic tests are not the signs of the proper road? I did not answer that question when, a few years ago, utilizing a clinical material which exceeds 500 patients, I undertook to make an inquiry into the cause of ophthalmia eczematosa.

In presenting to the profession the results of this investigation no claim is made that this report is exhaustive, nor is it the intention to submit a detailed recapitulation of the scientific labors on this subject available in contemporaneous literature; but the mere narration, it is believed, is sufficiently convincing to show that the evidence, new though it be, is based on undisputable facts.

To begin with, the doubt of the tuberculous origin of the disease, justified by the repeated observation that patients treated for tuberculosis fared no better than those given other routine therapy, should cause the

elimination of this theory from all calculations; but this, too, was included in the projected investigation.

Surgical therapy, and the use of vaccines and antitoxins have shown so conclusively negative results that these early were eliminated, not only as curative agents, but also as measures to indicate the causal factor or factors.

A plan was adopted to try with great care any drug at all likely to benefit the disease itself or at least its symptoms, on the presumption that if a really valuable agent be found an explanation of its rationale could possibly be arrived at and the desired goal reached. To prevent error, the routine practice of administering several drugs at one and the same time was abandoned, and each drug selected was studied singly. Boric acid, argyrol, protargol, silver nitrate, zinc sulphate, yellow mercuric oxid, mercuric chlorid, ethylmorphin hydrochlorid (dionin), atropin and similar drugs were subjected to thorough and prolonged therapeutic tests. It is evident that practically the entire ophthalmic, bactericidal and mydriatic armamentarium has been exhausted. The result of this inquiry can be given in one brief sentence: Not one of these drugs, with the single exception of atropin, had any real influence on the course of the disease. The findings are based on control observations, namely, that the drugs named, atropin excepted, had identically the same therapeutic results as those seen from the application of placebos. Atropin gave decided symptomatic relief, and in all probability was not without some curative influence.

It is interesting to note, however, that while the corneal phase of this disease does not respond to our classic bactericidal treatment, true corneal ulcer becomes clean and covered by epithelium; but this is as far as one can attain, for it continues to remain in a state of irritation, not unlike a nerve ending disturbance, similar to herpes zoster ophthalmicus, but milder than in this condition until the clinical entity has run its course.

In view of these results, all drugs were discarded and topical treatment restricted to the use of atropin irrespective of whether the cornea, conjunctiva or limbus was involved.

The evidence of the ineffectiveness of topical therapy clearly shows that the condition under consideration is not a localized disease, but a local manifestation of a systemic disturbance. The finding of the true underlying systemic cause, therefore, became the most important problem. The question which presented itself was by what method the desired result might be attained, since the aimless employment of the entire diagnostic armamentarium could have led only to confusion. Physical examination of the patients yielded the following data, which gave some index as to the best manner of search:

The greatest prevalence of the disease is between the second and twelfth years of life. Patients under 2 years of age are rarely seen. Seventy-five per cent. of the children were fairly well nourished. It is noteworthy that some of the most severe cases seen were in plump, ruddy children, to all appearances presenting an excellent degree of health.

This does not bear out the assertions of certain authors that phlyctenular disease is usually observed in anemic children with what is best described as a tuberculous habitus. The sexes were equally represented. The majority of the patients belonged to the poorer classes in which the families are large, and in which the environment from a hygienic standpoint is not wholly bad, except that individual parental control is exceedingly limited, the children being left almost entirely to their own resources. Adenopathy was manifest in the submaxillary, anterior and posterior cervical regions, the lymph glands being plainly discernible on palpation but rarely painful on pressure. The tonsils and adenoids were frequently enlarged and diseased. Skin and hair were characteristically dry. Erosions around the nostrils and behind the ears, espe-

cially where the two skin surfaces come in contact, were seen frequently. These erosions, which proved to be an intertriginous eczema, were often observed also in the inguinal folds, especially in very young children. Seborrhea of the scalp was not infrequent in the same class of children. A history of cough and loss of weight was seldom secured. Urinalysis, with special reference to the presence of albumin, sugar and indican, invariably gave negative results.

The routine eye examinations for this class of cases revealed nothing unusual as regards the clinical picture of the conjunctiva, limbus or cornea, and presented no statistical data of special significance. Could these manifestations be explained by tuberculosis? The most thorough search to sustain such a diagnosis by all available clinical agents proved futile. While there is no lack of ophthalmologists who still believe tuberculosis or tuberculous toxins to be the cause of phlyctenular disease, there are authorities who have emphatically rejected this theory.

Macnab,[1] quoting Axenfeld, says:

> Axenfeld has thoroughly disposed of the contention that phlyctenules are true tubercular lesions, and Mueller, in a series of transplantation experiments in which fresh phlyctenules were introduced into the anterior chamber of rabbits, failed in every case to produce a typical tubercular lesion.

John W. H. Eyre,[2] hunterian professor and examiner in bacteriology of the Royal College of Surgeons, in his hunterian lecture on tuberculosis of the conjunctiva, says:

> That many of those subject to phlyctenules give evidence of the existence of tuberculous infection in some portion of their tissues is no evidence that the conjunctiva lesion is also tuberculous, and until some responsible observer has demonstrated the presence of the tubercle bacillus in an extended series of phlyctenules, I see no valid reason for regarding phlyctenulosis of the conjunctiva per se as a form of tuberculosis of the conjunctiva.

1. Macnab, Angus: Diseases of the Cornea, New York, William Wood & Co., 1907.
2. Eyre, J. W. H.: Lancet, London, 1912, **1**, 1319.

L. V. Hamman,[3] chief of the Phipps Tuberculosis Dispensary of Johns Hopkins Hospital, and co-author of Hamman and Wolman's work, "Tuberculin in Diagnosis and Treatment," says that if phlyctenulosis is a tuberculous manifestation it certainly some time should show the presence of the tubercle bacillus, and that the tuberculous toxin theory is fast losing ground.

George S. Derby[4] of Boston, who has treated a great many of these conditions with tuberculin, says:

> I have had considerable experience with tuberculin in the treatment of phlyctenulosis, and I feel doubtful whether any of these cases have been much benefited by it.

Brown and Irons[5] of Chicago recently reported on 100 cases of disease of the uvea, and in only two cases did they have a focal reaction with the subcutaneous test. This demonstrated the rarity of tuberculosis of the eye.

In common with the most universally accepted teachings, I have long abandoned the use of the skin test after the second year of life. In patients under that age only 35 per cent. gave a positive reaction, and that usually was a delayed local reaction, while the subcutaneous tests, which have been tried in a relatively small number of cases, rarely proved positive. Therapeutically the topical and subcutaneous use of tuberculin proved without effect. Tuberculosis as a factor in phlyctenular disease can therefore be absolutely set aside.

Attempts to secure cultures from phlyctenules have not given satisfactory results, owing to the great difficulty in securing unmixed material from the papules even before they ruptured. As a rule the same flora was encountered which is commonly found in all conjunctival sacs, namely, staphylococcus, pneumococcus, diplococcus, *Micrococcus catarrhalis,* xerosis bacillus and others. Reliable workers have found the contents of unruptured phlyctenules to be absolutely sterile.

3. Hamman and Wolman: Tuberculin in Diagnosis and Treatment, p. 209.
4. Derby, G. S.: Disease of the Optic Nerve in Myxedema, THE JOURNAL A. M. A., Sept. 21, 1912, p. 1045.
5. Brown and Irons: Paper read before the Chicago Ophthalmological Society, 1916.

While syphilis suggests itself as a possible factor, though no one has so far seriously considered it, this can be eliminated, since in the available material no data for such a diagnosis were even remotely present.

Finally, the removal of enlarged and diseased tonsils and adenoids for the purpose of influencing the ophthalmia, which had been strongly advocated some time ago, was subjected to a thorough test, but in spite of numerous radical enucleations no appreciable effect on the course of the disease could be demonstrated.

There remains the theory of intestinal putrefaction, which even at the present time has many advocates. The histories of all patients under observation show that their parents have seldom observed offensiveness of the stools. I made the Obermeyer test for indican in twenty-five cases, but no trace of this was discovered in the urine. This, however, need not be accepted as conclusive evidence for the absence of intestinal putrefaction. Though intestinal putrefaction could not be demonstrated through urinalysis, a number of patients were treated on the assumption that the intestinal disturbances actually existed. Suitable drugs, such as salol, calomel, acetylsalicylic acid, sodium salicylate, bismuth, sodium bicarbonate, liquid petrolatum and lactic acid bacillus either in pure bouillon culture, in tablet or in powder form, were administered, each single drug being subjected to a prolonged test.

It is interesting to note that, of all of the drugs used, calomel appeared to produce the most favorable results. This led to the adoption of an empiric routine, namely, atropin locally and calomel internally, as the most effective medicinal therapy.

It may be added that for some time the favorable results obtained from this combination encouraged the conviction that intestinal putrefaction was the solution of the problem, and the tentative acceptance pointed to the next step to carry the treatment to

logical finality through the addition of the appropriate dietetic measures.

When the parents of the afflicted children were questioned, it was learned that fully 98 per cent. of the patients consumed an excessive amount of carbohydrates, especially in the form of sweets. At first I was inclined to pay scant attention to this feature. Further questioning, however, elicited the statement that these very children had little desire for plain, nutritious food. With a view of ascertaining whether or not this faulty diet had any decisive influence on phlyctenular disease, a practically carbohydrate-free diet was instituted in a series of cases as the single and sole method of treatment, excluding even the topical use of atropin, with the gratifying result that the children so treated did far better than with calomel and atropin alone. Later medication and diet were combined with exceedingly satisfactory results. The diet prescribed consisted of plain foods, as bread, butter, milk and eggs. This dietary cannot be regarded as ideal, but was the best available for external reasons.

These results were so striking that many interested colleagues who were familiar with this series of experiments admitted the striking improvements, but questioned the direct relation of the dietary to the favorable phenomena. As I was convinced that the dietetic regimen alone and unaided was the principal cause of the favorable course of this disease, another series of experiments was made which left no room for further doubt. A number of children whose affections had been under complete control were taken off the dietetic regimen, and given foods rich in carbohydrates. Almost at once this disease reappeared with the acuity of a pronounced relapse. These experiments were repeated at frequent intervals, and invariably yielded the same results.

The experience became so convincing that whenever patients presented themselves with a relapse, faulty diet was assumed and invariably admitted. Again and

again the observation was made that relapses occurred when the parents ceased to exercise control over their children, enabling the latter to yield to the temptation to consume sweets. Naturally the question suggested itself whether the dietetic error, shown to exercise a decided influence on the course of the disease, was to be sought in the carbohydrates per se, or merely in the sweets. To answer this question, a control test was made in which ample doses of saccharin were administered as a substitute for the sweets. It was now uniformly observed that this coal tar product did not in the least show any unfavorable influence on the ocular manifestations, proving conclusively that the carbohydrates per se must be held responsible. Whether the unfavorable influence of carbohydrates is to be ascribed to faulty metabolism, or to a limited ability of assimilation of the carbohydrates, the excess probably undergoing chemical changes, which in turn yield toxic or irritating agents sufficiently effective to produce eczematous manifestations, must for the present remain an open question. As regards the modus operandi of whatever processes are responsible, we must look to physiology for an explanation.

It will be recalled that some time before the investigation was completed, the routine treatment of atropin topically, calomel internally and a carbohydrate-poor diet was found to yield the best results. The rationale of the administration of calomel needs no defense; but this drug has no bearing on the favorable influence of atropin, the latter having proved effective even without calomel. How can this be explained? Might it not be that in phlyctenular disease we have actually to deal with a part of the picture of exudative diathesis as propounded by Czerny,[6] and that the external phenomena are the result of irritability or hypertonia of the vagus system?

If this is the case, atropin, and not calomel, is therapeutically the drug best suited. Accordingly, calomel

6. Czerny: Exudativa Diathesa, Jahrb. f. Kinderh., 1905, p. 199; Scrofulosa and Tuberculosa, ibid., 1909, p. 529.

was abandoned and atropin administered internally instead, in a series of cases, as suggested by the practice of Krasnogorski[7] and Leopold,[8] who prescribe a solution of atropin of 1 grain to the ounce of water in doses of 3 drops every three hours, and daily increase the dose by 1 drop until the physiologic effect is reached. A number of patients were treated by this method without stopping the topical applications of atropin and restriction of diet. The tolerance manifested by these children for this powerful drug is quite remarkable, some consuming as high as ⅓ grain daily.

This form of treatment has now been extensively followed for the past eight months, and the results have been so greatly superior to any routine followed heretofore that, with the present experience as a basis, one cannot but say that absolute control of the symptoms of phlyctenular disease is assured by it. It must be emphasized, however, that while atropin best controls the symptoms, permanence of therapeutic results will depend on a strict adherence to a suitable diet even long after all the symptoms have disappeared.

It is realized that, in the presentation of the observations, workers accustomed to exact laboratory methods will find much that is not free from objections. It is conceded that in the further investigation of phlyctenular disease laboratory workers can find many unexplored avenues for scientific research. The investigation of the blood for the presence of acetone alone presents quite a problem. Chemical studies of carbohydrate metabolism and neurologic study of the vagus system are other fields of interesting activity. But the evidence so far submitted, clinical though it be, is too persistent and too extensive to be ignored.

I desire to express my sincere appreciation to the staff of the Illinois Charitable Eye and Ear Infirmary of Chicago for its courtesy in referring many patients to me for the purpose of furthering this investigation, and to Dr. A. Levinson, pediatrician to Michael Reese Hospital for Chicago, for many valued suggestions.

7. Quoted by Leopold (Note 8).
8. Leopold, J. S.: Atropine Treatment for Exudative Diathesis in Infancy, Am. Jour. Dis. Child, October, 1915, p. 288.

SUMMARY

1. Ophthalmia eczematosa or phlyctenular disease is not a true pathologic entity but symptomatic manifestation of a systemic disturbance.

2. Tuberculosis, syphilis and sepsis can be excluded with certainty as causal factors.

3. Phlyctenular disease is in all probability one of the expressions of vagus system irritability produced by some toxic agent, resulting from faulty carbohydrate chemism.

4. Correction of the chemism by carbohydrate-free diet and control of the vagus hypertonia through the topical and internal use of atropin yield the best and quickest possible therapeutic results.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. GOLDBACH AND GOLDENBURG

DR. RICHARD J. TIVNEN, Chicago: Dr. Goldenburg has given us a splendid report on his study of 500 cases of phlyctenular disease, covering a period of seven years, and has advanced an interesting theory to explain the cause of the malady. His report on these cases is a preliminary one, and as such, therefore, we find omitted much exact information which one needs in a scientific discussion. I gather from the paper that his conclusions are briefly as follows:

1. Phlyctenular trouble is not a true pathologic entity, but a symptomatic manifestation of a systemic disturbance.

2. Tuberculosis and syphilis can be excluded with certainty as causal factors.

3. Phlyctenular disease is in all probability one of the expressions of vagus system irritability produced by some toxic agent, resulting from faulty carbohydrate chemism—a part of the picture of "exudative diathesis" as propounded by Czerny.

4. Correction of the chemism by carbohydrate-free diet, and control of the vagus hypertonia through the topical and internal use of atropin, yield the best and quickest possible therapeutic results.

Taking up Dr. Goldenburg's conclusions, I feel that I am in harmony with prevailing opinion in stating that we all agree that phlyctenular trouble is not a true pathologic entity but a symptomatic manifestation of a systemic disturbance.

As to Dr. Goldenburg's second conclusions, that tuberculosis can be excluded with certainty as a causal factor, I must

differ with him. Personally, I believe that phlyctenular trouble is an ocular manifestation of tuberculosis, basing my opinion on a study of fifty cases treated with tuberculin diagnostically and therapeutically, as well as a study of the observation of numerous observers.

Dr. Goldenburg has not, in my opinion, submitted any precise scientific data, nor does such research work and observation as he has presented warrant him, in my judgment, in making such a sweeping, positive assertion. It is admitted by those favoring the tuberculous theory that their contentions are far from being proved. It is true, as Dr. Goldenburg says, that the tubercle bacilli have not been found in the phlyctenule; that transplantation experiments with fresh phlyctenules into the anterior chamber of the eyes of rabbits have failed to produce a typical tuberculous lesion. These are the two evidences which at this present time prevent the acceptance of the tuberculous theory. It has been advocated that the failure to obtain success in these may be, perhaps, due to the peculiar vagary of the phlyctenule itself, a peculiar stage of its development or a faulty method of identification. Be that as it may, however, precise clinical observation cannot or should not be ignored. Certainly there is no class of patients which presents clinically more significantly the stigmata of a tuberculous process than these children with phlyctenular trouble. The majority are poorly nourished, of minus resistance, come from bad hygienic surroundings, many present, as Dr. Goldbach and others have shown, tuberculous foci in other parts of the body—the lungs, bones and glandular system. Their phlyctenular trouble itself is, like most tuberculous processes, stubborn, sluggish and prone to recur with each drop or remission in the body resistance. In addition to these clinical observations, tuberculin diagnostically and therapeutically does give a positive response in an astonishingly large number of cases and its therapeutic administration does, according to competent observers, give improvement. I use the word *improvement* advisedly, because I am sure I have the approval of all who have attempted a conscientious study of tuberculin administration in this class of cases, that the difficulties surrounding the proper scientific carrying out of the details of such treatment are enormous.

The treatment of an acute attack of phlyctenulosis admittedly has its own peculiar problems, which very often cause both patient and physician much distress and disappointment. It is more especially true, however, that it is not after all these "acute attacks," but the relapses, the "recurrences" of the disease, that baffle our efforts and cause us to grow discouraged. The prevention of these relapses or "recurrences" is the crux of the whole problem.

Only a limited experience is required to convince one that any treatment designed to accomplish this result must enlist

the utmost cooperation of patient and parents and be continued over a long period of time. This is especially, signally and emphatically true in the use of tuberculin. It must be given over a long period and its administration must be safeguarded with a superlative degree of care as regards the dosage, the frequency of administration and the reports of the reactions obtained from each treatment.

The vast majority of phlyctenular cases are ambulatory, ignorant, and their home conditions hygienically of the worst. It is exceedingly difficult to secure cooperation in the ways indicated and essential, under such circumstances, over such a comparatively long period as is necessary properly to carry on the tuberculin treatment. Again, the necessary "team work" of internist, rhinologist, dentist and ophthalmologist is frequently not to be had. The ideal plan for study would be an organization comprising these individual units in conjunction with one who specializes in tuberculin administration and a settlement nurse assistant. From my own experience I feel sure that much of the failure and disappointment in the administration of tuberculin is due to these causes, and I feel also that when such cases are studied and the patients treated under the favorable plan of management outlined, that phlyctenular trouble will undoubtedly finally be classified as an ocular manifestation of tuberculosis. The exudative diathesis, suggested by Dr. Goldenburg as a possible explanation of the phlyctenulosis, is to my mind an uncertain, nebulous affair, admitted as such by the pediatricians. They are not clear as to its cause, and differ widely as to the explanation of its several manifestations. Czerny, for example, who propounded the theory, regards the phlyctenular manifestation which often accompanies the condition as eczema, while Heubner and Finkelstein deny the relationship of this condition to the exudative diathesis. Indeed, I am constrained to venture the statement, after study of this exudative diathesis syndrome, that it is likely that it is itself a manifestation of tuberculosis.

The doctor's plan of treatment, the use of atropin locally, with restriction and modification of diet, commends itself and is excellent, whatever the etiologic factor may be. As to atropin internally, I have had no experience, but, on Dr. Goldenburg's suggestion, I shall be glad to try it. I trust Dr. Goldenburg will continue his observations and will at our next meeting follow this, his preliminary report, with a detailed presentation of the cases studied.

Dr. H. H. Turner, Pittsburgh: The final solution of the etiology of phlyctenulosis must explain certain things: (1) Why does the initial attack of ophthalmia so frequently follow the acute infectious diseases of childhood; (2) why does the process confine itself so constantly, in many cases, to only one and always the same eye; (3) why do some of

the most violent cases occur among children who appear robust and healthy; (4) why does the removal of pathologic tonsils and adenoids stop the tendency in some cases but not in others; (5) why does restriction of diet benefit but not cure these cases; (6) how does a dietetic indiscretion produce a lymphatic disturbance in an organ relatively remote; (7) why does the tendency disappear at adolescence? Any theory which cannot answer these queries is not based on facts.

Can perversion of carbohydrate metabolism or the tubercular theory explain these things? We all know they cannot.

It is generally conceded that the ocular manifestation is secondary. Where, then, and of what character is the primary lesion? The fact that the process remains limited in many cases to only one and always the same eye, over a period of years, is proof positive of a tissue pathology, if not of the eye, then of some contiguous structure, which may be unilateral. What is the answer?

The relation of ocular pathology to diseases of the accessory nasal sinuses has been a study with me for the past twelve years, and I would like to place on record before this section a brief résumé of my deductions as to the etiology of phlyctenulosis, which have no mention in the literature, and which, to my mind, definitely explain all the queries just made.

In clinical service at the Mercy Hospital, Pittsburgh, over a period of ten years, where we have a large clinic, and in the capacity of ophthalmologist to St. Paul's Orphanage, which cares for 1,300 children, the field for observation has been fertile.

I am firmly convinced that the essential lesion in these cases is a low grade, chronic infection of the ethmoidal labyrinth *with obstruction to drainage.* The outer wall of this sinus, the lamina papyracea, forms the inner third of the bony orbit, and is frequently the seat of defects in continuity. Different theories advanced by various observers from time to time bear an important relationship, in that they are based on elements which excite or influence unfavorably the local focus of infection in the sinus.

Skillern says, in discussing the frequency of sinusitis in children, that it is a very common thing for this infection to occur during the course of measles, scarlet fever, pertussis, influenza, pneumonia, etc., and that the tissue devitalization resulting favors a secondary infection of chronic character, which persists indefinitely. All ophthalmologists know by experience with what frequency the initial attack of phlyctenulosis follows these acute infectious diseases. Skillern quotes Hajec to the effect that indiscretions in diet will produce an acute exacerbation of a chronic sinusitis. The excoriated, fissured nares so constantly seen in these cases are

largely the result of an excessively acrid discharge from the ostium of the ethmoidal labyrinth, the turgid nasal tissues above the orifice of the nasolacrimal duct being bathed in a sanious discharge, which gives a peculiar grayish appearance, and produces more or less odor. Many of these children develop marked ozena.

As the child with the phlyctenular history approaches the age of adolescence the facial and cranial bone spaces develop, the nasal cavities widen, and the impediment to sinus drainage becomes less marked and possibly entirely disappears. Therefore the tendency to phlyctenular ophthalmia usually subsides in the vast majority of cases at about this age, and remains quiescent, except at such times as the drainage may become blocked by an acute intumescence of the local soft parts.

The successful treatment of this disease requires a broad grasp of the etiologic factors and the consideration and elimination of *all* the elements which may influence unfavorably the local sinus lesion. The several influencing elements are: Diseased tonsils and adenoids; obstruction to ethmoidal drainage by deflected septum, spurs, or tight nostrils, which in children usually result from faulty eruption of the teeth; any general toxemia, of which the chief is intestinal in origin.

Hypertrophied, pathologic tonsils and adenoids produce a stasis in the lymphatic flow, with intumescence and bogginess of the intranasal and intrasinus tissues, and interference with sinus drainage. The retained, infected sinus contents accumulate under pressure and are forced into lymphatic channels, being carried along lymphatic pathways to the tissues of the conjunctiva or cornea, where they lodge and produce the typical phlyctenular lesions. The removal of such diseased tonsils will effect a relief of the condition only when they are the sole exciting factor.

As is well known, the separation of the superior maxillae is produced largely by the wedging action of the upper teeth. When the teeth are badly decayed or erupt abnormally, this wedging action is lost, and we find a dental arch, flattened laterally, with the incisors projecting to varying degrees, a contracted, highly arched palate, and narrowed nasal chambers, with deflected septum produced by the vertical encroachment of the highly arched palate. These cases, almost without exception, have obstruction to sinus drainage on one or both sides, and if a chronic infection be present, are very likely to develop a typical phlyctenular ophthalmia. I have in many cases secured beautiful results by referring these patients to a skilful orthodontist, who, by spreading the superior dental arch, has caused all these structures to be restored to near normal, the relief from obstruction to drainage causing the phlyctenular tendency to disappear.

Given hyperplasia of the middle turbinates or accessory sinus tissues, the ethmoidal labyrinth especially, any form of constitutional toxemia will produce marked circulatory disturbances in the nasal and orbital tissues of the same side, the opposite side remaining normal, if the nasal pathology be unilateral. The toxemias most frequent originate in an active tuberculous focus, or in the gastro-intestinal tract.

There is a peculiar, reflex, perversion of the digestive function, which is found in the entire group of sinus disease, including phlyctenulosis. The relation is vicious, in that the auto-intoxication intensifies the pathologic process which produces the reflex. I have seen many of these cases in which the chronic digestive trouble disappeared entirely, or was vastly improved following the correction of such a sinus lesion. The intestinal condition is characterized by hyperacidity, with fermentation, flatus, eructations, etc. The aim in diet should be to exclude those elements which already contain such acids as would add fuel to the fire, and those, which, because of indigestibility, would add to the existing status the acids and toxins formed during the process of intestinal fermentation. Personally, I do not believe that a carbohydrate-free diet is at all indicated, but do believe that certain of the carbohydrates, among other elements, should be excluded absolutely and for all time in these cases.

The Roentgen ray in these cases is not dependable. If the members of this section will study these cases, alone or in conjunction with an associate rhinologist, along the lines I have mentioned, I am certain that they cannot but reach the same conclusions as myself.

Dr. George F. Sullivan, Hoboken, N. J.: I do not agree with Dr. Goldenburg that we can immediately cast aside the theory of tuberculosis. According to the title of the paper this is an ophthalmia eczematosa. Almost all skin men agree at present that infantile eczema or eczema in the adult is a protein sensitization. That is, there is anaphylaxis to certain proteins. If such is the case, a high protein diet would increase the eczema instead of decreasing it, inducing anaphylaxis. In eczema Dr. Bulkley gives a diet of rice, bread and butter and water. He usually keeps the patients on this diet four to six days, and most of them clear up wonderfully. Twenty or thirty years ago Dr. Herman Knapp in his questionnaire usually gave as the first question, "Does the child eat cinnamon buns, fresh bread, tea or coffee?" The question was usually answered in the affirmative. He immediately placed them on a rigid protein diet, as one would patients with tuberculosis or adenopathy. There is a general adenopathy in 50 per cent. or more of the cases, which is indicative of tuberculosis, and the first thing you do is to place them on a tuberculosis regimen—give them

fresh eggs, milk, air baths and cod liver oil. I think cod liver oil is a great help, and I think tuberculin in long continued treatment is efficacious. If tuberculin therapy was carried out long enough we would see better results. Dr. Derby found that 88 per cent. responded to the von Pirquet test. In these cases I feel that it does not make much difference whether one gives atropin internally or in the eye, provided it is used weak enough; 1/100 of 1 per cent. locally will give practically the same results as if given internally in larger doses. Mercury is given empirically. We do not know whether it does any good or not, but atropin is an antispasmodic and the results have been through its influence on the ciliary muscle.

DR. H. W. WOODRUFF, Joliet, Ill.: It seems to me that the one fact we should bear in mind is that we should try not to become men of one idea. We set up a certain theory or idea and make everything focus in that direction. There are certain facts that stand out clearly in connection with this disease, and one is that these cases seldom occur where the children have the best surroundings. That of course fits in with tuberculosis, but at the same time it fits in with many other diseases. I do not see that we have gone far in taking up this question of the exudative diathesis. I have read what I have been able to find on that subject and I do not see that we have advanced any farther than when we talked about scrofula. We had no definite explanation about scrofula; then came the theory of tuberculosis. Now we are asked today to take up the theory of the exudative diathesis. It is fortunate indeed that we are still able to treat these diseases even when we do not know absolutely the cause; in fact, there is no one cause; it is a combination of conditions.

DR. A. E. DAVIS, New York: I want to speak of the treatment. I have been using the tuberculin treatment for some years and I am still more favorably impressed with it than when I began. I agree with Dr. Woodruff that we do not know what causes it; we are still guessing; but we do know the result of treatment. My belief is that we stop the tuberculin treatment too quickly in these patients. For instance, we have a child with phlyctenule and the other discrasia of scrofula. We put it on a diet, local and constitutional treatment, and perhaps tuberculin. As soon as these little phlyctens disappear and the child picks up some we turn him loose. What is the result? If you get him early in the spring, he has another attack before summer comes on. I have found that if we give them four months' treatment the tendency is not for a recurrence. That is the point I should like to impress on the section, that we do not give the treatment long enough. I also like to get some reaction from the tuberculin injections. I do not believe in keeping them too

low. We ought to know when we get the effect, and when we get a reaction, drop back a little and hold it there.

DR. LEO J. GOLDBACH, Baltimore: These patients had thorough physical examinations; blood tests and roentgenograms were made. They were referred to different departments for special examinations. Our social worker followed up each case, making them come back for treatments and observation by giving inducements. We did get some results. I agree with Dr. Davis that it is important to keep up the tuberculin treatment. We have some cases that have had sixty injections or more. With thorough examinations and team work we intend to follow up these cases. The phlycten is a manifestation, and the tubercle bacillus will not be found in the sections. It is an ocular manifestation of some systemic discrasia, probably tuberculosis.

DR. MICHAEL GOLDENBURG, Chicago: In reply to Dr. Tivnen, I would suggest that he try in his next fifty cases the régime he suggests, minus the use of tuberculin, and I am sure he will get just as good results. I have tried it, and if he will try the treatment I have outlined in a series of cases I will positively promise him better results and in half the time.

In regard to this condition occurring in poor people, it is true it is much more common in poor people, not because they are poor or because their food is poor, but because they are members of large families where the mothers are unable to give the children individual attention. In private practice or in adults, where I have been able to obtain intelligent cooperation, I have not had a relapse in the past three or four years. Dr. Tivnen quotes Finkelstein: "Finkelstein mentions an alimentary intoxication due to carbohydrate intolerance."

As to Dr. Turner's suggestion of sinusitis as a causal factor, I have examined the nasal passages of a great many of these children and could find no cause for thinking as he does. The rhinorrhea present is largely due to excessive lacrimation and patent tear ducts.

As to Dr. Sullivan's suggestion of anaphylaxis, I tried to account for this condition on these grounds a few years ago, but could make no headway. I went into their dietary thoroughly and found they consumed an excess of carbohydrates, not proteins. It is to be regretted that very little appears in the literature on carbohydrate metabolism after the second year of life; everybody seems to be working on proteins. As to the action of atropin, it has been my experience that in the severe cases, as soon as the pupils are thoroughly dilated, the local symptoms subside, and not before. In fact, if a child did not show improvement in a few days I could say that the pupils were not dilated, even before I separated the lids. Atropin at first has a simulating action, and during this stage it acts on the vagus.

In reply to Dr. Woodruff: The exudative diathesis theory was propounded to me by a pediatrician because it seemed to fit in with the excellent clinical results I obtained under this routine. It seemed to me the most rational theory by which I could explain this metabolic dyscrasia and the rapid control of irritative symptoms by the use of atropin. The fact that I have been able to clear up these cases in a much shorter time than under any other form of therapy, and that I could again precipitate the local symptoms almost at will, is too encouraging to go back to the therapy of yesterday.

In conclusion I want to suggest to the members of the section who have opportunity to see many of these cases that they try this simple procedure in a series of cases—you can do no harm—and let me hear what you have to say at the next meeting. You will have many relapses, because your greatest difficulty will be adherence to the rigid diet.

INTRACAPSULAR EXTRACTION OF SENILE CATARACT

AN ANALYSIS OF ONE HUNDRED AND FORTY-SIX CONSECUTIVE CASES *

A. S. GREEN, M.D.

AND

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SAN FRANCISCO

In the accompanying tables are statistics of the cases of senile cataract in which we have operated by the intracapsular method in this country during the past two and a half years. Since as far as practicable we have had each patient report at frequent intervals for examination, we have had an excellent opportunity to study carefully 146 cases of senile cataract, the periods of observation ranging from one month to two and one-half years after operation.

In order to pass a judgment on the value of any operative procedure, it is self-evident that enough cases should be observed, and also that a sufficient length of time should elapse to note the remote as well as the immediate results of the operation.

The statistics on cataract operations generally available are often meager in details, the operator being satisfied to report only the visual results obtained at some unspecified time after his operation, and to incorporate in his tabulation of each case a few unessential points. The lack of important details in such statistics makes a comparison of results difficult if not impossible, for while vision may be high in from three to six months after operation, subsequent inflamma-

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tion, glaucoma, epithelial downgrowth or the formation of after-cataract or membrane, all frequently attributable to the operative procedure, may materially reduce the visual acuity first obtained. Therefore, in the report submitted herewith, along with other important data, the date of operation is followed by the date, vision, and correction at the first examination after operation, side by side with the same data for the last examination; thus the changes in vision and refraction, as well as the increase or decrease of vision at the final examination, are shown at a glance.

As a number of operations in this series were performed at state or government institutions outside the city, it was impracticable to make a complete examination before and after operation, so that in several instances the blood pressure and tonometric readings were unobtainable. Finger palpation was relied on when the tonometer was not used, and if the tension found was apparently normal, the operation was performed. Both systolic and diastolic blood pressure readings were taken, but in the tables it was considered sufficient to give only the systolic pressure. With institutional patients and in some private cases from outside the city, only one refraction subsequent to operation was obtainable, and in a few of the latter the refraction and visual result given are those reported by the home oculist.

We considered it advisable also to separate the complicated from the uncomplicated cases in order to appraise the operation at its true value. It is unnecessary to call attention to the fact that an operation may be a success from a surgical standpoint but a functional failure, and that this may be due to causes other than the operation and beyond the control of the operator, such as preexisting fundus lesions, corneal and vitreous opacities, general systemic affections, or conditions arising subsequent to the operation. In some of the cases in this series, the operation was under-

taken with a full realization of the possible untoward results, for such cases were diagnosed as complicated before the operation; under this category come most of the cases with diabetes, chronic glaucoma and detachment of the retina. Fundus lesions, however, were frequently not diagnosed until later when the backgrounds could be seen after the removal of the cataract.

DATA

Method.—The method employed, except in twelve cases done by the Stanculeanu or the Knapp methods, has been in the main that known as the Smith Indian intracapsular cataract operation, the technic having been learned by us while in India under the personal instruction of Colonel Henry Smith.

Sex.—The sexes were divided about equally, there being no appreciable difference between them in tractability, convalescence or final results.

Age.—This varied from 35 to 95 years, and it seems to have had little or no bearing on healing or on recovery, but it did have a distinct influence on the delivery of the cataract. The older the patient, the weaker the suspensory ligament and the greater the ease of delivery. Again, age seems to have an influence on the maturation of the cataract; thus at 50 or under, only one was immature and bilateral, while six were mature and unilateral. The conclusion may be drawn, therefore, that the younger the patient the more likely it is for the cataract to be one-sided and mature when the patient comes under observation, while the older the patient, the greater the relative number of bilateral and immature cataracts presented. In patients under 50, and especially when only one eye was involved, the lens was harder to deliver within the capsule. These cataracts were probably secondary to some systemic affection or to a "quiet" form of uveitis. Vascular changes of the fundus were found more frequently in older patients.

TABLE 1.—UNCOMPLICATED CASES*

No.	Name	Age	Eye	Duration	Type	Tension	Syst. Blood Pressure	Vision before Operation	Method, Section, vitreous loss, and Complications	Date of Operation
										1914
1	Mr. J. H.	70	R	3 yrs.	Im.	20	170	3/100	Comb. corn.	Sept. 15
2	W. W. B.	45	R	7 yrs.	T.	N.	...	H. M.	Comb. corn. Van Lint flap...	Sept. 17
3	Mrs. A. H.	57	R	1 yr.	M.	20	...	H. M.	Simple, corn. Vitr. lost while sewing Van Lint flap	Oct. 5
4	Mrs. H. R.	65	L	1 yr.	Im.	N.	185	1/200	Comb. corn.	Oct. 6
5	C. P. H.	74	R	2 yrs.	Im.	N.	160	H. M.	Corn. Squeezed on completion of incision; vitr. lost. Simple	Oct. 7
6	Mrs. A. E. W.	70	L	1 yr.	Im.	N.	172	3/100	Simple; corn.	Oct. 8
7	J. W. W.	65	L	1½ yrs.	M.	N.	...	H. M.	Simple; corn.	Oct. 14
8	Mrs. K. O'N.	75	R	20 yrs.	Im.	32	...	20/150	Prelim. iridect. Nov. 5, 1914. Corn.	Nov. 25
9	M. McC.	68	R	3 yrs.	Im.	N.	...	3/100	Prelim. iridect. Nov. 6, 1914. Corn.	Nov. 27
10	Mrs. A. E. W.	70	R	6 yrs.	Im.	20	...	H. M.	Simple; corn.	Dec. 16
										1915
11	C. B.	75	L	2 yrs.	Im.	20	...	Fing. 2 ft.	Simple; corn.	Jan. 6
12	J. W. W.	65	R	1 yr.	Im.	N.	...	20/150	Simple; corn. Vitr. lost.....	Jan. 20
13	P. H. F.	75	R	5 yrs.	Im.	20	...	1/20	Prelim. iridect. Feb. 5, 1915. Corn.	Feb. 20
14	Mrs. C. S.	70	L	2 mos.	T.	23	175	H. M.	Prelim. iridect. Feb. 15, 1915. Limbal.	Feb. 26
15	T. R.	64	R	6 mos.	Im.	10	130	H. M.	Comb. corn.	Apr. 16
16	L. M. R.	73	R	4 mos.	M.	22	185	H. M.	Prelim. iridect. Mar. 15, 1915. Corn.	May 7
17	Mrs. H. F. S.	78	L	35 yrs.	Im.	28	220	H. M.	Prelim. iridect. Apr. 27, 1915. Corn. Hard delivery. Healing delayed	May 8
18	C. McC.	72	L	1 yr.	Im.	14	160	H. M.	Prelim. iridect. May 6, 1915. Corn.	May 19
19	Mrs. C.	72	L	9 yrs.	Im.	18	180	20/150	Prelim. iridect. May 10, 1915. Corn.	May 22
20	S.	..	L	.....	M.	N.	...	.....	Comb. corn. Entropion; photophobia	June 1
21	H. S.	75	L	8 yrs.	Im.	N.	...	Fing. 10 in.	Comb. corn.	June 18
22	J. W.	68	L	2½ yrs.	M.	N.	...	Fing. 16 in	Comb. corn.	June 18
23	I. J. A.	73	R	4 yrs.	Im.	N.	...	4/200	Comb. corn. Delayed healing	June 18
24	S.	..	R	.....	Im.	N.	...	.....	Comb. corn.	June 27
25	J. A. S.	78	R	10 yrs.	Im.	N.	...	H. M.	Comb. corn.	June 27
26	Mrs. M. P.	70	R	1 yr.	M.	N.	...	H. M.	Comb. corn. Wound open, delayed healing. Marked iridocyclitis	June 27
27	Mrs. B. M. S.	74	L	3 yrs.	M.	22	172	H. M.	Prelim. iridect. June 15, 1915. Corn.	June 29
28	P. H.	65	R	6 mos.	Im.	17	...	H. M.	Prelim. iridect. May 13, 1915. Corn.	July 1
29	Mrs. A. H.	81	R	2 yrs.	Im.	20	150	Fing. 1 ft.	Prelim. iridect. June 21, 1915. Corn.	July 5
30	T. K.	70	R	1 yr.	Im.	12	162	3/100	Prelim. iridect. July 1, 1915. Corn.	July 14
31	J. O.	60	R	6 mos.	Im.	N.	...	3/100	Prelim. iridect. July 19, 1915. Corn. Point of knife broke within eye during incision; not found. Iritis	July 30

* Comb. = combined. Corn. = corneal section. Fing. = fingers. H. M. = hand movements. N. = normal. Vitr. = vitreous. C. = complicated.

TABLE 1.—UNCOMPLICATED CASES*—*Continued*

Condition of Vision and Refraction		Remarks
At First Examination	At Last Examination	
Oct. 9, 1914 +6 ⌒ +5.50 ax. 155 = 20/100	Jan. 22, 1915 +8 ⌒ +2 ax. 160 = 20/25	
Oct. 7, 1914 +9 ⌒ +3 ax. 180 = 20/60	Oct. 19, 1914 +11 = 20/15	Border of flap became incarcerated in nasal side of wound
Nov. 24, 1914 +8 ⌒ +3 ax. 90 = 20/60	Feb. 24, 1917 +12 ⌒ +1.50 ax. 110 = 20/30	Pupil markedly drawn up. Iridectomy from below Feb. 14, 1916
Nov. 16, 1914 +2 ⌒ +4 ax. 180 = 20/60	Feb. 13, 1916 +5 ⌒ +1 ax. 170 = 20/15	High myope. Post. staphyloma
Nov. 9, 1914 +10 = 20/150	Mar. 1, 1915 +11 ⌒ +1.50 ax. 90 = 20/30	†
Oct. 19, 1914 +7 ⌒ +4 ax. 45 = 20/80	March 1, 1917 +2 ⌒ +8 ax. 30 = 1/200	Looked down repeatedly during healing. Upper part of iris adherent to wound. Glaucoma. Disk cupped. Dec. 11, 1914 vision = +2 +8 ax. 30 = 20/15
Nov. 20, 1914 +7 ⌒ +5 ax. 30 = 20/60	July 1, 1916 +10 ⌒ +2.50 ax. 35 = 20/40	Upper part of iris adherent to wound; pupil slightly drawn up. At first iris was free and pupil central
Dec. 30, 1914 +4 ⌒ +7.50 ax. 150 = 20/60	Feb. 23, 1917 +9.50 ⌒ +2.75 ax. 165 = 20/20	Dec. 5, 1914, wound closed, pupil regular. Dec. 21, 1914, wound slightly opened above with slight incarceration of nasal pillar. Disobeyed orders—cooked and swept
Feb. 11, 1915 +9 ⌒ +2 ax. 180 = 20/20	June 16, 1915 +9 ⌒ +1.50 ax. 180 = 20/20	
Jan. 5, 1915 + 2.50 ⌒ +5.50 ax. 165 = 20/40	Feb. 13, 1917 +6 ⌒ +5 ax. 180 = 20/15	Pupil round at first but became drawn up within two weeks after operation; looked down repeatedly during healing
Feb. 24, 1915 +9 ⌒ +5 ax. 165 = 20/25	Oct. 19, 1916 +13 = 20/25	Upper part of iris adherent to wound
Feb. 13, 1915 +10 = 20/40	July 1, 1916 +12 = 20/15	Pupil horizontally oval. Upper part of iris became adherent to corneal wound
Mar. 12, 1915 +6 ⌒ +7 ax. 170 = 20/60	Feb. 6, 1917 +10 ⌒ +1.25 ax. 165 = 20/15	Pupil drawn up. Feb. 12, 1916, iridectomy from below; epithelial downgrowth in wound
Mar. 31, 1915 +10 ⌒ +1.50 ax. 15 = 20/15	Dec. 17, 1915 +10 ⌒ +1.50 ax. 15 = 20/15	
May 17, 1915 +7 ⌒ +3 ax. 20 = 20/60	Jan. 29, 1917 +5.50 ⌒ +1 ax. 150 = 20/20	Diffuse floating opacities; epithelial downgrowth from incision
June 5, 1915 +11 ⌒ +1.50 ax. 165 = 20/20	June 16, 1916 +12 ⌒ +1 ax. 135 = 20/15	Feb. 15, 1917, central retinal hemorrhages. Vision markedly reduced. Other eye had only light perception from cataract operation 3 yrs. prior by another surgeon
Aug. 31, 1915 +5 ⌒ +7 ax. 95 = 20/30	Mar. 3, 1916 +8 ⌒ +3 ax. 90 = 20/40	Very drawn up pupil
June 3, 1915 +9 ⌒ +3.50 ax. 165 = 20/15	Mar. 12, 1917 +9 ⌒ +1.50 ax. 5 = 20/15	
June 9, 1915 +7.50 ⌒ +2 ax. 180 = 20/25	Jan. 23, 1917 +8 ⌒ +3.50 ax. 180 = 20/15	
.....	Vision apparently good when last seen	Patient left hospital in good condition before being refracted
July 12, 1915 +10 ⌒ +2 ax. 180 = 20/20	Aug. 19, 1915 +11 ⌒ +1.50 ax. 180 = 20/15	
Aug. 2, 1915 +10 = 20/200	Feb. 26, 1917 +77 = 20/25	Other eye lost at cataract operation 10 yrs. before, by another operator
Aug. 20, 1915 +8 ⌒ +2 ax. 180 = 20/60	Feb. 26, 1917 +9 ⌒ +1 ax. 15 = 20/30	
.....	Vision apparently good when last seen	Patient left hospital apparently in good condition before being refracted
Oct. 9, 1916 +11 ⌒ +1.50 ax. 180 = 20/40	Feb. 26, 1917 +11 ⌒ +1.50 ax. 180 = 20/20	
July 19, 1915 Light perception	Feb. 26, 1917; light perception but tension subnormal	Moved by mistake to another ward; handled roughly. Had severe cough followed by pain in eye
July 19, 1915 +11 ⌒ +2 ax. 15 = 20/20	July 11, 1916 +12.50 ⌒ +0.50 ax. 180 = 20/15	R. E. lost 2 yrs. prior from cataract operation, by another operator
July 17, 1915 +10 ⌒ +1 ax. 155 = 20/25	Jan. 21, 1916 +11 ⌒ +0.75 ax. 170 = 20/15	L. E., +2 ⌒ +1.25 ax. 180 = 20/15. Has binocular single vision with both eyes
July 23, 1915 +10 ⌒ +3 ax. 180 = 20/25	Mar. 10, 1916 +10 ⌒ +0.75 ax. 180 = 20/15	
Aug. 9, 1915 +8 ⌒ +3 ax. 20 = 20/25	Sept. 27, 1916 +12 ⌒ +1 ax. 120 = 20/15	Patient very unruly
Aug. 25, 1915 +5 ⌒ +4 ax. 150 = 20/25	Mar. 1, 1917 +11 ⌒ +2 ax. 180 = 20/120	Chronic uveitis; numerous vitreous opacities

TABLE 1.—UNCOMPLICATED CASES*—*Continued*

No.	Name	Age	Eye	Duration	Type	Tension	Syst. Blood Pressure	Vision before Operation	Method, Section, vitreous loss, and Complications	Date of Operation
32	Mrs. J. A. D.	55	L	6 yrs.	Im.	18	...	Fing. 2 ft.	Prelim. iridect. July 23, 1915. Corn. Large amt. vitr. loss; delivery difficult. Healing delayed	1915 Aug. 6
33	Mrs. G.	..	L	.....	M.	N.	...	.....	Prelim. iridect. Corn.	Aug. 7
34	R. P.	75	R	5 mos.	Im.	15	178	1/20	Prelim. iridect. July 28, 1915. Corn. Vitr. lost during removal of speculum. Healing delayed	Aug. 10
35	J. F.	63	L	1 yr.	Im.	16	...	Fing. 18 in.	Prelim. iridect. Aug. 14, 1915. Corn.	Aug. 28
36	Mrs. S. B.	77	L	10 yrs.	M.	N.	...	H. M.	Comb. corn. Pupillary exud.	Sept. 15
37	Mrs. M. M.	70	R	2 yrs.	M.	N.	...	H. M.	Prelim. iridect. Sept. 16, 1915. Corn. Delayed healing	Sept. 28
38	J. C.	62	L	2 yrs.	Im.	25	144	1/30	Prelim. iridect. Sept. 20, 1915. Corn. Iris prolapse	Oct. 1
39	G. C.	60	R	2 yrs.	M.	N.	...	H. M.	Prelim. iridect. Sept. 3, 1915. Corn. Iris prolapse	Oct. 6
40	J. W. E.	66	L	6 yrs.	Im.	22	120	1/40	Prelim. iridect. Oct. 2, 1915. Corn. Stanculeanu forceps and method	Oct. 15
41	Mrs. F. R. T.	80	R	2 yrs.	Im.	17	174	3/100	Prelim. Oct. 26, 1915. Corn.	Nov. 10
42	F. V. U.	70	L	2 yrs.	Im.	17	160	20/60	Prelim. iridect. Nov. 3, 1915. Corn. Iris prolapse	Nov. 17
43	A. S.	81	R	6 yrs.	T.	N.	...	Light percep.	Prelim. iridect. Nov. 22, 1915. Corn.	Dec. 6
44	N. N.	80	R	7 yrs.	Im.	N.	...	Fing. 10 in.	Prelim. iridect. Nov. 22, 1915. Corn. Vitr. loss. Pupil up. Epithelial downgrowth. Iritis	Dec. 6
45	Mrs. F. M. F.	60	L	1½ yrs.	Im.	17	...	H. M.	Prelim. iridect. Nov. 12, 1915. Corn.	Dec. 7
46	Mrs. C. R.	79	R	2 yrs.	T.	22	170	H. M.	Prelim. iridect. Nov. 24, 1915. Corn. Stanculeanu forceps and method. Capsule rupt.	Dec. 8
47	Mr. J. G.	75	R	1 yr.	Im.	19	176	H. M.	Prelim. iridect. Dec. 8, 1915. Corn.	Dec. 28 1916
48	A. T.	62	L	3 yrs.	Im.	N.	...	3/100	Comb. corn.	Jan. 26
49	Miss J. A. D.	55	R	1 yr.	Im.	N.	...	3/100	Comb. corn. Vitr. loss. Difficult delivery	Jan. 31
50	J. K.	38	R	15 mos.	M.	21	...	H. M.	Comb. corn. Iris prolapse...	Feb. 4
51	S. L.	72	L	25 yrs.	T.	18	175	H. M.	Prelim. iridect. Dec. 30, 1915. Comb. Limb. Capsule ruptured. Iritis	Feb. 9
52	C. R. T.	68	L	2 yrs.	M.	N.	...	H. M.	Comb. corn.	Feb. 14
53	Mrs. C. B.	60	R	8 mos.	M.	16	135	H. M.	Comb. corn. Slight iris prolapse. Vitr. loss	Mar. 16
54	W. D.	62	R	3 yrs.	Im.	N.	...	1/200	Comb. corn.	Mar. 27
55	Mrs. F. B.	71	R	5 yrs.	M.	N.	...	H. M.	Comb. corn.	Mar. 29
56	F. D. S.	35	L	5 mos.	Im.	N.	108	Fing. 3 ft.	Comb. Limbal. Capsule ruptured	Apr. 6
57	F. B.	84	L	10 yrs.	Im.	N.	...	Fing. 2 ft.	Comb. corn. Epithelial downgrowth	Apr. 10
58	T. K.	70	L	1 yr.	Im.	17	162	20/100	Prelim. iridect. Nov. 5, 1915. Corn.	Apr. 11
59	O. W.	70	L	8 mos.	Im.	15	130	H. M.	Comb. corn.	May 11
60	O. W.	70	R	8 mos.	Im.	12	130	H. M.	Comb. corn.	May 20
61	J. E.	64	L	1½ yrs.	M.	N.	...	H. M.	Comb. corn. Bulging of corneal wound	May 22
62	S. L.	56	R	3 yrs.	Im.	15	120	2/30	Comb. Limbal. Slight vitr. loss	June 20
63	D. V. R.	57	R	10 yrs.	Im.	16	144	3/100	Comb. Conjunctival flap....	July 6

TABLE 1.—UNCOMPLICATED CASES*—*Continued*

Condition of Vision and Refraction		Remarks
At First Examination	At Last Examination	
Sept. 1, 1915 +10 ⌒ +4 ax. 45 = 20/100	Feb. 8, 1917 +5 ⌒ +3.50 ax. 15 = 20/30	Jan. 9, 17, iridectomy from below for drawn up pupil
Oct. 20, 1915 +11 ⌒ +1 ax. 180 = 20/30	Feb. 28, 1917; patient wrote that vision is good	In reply to a letter, patient wrote Feb. 28, 1917 that she reads newspapers, threads needles and sees better than at previous examination
Sept. 7, 1915 +10 = 1/20 Sept. 13, 1915 +8 ⌒ +4 ax. 180 = 20/200	May 10, 1916 +10 = 20/40 Jan. 26, 1916 +12 ⌒ +2 ax. 180 = 20/20	Epithelial downgrowth from corneal wound
.....	Mar. 20, 1916 +14 = 10/200	Noninflammatory exudate in pupillary area. Mar. 18, 1916, iridectomy from below to lower pupil
Oct. 23, 1915 +8.50 ⌒ +1.50 ax. 15 = 20/20	Mar. 10, 1916 +12 = 20/15	
Mar. 10, 1916 +10 = 20/100	Mar. 10, 1917 +11 ⌒ +1.50 ax. 30 = 20/30	Mar. 4, 1916, excised prolapsed iris, and lowered pupil
Oct. 25, 1915 +12 ⌒ +2 ax. 45 = 20/30	Feb. 9, 1917 +12 ⌒ +2 ax. 45 = 20/30	Nov. 10, 1915, cauterized iris prolapse
Nov. 2, 1915 +5 ⌒ +6.50 ax. 40 = 20/25	Aug. 19, 1916 +7.25 ⌒ +1.75 ax. 20 = 20/15	
Nov. 30, 1915 +3 ⌒ +8 ax. 10 = 20/30	Nov. 9, 1916 +9 ⌒ +1.50 ax. 180 = 20/15	Other eye lost 2½ yrs. prior from cataract operation by another operator
Dec. 8, 1915 +14 ⌒ +0.75 ax. 180 = 20/15	Jan. 10, 1917 +12 ⌒ +1.75 ax. 180 = 20/15	Dec. 1, 1915, cauterized and cut prolapsed iris
.....	Feb. 19, 1917 +10 ⌒ +2.50 ax. 180 = 20/15	
.....	Feb. 26, 1917 +10 = 20/60	Unruly during and after operation. Should have iridectomy from below to improve vision
Dec. 29, 1915 +6 ⌒ +5 ax. 30 = 20/20	Feb. 6, 1917 +7.50 ⌒ +5 ax. 15 = 20/30	Floaters in vitreous
Dec. 29, 1915 +5 = 20/150	Nov. 1, 1916 +7.50 ⌒ +3 ax. 90 = 20/30	
Jan. 12, 1916 +9 ⌒ +5.50 ax. 170 = 20/25	Jan. 26, 1916 +11 ⌒ +2 ax. 180 = 20/20	
Feb. 26, 1916 +11 ⌒ +3 ax. 45 = 20/20	Jan. 10, 1917 +10 ⌒ +2 ax. 40 = 20/20	
Feb. 28, 1916 +9 ⌒ +3.50 ax. 180 = 20/30	Feb. 8, 1917 +11 ⌒ +1.50 ax. 5 = 20/15	
Feb. 21, 1916 +2.50 ⌒ +8 ax. 165 = 20/60	May 25, 1916 +2.50 ⌒ +8 ax. 165 = 20/60	
Apr. 17, 1916 +10 = 1/20	Feb. 28, 1917 +9.50 ⌒ +3 ax. 60 = 20/20	July 13, 1916, capsule removed
Mar. 6, 1916 +9 ⌒ +3.50 ax. 180 = 20/200	Jan. 8, 1917 +11 ⌒ +2 ax. 180 = 20/20	
Apr. 6, 1916 +8 ⌒ +2 ax. 145 = 20/25	May 31, 1916 +11 ⌒ +1.50 ax. 45 = 20/15	Looked down after delivery, causing vitreous loss
.....	Apr. 6, 1916 +10 = 20/40	Wound healed. Eye in good condition. Died 12 days after operation; heart failure
Apr. 27, 1916 +6 ⌒ +6 ax. 155 = 20/40	Dec. 30, 1916 +7.50 ⌒ +4 ax. 120 = 20/20	
Apr. 19, 1916 +7 ⌒ +8 ax. 165 = 20/150	Feb. 3, 1917 +12 ⌒ +1 ax. 165 = 20/15	
.....	Feb. 26, 1917 +10 ⌒ +2 ax. 180 = 20/80	
May 22, 1916 +10 = 20/80	Sept. 27, 1916 +10 ⌒ +4 ax. 180 = 20/20	
June 1, 1916 +10 = 20/20	Sept. 22, 1916 +10 = 20/20	
June 1, 1916 +10 = 20/40	Sept. 22, 1916 +10 = 20/20	
.....	Feb. 26, 1917 +5 = 20/100	Patient unruly; too deficient mentally to give accurate visual data
July 15, 1916 +9 ⌒ +2 ax. 165 = 20/40	Dec. 21, 1916 +11.50 ⌒ +2 ax. 180 = 20/15	
July 22, 1916 +11 ⌒ +2 ax. 135 = 20/80	Jan. 9, 1917 +13.50 ⌒ +1.50 ax. 135 = 20/15	

TABLE 1.—UNCOMPLICATED CASES*—*Continued*

No.	Name	Age	Eye	Duration	Type	Tension	Syst. Blood Pressure	Vision before Operation	Method, Section, vitreous loss, and Complications	Date of Operation
										1916
64	Mrs. A. M.	56	R	1 yr.	Im.	20	124	20/150	Comb. Limbal. Considerable vitr. loss; lens delivered with Weber loop. Conjunctiva red and edematous. Photophobia	July 11
65	C. F.	58	L	1½ yrs.	Im.	16	130	20/200	Comb. Conjunctival flap....	July 12
66	G. S.	60	R	2 yrs.	Im.	24	138	20/150	Comb. Limbal. Kalt forceps; Knapp method. Capsule ruptured	July 12
67	Mrs. M. L.	82	L	2½ yrs.	M.	20	145	H. M.	Comb. Conjunctival flap....	July 13
68	Mrs. L. P.	58	R	15 yrs.	Im.	21	170	20/150	Comb. Conjunctival flap....	July 19
69	Mrs. A. M.	56	L	1 yr.	Im.	17	124	4/30	Comb. Limbal. Kalt forceps; Knapp method. Hard to dislocate lens; considerable loss of vitreous. Delayed healing	July 28
70	J. F.	63	L	2 yrs.	Im.	14	140	H. M. at 16 in.	Comb. Conjunctival flap....	July 29
71	Mrs. E. M.	65	L	2 yrs.	M.	20	178	H. M. at 1 ft.	Comb. Conjunctival flap....	Aug. 4
72	Mrs. U. C. H.	52	R	3 yrs.	Im.	15	168	H. M. at 16 in.	Comb. Limbal. Tough suspensory ligament; small amt. vitreous lost	Aug. 8
73	S. P.	81	L	2 yrs.	Im.	N.	...	3/100	Comb. Conj. flap.....	Aug. 12
74	C. W.	80	R	1½ yrs.	Im.	12	145	H. M. at 3 ft.	Comb. Conj. flap. Kalt forceps; Knapp method. Iris prolapse	Aug. 15
75	Mrs. H. B.	66	R	2 yrs.	Im.	24	172	20/200	Comb. Conj. flap. Kalt forceps Knapp method. Vitr. loss. Delayed healing	Aug. 17
76	Mrs. E. A.	72	L	5 yrs.	M.	20	178	H. M.	Comb. Conj. flap.....	Aug. 19
77	Mrs. G. G.	..	R	3 mos.	M.	26	190	Light percep.	Comb. Conj. flap.....	Aug. 22
78	Mrs. L. P.	58	L	15 yrs.	Im.	20	170	3/100	Comb. Conj. flap. Kalt forceps. Knapp method. Vitr. lost. Capsule ruptured. Prolapse of iris	Sept. 5
79	J. P.	73	L	6 yrs.	Im.	21	180	Fing. 12 in.	Comb. Limbal. Lens hard to dislocate. Iris prolapse	Sept. 16
80	G. H. M.	48	L	1½ yrs.	T.	10	120	Light percep.	Comb. Conj. flap. Capsule ruptured and removed	Oct. 1
81	Mrs. J. I.	64	L	6 mos.	Im.	19	176	1/200	Comb. Conj. flap.....	Oct. 7
82	L. W.	56	R	6 yrs.	Im.	N.	...	10/200	Comb. Conj. flap.....	Oct. 8
83	Mrs. M.	65	R	2 yrs.	M.	20	178	H. M. at 1 ft.	Comb. Conj. flap.....	Oct. 11
84	Mrs. W. P. B.	..	R	1 yr.	Im.	13	170	H. M.	Comb. Conj. flap. Kalt forceps; Knapp method. Capsule ruptured, removed	Oct. 17
85	Mrs. G. A. E.	50	L	6 mos.	M.	16	106	Light percep.	Comb. Conj. flap. Kalt forceps. Knapp method. Small ulcer devel. at lower quarter cornea, found on first dressing, 10th day. Recovery in 3 weeks	Oct. 17
86	J. L.	61	L	8 yrs.	M.	28	160	H. M. at 5 in.	Comb. Conj. flap. Kalt forceps; Knapp method	Oct. 18
87	T. D.	84	R	5 yrs.	M.	28	160	H. M. at 3 ft.	Comb. Conj. flap. Large amt. vitr. lost by carelessness in removing speculum. Delayed healing. Epithelial downgrowth	Oct. 18
88	Mrs. M. H.	54	R	3 yrs.	T.	13	200	H. M. at 1 ft.	Comb. Conj. flap. Patient stupid, and squeezed after completing toilet, losing considerable vitreous	Oct. 25

TABLE 1.—UNCOMPLICATED CASES*—*Continued*

Condition of Vision and Refraction		Remarks
At First Examination	At Last Examination	
Aug. 10, 1916 +6 ⌒ +4 ax. 180 = 20/60	Feb. 10, 1917 +8 ⌒ +4 ax. 165 = 20/30	Pressure with hook failed to deliver lens; photophobia persisted for three months
July 26, 1916 +9 ⌒ +1.50 ax. 180 = 20/20	Jan. 20, 1917 +10 ⌒ +2 ax. 10 = 20/15	
Aug. 19, 1916 +10 ⌒ +3 ax. 180 = 20/60 Aug. 1, 1916 +9 ⌒ +4 ax. 180 = 20/60 Aug. 2, 1916 +8 ⌒ +3 ax. 180 = 20/30 Aug. 15, 1916 +7 ⌒ +3 ax. 180 = 20/40	Aug. 25, 1916 +8 ⌒ +4.50 ax. 180 = 20/25 Oct. 17, 1916 +10 ⌒ +3.50 ax. 180 = 20/30 Feb. 15, 1917 +10 ⌒ +1 ax. 90 = 20/15 Feb. 10, 1917 +9 ⌒ +1 ax. 10 = 20/15	Aug. 18, 1916, capsule removed
Aug. 19, 1916 +8 ⌒ +1.50 ax. 180 = 20/25	Jan. 9, 1917 +8 ⌒ +1.5 ax. 180 = 20/30	
.....	Mar. 1, 1917 +11.50 ⌒ +2 ax. 30 = 20/30	Dacryocystitis; removed left sac July 11, 1916
Sept. 2, 1916 +6.50 ⌒ +3.50 ax. 150 = 20/15 Dec. 30, 1916 +8 ⌒ +3.50 ax. 5 = 20/15	Feb. 23, 1917 +7 ⌒ +3.50 ax. 165 = 20/15 Feb. 13, 1917 +9 ⌒ +2.50 ax. 5 = 20/15	
Sept. 21, 1916 +9.25 ⌒ +1.25 ax. 60 = 20/40	Feb. 8, 1917 +11 ⌒ +0.75 ax. 80 = 20/15	Sept. 15, 1916, prolapsed iris excised
Sept. 7, 1916 +8 ⌒ +3.50 ax. 105 = 20/30	Oct. 17, 1916 +10 ⌒ +3.50 ax. 150 = 20/20	Diabetic
Sept. 7, 1916 +11 ⌒ +4 ax. 165 = 20/80 Sept. 19, 1916 +3 ⌒ +9 ax. 165 = 20/30 Oct. 27, 1916 +10 ⌒ +3 ax. 15 = 20/30	Mar. 2, 1917 +13 = 20/15 Mar. 10, 1917 +5 ⌒ +7 ax. 165 = 20/30 Feb. 15, 1917 +10 ⌒ +2 ax. 45 = 20/60	R. E. lost 5 yrs. prior following cataract operation by another surgeon Oct. 19, 1916, excised prolapsed iris and removed secondary cataract
Oct. 3, 1916 +6 ⌒ +6 ax. 30 = 20/30 Oct. 24, 1916 +10 = 20/30 Nov. 20, 1916 +8 ⌒ +8 ax. 175 = 20/30 Dec. 14, 1916 +11 ⌒ +4 ax. 180 = 20/40 Oct. 31, 1916 +10 ⌒ +3 ax. 105 = 20/60	Feb. 26, 1917 +10 ⌒ +3 ax. 180 = 20/100 Feb. 10, 1917 +12 ⌒ +1 ax. 10 = 20/15 Dec. 14, 1916 +12 ⌒ +2.50 ax. 160 = 20/15 Jan. 12, 1917 +10 ⌒ +3.50 ax. 180 = 20/20 Mar. 1, 1917 +13 ⌒ +1.50 ax. 105 = 20/60	Dec. 26, 1916, developed glaucoma. Jan. 15, 1917, released iris from angle of wound Dacryocystitis. Aug. 30, 1916, extirpated right lacrimal sac. Floating opacities in vitreous
Nov. 2, 1916 +4 ⌒ +5 ax. 180 = 20/80 Nov. 7, 1916 +8 ⌒ +5.50 ax. 180 = 20/15	Feb. 13, 1917 +5 ⌒ +1.75 ax. 10 = 20/25 Jan. 15, 1917 +9 ⌒ +4 ax. 180 = 20/15	
Nov. 4, 1916 +10 ⌒ +2 ax. 165 = 20/60 Nov. 11, 1916 +8 ⌒ +7 ax. 135 = 20/150	Dec. 20, 1916 +12 ⌒ +0.75 ax. 15 = 20/30 Jan. 4, 1917 +11 ⌒ +4.50 ax. 150 = 20/100	
Jan. 9, 1917 +9 ⌒ +2 ax. 105 = 20/40	Feb. 23, 1917 +9.50 ⌒ +2 ax. 105 = 20/25	L. E. operated on 2 yrs. prior by another surgeon, resulting in failure

TABLE 1.—UNCOMPLICATED CASES*—*Continued*

No.	Name	Age	Eye	Duration	Type	Tension	Syst. Blood Pressure	Vision before Operation	Method, Section, vitreous loss, and Complications	Date of Operation
										1916
89	Mrs. S. F. S.	62	R	4 yrs.	Im.	N.	...	Fing. 2 ft.	Comb. Conj. flap.....	Nov. 1
90	Mrs. B. A. S.	59	R	18 yrs.	M.	N.	...	Faulty project.	Comb. Conj. flap.....	Nov. 3
91	N. L.	74	R	2 yrs.	M.	N.	...	H. M.	Comb. Conj. flap.....	Nov. 8
92	Mrs. J. S.	62	L	3 yrs.	Im.	17	210	1/90	Comb. Conj. flap. Kalt forceps; Knapp method	Nov. 9
93	L. W.	56	L	5 yrs.	Im.	N.	...	5/60	Comb. Conj. flap.....	Nov. 20
94	H. V.	78	R	8 yrs.	Im.	13	150	1/60	Comb. Limbal. Delayed healing	Nov. 21
95	N. L.	74	L	3 yrs.	M.	N.	...	H. M.	Comb. Conj. flap.....	Nov. 22
96	Mrs. B. A. S.	59	L	3 yrs.	Im.	N.	....	20/200	Comb. Conj. flap.....	Nov. 24
97	M. DeL.	95	L	4 yrs.	M.	N.	...	Light percep.	Comb. Conj. flap.....	Nov. 25
98	J. T.	57	L	2 yrs.	Im.	N.	138	3/100	Comb. Conj. flap. Capsule ruptured; vitr. lost; lacrimation for six weeks	Dec. 14
99	W. H.	38	L	1½ yrs.	M.	N.	...	H. M. at 1 ft.	Comb. Conj. flap. Small iris prolapse, excised two weeks later	Dec. 15
100	G. McC.	65	L	1 yr.	Im.	17	192	20/100	Comb. Conj. flap.....	Dec. 29
										1917
101	Mrs. M. A. W.	63	R	3 yrs.	T.	14	150	Light percep.	Comb. Conj. flap.....	Jan. 3
102	Mrs. S. A. C.	74	L	2 yrs.	T.	15	105	H. M.	Comb. Conj. flap.....	Jan. 11
103	Mrs. J. W. S.	71	R	1 yr.	Im.	18	114	20/150	Comb. Limbal.	Jan. 12
104	S. H.	60	L	6 mos.	M.	28	225	H. M.	Comb. Limbal. Large amt. of vitreous lost	Jan. 13
105	W. F. T.	60	L	5 yrs.	Im.	N.	...	6/100	Comb. Conj. flap.....	Jan. 15
106	A. A.	72	R	1 yr.	Im.	22	160	Fing. at 10 in.	Comb. Conj. flap.....	Jan. 18
107	J. T.	78	L	1 yr.	Im.	7	150	6/100	Comb. Conj. flap.....	Jan. 20
108	Mrs. E. O. R.	60	R	20 yrs.	Im.	19	145	1/120	Comb. Conj. flap.....	Jan. 23
109	G. I. Y.	57	R	2 yrs.	Im.	N.	...	20/80	Camb. Conj. flap.....	Jan. 24
110	Miss M. B.	50	L	.....	M.	22	132	H. M.	Comb. Conj. flap.....	Feb. 7
111	P. K.	66	L	3 yrs.	Im.	N.	...	.....	Camb. Conj. flap.....	Feb. 9
112	Mrs. M. P.	48	R	6 yrs.	M.	13	174	Light percep.	Comb. Conj. flap.....	Feb. 13

TABLE 1.—UNCOMPLICATED CASES*—*Continued*

Condition of Vision and Refraction		Remarks
At First Examination	At Last Examination	
Dec. 16, 1916 +9 ⌒ +4 ax. 160 = 20/50	Mar. 7, 1917 +11.50 ⌒ +3.50 av. 150 = 20/20	Corneal epithelium desquamated from use of 10% cocain. Bed ozena
.....	Feb. 3, 1917 +13 ⌒ +1 ax. 30 = 20/15	Eye injured 18 yrs. ago; lower half only gave response to light. Stupid patient
Dec. 7, 1916 +11 ⌒ +3 ax. 10 = 20/60	Mar. 6, 1917 +12 ⌒ +2 ax. 165 = 20/40	Media clear, fundus normal. Unable to account for reduced vision
Nov. 24, 1916 +10.50 ⌒ +2 ax. 15 = 20/20	Feb. 20, 1917 +10.50 ⌒ +4.50 ax. 15 = 20/15	
Dec. 14, 1916 +11 ⌒ +3 ax. 30 = 20/30	Jan. 12, 1917 +12.50 ⌒ +1 ax. 40 = 20/20	
Dec. 6, 1916 +0 ⌒ +1 ax. 165 = 20/60	Feb. 23, 1917 +9 ⌒ +3.50 ax. 180 = 20/20	Note change in amount and axis of astigmatism in the various examinations
Dec. 11, 1916 +8 ⌒ +7 ax. 15 = 20/60		
Dec. 7, 1916 +11 ⌒ +3 ax. 15 = 20/30	Mar. 6, 1917 +11 ⌒ +2 ax. 15 = 20/20	
.....	Feb. 3, 1917 +11 ⌒ +1.50 ax. 180 = 20/15	
Dec. 15, 1916 +11 ⌒ +5 ax. 180 = 20/30	Jan. 31, 1917 +12.50 ⌒ +1.25 ax. 180 = 20/25	
Dec. 27, 1916 +7 ⌒ +4 ax. 150 = 20/40	Feb. 20, 1917 +10 = 20/15	
.....	Feb. 4, 1917 +11 ⌒ +3 ax. 180 = 20/20	
Jan. 12, 1917 +4 ⌒ +10 ax. 25 = 20/30	Mar. 2, 1917 +8.50 ⌒ +5.00 ax. 15 = 20/20	R. E. lost by another operator 9 months before
Jan. 18, 1917 +6 ⌒ +4 ax. 150 = 20/80	Feb. 20, 1917 +6 ⌒ +7.50 ax. 150 = 20/15	
Jan. 30, 1917 +12 ⌒ +2.50 ax. 165 = 20/25	Feb. 21, 1917 +12.50 ⌒ +1.50 ax. 165 = 20/25	
Jan. 27, 1917 +12 = 20/60	Mar. 9, 1917 +12 ⌒ +0.50 ax. 180 = 20/25	
Feb. 12, 1917 +10 = 3/100	Mar. 6, 1917 +10 = 20/200	Patient very unruly; squeezed. Other eye lost 2 yrs. before by another operator
Feb. 3, 1917 +8 ⌒ +5 ax. 180 = 20/20	Feb. 15, 1917 +9 ⌒ +4 ax. 180 = 20/15	
Feb. 5, 1917 +10 ⌒ +1 ax. 180 = 20/40	Mar. 2, 1917 +11 ⌒ +1 ax. 180 = 20/40	
Feb. 3, 1917 +10 ⌒ +4.50 ax. 180 = 20/30	Mar. 8, 1917 +10 ⌒ +4 ax. 15 = 20/20	
Feb. 7, 1917 +6 ⌒ +2 ax. 180 = 20/60	Mar. 12, 1917 +9 ⌒ +3 ax. 15 = 20/20	
Feb. 10, 1917 +10 = 20/65	Feb. 27, 1917 +11 ⌒ +2 ax. 175 = 20/15	
Feb. 27, 1917 +13 ⌒ +2 ax. 45 = 20/65	Mar. 6, 1917 +12 ⌒ +4.00 ax. 15 = 20/30	Small macula on cornea
Feb. 26, 1917 +6 ⌒ +5.50 ax. 180 = 20/80	Mar. 10, 1917 +5 ⌒ +5.50 ax. 180 = 20/15	

Duration.—From the most reliable information we were able to obtain from the patient in cases in which the diagnosis of cataract had been made at an earlier period, some, we found, were ripe in six months or less, while others were still immature after thirty years; consequently it must frequently happen that a cataract does not become ripe within the lifetime of the patient.

Type.—For operative purposes we have classified cataracts into immature, mature and morgagnian or "tumblers." The immature includes cases of opacity from that degree at which the fundus is still to be seen to the stage at which no reflex can be obtained, but clear cortex remains; they constituted 63 per cent. of the entire series. The mature comprises cases in which no clear cortex remained; these constituted 29 per cent. Morgagnian or "tumblers" are the cases in which the lens substance has become partially or almost completely liquefied; these constituted 8 per cent. of our cases. This percentage of "tumblers" is higher than that usually reported, the difference, we believe, being due to the fact that in capsulotomy operation the diagnosis, from the very nature of the case, is difficult to make as the capsule is lacerated before the cataract is delivered, and it is likely to pass unnoticed unless the liquefaction is far advanced. In the intracapsular operation the diagnosis is readily made after the delivery of the liquefied lens in its intact capsule. In India the percentage of "tumblers" runs even higher, being from 25 to 30 per cent., owing to procrastination on the part of the native patient, which results in more advanced liquefaction of the lens.

Tension.—The intra-ocular tension ranged from 7 to 40, and was taken with the tonometer in all cases in which it was possible to do so. In the two hemorrhagic cases to be discussed later, Cases 2c and 17c, unfortunately, only finger palpation was possible, but the tension was apparently normal. Low tension

seemed to have no bearing on operation or on results; high tension was not so innocuous apparently, for out of ten patients with a tension above 25, two had bad results, but in only one, Case 19, are we able to attribute this to the high tension. This patient had a chronic glaucoma with a tension of 32 and general arteriosclerosis. The other patient, Case 7c, had diabetes, arthritis deformans, and pus in the accessory nasal sinuses. In Case 32c, although trephining and an iridectomy both failed to reduce the tension below 40, the tension was reduced to 16 after extraction of the cataract, and vision was raised from hand movements to $^{2}/_{150}$.

Blood Pressure.—The systolic pressure varied from 105 in Case 102 to 300 in Case 34c. As a prophylactic measure against intra-ocular hemorrhage, the high pressure was reduced by treatment before operation as far as possible in each case. In one, Case 2c, with intra-ocular hemorrhage, we have no record of the blood pressure; the other, Case 17c, had a systolic pressure of 156. Seven patients had a systolic pressure of 200 or over on the day of operation, so that it would seem that blood pressure has no very important bearing on the results.

Vision Before Operation—This ranged from $^{2}/_{40}$ to uncertain perception of light or faulty projection. Vision obtained before operation does not always give a positive indication of what it will be after operation. Thus Case 1c, with uncertain light perception before operation, ultimately had a vision of $^{2}/_{30}$, and Case 90, with a prior faulty projection, had ultimate vision of $^{2}/_{15}$; conversely, Case 25c had $^{2}/_{150}$ before operation and $^{1}/_{200}$ afterward, the reduced vision in this case being due to intercurrent retinal hemorrhages of diabetic origin.

Iridectomy.—Until January, 1916, we performed preliminary iridectomy in most cases, doing eight extractions, however, during the early part of that

period without iridectomy; the latter procedure we found undesirable because in each instance the iris ultimately became adherent to the upper lip of the corneal wound, resulting in glaucoma and loss of useful vision in one instance, Case 6. Preliminary iridectomy was abandoned in January, 1916, in favor of the combined operation, as the former offered no advantages that were not more than offset by the added risk and inconvenience of a two-stage operation, and also because we have found that a limbal incision, with or without a conjunctival flap, such as we have been doing since that time, enables us to make a basal iridectomy. This we consider highly desirable and difficult to execute with the corneal section. A preliminary basal iridectomy interferes with the making of a good conjunctival flap subsequently.

The Van Lint flap was used twice, but was given up, as it required unnecessary manipulation, produced great trauma, and also subjected the eye to possible loss of vitreous while the sutures were being tied.

The Section.—In seventy-six of our cases a corneal section was used; in fifteen the section was limbal, while in fifty-five the usual conjunctival flap was made.

In studying the first half of the cases of this series, we found that a serious objection to the intracapsular operation as we then performed it was with the corneal incision; when the section must be so large as it is required to be for the intracapsular operation, it is certainly easier to make a corneal incision. This offers less impediment to the delivery of the lens, while at the same time it probably slightly reduces the danger from vitreous loss. But there are two serious objections to it: first, the greater frequency with which the iris adheres to the lips of the wound, and second, the liability of the downgrowth of epithelium from the wound into the anterior chamber. The latter factor, aside from other possible untoward results, usually materially reduces the vision, although a good reflex may be obtained with the ophthalmoscope. We believe

that this downgrowth of epithelium is of more frequent occurrence in the capsulotomy operation than is realized, but is not so readily discernible here because the epithelium merges with the posterior capsule, or after-cataract.

This epithelial downgrowth occurred in five of our cases in which a corneal section was made, but in only one case in which the limbal section with conjunctival flap was employed. In the latter, Case 87, a large amount of vitreous was lost because of carelessness on the part of the assistant in handling the speculum; healing was delayed, a factor in itself conducive to epithelial downgrowth.

Complications During Operation.—In Case 31 the extreme tip of the knife was broken off while making the section, and was so small that it could not be found, nor would it respond to the Giant magnet. Vision was $^{20}/_{25}$ three weeks after the operation, but was reduced to $^{20}/_{120}$ seven months later by a "quiet" uveitis, which persisted for some time. We are unable to determine whether or not the accident was the cause of the uveitis. In the past few months the vision has gradually improved as the vitreous opacities have begun to clear up.

Iris Prolapse.—This occurred in ten cases, or 6.8 per cent.; of these, 4.1 per cent. occurred in those in which the corneal section was used, and 2.7 per cent. in the cases of limbal section with conjunctival flap. These results would also speak in favor of a limbal incision with conjunctival flap as opposed to the corneal incision.

Case 79 had iris prolapse with an attack of secondary glaucoma which was controlled by treatment, but nevertheless resulted in reduction of vision; no untoward results have occurred in the other nine cases. In eight of these ten cases of prolapse, excision has been done.

TABLE 2.—COMPLICATED CASES

No.	Name	Age	Eye	Duration	Type	Tension	Syst. Blood Pressure	Vision before Operation	Method, Section, vitreous loss, and Complications	Date of Operation
										1914
1	C. B.	74	R	10 yrs.	T.	13	...	Light percep. uncertain	Simple; corn.	Nov. 11
										1915
2	E. M.	68	L	2 yrs.	Im.	N.	...	Fing. 10 in.	Prelim. iridect. Mar. 6, 1915, intra-ocular hemorrhage on table. Difficult delivery. Corn.	Mar. 18
3	A. B.	60	R	2 yrs.	T.	20	130	H. M.	Prelim. iridect. Apr. 5, 1915. Corn.	May 12
4	Mrs. J. B.	72	L	2 yrs.	Im.	N.	...	3/100	Comb. Corn.	May 13
5	M. B.	52	R	3 yrs.	Im.	N.	...	3/100	Comb. Corn.	May 26
6	E. H.	72	L	24 yrs.	Im.	N.	...	H. M.	Comb. Corn.	June 18
7	Mrs. B. H.	58	L	1 yr.	Im.	29	...	Light percep. good	Prelim. iridect. June 22, 1915. Corn. Severe iritis	July 6
8	Mrs. P. T.	60	L	2 yrs.	Im.	N.	...	Fing. 14 in.	Prelim. iridect. July 10, 1915. Corn.	July 21
9	Mrs. I.	73	R	.....	Im.	N.	...	Fing. 10 in.	Prelim. iridect. July 6, 1915. Corn.	July 23
10	Mrs. M. C.	70	R	15 yrs.	Im.	13	135	4/20	Prelim. iridect. July 28, 1915. Corn. Eye inspected Aug. 12, wound closed, pupil and chamber normal. Aug. 14, eye struck by hard brim of visitor's hat, wound opened, vitr. lost, pupil distorted. Subsequent healing delayed	Aug. 7
11	Mrs. H. J.	64	R	10 yrs.	Im.	27	202	Fing. 10 in.	Noninflammatory pupillary exudate. Prelim. iridect. Aug. 5, 1915. Corn.	Aug. 18
12	H. S.	75	L	1 yr.	M.	N.	...	H. M.	Prelim. iridect. Oct. 26, 1915. Corn. Iritis for several wks.	Nov. 9
13	T. O. D.	65	L	3 yrs.	M.	16	165	H. M. at 3 ft.	Prelim. iridect. Sept. 2, 1915. Corn.	Nov. 10
14	Mrs. F. E. K.	74	L	12 yrs.	Im.	28	130	H. M.	Iridectomy 9 yrs. prior for glaucoma. Corn.	Nov. 18
										1916
15	Mrs. F. E. K.	74	R	15 yrs.	Im.	21	130	Fing. 18 in.	Iridectomy 9 yrs. prior for glaucoma. Limbal.	Mar. 2
16	Miss M. G. G.	60	L	2 yrs.	M.	18	...	Light percep.	Comb. Limbal.	Apr. 5
17	Mrs. C. S.	78	R	6 yrs.	Im.	N.	156	Fing. 4 in.	Comb. Limbal. Choroidal hemorrhage on table	May 5
18	A. R.	67	L	30 yrs.	M.	24	165	Light percep.	Comb. Corn. Capsule ruptured; removed	May 9
19	Miss K. McL.	65	L	2 yrs.	Im.	32	160	Fing. 10 in.	Comb. Corn. Iris prolapse. Bled freely from iridectomy. Wound remained open for 4 weeks. Prolapse excised June 13, 1916	May 18
20	Mrs. J.	71	L	15 yrs.	M.	N.	...	Fing. 8 in.	Comb. Conj. flap.....	July 13
21	Mrs. M. J. O'F.	61	R	3 yrs.	M.	N.	...	H. M. at 6 in.	Old prelim. iridect. Conj. flap	Aug. 3
22	E. M. B.	70	R	2 yrs.	Im.	16	148	6/100	Comb. Conj. flap. Kalt forceps. Knapp method. Lens dropped down; delivered with Webber loop. Vitr. lost	Aug. 30
23	L. L. B.	67	L	2 yrs.	Im.	11	150	6/100	Comb. Conj. flap.....	Aug. 31

TABLE 2.—COMPLICATED CASES—*Continued*

Condition of Vision and Refraction		Remarks
At First Examination	At Last Examination	
Nov. 24, 1914 +7 ⌒ +7.50 ax. 160 = 20/150	Oct. 19, 1916 +13 = 20/30	Upper part of iris adherent to wound; refused operation by others previously
.....	Light perception lost	L. E., phthisis bulbi. R. E. showed marked cupping, glaucoma simplex; floating opacities in vitr. V. R. = 20/150
June 7, 1915 +6 ⌒ +3 ax. 90 = 20/80	Sept. 20, 1915 +9.50 ⌒ +1.50 ax. 150 = 20/15	16 days after operation was struck in eye with chair; wound opened, vitr. lost. Nov. 23, 1915, retinal detachment. Vision = L. P.
June 4, 1915 +6 ⌒ +8 ax. 165 = 20/15 June 12, 1915 +5 ⌒ +2 ax. 165 = 20/25 July 20, 1915 Fingers 20 ins.	June 29, 1915 +6 ⌒ +2.50 ax. 150 = 20/15 Feb. 16, 1917 +8 ⌒ +2 ax. 135 = 20/30 Feb. 26, 1917 +10 = 1/200	Adherent leukoma from penetration of eye with stick 8 yrs. prior to operation Rigid pupil; partial atrophy of nerve. Wassermann +++ Old iridocyclitis; adherent leukoma. Vessels contracted, disk pale. Extensive retinochoroidal changes probably sympathetic from other eye, lost from cataract operation 15 years prior
Sept. 16, 1915 +10 ⌒ +5.50 ax. 10 = 20/200	Feb. 16, 1916 +12 ⌒ +4 ax. 10 = 10/200	Diabetes and pansinusitis. R. E. lost 2 yrs. before by another operator from glaucoma. L. E., chronic iridocyclitis
.....	Nov. 10, 1916 +10 = 20/100	Diabetes, bedridden, rheumatism. Chronic dacryocystitis. Sac extirpated
Sept. 21, 1915 −5 ⌒ −2 ax. 90 = 20/100 Aug. 24, 1915 +1 ⌒ +5 ax. 115 = 20/100	Jan. 5, 1916 −5.50 ax. 90 = 20/80 Dec. 14, 1916 +7 ⌒ +5.50 ax. 115 = 20/25	Myope of over 24 D. Large areas of choroidal atrophy Jan. 26, 1917, detachment of retina. Good vision over a year. Feb. 7, 1917, Vision = L. P.
Oct. 29, 1915 +5 ax. 45 = 10/200	Dec. 22, 1915 +5 ax. 40 = 20/200	20 D. myope. Myopic fundus changes. Vitreous opacities before operation
Dec. 22, 1915 +4 ⌒ +9.50 ax. 65 = 20/60	July 19, 1916 +12 = 20/80	Floaters in vitreous. Sympathetic inflammation from other eye which had been lost by another operator
Nov. 26, 1915 +6 ⌒ +6 ax. 25 = 20/150 Dec. 18, 1915 +8 ⌒ +5.50 ax. 15 = 20/30 Apr. 4, 1916 +9 ⌒ +6 ax. 180 = 20/30 Light perception	Oct. 18, 1916 +12 ⌒ +1 ax. 90 = 20/60 Sept. 11, 1916 +10 ⌒ +4 ax. 15 = 20/30 Sept. 11, 1916 +10.50 ⌒ +4.50 ax. 180 = 20/30 Light perception	Medullated nerve fibers Disk cupped from old glaucoma Disk not cupped Retinal detachment diagnosed before operation; verified afterward
Light perception lost	Light perception lost	Diabetic. Had cerebral hemorrhage with partial recovery one year prior
Light perception June 7, 1916 +4 ⌒ +1 ax. 135 = 20/40	June 19, 1916 Light perception Feb. 15, 1917; hand movements	Retinal detachment suspected before operation; verified after operation Increased intra-ocular tension prior to operation. Fundus could not be seen to determine state of optic nerve. Chronic glaucoma
Aug. 1, 1916 −7 ax. 75 = 20/40	Jan. 23, 1917 −7 ax. 75 = 20/40	High myope. Circumpapillary atrophy; choroidal changes
.....	Dec. 8, 1916 +10 = 3/100	Large leukoma adherens from ophthalmia neonatorum with perforation
Sept. 19, 1916 +8 ⌒ +4 ax. 165 = 20/100	Feb. 19, 1917 +9 ⌒ +4.50 ax. 165 = 20/60	Other eye lost from iridocyclitis following cataract operation by another operator. R. E. shows evidences of sympathetic involvement. Numerous floaters in vitr.
Sept. 27, 1916 +11 ⌒ +3 ax. 180 = 20/120	Oct. 30, 1916 +11.50 ⌒ +4 ax. 15 = 20/20	Diabetes, chronic Bright's disease and bronchitis. Heavy consumer of alcohol

TABLE 2.—COMPLICATED CASES—*Continued*

No.	Name	Age	Eye	Duration	Type	Tension	Syst. Blood Pressure	Vision before Operation	Method, Section, vitreous loss, and Complications	Date of Operation
24	Mrs. L.	65	R	2 yrs.	Im.	24	300	Fing. 2 ft.	Camb. Conj. flap.....	1916 Sept. 13
25	Mrs. L. J. C.	60	L	1 yr.	Im.	22	160	20/150	Comb. Conj. flap.....	Oct. 5
26	Mrs. E. M.	81	R	5 yrs.	T.	N.	...	Light percep.; project. poor	Comb. Corn. Iris adherent to upper lip of corneal wound	Oct. 6
27	Mrs. L. H.	48	R	16 yrs.	Im.	20	130	Fing. 14 in.	Prelim. Oct. 2, 1916. Conj. flap	Oct. 13
28	Mrs. S.	78	R	5 yrs.	M.	N.	...	Light percep.	Comb. Conj. flap.....	Oct. 16
29	Mrs. C. R. C.	64	L	2 yrs.	M.	14	135	H. M. at 2 in.	Comb. Conj. flap. Capsule ruptured; removed	Oct. 24
30	Mrs. M. J. O'F.	61	L	Uncertain	Im.	N.	...	.....	Comb. Conj. flap.....	Oct. 27
31	J. C. B.	81	L	20 yrs.	Im.	N.	...	3/100	Comb. Conj. flap.....	Oct. 30
32	J. S.	55	L	9 yrs.	M.	40	...	H. M. at 10 in.	Prelim. iridect. Conj. flap.	Dec. 13
33	Mrs. M. R.	80	L	Uncertain	M.	25	270	H. M.	Delayed healing Comb. Conj. flap.....	1917 Jan. 23
34	Miss L. M.	64	R	4 yrs.	Im.	24	160	3/100	Comb. Conj. flap.....	Jan. 19

TABLE 2.—COMPLICATED CASES—*Continued*

Condition of Vision and Refraction		Remarks
At First Examination	At Last Examination	
Oct. 7, 1916 +9 ⌒ +3 ax. 165 = 20/40 Nov. 14, 1916 +10 ⌒ +3.50 ax. 165 = 10/150	Dec. 19, 1916 +12 ⌒ +3 ax. 150 = 20/30 Feb. 10, 1917 +10 ⌒ +3.50 ax. 165 = 10/200	Blood pressure reduced to 220 after six weeks' treatment before operation Diabetes; hemorrhage in macular region
.....	Nov. 2, 1916; light perception	Optic atrophy. Diagnosed as soon as media were clear enough
Nov. 16, 1916 +10 ⌒ +4 ax. 165 = 20/150	Jan. 20, 1917 +9 ⌒ +4 ax. 90 = 20/20	Other eye lost from previous operation by another operator. R. E. gave signs of sympathetic iritis by synechia
.....	.....	Patient died five days after operation from diabetic coma; also had cystitis. Eye in good condition at time of death
Nov. 7, 1916 +6 ⌒ +8 ax. 180 = 3/100	Nov. 16, 1916 +8 ⌒ +5 ax. 180 = 20/100	Left leg amputated 8 yrs. prior; diabetic gangrene. Diabetic white spots in macular region
Nov. 13, 1916 Fingers 4 feet Dec. 19, 1916 +7 ⌒ +4 ax. 180 = 20/60	Mar. 10, 1917 Fingers 4 feet Dec. 19, 1916 +7 ⌒ +4 ax. 180 = 20/60	Large leukoma adherens from ophthalmia neonatorum with perforation Circumpapillary atrophy; central retinal degeneration. R. E. lost from cataract operation by another operator 25 yrs. ago
Dec. 28, 1916 +11 = 1/30	Feb. 7, 1917 +6 ⌒ +5 ax. 60 = 20/150	Iridectomy to reduce tension Oct. 12, 1916; trephine for persistent glaucoma Nov. 13, 1916. Other eye blind from glaucoma. Dec. 27, 1917, Tn. L. E. = 16
Jan. 18, 1917 −5 ax 105 = 3/200	Feb. 14, 1917 +8 ⌒ +5 ax. 60 = 20/150	High myope; extensive choroidal changes. Circumpapillary atrophy. Systolic pressure reduced to 210 before operation
Feb. 1, 1917 +8 ⌒ +4 ax. 180 = 20/120	Mar. 1, 1917 +9 ⌒ +3.50 ax. 180 = 20/100	Divergent strabismus of right eye since childhood

Vitreous Loss.—Vitreous was lost in nineteen cases, or 13 per cent.

Seven had vision of $^{20}/_{15}$.
One had vision of $^{20}/_{20}$.
One had vision of $^{20}/_{25}$.
Four had vision of $^{20}/_{30}$.
One had vision of $^{20}/_{40}$.
Three had vision of $^{20}/_{60}$.
One had vision of $^{20}/_{100}$.
One had vision of $^{20}/_{200}$.

While vision was greatly reduced because of the loss of a large amount of vitreous in Cases 87 and 104, there was no complete failure on that account. In these two instances neither the operation nor the operator was to blame for the excessive vitreous loss. In the one case it was due to carelessness on the part of the assistant, and in the other to the extreme misbehavior of the patient. The loss of vitreous occurring through the operator's manipulation in an operation is much less to be feared than that resulting from the patient's squeezing, or to the assistant's carelessness, because in the first event a smaller quantity is usually lost.

Of the nineteen cases in which vitreous was lost, 63 per cent. were in patients 60 years of age or under, thus tending to show that the older the patient the more easily the lens is delivered, and bearing out the assertion already made that the suspensory ligament weakens progressively with age; in fact, our experience has taught us that patients may be too young for the intracapsular operation, but not too old.

Iritis.—Iritis occurred in five cases, or about 3.5 per cent. In Case 31, as already mentioned, the point of the knife was broken off and never found; Case 51 was a morgagnian cataract with a ruptured capsule; Case 7c was in a diabetic and there was an accessory sinusitis; and in Case 12c there was a sympathetic ophthalmia from a previous cataract operation on the other side. This leaves only Case 44 with an unexplained primary iritis following operation.

Iridocyclitis.—This occurred once in the series, in Case 26. This patient was one among several exhibited during the American Medical Association session in San Francisco. She was handled carelessly while being moved by mistake from one ward to another, during which time she had also a severe coughing spell. The wound was opened and vitreous extruded; the result was a soft eye with vision reduced to light perception.

Suppuration.—Despite the prevalent belief that loss of vitreous is conducive to infection, suppuration did not occur in any of our cases, with or without the loss of vitreous.

Intra-Ocular Hemorrhage.—This occurred in two cases. In Case 2c, the tension by finger palpation was apparently normal, but there was glaucoma simplex of the other eye. In Case 17c, the patient was a diabetic, and had had a cerebral hemorrhage one year prior to this with partial recovery. In both of these cases light perception was lost.

Glaucoma.—Glaucoma occurred twice. Case 6 was a simple extraction with a round central pupil two weeks after operation. This patient repeatedly showed the wound to friends by drawing up the upper lid and forcibly looking downward; this caused a stretching of the wound and the development of an astigmatism of 8 D.; two months after operation the vision was $^{20}/_{15}$. In time the upper part of the iris became adherent to the wound, and when the patient returned after an absence of six months the tension in that eye was 52 with vision reduced to perception of light; the disk was deeply cupped. Under treatment, vision was improved to $^1/_{200}$, March 1, 1917.

In Case 79, there was a prolapse of the iris and a lens that was hard to dislocate; three weeks after the operation, vision was $^{20}/_{30}$. Three months later an attack of acute glaucoma developed; the iris was released from the angle of the wound, but vision had

deteriorated to $^{2}/_{100}$. Tension is gradually going down and vision improving.

Retinal Detachment.—This occurred in two cases. In Case 3c, after an uneventful operation and convalescence, the patient was accidentally struck in the eye with a chair; the wound was opened, vitreous was lost, and the pupil distorted. After a delayed recovery, the patient's vision was $^{2}/_{15}$ for four months; then suddenly the retina became detached. The retinal tear could be seen on the lower nasal side; vision now amounts to light perception.

In Case 10c, the patient was struck in the eye with the hard brim of a visitor's hat; the wound was opened, vitreous was lost, and the pupil distorted. Healing was delayed, but the patient's vision fifteen months after the operation was $^{2}/_{25}$. The retina became detached, Jan. 26, 1917, vision now being limited to light perception.

We believe that the results in these two cases are to be attributed directly to the accidents which the patients sustained.

Ruptured Capsules.—The capsules were ruptured in ten cases, or 6.8 per cent., which included several in which the Kalt or the Stanculeanu forceps had been used to dislocate the lens; the capsules were subsequently removed in three of these cases.

Secondary Operations.—In seven cases, secondary operations were necessary to improve vision, being less than 5 per cent. Three of these were for the extraction of retained capsules, and four were cases in which an iridectomy was done to lower a drawn-up pupil.

Previous Operations.—Fourteen patients of this series had been previously operated on by other operators for cataract of the opposite eye, the results varying from mere light perception to total failure; the eyeball was usually shrunken and soft, and beyond all hope of redemption. The vision of the eye on which we operated is given in Table 3. The low vision in

some of these cases was due either to the results of sympathetic ophthalmia from the opposite side, or to extensive fundus lesions.

TABLE 3.—VISION OF EYE ON WHICH OPERATION WAS PERFORMED BY US

Case 16	20/15.
Case 27	20/15.
Case 41	20/15.
Case 76	20/15.
Case 100	20/20.
Case 27c	20/20.
Case 22	20/25.
Case 88	20/25.
Case 31c	20/60.
Case 22c	20/60.
Case 12c	20/80.
Case 104	20/200.
Case 7c	10/200.
Case 6c	1/200.

Diabetes.—This was a complication in eight cases. In all an attempt was made to put the patient in the best possible condition before operation, and the sugar reduced to the limits of safety. Patient 17c had an intra-ocular hemorrhage on the table a few moments after the delivery of the cataract, causing a complete failure. A year prior to the operation she had had a cerebral hemorrhage with partial recovery.

TABLE 4.—RESULTS IN DIABETICS *

Case 75, ultimate vision	20/20.
Case 23c, ultimate vision	20/20.
Case 29c, ultimate vision	20/100.
Case 8c, ultimate vision	20/100.
Case 7c, ultimate vision	10/200.
Case 25c, ultimate vision	10/200.
Case 17c, total failure from hemorrhage.	
Case 28c, death five days after operation from diabetic coma; eye in good condition.	

* Reference to Tables 1 and 2 will show additional complications.

The prognosis in the case of diabetic patients is poor, as a rule, for the visual results are often disappointing. In all but Case 17c, healing was uneventful; in four others, visual results were poor, owing to diabetic changes in the fundus or to uveitis. Two patients, however, had vision of $^{20}/_{20}$. These results would cause one to consider carefully the advisability of operating on a patient with diabetes, and the gen-

eral condition, the degree of visual disability, and the functional condition of the retina should be given careful consideration.

Refraction After Operation.—In studying the tables, one is struck by the change in refraction and the improvement in vision between the first and the last examinations recorded.

At the first examination, usually occurring within four weeks after the operation, astigmatism as high as 10 D. was found, the average being 4½ D.; after two years the highest astigmatism was 8 D., in Case 6, while many cases lost the astigmatic element entirely, the average being 2 D. The reason for the 8 D. persisting in Case 6 has already been explained; this is the only case in our series in which such a high degree of astigmatism persisted for an extended period.

With the decrease in the astigmatism, the hypermetropia usually increased. Contrary to the common conception, there was no appreciable difference in the amount of astigmatism that developed in the corneal incisions as compared with the limbal sections, or those with a conjunctival flap. In the majority of cases, astigmatism was against the rule, approximately at axis 165 for the right eye, and axis 15 for the left. The spherical error averaged 10 D.

A condition not shown by the tables was that the astigmatism markedly increased during the first week the patient used his eyes. On examination immediately after the removal of the dressings, which had been left on for ten days after operation, usually no astigmatism, or less than 2 D. was found, but it developed or increased during the first week, for we found that after the patient had used his eyes for a few days, astigmatism as high as from 8 to 10 D. was sometimes present; this we attribute mainly to the stretching of the wound by the downward pull of the depressor muscles. But the astigmatism decreased after the first few weeks as the wound grew stronger, until at the end of a year frequently no astigmatism remained.

In Cases 6 and 10, the right and left eyes of the same patient, 5 D. of astigmatism in one eye and 8 D. in the other were developed by the patients looking downward to show the operative scar to her friends; this astigmatism remained permanently. Since that experience we have cautioned our patients to refrain from looking downward, and enforce it by having them wear spectacles, the lower halves of which are covered with adhesive plaster, so that the tendency to look downward is reduced to a minimum. These are worn for about two weeks after the removal of the dressings.

Visual Results.—Only the visual results of the uncomplicated cases are summarized, in order to form a proper basis for a fair judgment as to the results obtained, the final vision in the complicated cases, as previously stated, being dependent on factors other than operative. The visual results of these complicated cases may be studied by referring to Table 2.

TABLE 5.—SUMMARY OF VISUAL RESULTS OF ONE HUNDRED AND NINE UNCOMPLICATED CASES

20/15	40 cases
20/20	24 cases
20/25	9 cases
20/30	15 cases
20/40	6 cases

A total of 94 cases, with vision from 20/40 to 20/15, equivalent to 86.2 per cent.

20/60	4 cases
20/80	1 case
20/100	3 cases
20/200	1 case
10/200	1 case
1/200	1 case
L. P.	1 case

Not included in the foregoing are three patients who left the county hospital without reporting for refraction, but whose vision on cursory examination was good.

Taking as our basis of comparison Herman Knapp's visual standard of

20/20 to 20/200.....first class,
18/200 to 1/200.....moderate results,
L. P. to loss of L. P. .....failure,

we have obtained the following results:

First class	97.25 per cent.
Moderate	1.83 per cent.
Failure	.92 per cent.

It is worthy of special note that sixty-nine, or 63 per cent. of the series, were cases of immature

cataracts. These patients would have been condemned to partial blindness if they had had to wait for their cataracts to become mature before operation. Another point of special interest is the fact that despite the greater frequency with which vitreous was lost in the immature cases, the visual results are higher than in the mature and morgagnian cases. Thus of the 69 cases of the immature, 68 patients, or 98.55 per cent., had vision of from $^{20}/_{120}$ to $^{20}/_{15}$, and 66 patients, or 95.65 per cent., had vision of from $^{20}/_{40}$ to $^{20}/_{15}$; of the 40 mature, including morgagnian, 38 patients, or 95 per cent., had vision of from $^{20}/_{200}$ to $^{20}/_{15}$, and 33 patients, or 82.5 per cent., had vision of from $^{20}/_{40}$ to $^{20}/_{15}$. Placed side by side for comparison these results are given in Table 6.

TABLE 6.—COMPARISON OF VISUAL RESULTS

	Immature		Mature	
Vision	No. of Cases	Per Cent.	No. of Cases	Per Cent.
From 20/120 to 20/15 68		98.55	38	95
From 20/40 to 20/15 66		95.65	33	82.5

CONCLUSIONS

1. A limbal section, preferably with a conjunctival flap, is more desirable than a corneal incision.

2. There is no appreciable difference in the amount of astigmatism resulting after a corneal section as compared with a limbal, with or without a conjunctival flap.

3. The simple extraction is not well adapted to the intracapsular operation.

4. The combined extraction has several advantages over a two-stage operation with a preliminary iridectomy.

5. While all forms of senile cataract can be successfully removed by the Smith Indian intracapsular cataract operation, the immature cataract is the type that lends itself best to this procedure, especially in patients past the age of 60.

210 Post Street.

ABSTRACT OF DISCUSSION

DR. HOWARD F. HANSELL, Philadelphia: The intracapsular extraction of cataract according to modern methods, that of Smith, that of Stanculeanu and that of Knapp, presents advantages so obvious that every operator feels inclined to give up his old ways and accept the modern.

The East Indian method has been made familiar to us all by Smith himself, his co-workers and the Americans who have operated under his supervision. The excellent results reported by the late Dr. D. W. Green, by Fisher and finally by the Drs. Green, and its availability for the immature cataract, ought to recommend it to the unprejudiced operator. But are we unprejudiced? And isn't our prejudice based on good ground? The operator of unlimited opportunities, such as Smith, is justified in adopting any method that appears to him to be best, for he can rapidly become an expert; but it is quite otherwise with the surgeon who has limited opportunities, and most of us come under that class. We may approve of the newer methods, but we hesitate to change because we have secured a moderate skill by pursuing the same method of operation for years, and the untried method induces a certain amount of nervousness that is hard to overcome, and at first, at least, leads to disaster.

I was enthusiastic about the Stanculeanu operation three years ago, but gave it up because I felt I had no right to expose my patients to danger that they were comparatively free from when I performed the standard operation. Again, Dr. Arnold Knapp's operation, a modification of the Stanculeanu, appealed to me strongly, and if I could perform it as safely and as well as he does, I would surely discard the old way and adopt his. His paper, published in the *Archives of Ophthalmology*, January, 1915, gives results that correspond favorably with any others that have been published of extraction by any method. Of seventy-six uncomplicated cataracts, sixty-eight patients obtained vision of $^{20}/_{15}$ to $^{20}/_{40}$. Prolapse of the vitreous, including the slightest loss, occurred sixteen times. Drs. Green obtained $^{20}/_{15}$ to $^{20}/_{40}$ in ninety-four cases, equivalent to 86.2 per cent. These results, recorded according to Dr. Herman Knapp's standard, were extremely good. First class, 97.25 per cent.; moderate, 1.83 per cent.; failure, 0.92 per cent. Drs. Green had loss of vitreous in nineteen cases, or 13 per cent. Knapp, after witnessing 2,616 operations by Smith, reported 6.8 per cent. loss of vitreous. Knapp's statistics of 1,032 operations by the usual methods, presented 7.32 per cent. loss of vitreous; hence less than by the intracapsular methods.

This accident is unfortunate. In many cases a slight loss of vitreous is not incompatible with full acuity of vision, as is generally agreed, but even when the loss seems insignificant I fear ultimate bad vision even after a year or more

of satisfactory use of the eye. The ruptured hyaloid membrane becomes partly opaque and floats around in and near the pupillary area. If even fixed and susceptible of improvement of vision by discission, the improvement is, as a rule, only temporary. Casey Wood says in *A System of Ophthalmic Operations,* p. 1233, "There can be no doubt but that the great majority of cases of vitreous loss during extraction of senile cataract do not materially endanger the success of the operation. On the other hand, however, many evil results follow from such an accident." With the first statement not all of us are in accord. For a time it is true the loss of vitreous seems not to interfere in any way with healing or with the prospect of good vision, but later I believe the majority of cases are the worse for it.

The advantages of the Stanculeanu operation, according to my experience in witnessing Stanculeanu and his assistants in its performance, is the inability of the operator to be sure that the ligament is ruptured. The horizontal and vertical movements of the lens while the forceps grasp the capsule, so extensive that the periphery of the lens is brought into view, would seem to be forcible enough to rupture the suspensory ligament. An unruptured suspensory ligament is a source of great danger and expulsion of the vitreous is nearly sure to follow attempts at expression. Knapp's method up to this point is practically identical. His next step is to press on the lower third of the lens, force it to evert and be expressed as in the Smith operation. By this maneuver the ligament is positively ruptured and the danger of vitreous loss from this cause is eliminated.

Drs. Green advise against preliminary iridectomy. The scar at the periphery resulting from this operation, unless the knife is introduced in the corneal edge of the limbus, will interfere with the conjunctival flap, as they assert. The advisability of dividing the operation of extraction has been threshed out and its advantages and disadvantages are known to all of us. Personally I prefer preliminary iridectomy and advise it when the circumstances seem to justify it, and I do not find that it prevents a satisfactory conjunctival flap. I quote agree that the Van Lint flap ought not to be used for the reasons stated, namely, unnecessary manipulation and the time consumed before the real operation is commenced. Stanculeanu recommends it and always performs it, claiming speedier union of the edges of the wound and fewer cases of infection.

Drs. Green's paper is a valuable contribution to the subject, and is especially valuable in that it goes into great detail, so that the reader is furnished with all data necessary for comparison.

Dr. H. D. Thornburg, Chicago: The essayists are to be congratulated not only on the brilliancy of their results but

also on the careful, painstaking manner in which they have tabulated their interesting and important data. Vitreous loss has not seemed frequent nor the grave complication it has been painted, if we are to judge by the visual results following its occurrence. Their figures, too, would seem to refute the contention that glaucoma and retinal detachment are more likely to occur following the intracapsular than the classical method of operating. They are especially to be congratulated for their extremely low percentage of capsular rupture and secondary operations.

On the whole, however, when one considers the similar published reports of the intracapsular operation by Millette, the late Greene, Vail, Knapp and Fisher, he is not surprised at the wonderfully favorable results reported by the essayists and is very much inclined to consider this the operation of choice. As superintendent of an eye hospital where both methods are practiced, I have been confirmed in this view, my only hesitancy being occasioned by the fact that all those I have mentioned except Millette are men who have been to India.

Since every one of them has modified and continues to modify the original technic as taught him by Smith to meet American conditions, I do believe it possible for myself and others to acquire the proficiency that makes such results possible without the Indian experience—a matter of vital interest to the members of this section.

I believe this can be accomplished by persistent practice of the method described by Fisher before the Academy of Ophthalmology and Oto-Laryngology at Memphis in December, 1916, and again in his recent monograph, making use of the eyes of 4-weeks-old kittens, which present anterior segment conditions analogous to those of the human eye. In addition to other work, I have now operated on forty-one kittens—eighty-two eyes—according to the technic described, and know I did the last one very much better than I did the first, and propose continuing the practice.

It would seem that with the numerous favorable reports and with the general adoption of this or some other new method of acquiring technic there can be no question that the time is fast approaching when the intracapsular method will be generally accepted as the operation of choice for senile cataract; but a word of warning should be given to those who would adopt it, and that is that the technic must be mastered before doing so, as should that for the classical cataract or any other eyeball operation, and which I firmly believe to be possible with the material described. The essayists are to be congratulated for their prominent part in the movement that is certain to establish this operation on the basis it deserves.

DR. L. D. GREEN, San Francisco: There is one important point that the ophthalmologist is apt to overlook, and that is that 63 per cent. of these cases were of immature cataract. The question is: Would we have gotten the same results by doing the capsulotomy operation in this 63 per cent.? We intend to run a series of cases of immature cataracts with the capsulotomy method so as to compare the results.

Another question is as to the small percentage of secondary operation with the intracapsular method, at least in our series, and I think the same applies to other operators. Dr. Thornburg suggests practice on animals. One thing you have to consider is that the pressure required in delivering the lens in an animal is not quite the same as in the human eye, and also, you are dealing with a normal lens and not a cataractous lens. It is not quite the same, although you undoubtedly will gain a great deal of knowledge in the various procedures.

REPORT OF THE COMMITTEE ON COLLECTIVE INVESTIGATION CONCERNING THE OCULAR MUSCLES

In the last report made by this committee, a brief review was given of what had been accomplished, and the plan of further study proposed. It was pointed out that, as convergence and all questions concerning the action of the extra-ocular muscles are more or less related to accommodation, it therefore seemed desirable, first of all, to separate as clearly as possible what we do know from what we do not know concerning the latter important function. We found we could all agree that accommodation was essentially the result of an effort made by the ciliary muscle. We recognized the changes which normal accommodation undergoes with advancing years, and saw how it was influenced by the "minimum doses" of certain miotics, and cycloplegics, the term "minimum dose" indicating the smallest amount which has been found sufficient, by repeated experiment, to produce the characteristic effect on a normal eye of a given amount of a given drug in a given number of minutes.

Having thus cleared up the ground, in part at least, by deciding as best we could certain mooted points, our committee recommended that the attention of the section be directed next to anomalies of accommodation.

The importance of this aspect of ophthalmology is well stated by Duane when he says:

> Considering the amount of attention devoted to refractive errors and their correction, the study of accommodative anomalies has received comparatively little attention from ophthalmologists. Yet those who deal day after day in their consulting-rooms with the problems of refraction work recognize, I am sure, that these anomalies often occasion considerable trouble, so that if we fail to diagnosticate and treat them, we are not doing our whole duty to our patients.

In spite of this we do not yet have any standard method by which to determine when excessive or insufficient accommodation does exist or for expressing the degree which is present. The result is that widely different views are held as to the frequency of anomalies of accommodation. Some writers, for example, consider that spasm of accommodation is comparatively rare, while Schmidt-Rimpler and others would have us believe that this spasm can be found in a quarter or, with certain groups of ametropia, in more than half our cases.

Evidently the reason for this difference is in our methods of examination and in the definitions of what we find.

Let us therefore consider, first, excessive accommodation, in order to agree, if possible, on the method to be followed, in principle at least, in making the successive tests, and also on the symptoms which we are to consider sufficiently characteristic to warrant that diagnosis.

A categorical statement of these points gives no opportunity for explanations, but it has the merit of brevity.

For the diagnosis of any anomaly of accommodation we probably agree that we should know: (*a*) The age of the individual; (*b*) the refraction of the eye as determined by placing at least 0.00025 gm. (1/250 grain) of atropin on the conjunctiva and making careful tests of the refraction not less than forty minutes nor more than two hours thereafter, and (*c*) any cause which materially affects the general nervous system of the patient.

For the diagnosis of excessive accommodation or spasm we shall probably also agree that:

1. The range of accommodation is restricted as compared with that of the otherwise normal eye. The near point is too near and the far point also may be approached—that is to say, an emmetropic eye may appear to be myopic. Under such circumstances if we apply a sufficient quantity of atropin to relax any existing excess of accommodation, the amount of this apparent myopia, which has disappeared under the influence of the cyclopegic, measures, of course, the degree of excessive accommodation which was present. This is a diagnostic symptom.

2. Measurement of the relative accommodation shows that the positive part is greater and the negative part less than normal. This symptom is also diagnostic, especially as concerns the relaxation of the accommodation to the convergence.

3. A minimum dose of certain cycloplegics usually causes the ciliary muscle to relax less rapidly than normally.

4. A minimum dose of certain miotics usually causes the ciliary muscle to contract more rapidly than normal. The curves for these minimum doses of certain cycloplegics and miotics have been published in previous reports of this committee and elsewhere.

The evidence furnished by the third and fourth tests is only corroborative, not diagnostic.

What has been said in regard to the diagnosis of excessive accommodation applies also to insufficient accommodation, if we understand that, *mutatis mutandis,* the conditions are the same.

One practical point may be mentioned, however. Since the normal eye in early life has the power of accommodation of about 3 diopters when the visual axes are in the primary position, that is, when they are parallel perpendicularly to the base line and in the horizontal plane, then a patient should be able to see a given test type in the distance as well with

a minus 3 spherical before the eye as without that glass—any ametropia of course which exists having been corrected. This has been already stated. In view of the simplicity of this test, our results could be more uniform if this use of a minus 3 diopter glass formed a part of routine examinations.

As the terms "excessive" or "supernormal" accommodation and "spasm" of accommodation are used interchangeably, and as the terms "insufficient," "subnormal," "paresis" and "paralysis" of accommodation are also used interchangeably, and as it is desirable for us to ascertain and also to express in our records, and in any published statement, not only the kind but also the degree of the anomaly of accommodation, therefore our committee recommends that we should use the terms describing these anomalies as follows:

1. *Excessive Accommodation.*—A generic term to describe any degree of accommodation greater than normal in a person of a given age.

2. *Supernormal Accommodation.*—An excessive accommodation of less than 1 diopter.

3. *Spasm of Accommodation.*—Excessive accommodation greater than 1 diopter.

4. *Insufficient Accommodation.*—A generic term to describe any degree of accommodation less than normal in a person of a given age.

5. *Subnormal Accommodation* or *Paresis of Accommodation.*—An insufficient accommodation of less than 1 diopter.

6. *Paralysis of Accommodation.*—Insufficient accommodation greater than 1 diopter.

It is too much to expect that every member of the section will take the trouble to make all or even a part of these tests in the routine of office work. Certainly not every one would care to indicate in his records the special variety of an anomaly of accommodation which might be found. But the approval by the section of the few recommendations here made would be at least a convenience to American ophthalmologists who are inclined to do exact work, and it would also be an expression of the desire of the section for even a little more scientific accuracy.

Several other points might be brought forward in this connection. But the principal object of such a committee as this is to act as a clearing house to bring into accord and unify terms and methods which we employ. It is desirable, therefore, to make these reports always conservative and submit for approval only a little at a time.

In concluding this report, acknowledgments should be made to members of the section and to others who are cooperating in this collective investigation. As they have not been studying excessive accommodation, however, due notice of their work must be deferred until later.

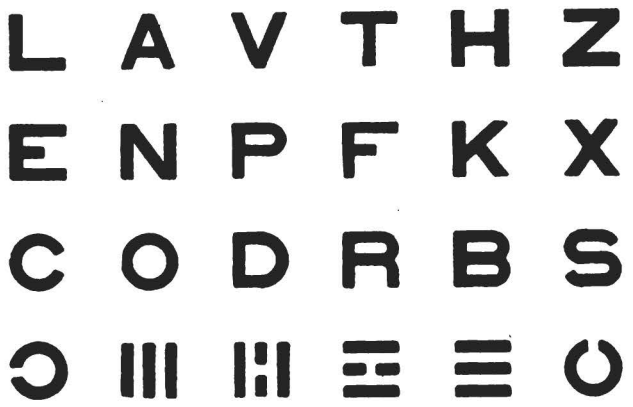
It is hoped that the present suggestion to establish a standard for the diagnosis of excessive action of the ciliary muscle may at least tend to lessen some of the confusion which exists in our discussions and in our literature. We appreciate that the recommendations are few and simple, and relate only to one small phase of a large subject. But with patience in clearing up thus one point after another, we hope that some substantial progress can ultimately be made in our knowledge of the ocular muscles.

LUCIEN HOWE,
HOWARD I. HANSELL,
THEODORE B. SCHNEIDEMAN,
Committee.

REPORT OF COMMITTEE ON STANDARDIZING TEST CARDS

In submitting the third report of this committee, it must again be emphasized that the most urgent need for the standardization of test cards arises from the fact that letters vary in visibility; yet they constitute the most generally useful of test objects for the subjective measurement of refraction. If, as has been generally done, the various letters are constructed and numbered on a fixed ratio of size to distance, they furnish an inaccurate measure of visual acuity.

To have test letters that will accurately test visual acuity, (*a*) the different letters on the same line must be made of different sizes; (*b*) the letters employed must be restricted



Gothic test letters for use at 5 meters.

to a very few, thus forfeiting the great advantage of using letters, or (*c*) the different letters must be standardized by comparing their visibility with that of some constant standard test. We have undertaken the latter method.

Last year a comparison was reported of eighteen block letters with the international broken ring test. We now report the results obtained by comparing eighteen Gothic letters with the same standard. The letters chosen are shown herewith, of the size subtending an angle of 5 minutes each, at the distance of 5 meters.

The results obtained show that if vision indicated by seeing the break in the broken ring at the standard distance

(5 meters) be taken as standard vision, V. = 1, the visual acuity indicated by the different letters is shown in Table 1.

TABLE 1.—COMPARATIVE VISUAL ACUITY INDICATED BY VARIOUS LETTERS

	V.=		V.=
L	0.62	F	0.81
A	0.71	C	0.85
T	0.72	K	0.88
V	0.74	O	0.88
H	0.74	D	0.88
Z	0.76	X	0.91
E	0.77	R	0.93
N	0.79	S	1.05
P	0.79	B	1.15

A new test of visual acuity proposed by Dr. A. E. Ewing[1] consists of squares, each composed of three lines. In some squares the center line has a break in it subtending the angle of 1 minute at the distance at which the test would be used to indicate standard vision. Samples of this test are shown in the last line of the accompanying illustration. Comparison of Ewing's test seems to show that it is slightly less visible than the broken ring, indicating at the standard distance V. = 1.13.

Your committee has also arranged two series of block letters with the sizes of the various lines proportioned in accordance with the results of comparisons reported last year, so that the reading of half the letters of the line, or all the letters of the line shall require, as exactly as may be, the visual acuity indicated therefore. These series are given in Table 2.

TABLE 2.—TEST CARDS OF BLOCK LETTERS FOR USE AT FIVE METERS

Proper Height for Letters	Letters to Be Used	Vision if Half Are Read	Vision if All Are Read
51.47	L		0.1
36.2	T B		0.2
22.3	V O H		0.3
16.7	C Y N G		0.4
12.9	L Y O E S		0.5
10.65	T U Y P R D		0.6
9.06	L C F N H S G B	0.7	0.8
6.67	V O P D N R G H	0.9	1
5.38	T U Y P Z E R S	1.1	1.2
4.44	L V U C D N E G	1.3	1.5
	SECOND CARD		
53.6	T		0.1
33.35	L H		0.2
24.2	D R B		0.3
16.1	T V Z S		0.4
13.34	C F D N G		0.5
10.77	V O F E S R		0.6
9.06	U P N E R S H B	0.7	0.8
6.67	T O Y D Z E G H	0.9	1
6.04	F D N E G H B S	1.1	1.2
4.44	L T V U P R H Z	1.3	1.5

1. Ewing, A. E.: Tr. Am. Ophth. Soc., 1916.

The test cards for illiterate patients obviously require standardization, as do also the color and surface of the cards on which the letters are to be printed. But these have not yet received adequate attention from your committee.

Respectfully submitted,

EDWARD JACKSON, Chairman,
ROBERT FAGIN,
WALTER B. LANCASTER.

ANNUAL REPORT OF THE AMERICAN BOARD FOR OPHTHALMIC EXAMINATIONS

The American Board for Ophthalmic Examinations since its organization at Washington, May 8, 1916, has spent a year of activity in the work for which it was formed. After discussion it was decided that all applications for examination should cover as far as practicable the applicant's professional life; and where public and definite records of his work give full guarantee of his fitness for ophthalmic practice, that the certificate of the board should be granted without such an examination as would be necessary for those whose professional record was yet largely to be made.

Announcements regarding requirements for applicants and times and dates for examinations were prepared and sent to members of our constituent organizations, and to intended applicants. Examinations have been arranged to be held at Memphis, Dec. 14 and 15, 1916, and at New York City, June 8 and 9, 1917. The total number of applicants up to this date is 121. Of these some have been rejected as ineligible for examination, and a certain proportion will be certificated on their professional records. At Memphis, in December, eleven appeared for examination. It is anticipated that a much larger number will appear for examination in New York City this week. It is proposed to publish the list of all who have been awarded the certificate of the board, after the results of the examinations in New York City have been decided.

Many letters of inquiry have been received, both with regard to the examinations, and also with regard to the course of study that would prepare for such examinations. The latter, coming both from individuals who desired to take such courses, and from institutions that propose to give them, show that the activities of the board have already awakened an important interest in the subject of training for ophthalmic practice. This correspondence has entailed a large amount of labor in the secretary's office, as well as on the members of the board in immediate charge of the examinations. The employment of an assistant secretary for the board, as contemplated in the plan of organization, was rendered necessary.

In December there was brought to the attention of the board the need for special examinations for applicants for fellowship in the American College of Surgeons, who claimed eligibility on the ground of being engaged in ophthalmic practice, and the difficulties experienced by the college in giving these applicants proper examinations. It was at once evident that if any special examination as to the training

for ophthalmic practice was to be given such applicants, it was extremely desirable that these examinations should conform closely to those given the applicants to this board. The establishment of a single standard of requirement in ophthalmology throughout this country, the lack of which is so keenly felt with regard to requirements for the general practice of medicine, could thus be secured, to the great advantage of the applicants, the profession and the public.

The board therefore immediately entered into negotiations with the authorities of the College, who agree to make its members a credentials committee of the College, in control of all examinations in ophthalmology conducted for the College, and to pay from its own receipts all the expenses of such work. The number of such applicants awaiting examinations in ophthalmology to determine their fitness for fellowship in the college is now several hundred. The opportunity for prompt and wide extension of the influence our examinations will exert on the teaching, study and practice of ophthalmology is very important. It is suggested that each of the organizations represented in the board should offer its representatives on the board as members of the Credentials Committee in Ophthalmology of the American College of Surgeons. It is believed that affiliation to that extent will be beneficial in every way.

The receipts of the board have been $3,225.50. Its expenditures have been $1,047.05.

During the year, the term of Dr. William H. Wilder, representing the American Ophthalmological Society, expired; and Dr. John E. Weeks succeeded him. Dr. Edward C. Ellett, representing the Section on Ophthalmology of the American Medical Association, and Dr. Frank C. Todd, representing the American Academy of Ophthalmology and Oto-Laryngology, whose terms also expired, were chosen to succeed themselves. In December, the board lost the services of one of its most active, earnest and enthusiastic workers, Dr. Wendell Reber. To fill the vacancy thus created, Dr. William H. Wilder of Chicago was appointed to represent the American Academy of Ophthalmology and Oto-Laryngology.

The organization of the board for the past year has been: chairman, Edward Jackson; vice chairman, Myles Standish; secretary, Frank C. Todd; Committee on Preliminary Requirements, Myles Standish, William H. Wilder and Frank C. Todd; Committee on Examinations, Hiram Woods, Walter B. Lancaster and Wendell Reber; Committee on Finance, Edward Jackson, Edward C. Ellett and Alexander Duane.

The important financial and other responsibilities resting on the board have induced it to secure incorporation under the laws of Minnesota. It will facilitate its proceedings to have this action ratified by each of the constituent organizations, and action to that effect is recommended.

EDWARD JACKSON, Chairman,
FRANK C. TODD, Secretary.

REPORT OF COMMITTEE ON KNAPP TESTIMONIAL FUND

To the Members of the Section on Ophthalmology, American Medical Association:

Again your Knapp Testimonial Fund Committee desires to express appreciation of the support given this enterprise by a representative number of the members of this section. Owing to the urgent appeal, the number of contributions for the year 1916 increased to over 200, thus enabling us to close the year with nearly as much cash balance on hand as we had at the end of the year 1915, even though our expenses were increased.

To each contributor of $10 or more, a volume of the "Ophthalmic Year Book" and a volume of the *Transactions* of the section have been furnished, and to each contributor of less than $10 but more than $1, a volume of the *Transactions* alone has been furnished. Aside from this the various activities promoted by the fund have been kept up, including the Knapp Medal and the encouragement and assistance of research work.

Owing to the uncertainty concerning the fate of the "Ophthalmic Year Book," your committee has not made any promises concerning the furnishing of that work to contributors to the Knapp Fund, though it is hoped that the arrangement of previous years may be continued. We are therefore soliciting a continuance of subscriptions for 1917 in the same amounts as heretofore subscribed, and promise the contributor that the funds so received will be employed to the best advantage in sustaining the various activities promoted by the fund. There is an especial need for funds to encourage and promote original research work, and for that reason, aside from all other considerations, your committee urges a liberal response to the request for a continuation of the Knapp Fund subscriptions in amounts the same as heretofore given.

Your committee desires to say that a number of requests have been made for appropriations from the Knapp Fund to assist various enterprises either directly or indirectly connected with ophthalmologic work, and while such enterprises were in every way deserving of encouragement, yet your committee has not deemed it good policy to depart from the regulations laid down at the time the fund was started. In short, the fund was created to promote and sustain activities and enterprises directly connected with the section's welfare,

and especially to encourage and promote scientific work and advancement in the field of ophthalmology. There is abundant opportunity in the latter field alone, and your committee believes that all surplus funds be employed in that channel.

The status of the fund since our published statement of last year is as follows:

Receipts				Disbursements	
1	at $15		$ 15.00	Printing	$ 66.50
187	at 10		1,870.00	Multigraph and Add.....	31.40
5	at 5		25.00	A. M. A.	218.00
4	at 2		8.00	Year Book	1,447.50
7	at 1		7.00	Postage	52.20
			$1,925.00	Tiffany & Co.	143.00
Balance from 1915			856.30	Research work	120.00
				Incidentals	4.25
					$2,082.85
				Balance	698.45
Total			$2,781.30	Total	$2,781.30

Respectfully submitted,

KNAPP TESTIMONIAL FUND COMMITTEE.
ALBERT E. BULSON, JR., Treasurer.

REPORT OF COMMITTEE ON AWARD OF KNAPP MEDAL

To the Members of the Section on Ophthalmology, American Medical Association:

Your Committee on the Award of the Knapp Medal has been impressed by the excellence of a number of the papers that were presented at the meeting of the Section on Ophthalmology in 1916. An unusual feature of the session was the presentation of an address by the President which contained the results of much carefully planned, well executed and competently observed research work which has resulted in elucidating the causes for a phenomenon, namely, the variation of degree of choked disk in the two eyes in cases of increase of intracranial pressure, the reason for which has heretofore been unknown. While your committee was unanimously of the opinion that this communication should be placed on the same footing with other papers in regard to the award of a Knapp medal, it did not assume the responsibility of deciding the question. A ruling was asked of your Executive Committee through your secretary. This ruling was unanimously in favor of including the President's address, and it has been so considered.

In view of the excellence of three of the papers, it has been thought desirable to accord favorable mention to the papers by Dr. Lucian Howe on "The Fatigue of Accommodation," Dr. Mark J. Schoenberg, on "Intracranial Treatment of Syphilis and Parasyphilitic Optic Nerve Affections," and of Dr. George Slocum, entitled "A Study of Ophthalmoscopic Changes in Nephritis." The committee has separately and unanimously voted the award of the Knapp Medal to Dr. Walter R. Parker, the chairman of this Section in 1916, author of the paper entitled "The Relation of Choked Disk to the Tension of the Eyeball: An Experimental Study."

We trust that the award will receive the hearty approval of our fellow members.

JOHN E. WEEKS, Chairman,
CHARLES H. MAY,
WILLIAM C. POSEY.

IN MEMORIAM

It is with deep regret that we record the death of Dr. Wendell Reber at his home in Philadelphia, Dec. 30, 1916. While attending the meeting of the American Academy of Ophthalmology and Oto-Laryngology in Memphis, he contracted a cold which rapidly developed into lobar pneumonia, the immediate cause of his death.

He was born in St. Louis, April 4, 1867; was educated in the city of his birth, and entered Washington University, from which he graduated with the degree of Doctor of Medicine in 1889.

For a few years he practiced general medicine in St. Louis, and preparatory to his entrance into ophthalmology he spent a year in the State Hospital for the Insane at Norristown, Pa. During these early years he laid a broad foundation in general medicine and nervous and mental diseases which accounted in a large measure for the great success which he ultimately achieved in his chosen field of ophthalmology.

His brilliant career as an oculist was begun in Pottsville, Pa. After a few years of practice in this restricted field, however, he located in Philadelphia and entered on his life's work.

The energy and enthusiasm with which he entered into his new field of labor rapidly gained for him the recognition of the profession as an earnest, conscientious and capable ophthalmologist. He became identified with the German, Jefferson, and Polyclinic hospitals. In the latter two teaching institutions he gave early evidence of special qualifications for teaching, and finally was rewarded with an appointment as Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine.

His early struggles for recognition were many, but his buoyancy, optimism, conscientious work and genial disposition after a time won for him the recognition which he so well merited. Shortly after his election to the chair of Ophthalmology in the Philadelphia Polyclinic, he was honored by being elected to the chair of Diseases of the Eye in the Medical Department of Temple University. At the time of his death he was Professor of Ophthalmology in the Philadelphia Polyclinic and College for Graduates in Medicine, Professor of Diseases of the Eye of Temple University Medical School, Ophthalmologist to the Philadelphia General, Samaritan, Garretson and Polyclinic hospitals, Consulting Ophthalmologist to the Rush Hospital for Consumption and Allied Diseases and the Friends' Hospital for Nervous and Mental Diseases.

Dr. Reber had numerous society affiliations. In addition to membership in most of the local Philadelphia societies he was a fellow of the American College of Surgeons; a fellow and enthusiastic worker and ex-president of the American Academy of Ophthalmology and Oto-Laryngology; a member and ex-chairman of the Eye, Ear, Nose and Throat Section of the Medical Society of the State of Pennsylvania, and an active member of the Section on Ophthalmology of the American Medical Association. He was one of the eight honorary members of the Chicago Ophthalmologic Society. His society affiliations were not limited to this country. He was for many years a member and a frequent attendant of the Oxford Ophthalmologic Congress, England; in 1914 he was the only American representative to that distinguished body, and in the same year, by invitation, delivered an address on "The Present Status of Heterophoria."

He possessed a facile pen. American and English ophthalmic literature have been much enriched by his contributions. Among the best of his writings was the volume on "Muscular Anomalies of the Eye," which he produced jointly with a colleague, Dr. Howard F. Hansell. This volume is well known to all ophthalmologists. It passed through its second edition and was about to appear in a third at the time of his death. He contributed the chapter on "Anesthesia" in "Wood's Ophthalmic Operations," and was one of the assistant editors of the "Ophthalmic Year Book." He also was one of the contributors to the "American Encyclopaedia of Ophthalmology." In addition to these recognized contributions, ophthalmic literature is replete with shorter papers of great merit from his pen.

The historian would be remiss if he did not pause for a moment to pay a tribute to Dr. Reber as a teacher. His devotion to his students and to the work of teaching was carried almost to a fault. He was clear, concise, had due regard for the views of others when they differed from his own, and was always eager to impart knowledge to anyone who showed a desire to learn. If there was one thing which contributed more than others to the undermining of his general resisting powers so that he became an easy prey to the disease which claimed him, it was his devotion to his teaching and the burning up of energies in his efforts to instruct his "boys" and to make them finished ophthalmologists. Postgraduate ophthalmologic education in America owes much to Wendell Reber. As an educator, he had a keen interest in the raising of standards and in exacting greater efficiency. One of the most recent of his contributions was "The Future of Postgraduate Ophthalmologic Education in America," a paper which he read before the Chicago Ophthalmologic Society. He was one of the nine members of the National Board of Examiners for Advanced Standing in

Ophthalmology, and it was while in discharge of this official duty that he contracted the illness which caused his death.

As a man he was genial, artistic, enthusiastic and optimistic in temperament; loyal in his friendships and strong in his convictions; the champion of the weak and of right. These qualities, together with his keen sense of humor and cheerful disposition, attracted to him a host of loyal and devoted friends who today mourn in Wendell Reber a friend, an ophthalmologist of renown, a writer, and above all a teacher and educator of rare ability.

Dr. Reber is survived by his widow, who, before their marriage on Jan. 6, 1902, was Miss Jessie Dalrymple.

Dr. Wilbur Boileau Marple was born July 20, 1855, at Columbus, O., and died suddenly at Kennebunkport, Me., Aug. 30, 1916. He received his preliminary education in his native city, beside High School and State University. He left the latter to enter Amherst College in 1875, where he graduated in the class of 1877. After graduation he taught school until 1879, when he began the study of medicine at the Starling Medical College, Columbus, O. Graduating in 1881, he became associated with Dr. Foster at Washington Court House, Ohio, until 1883, when he came to New York City to do special eye work under Dr. Hermann Knapp.

In 1884 he joined the medical staff of the Immigrant State Hospital where he remained until 1890, when he went abroad for postgraduate study.

In 1892 Dr. Marple was appointed Assistant Ophthalmic Surgeon to the New York Eye and Ear Infirmary, and nine years later he became full Surgeon, which position he held at the time of his death.

From 1895 to 1901 he was Visiting Surgeon to the New York Polyclinic Hospital; Consulting Surgeon to the Babies' Hospital in 1900; Consulting Surgeon to the Central and Neurological Hospital, Blackwell's Island, in 1912.

Dr. Marple was a member of the American Medical Association, American Ophthalmological Association, American College of Surgeons, American Academy of Ophthalmology and Oto-Laryngology, American Therapeutical Society, New York Academy of Medicine and New York Ophthalmological Society.

The following are some of his contributions to opththalmic literature:

On the Pathology of Hypopyon Keratitis (1893); The Indications and Contraindications for the Use of Atropin in the Treatment of Diseases of the Eye (1897); Pemphigus of the Conjunctiva (1900); The Ocular Lesions of General Arteriosclerosis (1907); Extraction of Cataract in the Capsule (1908); Tubercle of the Choroid in Tuberculous Meningitis

(1912); Arteriosclerosis as Seen by the Ophthalmologist (1915).

A well known modification of the Electric Ophthalmoscope bears Dr. Marple's name.

Dr. Marple was a stanch friend, of pleasing and genial personality and ethical in his dealings with professional confrères and patients. He had a great fund of general information, beside being well versed in general medicine and in his specialty.

REPORT OF COMMITTEE ON ELLIOTT TREPHINE OPERATION

The committee on Elliott trephine operation has no conclusive report to make at this meeting. Due to lack of funds to cover the expense of printing and postage, circular letters were not sent out to members of the section.

Your committee would request that funds be provided for this purpose in order that a supplementary report may be made at the next meeting.

Respectfully submitted,

WALTER R. PARKER, M.D., Chairman.

REPORT OF COMMITTEE ON CONSERVATION OF VISION

DR. HIRAM WOODS, BALTIMORE

In the matter of the work of the Committee on Conservation of Vision, I received a communication during the past year from Dr. Allport, chairman, stating that owing to lack of funds, and for various other reasons into which it is unnecessary to enter, the Council on Health and Public Instruction could not make the usual appropriation this year for the continuation of conservation work, and that as the work could not be done, he offered his resignation as chairman. The letter is as follows:

CHICAGO, May 11, 1917.

Dr. Hiram Woods.

Dear Doctor:—At the Minneapolis meeting of the American Medical Association a Conservation of Vision Committee of the Council on Health and Public Instruction was formed, and I was requested to assume the chairmanship. This I reluctantly consented to do, for I was well aware of the fact that my other duties left no unoccupied time at my disposal for the performance of conservation of vision work. By making many sacrifices, however, and by the expenditure of about $600 a year out of my own pocket, I have done what I could and trust it has met with your approval. The work is before you and you know about what it is. Up to Jan. 1, 1917, the American Medical Association has given $600 a year for the support of the committee. Owing to financial reverses, this income stopped Jan. 1, 1917, and since then the support has proceeded from the Academy of Ophthalmology and Oto-Laryngology and myself. The American Medical Association has always paid for our printed matter which has, of course, been of great assistance. The American Medical Association has also furnished a box of stereopticon slides for each state for lecturing purposes.

We appointed state managers in each state, whose duty it has been to perfect a state organization and report once a year to our committee. Wherever possible, the state managers have also been made chairmen of the conservation of vision committee of the various state medical societies. In this way, harmony in work has been secured. These state managers have secured the services of well-known oculists in various portions of their states, who have agreed to go and lecture in neighboring cities whenever invited. Their expenses have usually been paid by the society issuing the invitation, although many men have paid their own expenses, thus sacrificing both time and money in the enterprise. The state managers have arranged for lectures before medical societies, women's clubs, teachers' institutes, normal schools, etc., and thus far about 500,000 people have listened to these lectures. We have positive assurances that much good has followed the lectures. Among such benefits may be mentioned medical school inspection, school nurses, schools for defectives, better shop conditions, the abolition of the "shop oculist" and common towel in many factories, active work in the passage of laws for ophthalmia neonatorum, trachoma, wood alcohol,

shop conditions, school children's eyes, etc. Our committee has also been instrumental in having lectures delivered in medical schools by the professors of ophthalmology on visual conservation. While the American Medical Association published its weekly *Health Bulletin*, and sent it broadcast all over the United States for use by papers, magazines, etc., our committee kept it constantly supplied with practical articles on the care of the eyes, avoidance of accidents, etc. We also published twenty pamphlets on subjects concerning visual conservation, that have had a large sale, and that have been gratuitously distributed to thousands of readers. These pamphlets were written by well-known ophthalmologists and no technical language was used in any of them. We have reason to believe that they have accomplished much good.

A very important work of our committee has been the collection of practically all the state laws in the United States concerning the eyes. This collection has been divided into seven papers and has appeared quarterly in *Ophthalmology*. The subjects have been as follows:

State Legislation Concerning School Children's Eyes, etc.
State Legislation Concerning Ophthalmia Neonatorum.
State Legislation Concerning Trachoma.
State Legislation Concerning Wood Alcohol.
State Legislation Concerning Shop Conditions, etc.
State Legislation Concerning Optometry.
State Legislation Concerning the Blind.

These articles will be revised and soon will be published in book form by the American Medical Association. In this one volume can then be found practically every law in the United States on the subject of the eye. These are not the only accomplishments of our Committee, but it is enough to demonstrate the usefulness of the organization and its possibilities for the future. This committee has become a national institution and is highly regarded wherever its work is known. Much remains to be done, however, and it is a pity that the committee is compelled to close its labors and go out of existence, especially as it only requires about $1,500 a year to enable us to go on with the work. I am sure that the American Medical Association regrets most sincerely that owing to "lack of funds" this and other committees must cease from their beneficial labors; but under present conditions they have no alternative. I, therefore, desire to say to the members of our committee and to the state managers who have so bravely carried on the work in their several states, that I regretfully retire from the work as chairman of the committee and thank them from my heart for the kindness and generosity they have shown me, and for the work they have so cordially given to the cause of visual conservation.

Cordially yours,

FRANK ALLPORT.

Dr. Allport has done a tremendous amount of work and spent a good deal of money, I know, privately, in addition to the appropriation. The work is organized with a chairman in each state, slides are furnished for lecture purposes, literature written in a popular vein has been prepared, and it seems a pity to have this work stopped, with all the dangers that attend a renewal of it.

Now the knowledge of that state of things came to the Conservation Committee of the National Committee for the Prevention of Blindness located in New York, of which Mr. Edward M. Van Cleve, formerly of Columbus, is superintendent. Mr. Van Cleve was recently in Baltimore and outlined to me verbally a plan for continuing our work, which plan he subsequently put in writing in the form of the following letter:

NEW YORK, June 4, 1917.

Drs. Alger, Lewis and Woods.

Gentlemen:—Some weeks ago Dr. Allport wrote to me to say that the resources of the subcommittee of the council on Health and Public Instruction, of which he is chairman, were too limited to continue the service longer, and with the meeting of the American Medical Association in New York this month it would probably cease to exist. This seems to me so unfortunate that I wish some means might be found to continue the Committee on Conservation of Vision, in order that the results of its organization may not be lost. Throughout the country there are devoted state chairmen who are giving their services and influence to the cause in which we are all interested. There has been no conflict and no serious crossing of lines in the work done by Dr. Allport's Committee and this organization; in fact, there has been the utmost harmony of cooperation.

It occurs to me that out of the present crisis there may come a mutually satisfactory arrangement for even closer cooperation. I write this to you as the three members of our board of directors, who are also members of Dr. Allport's committee, and you will know whether or not such arrangement as I am about to propose will be agreeable to the Council. If it is not likely to prove agreeable, you will please disregard the suggestion; if you deem it worthy of presentation to the Council, you will know what steps should be taken to consummate the arrangement.

Let the committee be continued with its present membership, adding perhaps one of the younger New York ophthalmologists who could be the local acting chairman for Dr. Allport, and let it become by action of our executive committee an integral part of our committee, retaining its name and being known as "The Committee on Conservation of Vision, American Medical Association, Cooperating with the National Committee for the Prevention of Blindness." Our Executive Staff, in close association with the local representative of the American Medical Association committee, would conduct the work to the best of its ability, relying on the continued effective service of the state chairmen who have served at Dr. Allport's call, and contributing the work of our office in the task of directing and advising these men; contributing also our resources in the collection of lantern slides, the exhibits, etc.

From year to year, the Committee on Conservation of Vision would report to the American Medical Association, and when again the financial condition shall make it practicable the Committee would be in a position to resume its former activity, if it then seemed desirable, or it could continue in this proposed fellowship, making such financial contribution as it might deem proper; providing the arrangement proves mutually agreeable.

Perhaps a man like Dr. Walter B. Weidler, whom you know well, I think, and whom we have found to be most effective in the lecture work here in New York State and in New Jersey, might be induced to undertake the local acting chairmanship. Another very effective young man is Dr. Conrad Berens, Jr.; however, he is in the service of the federal government and is not now available.

The admirable service rendered by Dr. Allport in organizing his army of lecturers is surely worthy the appreciation of the Council, and some such arrangement as I suggest might serve to prevent the disintegration of this fine fighting force. What I offer is, of course, only a suggestion, but I hope it will not appear to you inapplicable to the present situation.

Sincerely yours,

EDWARD M. VAN CLEVE,

Managing Director, National Committee for the Prevention of Blindness.
130 East Twenty-Second Street.

So the situation is this: The conservation work as carried on since 1913 cannot be continued for the present because we have no money. The Conservation Committee of the National Association for the Prevention of Blindness expresses its willingness to take over that work and to continue it as part of its own, provided a New York man—they suggest Dr. Walter B. Weidler—be appointed to act as local vice chairman, so that they can have a man whom they can get at, and hand the work back to the Association at such time as the Council on Health and Public Instruction can finance it.

My recommendation is that we adopt this plan, with the appointment of a New York man.

The members of the committee are as follows: Frank Allport, chairman, Chicago; E. M. Alger, New York; W. E. Bruner, Cleveland; J. J. Carroll, Baltimore; E. C. Ellett, Memphis, Tenn.; George H. Kress, Los Angeles, Calif.; W. B. Lancaster, Boston; F. Park Lewis, Buffalo; J. A. Stucky, Lexington, Ky.; Wendell Reber [deceased], Philadelphia; George C. Schaeffer, Columbus, Ohio; W. H. Wilder, Chicago; Casey A. Wood, Chicago, and Hiram Woods, Baltimore.

For action on the foregoing report see the minutes of the section.

LIST OF FELLOWS OF THE AMERICAN MEDICAL ASSOCIATION REGISTERED IN THE SECTION ON OPHTHALMOLOGY

List of Fellows of the American Medical Association who registered in this Section at one or more of the last five Annual Sessions, together with Fellows who have subscribed to the Transactions of the Section for 1917. The figures following the names indicate the attendance at the Annual Sessions (7 indicates 1917, 6 indicates 1916, etc.). T follows names of nonattendant Fellows subscribing to the Transactions for 1917.

Corrections will be appreciated.

ALABAMA

ANNISTON

Huey, Thos. F., 1119½ Noble St., T.

BIRMINGHAM

Ledbetter, Samuel L., First Nat'l Bank Bldg., 6, 7, 8, 9, 0, 1, 3.
Nabers, Samuel F., First National Bank Bldg., 4, 7.
Sibley, B. D., 2150 Highland Ave., T.

MOBILE

Wright, Ruffin A., 11 N. Joachim St., 4.

MONTGOMERY

Thigpen, Chas. A., 13 S. Perry St., 8, 4, 6, 7.

TUSCALOOSA

Searcy, Harvey B., 807 Greensboro Ave., T.

ARIZONA

DOUGLAS

McLellan, Geo. H., U. S. Army, 5.

PHOENIX

Martin, Ancil, Goodrich Bldg., T.

ARKANSAS

EUREKA SPRINGS

Huntington, Robert H., 6th and Spring Sts., T.

HOT SPRINGS

Rowland, John F., Ark. Nat'l Bank Bldg., T.

LITTLE ROCK

Caldwell, Robt., State Bank Bldg., 0. 5.

TEXARKANA

Lanier, L. Herbert, 5.

CALIFORNIA

BAKERSFIELD

Hamlin, Francis A., Hopkins Bldg., T.

FRESNO

Trowbridge, D. H., 1, 5.
Walker, J. R., 5, 1, 5.

LOS ANGELES

Anderson, Helen O., Bradbury Bldg., 1, 5.
Bullard, Frank D., 1241 West 8th St., 1. 5.
Detling, Frank E., Investment Bldg., T.
Dudley, Wm. H., Brockman Bldg., 9, 1, 5.
Ellis, H. Bert, Bradbury Blk., 6, 8, 9, 0, 1, 2, 4, 5.
Graham, Richard W., 607 S. Hill St., 1, 5.
Kelsey, Arthur L., Brockman Bldg., T.
Kress, George H., Bradbury Bldg., 2, 3.
Libby, Arthur A., Bradbury Bldg., T.
MacLeish, Archibald L., Brockman Bldg., 7, 1, 2, 5, 7.
Mansur, L. W., Brockman Bldg., T.
McCoy, Thos. J., Security Bldg., 1, 5.
Miller, F. W., Merchants Nat'l Bank Bldg., 8, 1, 5.
Mills, Lloyd, 1669 West Adams St., 5.
Shaul, J. W., 1342 Elza Ave., T.
Thorpe, L. S., Marsh-Strong Bldg., 0, 1, 5.

MODESTO

Reamer, E. F., 8, 3.

OAKLAND

Brinckerhoff, Elmer E., 1st Nat'l Bank Bldg., 5.
Brinckerhoff, G. E., Delger Bldg., T.
Card, Egerton F., Broadway and 14th St., 1, 5.

Friedman, William L., 1706 Broadway, 5.
Hazen, E. H., 5539 Kales Av., T.
O'Connor, Roderic P., 148 Moss Ave., 5, 6.
Thomas, H. G., Dalziel Bldg., 7.

PACIFIC GROVE

Ritchie, A. M., 5.

PALO ALTO

Kirk, Josiah H., 4, 5.
Thomas, Jerome B., Frazer Bldg., 1, 5.

PASADENA

Dilworth, W. D., Citizens Savings Bank Bldg., 1, 5.
Roberts, W. H., 461 E. Colorado St., 6, 8, 9, 1, 4, 5.
McKellar, James H., Citizens Bk. Bldg., T.

REDLANDS

Church, Benj. F., 1, 3, 5.

REDWOOD CITY

McKee, Albert B., R. F. D., 5, 7.

SACRAMENTO

Briggs, Wm. E., 1005 K St., 1, 5, 6.
Spencer, G. A., Odd Fellows Temple, T.

SAN FRANCISCO

Alexander, Edgar W., Butler Bldg., 1, 5.
Barkan, Adolph, 704 Market St., 5.
Barkan, Hans, 506 Sutter St., 5.
Bixby, Edward M., Shreve Bldg., 1, 5.
Black, James A., Hotel Fairmount, 5.
Cohen, Albert, 146 Grant Ave., 5.
Cohn, Robert D., 126 Stockton St., 5.
Deane, Louis C., 350 Post St., 1, 5.
Dolman, Percival, Butler Bldg., 5.
Franklin, Walter S., Butler Bldg., 1, 5.
Fredrick, Marcus White, 135 Stockton St., 5.
Glaser, Edward F., 391 Sutter St., 5.
Green, Aaron S., Shreve Bldg., 5, 6.
Green, Louis D., 210 Post St., 5, 7.
Hopkins, Edward K., Butler Bldg., 5.
Howard, J. V., U. S. Navy, Mare Island, 5.
Hulen, V. H., 135 Stockton St., 6, 7, 8, 9, 0, 1, 5, 6.
Kirk, A. W., 681 Market St., 5.
Lewitt, Frederick C., St. Paul Bldg., T.
Lucchetti, Victor F., 916 Kearney St., 1, 5.
Martin, William A., 870 Fell St., T.
McMurdo, John R., 240 Stockton St., 5.
Nagel, C. S. G., 209 Post St., 1, 5.
Philip, John Harold, 133 Geary St., T.
Pischel, Kasper, 135 Stockton St., 5.
Shortlidge, E. D., 209 Post St., T.
Smith, James F., 86 Post St., 5, 7.
Stephens, W. B., Shreve Bldg., T.
Sumner, Percy, 111 Ellis St., 5.

SAN JOSE

Jordan, P. A., 84 S. 1st St., 1, 5.
Wagner, Edward R., 84 S. 1st St., 5.

SANTA BARBARA

Ryan, L. R., San Marcos Bldg., T.

SANTA ROSA

McLeod, James H., 1, 5.

WOODLAND

Fairchild, Chester H., T.
Ward, M. W., 530½ Main St., 1, 5.

COLORADO

BOULDER

Spencer, Frank R., Physicians' Blk., T.

CANON CITY

Orendorff, Otis, 506 Main St., 8, 0, 4, 5, 6.

COLORADO SPRINGS

Magruder, A. C., Burns Bldg., 6.
Neeper, E. R., Box 567, 7.
Patterson, J. A., Burns Bldg., 7.

DELTA

Smith, H. A., T.

DENVER

Bane, W. C., Metropolitan Bldg., T.
Black, Melville Metropolitan Bldg., 6, 9, 1, 3, 4, 7.
Burns, H. R., Mack Bldg., T.
Chase, John, Majestic Bldg., 7.
Conant, Edgar F., Metropolitan Bldg., 5.
Coover, David H., Metropolitan Bldg., T.
Grant, W. W., Mack Bldg., T.
Jackson, Edward, Majestic Bldg., 6, 7, 8, 9, 0, 1, 2, 3, 4, 5, 6, 7.
Sedwick, W. A., Metropolitan Bldg., 6.
Walker, Charles E., Jacobson Bldg., T.

PUEBLO

Pattee, James J., 1st National Bank Bldg., T.

TRINIDAD

Presnall, C. W., 1202 Arizona St., T.

CONNECTICUT

BRIDGEPORT

Miles, H. S., 417 State St., 6, 7, 9, 1, 2, 7.
Warner, George H., 849 Myrtle Ave., 7.

DANIELSON

Burroughs, Geo. M., 3 Broad St., 9, 0, 2, 4, 7.

HARTFORD

Waite, Robert L., 68 Pratt St., T.

HARTLAND

Gill, M. H., 36 Pearl St., 8, 2, 7.
Smith, E. T., 36 Pearl St., 9, 4, 7.
Waite, F. L., 68 Pratt St., T.
Weidner, C., 49 Pearl St., 9, 4.

MERIDEN

Pierce, E. W., 53 W. Main St., T.

NEW HAVEN

Blake, Eugene M., 55 Trumbull St., 4, 7.
Cohane, Jeremiah J., 59 College St., 7.
Ring, Henry W., Hotel Taft, 4.
Swain, Henry L., 232 York St., T.

NEW MILFORD

Wright, George H., 7.

SOUTH MANCHESTER

May, George W., Cheney Bldg., T.

STAMFORD

Avery, John Waite, 295 Atlantic St., T.

WATERBURY

Rodman, C. S., 6, 7, 2, 4.

DELAWARE

MILFORD

Marshall, S. M. D., 111 W. Front St., 7.

WILMINGTON

Ellegood, J. A., 9th and Market Sts., 7, 9, 2, 4.
La Motte, W. O., 2011 Monroe, 4, 7.

DISTRICT OF COLUMBIA

WASHINGTON

Butler, W. K., 1207 M St., N.W., 6, 7, 9, 2, 4.
Davis, William T., 927 Farragut Square, T.
Dufour, Clarence R., 1343 L St., N.W., T.
Greene, Louis S., 1624 I St., 0, 4.
Lamb, Robert S., Stoneleigh Court, 4.
Lyon, Martha M. Brewer, 48 V St., N.W., T.
Muncaster, S. B., 907 16th St., N.W., 6, 7, 8, 9, 4, 7.
Newell, William S., 1424 K St., T.
Phillips, Orlyn S., % Indian Office, 5.
Polkinhorn, Henry A., 1201 M St., 9, 4.
Wilkinson, Oscar, 1408 L St., N.W., 6, 7, 8, 9, 2, 3, 4, 7.
Wilmer, W. H., 1601 I St., N.W., T.

FLORIDA

DE LAND

Ingram, L. C., Dreka Bldg., 6.

JACKSONVILLE

Heggie, Norman M., Buckman Bldg., 7.
Manning, Wm. S., 215 Laura St., 4.

MIAMI

Hodsdon, B. F., 0, 4, 5.

TAMPA

Bird, U. S., T.
Taylor, Joseph W., Citizens Bank Bldg., 4.

GEORGIA

ATLANTA

Calhoun, F. Phinizy, Candler Bldg., 1, 2, 3, 4, 6, 7.
Lokey, Hugh M., Candler Bldg., 4.
Roy, Dunbar, Grand Opera House, T.

GRIFFIN

Austin, William H., 107½ Hill St., 7.

ROME

Cox, Ross P., T.

SAVANNAH

Martin, H. H., 247 Bull St., 6, 7, 9, 0, 3, 6, 7.
Osborne, Elton S., 19 E. Jones St., 6.

IDAHO

BOISE

Maxey, Ed. E., 5.
Nourse, Robert L., 5.

ILLINOIS

AURORA

Allen, George F., Frazier Blk., 6.
Pratt, John A., Coulter Blk., T.

BELLEVILLE

Auten, Frank E., 6.
Gunn, John C., 500 E. First St., 0, 4.

BLOOMINGTON

Smith, J. Whitefield, 1122 E. Grove St., 4.

BUSHNELL

Duntley, George S., 408 E. Main St., T.

CAIRO

Dunn, J. W., 8th and Washington Sts., 6, 8, 0, 3, 5, 6, 7.

CHICAGO

Abele, L. H., 209 S. State St., T.
Allport, Frank, 7 W. Madison St., T.
Andrews, A. H., 32 N. State St., T.
Barr, W. Allen, 32 N. State St., T.
Brawley, Frank E., 7 W. Madison St., 6.
Brown, Earl J., 7 W. Madison St., T.
Brown, E. V. L., People's Gas Bldg., 6, 8, 9, 0, 4.
Brown, Heman H., 31 N. State St., T.
Collier, Clinton C., 9119 Commercial Ave., T.
Crossley, E. R., Heywood Bldg., 7.
Dodd, Oscar, 30 N. Mich. Blvd., 8, 0, 1, 6.
Faith, Thomas, 31 N. State St., 6.

Fiske, David, 25 E. Washington St., 6.
Francis, Charles H., 30 N. Michigan Blvd., T.
Frank, Mortimer, 30 N. Michigan Blvd., 6, 9, 0, 2, 4, 7.
Fuller, C. G., 32 N. State St., T.
Gamble, Wm. E., 5535 Kenwood Ave., 8, 9, 0, 3, 4, 6.
Goldenburg, Michael, 4641 Vincennes Ave., 6, 7.
Gradle, H. S., 32 N. State St., 3, 6.
Harris, M. L., 25 E. Washington St., T.
Hawley, C. W., 7 W. Madison St., T.
Hayden, A. A., 25 E. Washington St., 0, 3.
Kraft, Oscar H., 2600 Hampden Ct., 3.
Lane, Francis, 25 E. Washington St., 6.
Lebensohn, Mayer H., 739 W. 12th St., 4, 6.
Mahoney, G. W., 30 N. Michigan Blvd., 8, 3.
Mann, William A., 30 N. Michigan Blvd., T.
Mundt, G. H., 432 Normal Parkway, T.
Nance, W. O., 5330 Hyde Park Blvd., 8, 0, 3.
Noble, W. L., Reliance Bldg., 2, 4, 5, 6.
Orcutt, D. C., 7 W. Madison St., 8, 3, 6.
Peterson, William A., 30 N. LaSalle St., 5.
Slavik, Edward F., 2202 S. Crawford Ave., T.
Suker, Geo. F., 25 E. Washington St., 3, 6.
Swan, C. J., 77 E. Washington St., 0, 2, 3.
Thornburg, H. D., 31 N. State St., 7.
Tivnen, Richard J., Hotel Metropole, 7, 8, 2, 4, 7.
Tydings, Oliver, 31 N. State St., 6.
Vonder Heydt, Robert, 25 E. Washington St., T.
Walker, Sydney, Jr., 25 E. Washington St., 7.
Walter, Will, 122 Michigan Ave., 7, 8, 9, 0, 3, 6, 7.
Wescott, C. D., 22 E. Washington St., 8, 9, 0, 1, 2, 3, 6, 7.
Wild, Theodore (Jr.), 803 Milwaukee Ave., T.
Wilder, Wm. H., 122 S. Michigan Ave., 6, 7, 8, 9, 0, 3, 6, 7.
Williams, T. J., 30 N. Michigan Ave., 6.
Wood, Casey A., 7 W. Madison St., 6, 7, 8, 9, 0, 2, 3, 6, 7.
Woodruff, Thomas A., 30 N. Michigan Blvd., 7, 8, 9, 0, 2, 3, 7.

CHICAGO HEIGHTS

Gorrell, T. J. H., 1661 Thorn St., T.

DIXON

Edgar, Thomas O., 6.

ELGIN

Gillette, Philip F., Sherwin Blk., T.

EVANSTON

Boot, G. W., 800 Davis St., T.

FREEPORT

Clark, J. Sheldon, 76 Stephenson St., T.

GALESBURG

Matheny, R. C., 306 E. Main St., 6, 7.

HOOPESTON

Earel, A. M., T.

JACKSONVILLE

Adams, A. L., 5, 8, 0, 3, 6.
Gregory, A. R. (Jr.), 326 W. State St., T.

JOLIET

Woodruff, H. W., 1201 Western Ave., 8, 1, 2, 6, 7.

MATTOON

Coultas, R. J., 1706 Broadway, 6.
Voit, C. B., 8, 0, 3.

MOUNT VERNON

Edmondson, Edward E., Box 43, 6.

PEORIA

Bradley, E. H., Woolner Bldg., T.
Brobst, C. H., Central National Bk. Bldg., 7, 8, 9, 0, 1, 2, 6.
Welton, C. B., Jefferson Bldg., 8, 3.
Williams, Wright C., 201 Rebecca St., 6.
Wyatt, W. W., Central Bank Bldg., 8, 0, 3.

PONTIAC

Middleton, A. B., 8, 0, 2, 3, 6.

QUINCY

Pendleton, F. M., Stern Bldg., 8, 1, 3.

ROCKFORD

Fringer, W. R., William Brown Bldg., 6.
Starkey, Horace M., Brown Bldg., T.

ROCK ISLAND

Ostrom, Louis, Peoples National Bank Bldg., T.

SPRINGFIELD

Hägler, E. E., Hägler Bldg., T.
Prince, A. E., 628 Capitol Ave., T.

STERLING

Brodrick, F. W., 6.

STREATOR

Hill, H. C., 317 Main St., T.
Lester, H. S., T.

URBANA

Scheib, Geo. F., 1007 Illinois St., 3.

INDIANA

ATTICA

Kelsey, T. W., Central Bank Bldg., 6, 7.

AUBURN
Stewart, Charles S., 6.

BEDFORD
Emery, C. H., 8, 2, 4, 6.
Heitger, Jos. D., Heitger Bldg., T.

BLUFFTON
Shoemaker, S. A., 6.

EVANSVILLE
Field, William H., 1440 S. Second St., 6.

FORT WAYNE
Bulson, Albert E., Jr., 406 W. Berry St., 6, 7, 8, 9, 0, 1, 2, 3, 4, 6, 7.
McBride, W. O., Gauntt Bldg., T.
Glock, H. E., 930 Calhoun St., 6.

HUNTINGBURG
Knapp, H. C., 6.

INDIANAPOLIS
Heath, F. C., Newton Claypool Bldg., T.
Hood, Thomas C., 226 Newton Claypool Bldg., 6.
Hughes, W. F., Hume-Mansur Bldg., 6.
Larkin, Bernard J., Hume-Mansur Bldg., 7.
Newcomb, John R., Hume Mansur Bldg., 7.
Pfafflin, Charles A., Newton Claypool Bldg., T.
Sharp, Walter N., Hume-Mansur Bldg., T.
Whitaker, Joel, Hume Mansur Bldg., 7.
Willis, Edward A., Hume-Mansur Bldg., 6.

JEFFERSONVILLE
Funk, Austin, 6.

LAFAYETTE
Keiper, Geo. F., 6, 7, 8, 9, 4, 7.

MARENGO
Grant, P. T., 6.

MISHAWAKA
Bostwick, James G., 117 S. Main St., 6.

MUNCIE
Miller, Charles E., 315 S. Jefferson St., 7, 9, 0, 2, 3, 4, 6, 7.

NEW ALBANY
Leach, W. J., 6, 7, 8, 9, 0, 3, 7.

RICHMOND
Hays, George R., 27 S. 8th St., T.
Stevenson, D. W., T.

SOUTH BEND
Hager, W. A., 103 N. Lafayette St., 2. 6.

TERRE HAUTE
Breaks, L. Z., Tribune Bldg., 6.

TIPTON
Read, Horace G., 3.

WARREN
Black, C. S., 6.

IOWA

ARMSTRONG
West, G. H., 8, 3.

BURLINGTON
Frantz, Charles P., Iowa State Bk. Bldg., T.
LaForce, E. Frank, Iowa State Bk. Bldg., 8, 0, 3, 6, 7.
Stutsman, Carl, Iowa State Bank Bldg., 3.

CEDAR RAPIDS
Bailey, Fred W., Security Bldg., 3.
Walker, H. L., 3.

CLARINDA
Matthews, R. J., 8, 3.

CLINTON
Weih, E. P., Wilson Bldg., T.

COUNCIL BLUFFS
Dean, Frank W., 424 Oakland Ave., 0, 3.
Henninger, L. L., City Nat. Bk. Bldg., T.

CRESCO
Connolly, Wm., 3.
Jinderlee, J. W., 3.

DAVENPORT
Elmer, A. W., 730 E. Locust St., 4.
Vollmer, Karl, Schmidt Bldg., 7.

DES MOINES
Cook, C. P., Utica Bldg., 3.
Downing, J. A., 417 Equitable Bldg., 6.
May, George A., Equitable Bldg., 7.
Parker, Ralph H., Fleming Bldg., T.
Pearson, W. W., Equitable Bldg., 8, 9, 4, 7.
Shore, F. E. V., Citizens Bank Bldg., 6.
Werts, C. M., Equitable Bldg., 3, 6.
Will, Frank A., Equitable Bldg., 6.

DUBUQUE
Gratiot, H. B., 256 10th St., 4.

ESTHERVILLE
Bradley, W. E., 5.

IOWA CITY
Boiler, W. F., Box 155, 3.
Dean, Lee Wallace, T.

IOWA FALLS
Pagelsen, O. H., 6.
Cobb, Edwin, Masonic Temple, T.

MARSHALLTOWN
Lierle, F. P., 3.
Singleton, E. M., 3.

MUSCATINE
Johnston, W. H., Hershey Bldg., T.

NEW HAMPTON
Fallows, H. D., 3.

NEWTON
Engle, Harry P., 4.

OTTUMWA
LaForce, Burt D., Ennis Bldg., 8, 0. 3, 4.

SIOUX CITY
Reeder, James E., Davidson Bldg., T.
Walters, B., Frank, Box 332, T.

VINTON
Griffin, Clark C,, 3.

WATERLOO
Small, W. B., Black Bldg., 4, 7.

KANSAS

ARKANSAS CITY
Zugg, C. L., T.

ATCHISON
Bribach, E. J., Simpson Bldg., 3.
Pitts, E. P., 5.

EMPORIA
Neighbors, C. A., 621 Commercial St., T.
Trimble, C. S., 621 Commercial St., T.

FORT SCOTT
Jarrett, M. F., 5.
Hopper, W. L., T.

INDEPENDENCE
Smith, Chas. L., 115½ W. Myrtle St., 3, 4.

KANSAS CITY
Lidikay, C. J., 6th and Minnesota Ave., T.
May, James W., Portsmouth Bldg., 8, 9, 0, 1, 3.

NORTON
Cole, Charles W., T.

PARSONS
Markham, H. C., T.

PITTSBURG
Graves, W. H., Commerce Bldg., T.

TOPEKA
Alkire, H. L., 614 Kansas Ave., T.
Esterly, Daniel E., 813 Kansas Ave., T.
Williams, C. L., 1605 Topeka Ave., 3, 4, 5.

WICHITA
Dorsey, J. G., 105 W. Douglas Ave., 8, 0, 1, 6.
Gsell, J. F., Beacon Bldg., 0, 1, 6.
Palmer, Edward M., Beacon Bldg., 6.

KENTUCKY

FRANKFORT
Coleman, J. S., T.

HOPKINSVILLE
Brown, F. Manning, 9th and Main Sts., 6, 7, 8, 0, 3, 6.

JACKSON
Wickliffe, T. F., 7.

LEXINGTON
McMullen, John, U. S. P. H. S., 3, 7.
Stuckey, J. A., Fayette Nat'l Bank Bldg., T.
Smith, Orrin Le Roy, Security Trust Bldg., T.

LOUISVILLE
Hall, Gaylord C., 1300 S. 6th St., 6.
Pfingst, Adolph O., Atherton Bldg., 7, 9, 0, 4, 6, 7.
Ray, J. M., Atherton Bldg., 7, 8, 0, 2, 4, 6.
Wolfe, Claude T., Atherton Bldg., 7.

OWENSBORO
Griffith, D. M., 207 W. 4th St., 7, 0, 4.

PADUCAH
Reynolds, H. G., City Nat'l Bank Bldg., T.

PARIS
Stern, Milton J., 436 Main St., 6, 7.

LOUISIANA

NEW ORLEANS
Blum, Henry N., Maison Blanche Bldg., T.
Bruns, H. Dickson, 211 Camp St., 7, 9, 2.
Feingold, Marcus, 124 Baronne St., 8, 9, 0, 2, 4, 6.
Landfried, Charles J., 5907 Garfield St., T.
Smith, V. C., Maison Blanche Bldg., 3, 7.

SHREVEPORT
Dowling, Oscar, Commercial Nat'l Bank Bldg., T.

MAINE

AUGUSTA
Beach, S. J., 283 Water St., 7.
Turner, Oliver W., Water St., 6.

CALAIS
Gilbert, Walter J., 7.

LEWISTON
Wakefield, F. S., 342 Main St., T.

PORTLAND
Bowers, J. W., 732 Congress St., 6, 9, 4.
Gilbert, F. Y., 148 Park St., 6, 7, 9, 2, 3, 4, 6, 7.
Haskell, Alfred W., 100 Winter St., 4.

Holt, E. E., Sr., 723 Congress St., 6, 7, 8, 9, 2, 4, 7.
Holt, Eugene E., Jr., 723 Congress St., 2, 4, 7.
Little, A. H., 188a State St., 9, 2, 7.

ROCKLAND

Gribbin, H. E., 9 Claremont St., T.

WATERVILLE

Hill, J. F., 111 Main St., T.

MARYLAND

BALTIMORE

Bordley, James, Jr., Professional Bldg., 7, 9, 0, 2, 4, 6, 7.
Carroll, J. J., Charles and Read Sts., 6, 7, 8, 9, 0, 1, 2, 7.
Clapp, C. A., 513 N. Charles St., 9, 2, 6, 7.
Clarken, James V., 529 N. Charles St., 7.
Cohen, Lee, 1820 Eutaw Pl., T.
Crouch, J. Frank, 1125 N. Charles St., 4.
Downey, Jesse W., Jr., 529 N. Charles St., T.
Fleckenstein, H. K., 700 N. Howard St., 4.
Fleming, G. A., 1018 Madison Ave., 6, 7, 9, 2, 4, 6.
Friedenwald, H., 1029 Madison Ave., T.
Boldback, L. J., Brehm's Lane, Belair Road, 4, 7.
Harlan, Herbert, 516 Cathedral St., 6, 7.
McConachie, A. D., 805 N. Charles St., 7, 8, 9, 2, 4.
Peterman, H. E., 114 W. Franklin St., 7, 9, 4.
Randolph, Robert L., 609 Park Ave., 7.
Reik, H. O., 506 Cathedral St., T.
Schaefer, Otto, 1105 Madison Ave., 6, 7, 8, 9, 2, 4.
Tarum, Wm., 605 Park Ave., 0, 2, 4.
Theobald, Samuel, 970 N. Howard St., 7, 9, 2, 4, 7.
Weinberg, M. A., 1804 Madison Ave., T.
Woods, Hiram, 842 Park Ave., 7, 8, 9, 0, 1, 2, 3, 4, 6, 7.

CUMBERLAND

Jones, Emmett L., First Nat'l Bank Bldg., 7, 8, 9, 0, 1, 2, 3, 6, 7.
O'Neil, Francis P., 22 Baltimore Ave., T.

HAGERSTOWN

Maisch, Aug. C., 128 W. Washington St., 7, 9, 4.

MASSACHUSETTS

BOSTON

Bossidy, John C., 419 Boylston St., 9, 0, 2, 7.
Carvill, Maud, 101 Newbury St., T.
Cheney, Frederick E., 126 Commonwealth Ave., 4.
Crosby, Leander M., 419 Boylston St., 7.
Derby, Geo. S., 7 Hereford St., 6, 9, 2, 3, 4, 5, 6.
Greenwood, Allen, 101 Newbury St., 6, 7, 0, 3, 4.
Hall, William D., 416 Marlboro St., T.
Haskell, Henry H., 29 Commonwealth Ave., T.
Hurley, Edward D., 575 Broadway, 7.
Jack, E. E., 215 Beacon St., 4.
Lancaster, W. B., 522 Commonwealth Ave., 3, 4, 6, 7.
Liebman, Wm., 1069 Boylston St., T.
Loring, Robert G., 2 Arlington St., T.
Lowell, W. H., 101 Newbury St., T.
Mansfield, James A., 90 Brighton Ave., 6, 7.
Quackenboss, Alexander, 143 Newbury St., T.
Riemer, Hugo B. C., 20 Beacon St., T.
Rood, Luther C., 434 Washington St., 7.
Spalding, Fred M., 39 Huntington Ave., Back Bay, T.
Standish, Myles, 6 St. James Ave., 7.
Stevens, H. B., 419 S. Boylston St., T.
Tenney, J. A., 419 Boylston St., 6, 7, 9, 2, 7.
Thompson, Peter H., 308 Commonwealth Ave., T.
Tingley, Louisa P., 9 Massachusetts Ave., 6, 7, 2, 4, 6, 7.
Verhoeff, F. H., 233 Charles St., 6, 9, 2, 4, 7.
Walker, C. B., 697 Huntington Ave., 3, 4, 5.
Wells, D. W., The Westminster, 8, 9, 2, 4, 7.
Williams, Chas. H., 1069 Boylston St., 6, 7, 8, 9, 0, 2, 4, 6.
Williams, Edward R., 1069 Boylston St., 7.

BROCKTON

Murdock, Frederick W., 54 W. Elm St., 2, 4, 7.

CAMBRIDGE

Clarke, Inez Louise, 825 Massachusetts Ave., 5.
Kelléher, P. F., 1636 Massachusetts Ave., T.
McIntire, Herbert B., 4 Garden St., T.

FALL RIVER

Abbe, Alanson J., 375 Rock St., 7.
Jackson, Oliver H., 34 N. Main St., T.
Pritchard, William P., 273 N. Main St., 7.

FITCHBURG

Rodrick, Albert Fowler, 4 Pleasant St., 4, 6.

FRAMINGHAM

Jessaman, L. W., 60 Concord St., T

HOLYOKE

Hussey, Edward J., 276 High St., 9, 2, 4, 7.

LAWRENCE

Conlon, Frank A., Bay State Bldg., 4, 6, 7.
Merrill, W. H., Bay State Bldg., T.

LOWELL

French, C. E., Wyman's Exchange, 7.
Leahey, George A., 128 Merrimack St., T.
Livingston, C. B., 67 Burtt St., T.
Meigs, R. J., 226 Merrimack St., 7.

NEW BEDFORD

Weaver, Harry V., 161 William St., T.

NORTHAMPTON

Dow, Frank E., 16 Center St., 7.
Gardner, C. R., 78 Main St., 7.

NORTHFIELD

Newton, Aaron L., 4, 7.

PALMER

Moore, George A., 7.

SALEM

Hennessey, William W., 333 Essex St., 7.

SOUTH BOSTON

Hurley, Edward D., 575 Broadway, T.

SPRINGFIELD

Carleton, Ralph, 121 Forest Park Ave., 4.
Irwin, V. J., 351 Main St., 6, 7.
McKechnie, F. J., 317 Main St., 4.

WARE

Miner, Worthington W., 37 Main St., T.

WESTFIELD

Clark, Fredk. T., T.

WORCESTER

Cahill, J. W., 390 Main St., T.
Cross, Albert E., Slater Bldg., T.
Dolan, Wm. E., 340 Main St., 7.
Estabrook, Charles T., 857 Pleasant St., 7.
Harrower, David, 4.
Lovell, David B., State Mutual Bldg., 7.
O'Connor, D. F., State Mutual Bldg., 4, 7.

MICHIGAN

ANN ARBOR

Slocum, George, 311 S. State St., 6.

BAD AXE

Conboy, Daniel, 6.

BATTLE CREEK

Haughey, Wilfrid, Post Bldg., 0. 2.
Stegman, Louie Vandervoort, Battle Creek Sanitarium, 6, 7.

BAY CITY

Andrews, George E., Crapo Blk., 6.
Baker, Charles H., Crapo Blk., T.
Morse, H. Beach., The Ridetto, 6.

DETROIT

Beattie, Robert, David Whitney Bldg., 6, 7.
Begle, Howell L., David Whitney Bldg., 6.
Bernstein, Edward J., Kresge Bldg., 4.
Campbell, Don M., J. H. Smith Bldg., 2, 4, 6, 7.
Connor, Ray, Washington Arcade, 8, 9, 2, 3, 4, 6.
Fowler, William, 319 Broadway, 6.
Frothingham, G. E., 706 Woodward Ave., 4, 6.
Gillman, Robert W., 33 Peterboro St., T.
Goux, Raymond S., David Whitney Bldg., 6.
Grant, L. E., 56 Midbury Ave., 6.
Jenne, Byron H., Fine Arts Bldg., 6.
Kahn, W. W., Fine Arts Bldg., 6, 7.
Kuhn, Charles F., 46 Warren Ave. E., T.
Maire, Lewis E., Kresge Bldg., 6.
Martin, Elbert A., 232 Horton Ave., 6.
Odell, Anna, 38 Adams Ave. W., T.
Parker, W. R., David Whitney Bldg., 6, 7, 8, 9, 0, 2, 3, 4, 5, 6, 7.
Pasternacki, B. W., Brietmeyer Bldg., 6, 7.
Renaud, George L., Fine Arts Bldg., T.
Robb, J. M., David Whitney Bldg., 6.
Rothschild, Douglas, Gas Bldg., 6.
Sanderson, Herman H., David Whitney Bldg., 6.
Smith, Eugene, 34 Adams Ave. W., 4, 6.
Thuner, Alois, Shurly Bldg., 6.
Waldeck, George M., David Whitney Bldg., 6.
Waltz, F. D. B., 723 Dix Ave., 6.
Watson, Edward C., David Whitney Bldg., 6.
Wilson, Harold, David Whitney Bldg., 7.

FLINT

Riker, E. V., Armory Bldg., 6.

GRAND RAPIDS

Huizinga, J. G., Widdicomb Bldg., T.
Paterson, Everard W., 45 Monroe Ave., N. W., 6.
Roller, Louis A., Gilbert Bldg., 6.
Tolley, E. W., 16-18 Monroe Ave., T.
Welsh, D. E., Powers Blk., 6.

GREENVILLE

Johnson, Fred A., 113 E. Grove St., 6.

IRONWOOD

Urquhart, John H., 3.

JACKSON

Bulson, A. E., Sun Bldg., 6.
Spicer, Walter E., Dwight Blk., T.

KALAMAZOO

Grant, Frederick E., 603 Kalamazoo Nat'l Bank Bldg., 6.
Wilbur, Edward P., Kalamazoo Nat'l Bank Bldg., 6.

LANSING

Burdick, A. F., Jenison Blk., 3.

LAWTON

Young, William R., 6.

MANISTEE

Lewis, Lee A., 6.

MARQUETTE

Hornbogen, H. J., 3.

MENOMINEE

Elwood, C. R., Spiers Bldg., T.

OWOSSO

Porter, Charles B., 311 Ball St., 6.

PORT HURON

Stockwell, C. B., 6.
Vroman, Mason E., Meisel Blk., 6.

SAGINAW

McKinney, A. R., Bearinger Bldg., 6, 7.
Rogers, A. S., 105 S. Jefferson Ave., T.
Watson, R. S., Graebner Bldg., 6.

STURGIS

Robinson, Fred W., 110 Pleasant Ave., T.

TECUMSEH

Conklin, H. R., 8, 9, 1, 6.

MINNESOTA

ALEXANDRIA

Boyd, L. M., 3.

BRAINERD

Nordin, C. G., T.

CENTER CITY

Gunz, A. N., 3.

DULUTH

Collins, Homer, New Jersey Bldg., 6, 7, 8, 1, 2, 3.
Conkey, C. DeW., Fidelity Bldg., 8, 3.
Gillespie, N. H., Fidelity Bldg., 3.
Lum, Clarence E., 1103 1st St., 2, 3.
Robinson, J. M., Providence Bldg., 8, 3.
Tilderquist, D. L., 7 E. Superior St., T.
Winter, J. A., Fidelity Bldg., T.

FARIBAULT

Lane, L. Arlene, 128 Central Ave., 4, 6, 7.

MANKATO

James, J. H., T.
Miller, V. I., 212 Liberty St., 1, 3.

MINNEAPOLIS

Aune, Martin, 2543 Emerson Ave., 3.
Benson, Geo. E., 3316 Irving Ave. S., 0, 3.
Bishop, C. Wesley, Pillsbury Bldg., T.
Brown, Edward J., 3027 Pleasant Ave., T.
Clark, Howard S., Syndicate Bldg., 6, 7.
Ericson, J. G., Syndicate Blk., 3.
Kerrick, S. E., Syndicate Bldg., 3.
Koller, L. R., Masonic Temple, 3.
Leavitt, H. H., 2015 James Ave., 3, 6.
Lewis, Jos. D., 312 Reid Corner, 4, 7.
Macnie, J. S., 2113 S. Bryant Ave., 3.
Morse, John H., Syndicate Bldg., 3.
Morton, H. McI., Metropolitan Bank Bldg., 3.
Murray, W. R., Physicians and Surgeons Bldg., T.
Newhart, Horace, Donaldson Bldg., T.
Nissen, H., Masonic Temple, 3.
Oberg, C. M., Syndicate Bldg., 3.
Pineo, W. B., Pillsbury Bldg., 3.
Pratt, F. J., 328 E. Hennepin Ave., T.
Schefcik, J. F., Masonic Temple, 3.
Simpson, J. D., Physicians and Surgeons Bldg., 7, 8, 0, 3, 6.
Spratt, C. N., Physicians and Surgeons, Bldg., 6, 8, 3.
Strout, Eugene S., 2838 James Ave. S., 8, 3, 5, 6, 7.
Thomas, G. H., Pillsbury Bldg., 3.
Todd, Frank C., Donaldson Bldg., 6, 7, 8, 9, 0, 2, 3, 4, 6, 7.
Wilcox, M. R., Donaldson Bldg., 3.
Wood, Douglas F., Donaldson Bldg., 3.
Wright, C. D., Met. Life Bldg., 3.

NEW ULM

Reineke, G. F., 3.

RED LAKE FALLS

Wilkinson, J. C., 3.

REDWOOD FALLS

Brand, W. A., 3.

ROCHESTER

Fisher, Carl, Y. M. C. A. Bldg., 4, 7.

ST. PAUL

Barsness, N. O. N., Lowry Bldg., 3.
Beaudoux, H. A., Lowry Bldg., T.
Boeckmann, Eduard, Lowry Bldg., T.
Bray, E. R., 237 Arundel St., 3.
Brown, John C., 2080 Commonwealth Ave., 3.
Burch, Frank E., 754 Linwood St., 8, 3, 6, 7.
Chamberlin, J. W., Lowry Bldg., 8, 0, 1, 2, 3, 4, 7.
Harding, J. C., American National Bank Bldg., 3.
Larsen, C. L., Lowry Bldg., 3.
Lewis, W. W., Lowry Bldg., 3.

McDavitt, Thos., Lowry Bldg., 6, 7, 8, 9, 0, 1, 7.
Maloney, T. J., Lowry Bldg., 3.
Nelson, L. A., Lowry Bldg., 3.
Van Slyke, C. A., Lowry Bldg., 8, 3.

SAUK CENTER

Lewis, E. J., 6, 8, 0, 2, 3, 6.

WILLMAR

Canfield, H. E., 3.

WINONA

Leicht, Oswald, Slade Blk., 3.
Lynch, J. L., 78 E. Third St., 6.

MISSOURI

CAPE GIRARDEAU

Yount, W. E., 0, 2, 5, 6.

CARTHAGE

Powers, Everett, Macon St., 8, 0, 2, 3, 7.

COLUMBIA

Noyes, Guy L., Univ. of Mo., 8, 0, 3.
Sneed, C. M., Guitar Bldg., 6, 7.

FARMINGTON

Smith, Owen A., Robinson Bldg., T.

JEFFERSON CITY

Summers, J. S., Central Trust Bldg., 0, 5.

KANSAS CITY

Beil, J. W., Argyle Bldg., 8, 0, 3, 6, 7.
Bellows, G. E., Rialto Bldg., T.
Blakesley, T. S., Lathrop Bldg., 8, 0, 3, 6.
Bourbon, Oliver P., 713 Lathrop Bldg., 6.
Curdy, R. J., Rialto Bldg., T.
Gosney, Chas. W., Lathrop St., 0, 1, 3.
Lichtenberg, J. S., Rialto Bldg., 2, 3, 6.
McAlester, A. W., Jr., Bryant Bldg., 8, 0, 2, 3, 5, 6.
Mott, J. S., Argyle Bldg., 3, 6.
Reyling, F. T., Argyle Bldg., T.
Schutz, W. H., Bryant Bldg., 7, 8, 0, 3.
Weaver, J. S., Rialto Bldg., T.

KIRKWOOD

Higbee, E. H., Rt. 12, 6.

NEVADA

McLemore, T., 6.

ST. JOSEPH

Leonard, P. I., 710½ Felix St., 9, 0, 3.

ST. LOUIS

Alt, Adolf, Metropolitan Bldg., 0, 1, 2, 3.
Charles, J. W., Humboldt Bldg., 8, 0, 3, 6.
Collasowitz, A., 3109 S. Grand Ave., 3.
Ewing, A. E., 5956 W. Cabanne Pl., 0, 4, 5, 6.
Green, John, Jr., Metropolitan Bldg., 6, 7, 8, 9, 0, 4, 6.
Gross, Julius H., Carleton Bldg., T, 6.
Hardy, Wm. F., Metropolitan Bldg., 6.
Henderson, F. L., Humboldt Bldg., T.
Hooss, Albert, 3643 Connecticut St., T.
Jennings, J. Ellis, Carlton Bldg., 8, 0, 4, 6.
Luedde, W. H., Metropolitan Bldg., T.
Napier, Amalie M., 5158 Ohio Ave., 6.
North, Emmett P., 3511 Washington Ave., 6.
Parker, Fredk. P., Times Bldg., T.
Post, M. H., Metropolitan Bldg., T.
Shahan, Wm. E., Metropolitan Bldg., 0, 3, 6, 7.
Skrainka, Philip, Metropolitan Bldg., 6.
Wiener, M., Carleton Bldg., 6, 7, 8, 9, 0, 2, 3, 5, 6, 7.
Woodruff, F. E., Metropolitan Bldg., T.

SEDALIA

Titsworth, Guy, 111 W. 4th St., 6, 7.

SPRINGFIELD

Love, Joseph W., Woodruff Bldg., T.

MONTANA

BUTTE

Donovan, John A., Phoenix Bldg., 7, 8, 9, 0, 1, 2, 3, 4, 5, 6, 7.

HELENA

Copenhaver, W. M., 5.

NEBRASKA

AUBURN

Dillon, I. H., 3, 6.

GRAND ISLAND

Sutherland, J. L., 109½ W. Third St., 6.

LINCOLN

Cook, S. E., Richards Blk., T.
Cowgill, Warwick M., Press Bldg., 4
Dayton, W. L., Funke Bldg., 7.
Hompes, J. J., Gauter Bldg., 6.
Williams, J. P., Funke Bldg., T.
Zemer, Stanley G., 2145 B St., 7.

OMAHA

Bushman, L. B., City National Bank Bldg., 3.
Gifford, Harold, 420 S. 36th St., 8, 9, 0, 5, 6.
Knode, A. R., Omaha Nat'l Bank Bldg., T.
Lemere, H. R., Brandeis Bldg., 8, 0, 2, 3, 5, 6.
Mick, W. H., Brandeis Bldg., 3.
Owen, F. S., Brandeis Bldg., T.
Patton, J. M., 916 Mercer Park Road, 3.
Rubendall, Clarence, Brandeis Bldg., T.
Wherry, W. P., 2444 Manderson St., 4, 7.

NEVADA

RENO

Brown, H. Alexander, 5, 7.
Robinson, J. LaRue, 4.

NEW HAMPSHIRE

CONCORD

Cook, George, 16 Center St., 2, 3.

MANCHESTER

Carvelle, H. DeW., T.
Fritz, E., 913 Elm St., T.
Varick, W. R., 1015 Chestnut St., T.

NASHUA

Nutter, C. F., 16 Amherst St., 6, 9, 2, 4.

PORTSMOUTH

Souter, W. N., 33 Market St., 2, 4.

NEW JERSEY

ASBURY PARK

Hill, John Augustus, 201 8th Ave., 2, 3, 7.
Upham, Helen F., 305 Third Ave., 7.

ATLANTIC CITY

Frisch, Fred, Galbraith Apts., 2, 4, 6, 7.
Gould, Geo. M., 215 Atlantic Ave., 2, 4
Mevay, James C. F., 707 Pacific Ave., T. 2.
Marvel, Philip, 1616 Pacific Ave., T.
Pollard, W. M., 25 S. South Carolina Ave., 7, 9, 2, 4.

BRIDGETON

Moore, J. H., 106 E. Commerce St., 7, 2, 4.

EAST ORANGE

Buvinger, Charles W., 50 Washington St., 4, 7.

ELIZABETH

Schlichter, C. H., 1024 E. Jersey St., 6, 9, 2, 4, 7.
Wilson, Norton L., 410 Westminster Ave., T.

GLASSBORO

Heritage, Charles S., 4.

GLEN RIDGE

Tully, Marcus E., 37 Forest Ave., 7.

HOBOKEN

Paganelli, T. Richard, 836 Garden St., 4, 7.
Sullivan, George F., 512 Hudson St., 7.

JERSEY CITY

Bull, Edward L., 2 Madison Ave., T.
Chambers, T. R., Exchange Pl., 6, 7, 9, 2, 7.
Pyle, Wallace, 612 Bergen Ave., 4, 7.

MORRISTOWN

Mial, Leonidas L., 2, 7.
Vaughan, Harry, 66 South St., 4.

NEWARK

Chattin, J. Franklin, 2 Lombardy St., 7.
Curtis, Elbert A., 334 High St., 7.
Dias, J. Lawrence, Broad St., 4, 7,
Quinby, William O'G., 14 James St., 4, 7.
Sherman, Elbert S., 310 Mt. Prospect Ave., 6, 7, 9, 2, 4, 7.
Sutphen, T. Y., 992 Broad St., 7.
Weiss, Louis, 544 Springfield Ave., 7.
Zehnder, A. Charles, 180 Fairmount Ave., 2, 7.

NEW BRUNSWICK

Howley, Bartholomew M., 421 George St., 7.
Sullivan, Charles J., 57 Paterson St., 7.

ORANGE

Emerson, Linn, Metropolitan Bldg., 7, 9, 0, 2, 4, 5, 6, 7.

PASSAIC

Chase, Wm. E., 137 Gregory Ave., 4.

PATERSON

Johnson, W. B., 170 Broadway, T.
Marsh, Elias J., 24 Church St., 7.

PLAINFIELD

Ard, F. C., 604 Park Ave., T.
Hubbard, H. V., 420 Central Ave., 7.
Van Horn, A. F., 514 Central Ave,. T.

RAHWAY

Orton, George L., 162 Irving St., 2, 4.

SALEM

Ewen, Warren L., 4, 7.

TOMS RIVER

Jones, R. R., 201 Washington St., 7, 9, 4, 7.

TRENTON

Adams, C. F., 52 W. State St., 6, 7, 8, 9, 2, 4, 6, 7.
Craythorn, C. J., 302 W. State St., 2, 4.
Schoening, G. A., 148 N. Clinton St., 4.
Yazujian, Dikran M., 230 N. Warren St., 7.

WESTFIELD

Laird, George S., 125 Central Ave., 7.

NEW YORK

ALBANY

Bedell, A. J., 354 State St., 8, 9, 0, 1, 2, 3, 7.

AMSTERDAM

Knapp, Raymond J., 23 Division St., 7.
Lyons, M. C., 21 Market St., 7.

BINGHAMTON

Branch, Frederick D., 86 Front St., 7.
Watson, H. D., 151 Front St., 7, 8, 2, 7.

BROOKLYN

Andrew, James H., 500 Madison St., 7.
Bailey, Fred D., 260 Hancock St., 7.
Bailey, John H., 694 Greene Ave., 7.
Deely, George E., 132 Montague St., 9, 4.
Hancock, J. C., 135 Cambridge Pl., T.
Ingalls, James W., 874 Lafayette Ave., 6, 7, 9, 2, 4.
Levitt, Marcus J., 258 Vernon Ave., 7.
Lombardo, M., 186 Meserole St., 7.
Ohly, John H., 22 Schermerhorn St., 7, 9, 2, 7.
Pond, Erasmus A., 1087 Dean St., 7.
Price, H. R., 435 Clinton Ave., 9, 4.
Reynolds, Willard G., 1165 Dean St., 7.
Smith, Henry M., 184 Jarolemon St., 7.

BUFFALO

Andrews, Herman D., 23 Allen St., 7.
Bennett, A. G., 26 Allen St., 6, 7, 0, 2, 6, 7.
Blaauw, Edmond E., 327 Franklin St., 6, 7.
Cowper, H. W., 543 Franklin St., T.
Francis, L. M., 636 Delaware Ave., 9, 0, 2, 3, 4, 6, 7.
Glosser, Herbert H., 448 Franklin St., 7.
Grove, B. H., 334 Pearl St., 5, 6, 7, 8, 9, 2, 7.
Hitzel, G. A., 49 E. Parade Ave., 6.
Howe, Lucien, 522 Delaware Ave., 6, 7, 8, 9, 0, 2, 3, 6, 7.
Lewis, F. Park, 545 Franklin St., 7, 8, 9, 0, 1, 2, 3, 6, 7.
Love, Frank W., 470 Linwood Ave., 9, 0, 2, 4, 6, 7.
March, Clara A., 465 Ashland Ave., 0, 1, 2, 3, 4, 5, 6, 7.
Phillips, William L., 759 Richmond Ave., 6.
Satterlee, Richard H., 187 Delaware St., 4.
Sernoffsky, I., 37 Allen St., 7.
Starr, Elmer G., 523 Delaware St., 4, 6, 7.
Weed, Harry M., 405 Franklin St., 0, 2, 3, 6, 7.

CORTLAND

Higgins, R. P., 20 Court St., 4, 7.

ELMIRA

Case, George M., 154 Main St., 9, 2, 4.

FLUSHING

Wiggers, A. F. A., 176 Central Ave., T.

GLENS FALLS

Dean, John Wyman, 6 Pine St., 2, 7.
Hunt, W. J., 21 Notre Dame, T.

HORNELL

Barney, B. A., 5 Center St., 6, 7, 9, 2, 4, 7.

ITHACA

Kirkendall, J. S., 315 N. Aurora St., 7, 9.
Wilson, Roscoe C., 208 E. State St., 2, 4.

KINGSTON

Gates, Aden C., 574 Broadway, T.

MAMARONECK

Steese, Edwin S., 7.

MT. VERNON

Coleman, Henry H., 15 Park Ave., 7.
Gould, George C., 267 S. Columbus Ave., 7.

NEW ROCHELLE

Beck, August L., 44 Centre Ave., 7.
Foster, Matthias Lanckton, 211 Centre Ave., 2, 4, 7.

NEW YORK CITY

Alger, Ellis M., 40 E. 41st St., 2, 7.
Bailey, John H., 694 Greene Ave., 7.
Bates, W. H., 40 E. 41st St., 2, 4, 7.
Beach, Bennett S., 144 W. 85th St., T.
Bell, George Huston, 40 E. 41st St., 4, 7.
Bridgman, T. Francis, 661 W. 180th St., 4.
Broughton, Wm. R., 348 Madison Ave., 6, 7, 9, 4, 7.
Callan, Lewis W., 461 W. 141st St., 7.
Callan, Peter A., 452 Fifth Ave., 6, 7, 9, 2, 4, 7.
Cohen, Martin, 1 W. 85th St., 2, 7.
Conner, Earl, 284 Lexington Ave., 2, 7.
Crigler, Lewis W., 40 E. 41st St., 7.
Curtin, Thomas H., 319 E. 149th St., 4, 5, 7.
Cutler, C. W., 24 E. 48th St., 7.
Davis, A. E., 50 W. 37th St., 6, 7, 8, 9, 0, 2, 3, 4, 7.
Decker, John J., 1939 Washington Ave., 7.
Diem, Oscar, 123 E. 60th St., 7.
Drake, Charles A., 17 E. 38th St., 7.
Duane, Alexander, 139 E. 37th St., 6, 7, 9, 2, 4, 7.
Friedenberg, Percy, 38 W. 59th St., 7, 9, 0, 2, 7.

Goodfriend, Nathan, 243 E. 72d St., 7.
Graef, Charles, 1125 Boston Road, 7.
Gulliver, F. D., 105 W. 72d St., 7.
Hallock, Silas F., 36 East 65th St., 7.
Hartshorne, Isaac, 105 W. 40th St., 7.
Heller, I. M., 450 E. Tremont Ave., T.
Holden, Ward A., 8 E. 54th St., 8, 9, 4, 7.
Hopkins, W. E., 515 Park Ave., 7.
Howley, B. M., 355 W. 28th St., T.
Irwin, Frank Newton, 527 Fifth Ave., 2, 7.
Jacobs, Simon M., 1187 Boston Road, 7.
Kalish, Richard, 36 W. 47th St., 7, 9, 2, 4, 7.
Kearney, James A., 127 W. 58th St., 7.
Keen, W. M., 85 Valick St., 7.
Key, Ben Witt, 7 W. 49th St., 7.
Knapp, Arnold, 10 E. 54th St., 6, 8, 9, 0, 2, 6, 7.
Koller, Carl, 681 Madison Ave., 7.
Krug, E. F., 12 W. 44th St., 7.
Lambert, W. E., 112 E. 35th St., 6, 7, 9, 1, 2, 4, 7.
Leshure, John, 423 Convent Ave., T.
Lesser, Henry R., 351 W. 114th St., 7.
Lester, John C., 432 Madison Ave., 7.
May, Chas. H., 698 Madison Ave., 6, 9, 4, 6, 7.
Medd, John C., 1 Madison Ave., 7.
Mittendorf, Alfred D., 399 Park Ave., 7.
Mittendorf, Wm. F., 399 Park Ave., 1, 2, 7.
Mulcahy, T. A., 143 W. 92d St., 7.
Oberdorfer, Archie L., 402 W. 145th St., 7.
Payne, S. M., 542 Fifth Ave., 6, 9, 2, 4.
Pooley, Thomas R., 40 E. 41st St., 7.
Power, James F., 137 W. 73d St., T.
Power, William T., 152 Greenwich St., 7.
Reese, Robert G., 50 W. 52d St., 4, 7.
Riedel, Alfred H., 220 W. 49th St., 7.
Rose, Malcolm Cameron, 130 Post Ave., 2, 4, 6, 7.
Samuels, Bernard, 50 W. 52d St., 7.
Schoenberg, M. J., 103 E. 81st St., 2, 4, 6, 7.
Shannon, John R., 17 E. 38th St., 9, 2, 6, 7.
Steese, Edwin S., 46 E. 57th St., T.
Stevens, Charles W., 40 E. 41st St., 7.
Stevens, George T., 350 W. 88th St., 7.
Strouse, Alfred N., 132 W. 58th St., 2, 7.
Taylor, T. M., 2 W. 45th St., 0, 2, 3, 4, 7.
Tenner, Arthur S., 120 W. 86th St., 4, 7.
Thomson, Edgar S., 405 Park Ave., 6, 7, 8, 0, 1, 2, 3, 7.
Torok, Erwin, 8 E. 54th St., 4.
Tyson, H. H., 11 E. 48th St., 5, 7, 9, 2, 4, 7.
Valk, Francis, 164 E. 61st St., 6, 7, 9, 2, 4, 7.
Virden, John E., 1443 Ferris Pl., Westchester St., 2, 4, 7.
Wandless, Henry W., 9 E. 39th St., 7.
Webster, David, 24 E. 48th St., 7.
Webster, David H., 24 E. 48th St., 7.
Weeks, J. E., 46 E. 57th St., 5, 6, 7, 0, 1, 4, 6, 7.
Weeks, Webb W., 46 E. 57th St., 7.
Weidler, Walter Baer, 131 E. 60th St., 2, 3, 4, 7.
Wheeler, John M., 80 Morningside Drive, 5, 7.
Wiener, Alfred, 616 Madison Ave., 9, 2, 7.
Ziporkes, Joseph, 135 W. 141st St., 7.

NIAGARA FALLS

Price, Norman W., 445 Third St., 7.
Teeter, L. H., 27 Gluck Bldg., 4.

NICHOLS

Cady, Geo. M., 4, 6, 7.

NORWICH

Smith, Homer E., 7, 2, 4, 7.

NYACK

Toms, S. W. S., 6, 9, 1, 3, 4, 7.

ONEIDA

Crockett, R. L., Devereux Blk., 6, 7, 8, 9, 7.

OSWEGO

Wallace, H. M., 9 W. Bridge St., T.

PLATTSBURG

Rogers, T. Avery, 42 Court St., 9, 7.

PORT WASHINGTON

Mortimer, W. Golden, T.

POUGHKEEPSIE

Cadwell, C. T., 285 Main St., 6, 8, 9, 3, 4, 7.
Dobson, William G., 278 Mill St., 7.

ROCHESTER

Barber, Frank, 31 S. Union St., 7.
Bissell, Elmer J., 75 S. Fitzhugh St., 6, 7.
Clark, Lloyd H., 337 Monroe Ave., 7.
Kennedy, E. W., 127 Genesee St., 7.
Ozmun, I. Davis, 137 East Ave., T.

ROME

Groff, J. E. 219 N. Washington St., 6, 7.

SCHENECTADY

Lord, Morris S., 24 State St., 9, 7.
O'Brien, John J., 11 Lafayette St., 9, 2, 7.

SOUTHAMPTON, L. I.

Allen, S. Busby, 7.

SYRACUSE

Lewis, G. Griffin, University Blk., 4, 7.
Marlow, F. W., 200 Highland St., 4, 7.

TROY

Smith, F. A., 3 Clinton Pl., T.
Sulzman, Frank M., 1831 5th Ave., 7.

UTICA

Beattie, William H., 252 Genesee St., 7.
Crumb, Charles W., 190 Genesee St., 4.
Hall, William P., 196 Genesee St., 4.

WATERTOWN

Atkinson, Walter S., 168 Sterling St., 7.
Cannon, Gilbert, Flower Bldg., 7.
Jones, Edward W., Charlebois Bldg., T.

NORTH CAROLINA

ASHEVILLE

Briggs, H. H., 73 Haywood St., 6, 7, 8, 9, 0, 4, 7.
Buckner, R. G., Medical Bldg., 4.
Russell, E. R., T.

GREENSBORO

Banner, C. W., Banner Bldg., 7, 8, 0, 2, 4.

LINCOLNTON

Sloan, Henry L., 150 E. 37th St., 7.

SALISBURY

Brawley, R. V., 126½ N. Main St., 7, 9, 4, 7.

SPRING HOPE

Brantley, H., 2, 4.

WILMINGTON

Murphy, J. G., 7.

WILSON

Saliba, N. M., T.

WINSTON-SALEM

Davis, Thos. W., 7, 2, 4.

NORTH DAKOTA

FARGO

Rindlaub, J. H., de Lendrecie Blk., T.

GARRISON

Stucke, E. C., 3.

GRAND FORKS

Marsden, C. S., 28 S. 3d St., 9, 3.

JAMESTOWN

Golseth, G., 7.

NEW ROCKFORD

MacLachlan, Charles, 7.

OHIO

AKRON

Grant, J. G., Everett Blk., 6, 8. 3.
Grubb, Edward W., 678 Hazel St., 6.
Seidel, U. D., 2d Nat'l Bk. Bldg., T.
Townsend, C. E., 308 Ohio Bldg., 6.
Weaver, Elizabeth M., 165 E. Market St., 6.

ALLIANCE

King, G. L., Box 983, 8, 9, 4.

ASHTABULA

Battels, Mary M., 126 Main St., 6.

BELLEFONTAINE

Harbert, John P., Canby Bldg., 7.
Stinchcomb, W. G., 129 E. Columbus St., 6.

BEREA

Knowlton, L. G., 24 Seminary St., 2, 3, 6.

CANTON

Crane, C. A., Electric Bldg., 6, 7.
Schild, Edw. H., The Koons Bldg., 4, 6.

CINCINNATI

Ayres, S. C., 3760 Clifton Ave., 7.
Castle, Chas. H., The Poinciana, 4.
Holmes, C. R., 10 E. 8th St., T.
Keller, Wm. S., Groton Bldg., 5.
Murphy, J. W., 1900 Humboldt Ave., E. Walnut Hills, T.
Rangly, John, 936 Clark, 6, 8, 0, 2, 5, 6, 7.
Sattler, Robt., The Groton, 3.
Vail, Derrick T., 24 E. 8th St., 6.
Wyler, Jesse S., Groton Bldg., 0, 3, 4, 6.

CLEVELAND

Brelsford, H. H., 636 Rose Bldg., 6.
Bruner, William E., Guardian Bldg., 6.
Chamberlain, W. P., 7205 Clinton Ave., 6.
Cogan, Jas. E., Rose Bldg., 2, 4.
Hartzell, H. J., 9402 Madison Ave., N. W., 6.
Kochmit, M. G., 4918 Broadway, 6.
Lauder, Edw., 1021 Prospect Ave., T.
Monson, S. H., Anisfield Bldg., 4.
Nelson, Chas., F., Schofield Bldg., 6, 7.
Shackleton, William E., Osborn Bldg., 6, 7.
Sherman, H. G., Rose Bldg., 6, 7, 2, 4, 6, 7.
Stotter, James, 2050 E. 88th St., 6.
Stuart, Charles C., Guardian Bldg., 6.
Tuckermann, W. C., 1021 Prospect St., 6.

CLYDE

Hunter, F. J., 6.

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Brown, John E., 239 E. Town St., T.
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Timberman, Andrew, 112 E. Broad St., 6, 7, 8, 2, 7.
Wright, John W., Central Nat'l Bank Bldg., 0, 4, 7.

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Harris, Harry B., Reibold Bldg., T.
Millette, John W., 58 Cambridge Ave., 9, 0, 2, 4, 6.

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Gill, George, 146 Middle Ave., 2, 7.

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Van Note, William B., 6.

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Bliss, Chester B., 415 Columbus Ave., 6.

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Hogue, D. W., Fairbanks Bldg., T.

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Mossgrove, James Ross, 426 Market St., T.

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Alter, Francis W., Colton Bldg., T.
Jacobi, Frank, Colton Bldg., T.
Landman, Otto, 230 Michigan St., T.
Leatherman, B. E., Cotton Bldg., 6.
Lukens, Charles, 218 Michigan St., 6.
Patterson, Paul M., 237 Michigan St., 6.
Snyder, W. H., 211 Ontario St., 7, 9, 0, 2, 6.

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Gibson, R. D., Dollar Bank Bldg., 6, 8, 9, 0, 2, 4.
Washburn, John L., Dollar Bank Bldg., 6.

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McCool, Joseph L., Stevens Bldg., 5.
Wright, S. E., Corbett Bldg., 8, 0, 3.

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Shope, S. Z., 610 N. 3d St., 7, 8, 9, 1, 2, 7.

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Baer, B. F., Jr., 2040 Chestnut St., T.
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Bush, Chas. B., 1421 Walnut St., 4.
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Chance, Burton, 235 S. 13th St., 7, 2, 4, 6, 7.
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CENTRALIA

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Haley, P. A., 108½ Capitol St., T.

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