

ARCHIVOS
DE LA
SOCIEDAD AMERICANA
DE
OFTALMOLOGIA Y OPTOMETRIA

VOLUMEN DEDICADO AL PROFESOR
IGNACIO BARRAQUER
CON MOTIVO DE SU LXXV^o. ANIVERSARIO

SECRETARIOS DE REDACCION: ENRIQUE ARIZA H., M.D., SALOMON REINOSO A., M.D.
APARTADO NACIONAL 700 CHAPINERO — BOGOTA - COLOMBIA

*ARCHIVOS DE LA SOCIEDAD AMERICANA
DE
OFTALMOLOGIA Y OPTOMETRIA*

ARCHIVOS
DE LA
SOCIEDAD AMERICANA
DE
OFTALMOLOGIA Y OPTOMETRIA

Vol. II

1959

Nos. 1-2-3-4

SECRETARIOS DE REDACCION:

ENRIQUE ARIZA H., M. D.
SALOMON REINOSO A., M. D.

APARTADO NACIONAL 700 CHAP.
BOGOTA - COLOMBIA

*HOMENAJE AL PROFESOR IGNACIO BARRAQUER
EN SU LXXV ANIVERSARIO*

*TODOS LOS TRABAJOS DE ESTE VOLUMEN HAN SIDO
EXPRESAMENTE ESCRITOS A TAL FIN*



*Al Profesor IGNACIO BARRAQUER,
quien tanto ha hecho por la visión hu-
mana, dedicamos este volumen haciendo
votos para que por largos años pueda con-
tinuar su fructífera labor.*

*“Pour le Jubilé d’Ignacio Barraquer, en
cordial hommage”.*

P. B.

Je pense souvent, et tous les oculistes du monde entier, j’en suis convaincu, font comme moi, à I. Barraquer; on peut dire qu’il a transformé l’opération la plus courante et la plus utile de l’ophtalmologie. Ses contemporains se rappellent la fréquence à l’époque, de la cataracte secondaire et leurs si nombreux déboires avec l’emploi des procédés anciens. Sans doute, Smith avait vers 1910 essayé de vulgariser l’extraction totale par le procédé qui porte son nom; quelques opérateurs, à l’école de E. Kalt remplaçant la vieille kystitomie par l’arrachement d’un lambeau de cristalloïde, enlevaient, le plus souvent sans le vouloir, la lentille tout entière, et déjà systématiquement quelques audacieux s’efforçaient d’enlever à la pince le cristallin dans sa capsule; déjà au début de ce siècle, étudiant le traitement chirurgical de la myopie, je parlais sans enthousiasme il est vrai, de ce procédé radical, de ses avantages, mais aussi de ses dangers. Bien peu d’entre nous avaient abandonné la Kystitomie, suivie des pressions et contre pressions classiques. Il faut savoir se retourner quelquefois vers le passé, regarder vers les débuts d’une audacieuse méthode qui devait bientôt inspirer à tous le goût de l’extraction totale. Ceux d’entre nous qui en 1920 puis en 1921, assistèrent au Congrès de la Société française d’ophtalmologie se rappellent l’émotion qu’ils éprouvèrent alors. José Barraquer nous présenta la phakoérésis que son fils Ignacio employait depuis 1917 “La technique est des plus simples, nous disait il” Suivit la projection d’un film, fort beau pour l’époque, qui fit passer dans l’assistance un peu d’enthousiasme et beaucoup de frissons. En quittant la salle, on se regardait, on disait son admiration pour l’adresse opératoire, mais beaucoup hochait la tête et ne songeaient guère à utiliser eux mêmes la méthode. Que devenaient nos chers procédés que nous avaient appris nos maîtres et nos habituelles discussions académiques sur l’emploi de l’iridectomie? Mais beaucoup qui firent le pèlerinage de Barcelone et virent Barraquer opérer revinrent convaincus.

Quand une méthode est bonne, elle fait son chemin, avec lenteur souvent parce qu’elle rencontre l’opposition venue de ceux qui préfèrent la douce routine au progrès. L’extraction totale dont I. Barraquer est, sans conteste, le grand promoteur s’est généralisée. Si tous ceux qui la jugent excellente n’utilisent pas l’irisiphague, quel que soit le procédé, c’est Barraquer qu’il en faut d’abord remercier. Voici qu’avec la troisième génération, un procédé nouveau nous vient pour vaincre très simplement la résistance zonulaire. Le nom des Barraquer, celui d’Ignacio surtout n’est pas près d’être oublié. Nous adressons à notre collègue un hommage admiratif et reconnaissant.

PIERRE BAILLIART

E' LA CREAZIONE DI UN IPERTONO DEL BULBO UN COADIUVANTE ALLA GUARIGIONE CHIRURGICA DEL DISTACCO DI RETINA?

(Breve sguardo panoramico e critico sui mezzi per provocarlo)

PER

ALFONSO APOLLONIO, M. D.

Varese, Italia

Se andiamo a rileggere, sui vecchi testi di oftalmologia, ad esempio sul classico Fuchs, il capitolo del distacco di retina, quando le rotture retiniche erano considerate un fenomeno secondario del distacco, troviamo indicato, quale terapia d'importanza, il bendaggio compressivo del bulbo. Era infatti convinimento che esso, aumentando la tensione bulbare, favorisse il riassorbimento del liquido subretinico e quindi il riaccollamento della retina. Dopo Gonin, trovata la vera causa del distacco, ossia la rottura retinica, tutti gli operatori si sono indirizzati alla ricerca di sistemi, sempre migliori, per ottenere la chiusura delle rotture lo svuotamento del liquido subretinico e nessuno si curò del tono bulbare. Solo sporadicamente, qualche autore, fa accenno alla tensione oculare come indizio pronostico sgradevole in casi di forte ipotonia. Si deve a Custodis, nel 1951, il primo richiamo al controllo sistematico della tensione bulbare nei distacchi di retina e ultimamente si giunge a Hensius che così si esprime: "A me sembra indispensabile prendere nota del tono oculare durante e dopo ogni operazione di distacco di retina, giacchè nei casi in cui il tono è normale, le probabilità di successo sono buone, mentre nei casi di ipotensione od addirittura di bulbo molle, esse sono molto scarse".

Anche Nicolato proiettando un suo film a colori sulla resezione sclerale in un congressino della S. Oft. Lomb. ebbe a dire: "Quando mi accingo ad operare un distacco di retina oso sperare nella guarigione solo se in precedenza ho potuto ottenere una buona midriasi con l'atropina, in caso contrario l'insuccesso è di regola". E' logico che egli si riferiva al fatto che nella forte ipotensione bulbare è ben difficile allargare la pupilla con i midriatici. Nel congresso oftalmologico svizzero del 1956, Dufour e Bianchi sul tema "Distacco di retina e glaucoma" esprimono il pa-

rere che di norma il distacco di retina è accompagnato da ipotensione e che la eventuale concomitanza di uno stato ipertensivo debba essere ricercata in complicanze uveitiche ed inoltre che un tono elevato del bulbo favorisce la guarigione mentre la ipotensione la contrasta. Nella discussione di questa comunicazione Rintelen porta il caso di un occhio già glaucomatoso affetto da distacco di retina con due rotture che si riaccollò spontaneamente perchè spontaneamente la tensione bulbare da 15 mill. Hg era salita a 50 mill. Hg; ed un altro che da 50 mill. era scesa con la Elliot a 14 mill. Hg, in cui egli aveva avuto un distacco di retina con rotture. Nella letteratura recente ho trovato diversi casi di distacchi di retina in glaucomi, specie infantili, per caduta repentina della tensione da alte quote, mediante colliri ipotensivi molto forti. Bonavolontà in una ampia disamina della tensione bulbare in distacchi monoculari nel periodo preoperatorio, trova invero solo nella metà dei casi una ipotensione nell'occhio malato in confronto al sano, mentre nell'altra metà il tono era pari in ambedue gli occhi. Lo Cascio nel 1958, in quattro casi esaminati per rotture di retina, senza distacco, misurò in un solo occhio la tensione oculare ed essa era normale, questo caso, osservato per lungo tempo, non ebbe distacco di retina; è un vero peccato che egli non abbia preso nota della tensione in quei casi che poi sfonciarono in distacco. In una mia comunicazione alla Soc. Oft. Lombarda del 1952 trattando "Lo scoppio della borsa retinica nel distacco di R." accennai al caso di una signora di Trieste F.K. da me controllata per una decina di anni per un foro retinico paracentrale, con opercolo fluttuante nel vitreo, senza che si fosse formato il distacco di R., ora aggiungo che in tutto quel tempo la tensione del bulbo era stata normale (pari all'altro occhio).

Da quanto esposto appare che il distacco di retina con rotture, sia di norma congiunto ad ipotensione bulbare e ciò per reazioni funzionali della coriocapillare in diretto contatto con il vitreo, attraverso la deicenza retinica (Duke Elder, Arruga). Del resto, assistiamo ad un fenomeno analogo quando con la sclerotomia "posterior", nei glaucomi assoluti, otteniamo la riduzione della tensione mediante un taglio che investe non solo la sclera e la coroidea ma pure la retina.

Ora, messo sullo stesso piano distacco di retina con rotture e ipotensione bulbare, dobbiamo osservare il comportamento del distacco di R. quando si riesce ad imporgli un ipertono, anche modico, del bulbo. Ho già accennato al parere dei vecchi testi di oculistica, che per ottenere un tono elevato del bulbo nel distacco usavano il bendaggio compressivo; al parere di Dufour-Bianchi, al caso di Rintelen, alle vedute di Custodis e di Hensius, e potrei aggiungere giudizi di molti autori, che si dichiarano convinti che lo stato ipertensivo del bulbo, operato di distacco, rende più probabile la sua guarigione, e ciò anche senza ricorrere alla perforazione sclerale per svuotare il liquido subretinico. Infantti, secondo essi, questo liquido viene riassorbito molto più facilmente e rapidamente, quando con

l'intervento, dopo aver curato la chiusura delle rotture retiniche, si porta il bulbo ad un ipertono. Come si può spiegare tale fenomeno? E' probabile che, mediante la ipertensione, il liquido subretinico sia costretto a ridursi di spessore e ad estendersi in superficie, sotto la retina, ed entrare in contatto con una più vasta porzione di coriocalpillare, che quindi si mette in condizione di un più intenso riassorbimento. E' però verosimile che altri processi, basati forse su reazioni da choc pressorio sul sistema vasale oculo-uveale, vi entrino in funzione. In ogni modo anche senza una esatta spiegazione delle cause del fenomeno, ci conviene considerare il dato di fatto e sfruttarlo nel tentativo di aumentare le probabilità di guarigione del distacco di retina. E' utile poi porre il problema se la creazione di un ipertono, ci disimpegna dallo svuotare il liquido subretinico. E' chiaro che questa possibilità ci renderebbe un gran servizio, sebbene la eventuale perforazione sclerale (per ottenere lo svuotamento del liquido subretinico) abbassi la tensione bulbare per breve tempo, giacchè la ferita si chiude rapidamente mentre le misure ipertonizzanti presse possono rientrare in funzione.

Ora è logico che per avere esatta conoscenza del tono bulbare, è necessario che noi lo si debba controllare o con il tonometro di Schiötz o con quello a molla di Bailliart, mentre il controllo digitale non è nè sufficiente, nè consigliabile.

Passiamo ora a vedere quali mezzi sono a nostra disposizione per ottenere un rialzo del tono oculare: anzitutto i comuni midriatici, atropina e scopolamina; il loro uso è generale e ogni considerazione critica è superflua.

Il bendaggio compressivo, buono solo se tollerato dalla cornea, già il Fuchs fa riserve appunto per il pericolo di complicanze corneali, io a volte ho usato il bendaggio compressivo su una ciambrella di materia onde risparmiare la pressione sulla cornea.

Il colmotage di Lagrange, di azione incerta, è attualmente abbandonato.

Il focolai di coagulazione piana sulla sclera, anche essi usati da tutti gli operatori sono di azione indiscutibile.

Le iniezioni di aria nel vitreo, oppure nel previtreo, secondo la mia pubblicazione del 1947, pratiche sempre utili, ma da adottarsi solo in certi casi e con tutte le cautele degli interventi penetranti nel bulbo.

La riduzione del bulbo con asportazione sclerale a tutto spessore secondo Lindner o lamellare secondo Paufigue operazioni fortemente attive, quindi preziose, ma pur esse invadenti e alquanto pericolosa la prima, ossia la tecnica di Lindner.

Il cerchiaggio del bulbo all'equatore, secondo le tecniche di Scheppens, di Grignolo e da ultimo secondo Arruga. Questi cerchiaggi determinano sempre una ottima elevazione del tono oculare, ma, mentre riducono il diametro bulbare all'equa-

tore, allungano il bulbo in senso anteroposteriore e ciò rappresenta un fattore negativo del sistema. Che detto allungamento sia una realtà, lo si può constatare con la misurazione, prima e dopo l'intervento con l'esofalmometro, ma esso risulta dimostrabile su una palla di gomma della stessa rigidità del bulbo oculare: si vede che essa, mentre si riduce di diametro nel senso della legatura cerchiante, si allarga in senso opposto. Per legge fisica la stessa cosa deve avvenire anche nel bulbo oculare e pertanto la retina verrà a trovarsi in condizione di sfavore nella sua parte posteriore, ossia centrale.

Tutte le varianti della resezione sclerale con infossamento di parti sclerali: tra queste citerò la recente tecnica di Hensius, basata sulla pergamenizzazione della sclera, suo assottigliamento ed introflessione con suture. Dirò soltanto che la pergamenizzazione si ottiene attraverso un'azione troppo intensa della diatermocoagulazione e quindi si possono determinare delle gravi complicanze profonde.

Le immissioni retrobulbari di tessuti: placenta, amnios ecc. pur esse a volte attive ma spesso del tutto inutili.

Da ultimo citerò il metodo dei piombi di Custodis. Io ho trovato questo sistema il più utile ed il più importante, sul terreno pratico, fra tutti i citati. Già da una considerazione logica esso risulta il più idoneo sia a determinare un rialzo del tono oculare, sia a creare condizioni fisiche ed ambientali le più favorevoli al rapido chiudersi delle rotture retiniche. E, cosa importante arriva a ciò senza provocare deformazioni bulbari inutili e fuori zona, in quanto con esso si pratica un infossamento sclerale là soltanto, ove risiedono le rotture retiniche e si avvicina la corioidea coagulata alla retina lacerata, aiutando la saldatura tra le due membrane. Custodis molto sovente traslascia la puntura diasclerale per lo svuotamento del liquido subretinico in quanto esso, a rottura chiusa, ingrazia del tono oculare elevato si riassorbe rapidamente: egli segue il tono dopo l'intervento e se esso è troppo alto o dura troppo a lungo, lo riduce con il diamox anche per via endovenosa. Regola da tener presente nell'esecuzione della tecnica di Custodis, è che le diatermocoagulazioni sclerali sieno leggere e che i piombi vengano adagiti esattamente contro le rotture retiniche.

Terminerò questa rassegna parlando brevemente di due pratiche da me usate, una consiste nell'accorciamento dei quattro muscoli retti per doppiaggio e nella tenotomia dei due obliqui, intervento che qualche volta ho aggiunto al cerchiaggio equatoriale in casi disperati ed in monocoli, onde evitare l'allungamento del bulbo per il cerchiaggio; detta pratica prolunga il già lungo intervento del cerchiaggio e tutto l'insieme diviene poco raccomandabile.

La seconda, che non rappresenta un vero atto chirurgico ed è di facile esecuzione, consiste nell'applicazione di un anello elastico, sottile e bucherellato di plexi-

glas, applicato dietro al limbus e stirato posteriormente da quattro suture saldate, sopra la congiuntiva, ai muscoli retti. Con la trazione delle suture si può determinare un aumento della pressione bulbare, regolabile a volontà. La uso a volte come mezzo preoperatorio, assieme al bendaggio binoculare ed al riposo a letto, per ottenere meglio il riassorbimento del liquido subretinico ed insieme il riavvicinamento della retina alla coroidea.

La esposizione critica dei sistemi suddetti risponde a mie osservazioni pratiche su una discreta casistica, che qui non credo sia il caso di riportare; dirò soltanto che essa, in linea di massima, mi autorizza a rispondere in senso affermativo alla domanda che mi sono posta con il titolo del tema svolto.

Concludo dunque, unendomi a Linnen, nel giudizio che la creazione di un tono elevato od almeno di grado normale, controllato tonometricamente, rappresenta un coadiuvante utile alla guarigione chirurgica del distacco di retina.

Naturalmente l'ipertono non deve eccedere nè per entità, nè per durata di tempo e verificandosi una tale evenienza si dovrà correre ai ripari e precisamente con la puntura sclerale durante l'intervento, con il diamox a dosi elevate, e per via endovenosa dopo l'intervento.

Varese, Italia

BIBLIOGRAFIA

- APOLLONIO A., Boll. Soc. Medico chir. Varese, 1947-48.
 APOLLONIO A., Annali di Ottalmol. e Clin. ocul., 1952.
 ARRUGA H., Modal. técn. recientes de las operac. del Depr. ret. Barcelona, 1952.
 BARRAQUER J., MUIÑOS A. Rass. Ital di Ottalm., 1954.
 BERHARD-MAGISTRETTI, Boll. Ocul., 1958.
 BIETTI G. B., Congresso Latino di Oftalm., 1956.
 BONAVOLONTA, Boll. d'Ocul. 1952. Giorn. Ital. Ottalm. 1953 e 1958.
 CHAMLIN, RUBNER, Amer. J. d'Ophthal., 1956.
 CUSTODIS E., Ber. Deutsch. Ophtalm. Ges., 1951-53.
 DUFOUR-BIANCHI, Congr. Sviz. di Oftalm., 1956.
 GONIN, Le décol. de la retine Pajot u. Co., 1934.
 GRIGNOLO A. Boll. d'Ocul., 1956.
 JESS A. Klin. Monatsbl. f. Augenheilk., 1957.
 LINDNER, Ophtalm. Operationslehre, 1942.
 LINNEN, Klin. Monatsbl. f. Augenheilk., 1958.
 LO CASCIO G., Giorn. di Ottalmol. e Clin. Oc., 1958.
 OKAUNN J. D., - SCHEPPENS C. L., Leg. Saw. Rev., 1955.
 PAUFIQUE L., Bullet. Soc. Franc. d'Ophtalm., 1951.
 REMKJ H., Deutsch. Ophtalm. Gesel., 1956.
 SCHEPPENS C. L., Meeting Soc. Opt. I., 1952-1955.
 SCHIFF, Wertheimer Arch. Ophtalm. 155 Bd 15 Heft.
 SOFOD K., Klin. Monatsbl. f. Augenheilk., 1959.
 STRAMPELLI B., Ann. Oftalm. e Clin. Ocul., 1954.
 VETTER I., Klin. Monatsbl. f. Augenb., 1959.

PLEGAMIENTOS ESCLERALES EN EL DESPRENDIMIENTO DE LA RETINA

POR

ENRIQUE ARIZA H., M. D.

Bogotá, Colombia

Dentro de las actuales tendencias quirúrgicas en el tratamiento del desprendimiento de la retina, las técnicas que pretenden reducir el volumen del globo ocular, la superficie del mismo, o ambas cosas simultáneamente, constituyen el grupo más discutido a la vez que el más rico en modificaciones y variantes.

Es nuestro propósito revisar esquemáticamente las técnicas de los plegamientos esclerales y presentar una descripción gráfica de la que básicamente empleamos en la actualidad: una variante del plegamiento escleral interno por inclusión de esclera.

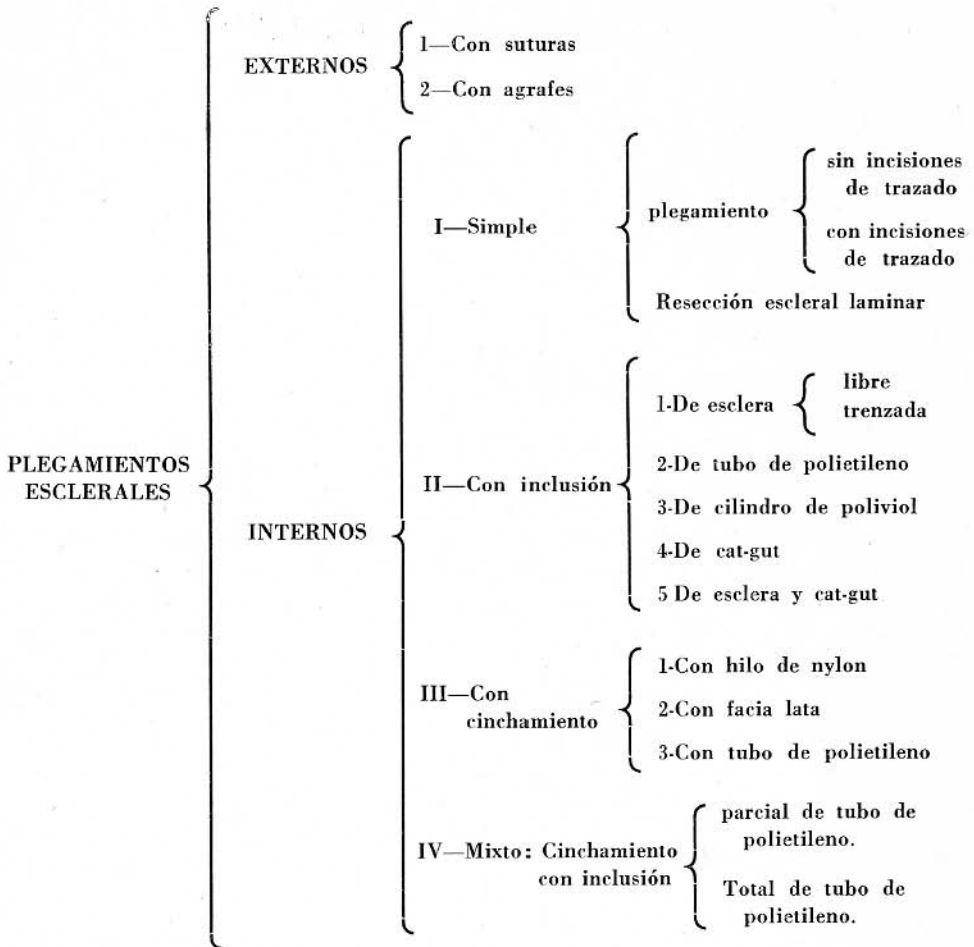
Con objeto de comprender mejor las modificaciones que los plegamientos esclerales determinan en el ojo y por consiguiente hallar un fundamento a su indicación quirúrgica en el desprendimiento de la retina, es conveniente tener en cuenta los interesantes experimentos de G. CLARK en el ojo esquemático. Si bien es claro que tales cálculos no pueden aplicarse estrictamente en el terreno clínico, representan sin embargo, valiosas orientaciones en el aspecto práctico.

Las cifras de la figura 1 han sido calculadas de cirugía practicada sobre un cuadrante a nivel del ecuador del globo ocular (14-16 mm.).

El cuadro que ofrecemos en la página siguiente es la síntesis que hemos escogido como guía para presentar la revisión en cuestión.

<i>OPERACION (un cuadrante)</i>	<i>Volumen de la Reducción ocular</i>	<i>Superficie de la Reducción ocular</i>
Resección escleral de 3 m.m.	6.5%	4.5%
Resección escleral laminar con plegamiento por tubo de polietileno de 1.25 m.m.	8.5%	0

Fig. 1. Cambios mecánicos en el ojo consecutivos a resección escleral laminar y a plegamiento escleral interno por inclusión del tubo de polietileno (un cuadrante). (Cortesía de G. Clark).



En los pliegues esclerales internos varían especialmente las cifras relativas a la reducción del volumen ocular, puesto que ello depende de cuan profundo quede el material empleado. El plegamiento escleral interno mixto por inclusión de tubo de polietileno, es ejemplo típico de las técnicas que permiten obtener grandes modificaciones volumétricas.

OPERACION (media circunferencia)	Volumen de la Reducción ocular	Superficie de la Reducción ocular
Resección escleral de 3 m.m.	13 %	9 %
Resección escleral laminar con plegamiento por tubo de polietileno de 1.25 m.m.	17 %	0

Fig. 2. Cambios mecánicos en el ojo consecutivos a resección escleral laminar y a plegamiento escleral interno por inclusión de tubo de polietileno (media circunferencia). (Cortesía de G. Clark).

PLEGAMIENTOS ESCLERALES

Las cifras de la figura 2 han sido calculadas de cirugía practicada sobre la mitad de la circunferencia ocular y las de la figura 3 señalan las modificaciones de cirugía sobre toda la circunferencia ocular.

<i>OPERACION (toda la circunferencia)</i>	<i>Volumen de la Reducción ocular</i>	<i>Superficie de la Reducción ocular</i>
Resección escleral. No se aconseja	26 %	18 %
Resección escleral <i>parcial</i> con plegamiento por tubo de polietileno de 1.25 m.m.	34 %	0

Fig. 3. Cambios mecánicos en el ojo consecutivos a resección escleral laminar y a plegamiento escleral interno por inclusión de tubo de polietileno (toda la circunferencia). (Cortesía de G. Clark).

Los ingeniosos esquemas de las figuras 4 y 5, muestran claramente los diferentes grados de modificaciones y permite observar que los plegamientos esclerales internos obran primariamente sobre el volumen ocular.

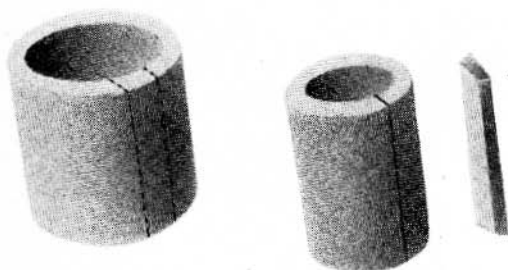
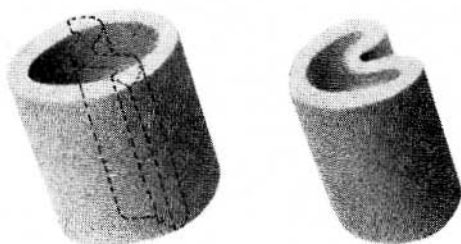


Fig. 4. Esquema de Clark para mostrar las modificaciones mecánicas que sufre el globo ocular en las resecciones esclerales laminares con lecho muy delgado. Es aplicable a los pliegues esclerales externos.

Fig. 5. Esquema de Clark para mostrar las modificaciones mecánicas que sufre el globo ocular en los plegamientos esclerales internos. Obsérvese su acción sobre el volumen ocular.



PLEGAMIENTOS ESCLERALES EXTERNOS

Este grupo se halla representado por dos técnicas que similares en su principio difieren considerablemente en su aplicación: El plegamiento mediante hilos de sutura y el plegamiento empleando agrafes de titanio.

1) *Plegamiento escleral interno con hilos de sutura.*

Técnica: Apertura y exposición escleral a nivel de los desgarros retinianos. Punción y drenaje del líquido subretinal. A continuación se practica un pliegue exclusivamente escleral con objeto de no herir la coroides al colocar los hilos de sutura. Para ello se pliega la esclera con una pinza de St. Martin y sin soltar la primera toma se realiza (con otra pinza) una segunda, por debajo de la primera repitiendo la maniobra hasta lograr un pliegue de 3 a 4 mm. de altura. El pliegue así obtenido se fija con un punto de sutura en U. El procedimiento descrito se continúa bajo control oftalmoscópico hasta obtener la máxima adaptación retinal posible. Bloqueo diatérmico de los desgarros y reconstrucción por planos (Fig. 6).

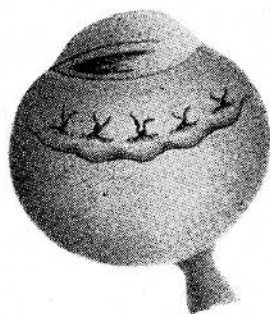


Fig. 6. Plegamiento escleral externo empleando hilos de sutura.

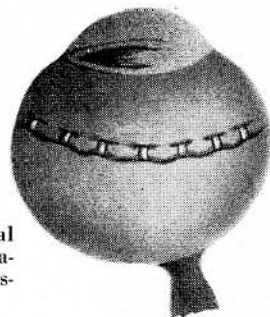


Fig. 7. Plegamiento escleral externo empleando agrafes de titanio (Castroviejo).

2) *Plegamiento escleral externo con agrafes.*

Técnica: Apertura y exposición de la esclera a nivel de los desgarros retinales. Punción y drenaje del líquido subretinal. A diferencia de la técnica anterior, se practica un pliegue escleral total que incluye la coroides dentro de él, sin peligro de hierirla puesto que los agrafes de titanio empleados en su fijación solo penetran 0.25 mm. en la escleral. El plegamiento se continúa bajo control oftalmoscópico hasta obtener una buena adaptación retinal. Bloqueo diatérmico de los desgarros y reconstrucción por planos (Fig. 7).

Los métodos anteriormente reseñados obran mecánicamente disminuyendo la superficie y el volumen del globo sin crear, per se, ningún tipo de barrera endocular y en grado diferente como se concluye del tamaño del pliegue que puede obtenerse: con hilos de sutura, el pliegue equivale aproximadamente a una resección laminar de 7 mm. de anchura; con agrafes, equivale a una resección laminar de 4 mm. de anchura.

Es precisamente esta acción mecánica la que nos permite la orientación quirúrgica de los plegamientos externos. Por ejemplo, estarían indicados, cuando es de

seable una buena reducción superficial del globo y no es conveniente correr el riesgo de ocultar parcial o totalmente los contornos de él, o los, desgarros retinianos.

Castroviejo aconseja su técnica con agrafes en los ojos miopes en los que la reducción superficial del globo es importante y la esclera adelgazada hace difíciles las resecciones laminares. Algunos casos de desprendimientos miópicos plantean el problema de grandes desgarros situados muy posteriormente y por esa razón difíciles de bloquear con plegamientos internos o diatermia sin provocar a su vez grandes irritaciones uveales y/o lesiones maculares irreversibles. En tales casos hemos visto que un pliegue suficiente para permitir la completa adaptación retinal pero que no oculte los límites de él, o los desgarros, puede complementarse con fotocoagulación y curar anatómica y funcionalmente el ojo.

PLEGAMIENTOS ESCLERALES INTERNOS

Este grupo se halla constituido por una serie de intervenciones que difieren entre sí por el procedimiento empleado en la creación del pliegue escleral: a) Pliegues simples; b) Pliegues por inclusión; c) Pliegues por cinchamiento y d) Pliegues mixtos.

I. *Plegamiento escleral interno simple.*

En los plegamientos internos simples la esclera puede dejarse in situ, o previa resección laminar y ello, a su vez, permite algunas variantes.

El más sencillo de los procedimientos de inclusión escleral sin resección consiste en practicar una barrera de diatermia superficial, colocar en sus bordes puntos en U con trayecto intraescleral de 2 mm. a cada lado, evacuar el líquido subretinal, anudar las suturas y bloquear los desgarros en la forma habitual. Si previamente se han trazado los bordes del pliegue con bisturí o similares, la coaptación de los bordes mejora sensiblemente.

Los plegamientos esclerales internos sin resección, hallan excelente indicación en casos con escleras degenerativamente adelgazadas, porque en ellos la diatermia superficial tiene acción comparable a la practicada sobre lechos laminares, sin los riesgos de ese procedimiento en tales casos.

La resección escleral laminar, dejando el lecho constituido por una capa muy delgada, es el plegamiento interno simple más empleado y versátil, por la bondad y constancia de sus resultados en la mayoría de los desprendimientos retinales recientes (Fig. 8).

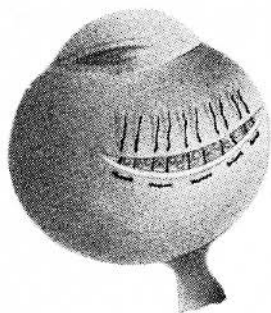


Fig. 8. Resección laminar simple. Los puntos en U permiten mejor afrontamiento de los bordes.

La técnica es como sigue: Apertura y exposición a nivel de los desgarros retinianos. Trazado y disección del fragmento escleral a resecar empleando para ello y según la preferencia del cirujano: escarificador de Desmarres, espátula cortante de Schepens, Bard-Parker bisturí o un fragmento de cuchilla de afeitar montado en portacuchillas especial. Colocación de puntos de sutura en U empleando seda 4-0 encerada. Diatermia superficial sobre el lecho de la resección. Punción evacuadora a nivel de la bolsa retinal. Cierre de la resección bajo repetido control oftalmoscópico. Bloqueo diatérmico de los desgarros y reconstrucción por planos.

El hecho de que éste procedimiento se haya enriquecido con numerosas modificaciones y variantes debe atribuirse a que las resecciones laminares profundas presentan dificultades y riesgos de orden técnico, especialmente en ojos con escleras muy adelgazadas. Estas modificaciones han convertido el pliegue simple en plegamiento escleral interno por inclusión, técnicas que pasamos a reseñar.

II. Plegamiento interno con inclusión de esclera.

Los plegamientos internos por inclusión de esclera son el producto de la necesidad de suprimir o disminuir los riesgos de las resecciones laminares clásicas y de ampliar sus indicaciones.

Pueden clasificarse en dos grupos básicos según incluyan esclera parcial o completamente libre de su lecho, o sujeta a él por uno de sus extremos y tensada para crear un pliegue artificialmente aumentado.

a) *Esclera libre*

Los pliegues por inclusión de esclera dejando el trozo resecao parcialmente fijo al lecho, tienen la siguiente técnica: Trazado de los bordes de la resección, disección laminar a partir de todo el borde superior de la resección hasta un poco por delante de su límite posterior para evitar que ella se libere completamente, colocación de puntos de sutura en U, diatermia plana sobre

el lecho y punción evacuadora. Cierre, bloqueo y reconstrucción habituales (Fig. 9). Una variación a la técnica descrita consiste en disecar laminarmente a partir de ambos bordes dejando el fragmento fijo al lecho a nivel de su línea media (Fig. 10).

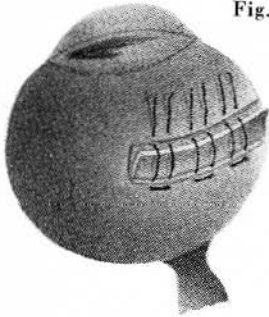


Fig. 9. Plegamiento interno por inclusión de esclera fija al lecho por su borde inferior (Lemoine, Robinson, Calkins).

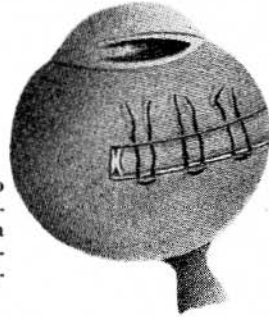


Fig. 10. Plegamiento interno por inclusión de esclera fija al lecho a nivel de su línea media (Chamlin, Rubner).

El plegamiento interno por inclusión de esclera completamente libre difiere poco de los métodos anteriores. Se procede como en una resección laminar típica hasta realizar la punción evacuadora. A continuación se repone el trozo resecaado y se cierra y reconstruye en la forma habitual. El accidente más frecuente es la sección o ruptura de la lámina escleral antes de completar su disección, contingencia que hace necesaria la inclusión fragmentaria o la elección del pliegue interno simple.

b) *Esclera tensada.*

Entendemos por plegamiento interno con inclusión de esclera tensada, al procedimiento que mediante disección laminar, provoca un pliegue artificialmente aumentado. La técnica, desarrollada por José I. Barraquer, es la siguiente:

1. Incisión conjuntival a 6 mm. del limbo corneo-escleral. Reparo y desinserción de él, o los músculos necesarios para lograr un campo operatorio suficiente. Exposición de la esclera a nivel de los desgarros retinianos.
2. Trazado de la resección escleral. Se emplea, por su sencillez, un fragmento de hoja de afeitar montada en porta-cuchillas especial. La resección debe quedar localizada por detrás de los desgarros, pues de lo contrario compromete o dificulta su bloqueo (Fig. 11).
3. Disección laminar conservando el trozo resecaado fijo a su lecho por uno de sus extremos. El lecho debe tener espesor suficiente para prevenir los riesgos inherentes a este tiempo quirúrgico (Figs. 12 y 13).

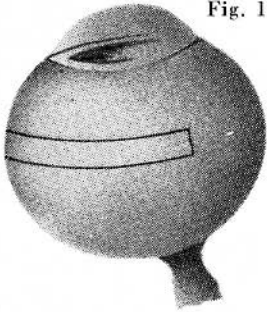


Fig. 11. Trazado de la resección escleral. Se emplea un fragmento de cuchilla de afeitar montado en portacuchillas especial (J. I. Barraquer M.).

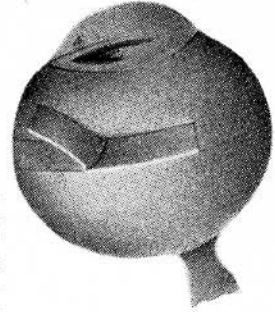


Fig. 12. Disección escleral laminar. Un lecho de espesor suficiente previene de los riesgos inherentes a este tiempo quirúrgico (J. I. Barraquer M.).

4. Diatermia plana sobre el lecho. La diatermina se extiende hasta la ora serrata desde ambos extremos de la resección (Fig. 14).
5. Punción y evacuación del líquido subretinal a nivel de la bolsa.
6. Tensamiento y fijación del extremo libre de la lámina escleral con punto en U, permitiendo que sobrepase el borde del lecho en 1 cm., aproximadamente (Fig. 15).

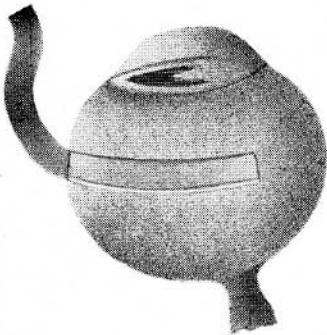


Fig. 13. Disección finalizada. El fragmento resecaado se conserva fijo a su lecho por uno de sus extremos. (J. I. Barraquer M.).

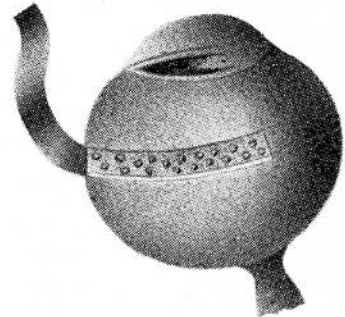


Fig. 14. Diatermia plana sobre el lecho. Se practica superficialmente y se extiende hasta la ora serrata desde ambos extremos de la resección. Punción y evacuación de líquido subretinal. (J. I. Barraquer M.).

7. Cierre de la resección mediante puntos en U, de algodón encerados (Fig. 16). El autor no considera necesario colocar los puntos previamente, como se aconseja en la mayoría de los plegamiento. El trozo de esclera que sobresale del lecho se recorta con tijeras.
8. Reconstrucción por planos.

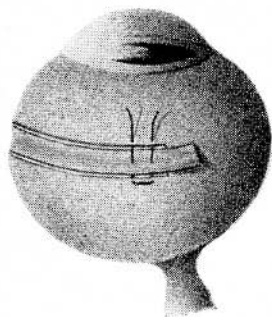


Fig. 15. Tensamiento de la esclera reseçada. El extremo libre de la esclera a incluir se fija con un punto en U permitiendo que sobrepase el borde del lecho (J. I. Barraquer M.).

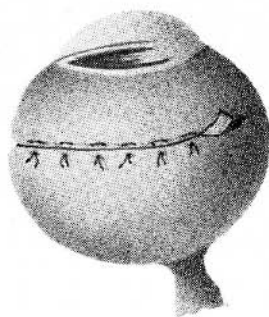


Fig. 16. Cierre de la resección. Los puntos en U realizan un excelente afrontamiento de los bordes. Se reseca el fragmento escleral que sobresale del lecho. (J. I. Barraquer M.).

Los plegamientos por inclusión de esclera crean mayores barreras endoculares que las resecciones laminares superficiales, sin todos los inconvenientes y riesgos de incluir sustancias extrañas al ojo. Las consideramos de elección cuando son necesarias reducciones volumétricas y pliegues de tipo medio. Debe pensarse en inclusiones esclerales libres, cuando es más cómodo resecar todo el fragmento, o cuando ello se ha realizado accidentalmente al tratar casos en que descamos pliegues internos de tamaño medio. No hemos visto pliegues retinales verticales a nivel de la mácula, complicación que Lemoine y colaboradores señala haber observado en su técnica de esclera fija al lecho y creemos que ello puede presentarse por exceso de diatermia.

Con la técnica de pliegue interno por esclera tensada, desarrollada por José I. Barraquer, hemos comprobado barreras mayores que las provocadas por cualquier tipo de inclusión escleral.

Como inconveniente puede señalarse que su ejecución es más difícil que la de otros procedimientos, pero en caso de ruptura accidental del fragmento, puede transformarse fácilmente en alguna de las variedades descritas.

III) *Plegamiento interno con inclusión de tubo de polietileno.*

Las técnicas de plegamientos internos por inclusión de tubo de polietileno han sido desarrolladas por la escuela de Schepens con el propósito fundamental de crear grandes barreras endoculares. Hemos sintetizado las múltiples variaciones de estos procedimientos en dos grupos: a) inclusiones sin resección escleral y b) inclusiones con resección escleral.

En la técnica sin resección escleral se procede como en los plegamientos internos con esclera in situ, aumentando el tamaño de la barrera mediante inclusión de tubo de polietileno. En el segundo grupo se practica resección laminar previa a la inclusión de él o los tubos.

Estas técnicas hallan excelente indicación en casos con esclera normal ("con resección escleral") o con escleras degenerativamente adelgazadas ("sin resección escleral") en los que se deseen grandes barreras.

IV. *Plegamiento interno con inclusión parcial de cilindro de poliviol.*

Desarrollado por Custodis, éste procedimiento consiste en provocar una gran barrera endocular mediante la inclusión parcial de un cilindro de poliviol de 5 mm. de diámetro. Ese material inerte y depresible, se fija (mediante puntos en U) a nivel del desgarro sobre esclera previamente tratada con diatermia plana.

Esta técnica ha sido empleada con éxito en una re-intervención en la cual el desgarro (pequeño y bastante posterior) no se había logrado aplicar y bloquear con otros procedimientos.

V. *Plegamiento interno con inclusión de cat-gut.*

Este procedimiento practicado por Bangertter, y Graemiger, persigue los mismos objetivos de las técnicas de Schepens, mediante el empleo de inclusiones reabsorbibles.

Idéntico a las técnicas de inclusión con resección laminar, presenta como novedad que la sustancia a incluir es cat-gut (Fig. 17).

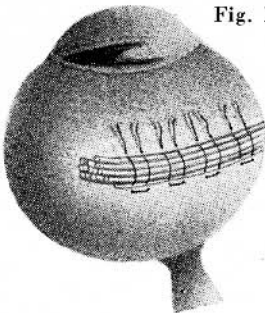


Fig. 17. Plegamiento interno por inclusión de hilos de cat-gut según técnica practicada por Bangertter-Graemiger.

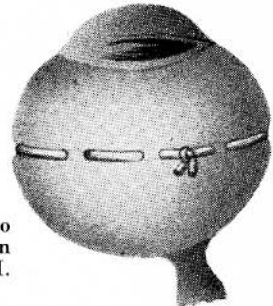


Fig. 18. Plegamiento interno por cinchamiento con hilos de nylon (H. Arruga).

Hemos empleado cat-gut plain 3-0 doblado para formar un cilindro con un término medio de cinco hilos que se coloca en el lecho de la resección laminar y se fija en la forma habitual. Como quiera que el cat-gut se hincha al contacto con líquidos debe tenerse en cuenta ese factor al calcular el número de hilos a incluir.

Si una vez colocado el rollo, el número de hilos resulta excesivo para el pliegue tallado, simplemente se retiran los sobrantes mediante maniobra que no presenta dificultad.

Una variación a ésta técnica consiste en dejar incluido el trozo escleral resecaado y aumentar el tamaño del pliegue mediante la adición de cat-gut según técnica descrita.

Hemos ideado y empleado esta variación, con el objeto de crear un gran pliegue interno sin tener que apelar a la inclusión permanente de cuerpos extraños.

3) *Plegamiento interno con cinchamiento.*

Las técnicas de cinchamiento tienen por objeto producir grandes modificaciones volumétricas con mínimo trauma uveal.

Están representadas por los métodos que emplean para ello hilo de nylon, facia lata o tubo de polietileno que, para fijarlo, lleva en su interior hilo de material no reabsorbible (nylon, dermalon, perlon, etc.).

La técnica consiste en incisión conjuntival alrededor de la circunferencia ocular. Disección de cada cuadrante, entre los músculos rectos, hasta dejar la esclera al descubierto. Bloqueo diatérmico de los desgarros. Colocación de la cincha a nivel ecuatorial y por detrás de los desgarros. El hilo de nylon (Arruga), enhebrado en agujas Grieshaber 82/10, se fija a la esclera sobre los espacios intermusculares, pasando bajo los cuatro rectos. Esta maniobra es idéntica si el material empleado es facia lata (Clark) o tubo de polietileno (Schepens), excepto que la cincha debe asegurarse a la esclera mediante puntos de sutura para evitar que cambie de situación.

Las técnicas de cinchamiento obran modificando considerablemente el volumen ocular, favoreciendo la adaptación retinal en casos con grandes bolsas, sin provocar fuertes irritaciones uveales y ofreciendo por su sencillez un recurso eficaz en la mayoría de los casos señalados.

4) *Plegamiento interno mixto*

Denominamos plegamiento mixto a la técnica de una gran barrera, mediante cinchamiento e inclusión parcial o total del material empleado como cincha.

Su técnica similar a la de los cinchamientos difiere de ellos en que el tubo (Schepens) se incluye parcial o totalmente a nivel del área retinal desgarrada, ya sea en esclera íntegra tratada con diatermia plana, o en el lecho de una resección laminar tratado de igual forma (Fig. 19).

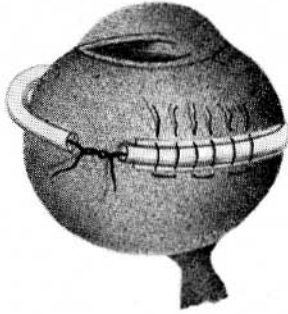


Fig. 19. Plegamiento mixto por inclusión parcial y cinchamiento con tubo de polietileno (Schepens).

La técnica mixta es el más traumatizante de los plegamientos esclerales y suele indicarse en los casos peores. En la variedad de inclusión total con resección escleral laminar, son frecuentes las lesiones maculares, (hemorragias, pliegues, etc.), como resultado de la violenta reacción uveal.

RESUMEN

1. Se presenta una clasificación de los plegamientos esclerales empleados en el desprendimiento de la retina.
2. Se revisan y comentan los procedimientos en cuestión.
3. Se presentan dos nuevas modificaciones:
el plegamiento interno por inclusión de esclera tensada (José I. Barraquer M.)
y el plegamiento interno por inclusión de esclera y cat-gut (E. Ariza H.).

Clínica de Marly

BIBLIOGRAFIA

- ARRUGA H., (1957) XXXV Arch. Soc. Oftal. Hispan. Amer.
- BARRAQUER M., JOSE I., (1959) Comunicación personal.
- CASTROVIEJO R., (1958) High. Ophthalm., 3, 158.
- CHAMLIN MAX, y RUBNER K., (1956) Amer. J. Ophthalm., 41, 633.
- CLARK GRAHAM, (1957) Amer. Acad. Ophthalm.
- LEMOINE A. N., ROBINSON J. T., y CALKINS LARRY (1958) Arch. Ophthalm., 60, 237.
- GRAEMIGER A., (1959) Comunicación personal.
- SCHEPENS C. L., OKAMURA I. D., y BROCKHURST R. J., (1958) Arch. Ophthalm., 60, 84, 1003.

L'ACTION DES EXERCICES ORTHOPTIQUES SUR L'EVOLUTION DE CERTAINES PARALYSIES OCULO-MOTRICES

PAR

NOEL BAILBE, M. D.

Perpignan, France

C'est en janvier 1955 que nous entreprîmes fortuitement des exercices orthoptiques sur les instances d'un jeune malade atteint depuis cinq mois d'une paralysie oculo-motrice non régressive.

Aujourd'hui, nous pouvons présenter II cas de paralysie oculo-motrice ayant régressé à la suite d'applications de ces mêmes exercices.

PREMIER CAS

Il s'agit de A.P. âgé de 20 ans qui fin juillet 1954 présenta soudainement:

Une paralysie faciale droite périphérique.

Une paralysie du moteur oculaire externe droit.

Une paralysie de l'horizontalité du regard vers la droite aussi bien volontaire qu'automatico-reflexe, associées à un nystagmus horizontal accompagné d'une composante rotatoire.

Aucune étiologie précise ne put être déterminée tout au plus pouvait-on dire qu'il s'agissait d'une paralysie oculo-motrice protubérancielles par poliencéphalite virale. Après de multiples examens et recherches de laboratoire nous reprîmes contact cinq mois plus tard avec le malade qui présentait le même degré de limitation des mouvements oculaires malgré toute sorte de medication.

Le 17. I. 55 nous començons les exercices orthoptiques au synoptophore avec les tests de fusion du 1er. degré (Soldat, guérite.).

L'angle est de + 12 degrés $A^0 = A^s$

A partir de ce jour et à raison de 3 fois par semaine nous essayons à l'aide du synoptophore de forcer le regard vers la droite en maintenant la fusion, et, en de-

hors de ces exercices nous demandons au malade d'effectuer chez lui, trois fois par jour les exercices de provocation du regard vers la droite.

Malgré une diplopie parfois gênante nous conseillons au malade de continuer à ne pas faire l'occlusion d'un oeil.

Il a fallu trois mois (janvier, février, mars) pour obtenir la normalisation des mouvements du regard.

Actuellement, l'état oculo-moteur est toujours aussi satisfaisant. Il importe de signaler deux faits en faveur de l'atteinte virale et des poussées virales.

1. La persistance plus longue du nystagmus au cours des années 1955 et 1956, malgré la guérison apparente des paralysies oculo-motrices.
2. La manifestation un an plus tard d'un ptosis bilatéral de courte durée.

DEUXIEME CAS.

Il s'agit d'un homme de 42 ans examiné pour la première fois le 27. IX. 1955 et atteint d'une paralysie du moteur oculaire externe gauche depuis plus d'un mois.

Un traitement à base de Cyanure de Hg. et Vitamine B₁ avait été entrepris.

Aucune étiologie ne put être retenue.

La seule anomalie associée à cette paralysie du VI était une hypoglycémie à 0,68.

La paralysie était survenue le soir après un gros effort physique.

Le 3. X. 55, c'est à dire deux mois après l'apparition de la paralysie nous commençons les exercices au synoptophore. La fusion s'établit à +15 degrés.

Nous demandons au malade de faire chez lui, trois fois par jour des exercices de provocation du regard vers la gauche et d'abandonner l'occlusion de l'oeil gauche le plus possible, sinon, de fermer l'oeil non paralysé (oeil droit) pour obliger l'oeil gauche à regarder.

Après une série d'exercices au synoptophore qui durèrent deux mois à raison de deux à trois séances par semaine la normalisation des mouvements du VI gauche fut obtenue.

TROISIEME CAS

S.B. âgée de 16 ans présente le 23. XI. 56 une paralysie du moteur oculaire externe droit associée à certains troubles parasthésiques des membres gauches.

Après plusieurs recherches et examens nous reprenons contact avec la malade qui le 28. XII. 56, soit plus d'un mois après, commence la série des exercices orthoptiques, l'angle de fusion est de +18 degrés.

Il s'agit encore d'une atteinte virale.

La paralysie du VI droit a la même importance qu'au 1er. jour. La cure médicamenteuse est abandonnée.

L'occlusion monoculaire n'est pas faite.

Les exercices de provocation du regard vers la droite sont entrepris à domicile.

Le 30 janvier 1957 les mouvements du regard sont normaux.

Il a fallu un mois pour les normaliser.

Ce cas s'apparente étrangement au 1er avec cette similitude aussi qu'au cours du mois de février 1957, cette jeune malade subit une poussée virale qui se manifesta par un ptosis gauche associé à une paralysie du droit supérieur gauche qui dura quatre jours (début février).

QUATRIEME CAS.

Femme de 70 ans examinée pour la 1ere fois le 28.XII.56 et qui depuis plus d'un mois accusait une diplopie par parésie du moteur oculaire externe droit.

L'action des médications vasculaires entreprises jusqu'à ce jour avait été sans résultat.

La paralysie est isolée malgré une sclérose nette mais non compliquée des vaisseaux rétinien.

L'atteinte nucléaire du VI droit est fortement suspectée et semble due à un trouble circulatoire.

Le 31. XII. 56 nous entreprenons les mêmes exercices signalés précédemment. L'angle de fusion est de + 3 degrés, le 1.II.57 les mouvements de l'oeil droit sont normaux après que la diplopie ait disparu bien avant.

CINQUIEME CAS

J. B. âgé de 71 ans venant consulter le 2 mai 1957 pour diplopie horizontale datant de 15 jours. Il s'agit d'une parésie du VI droit associée à un nystagmus horizontal avec composante rotatoire chez un sujet artérioscléreux faisant suspecter une atteinte circulatoire protubérancielle. Le 3.VI.57 des exercices orthoptiques sont entrepris avec un angle de fusion à +8 degrés et le 10. VII. 57 la normalisation des mouvements oculaires est obtenue.

SIXIEME CAS

J. V. âgée de 34 ans myope et souffrant depuis 4 mois d'une diplopie post-traumatique. Il s'agissait d'un traumatisme crânien ayant entraîné perte de connaissance, coma et paralysie complète et totale du III gauche. Celle-ci avait régressé au point que le 7.XI.57 date à laquelle nous examinons pour la première fois le malade, il persistait seulement une mydriase gauche et une paralysie de l'oblique inférieur gauche. Au Lancaster (paralysie de L'O.G. avec hyper-

phorie du D.S.D.) Pour éviter la diplopie la malade portait un obturateur sur le verre gauche de ses lunettes. Le 14.XI.57 les exercices sont entrepris avec les angles de fusion suivants: — 6 degrés horizontalement avec hyperphorie droite de 7 degré. Le 4. XII. 57 la fusion s'établit à — 1,50 horizontalment et pour la première fois les lunettes sont supportés sans obturateur.

Le 16.I.58 la fusion va de l'angle 0 à + 1 degré et la mydriase paralytique gauche a diminué de moitié. Le 15. II. 58, les exercices sont arrêtés, la normalisation des mouvements oculaires étant presque complète et la mydriase paralytique n'existe plus.

SEPTIEME CAS

M. L. âgée de 65 ans atteinte depuis un mois d'une paralysie isolée du VI droit.

Etant donné que 12 ans auparavant, puis 4 ans auparavant cette malade présenta une diplopie horizontale qui dura quelques jours, nous pensons qu'il s'agit d'un cas d'ophtalmoplegie récidivante. Toutefois cette deuxième récidence qui était la troisième atteinte vraisemblablement du même nerf oculomoteur traînait en longueur.

Nous entreprîmes les exercices 1 mois $\frac{1}{2}$ après l'apparition de cette paralysie, le 28.I.58, pour obtenir la normalisation des mouvements oculaires le 21.II.58, soit trois semaines plus tard.

HUITIEME CAS

M. T. R. âgée de 53 ans présentant une paralysie du VI gauche type ophtalmoplégie récidivante et qui après la troisième récidence traînait en longueur. La première manifestation de l'atteinte du VI gauche eut lieu le 28.I.57 et dura quelques jours, la seconde, le 17.II.57 et dure quatre semaines; la troisième, le 18.III.58 durait encore 8 semaines après, c'est pourquoi les exercices furent entrepris.

Le 19.V.58. 1er exercice angle de fusion + 15 degrés.

Le 31.VII.58. angle de fusion + 7 degrés.

NEUVIEME CAS

A. S. âgé de 52 ans présente en avril 1958 une paralysie totale mais incomplète du III droit avec ptosis et déviation oculaire externe sans atteinte pupillaire. Il s'agissait d'un vasculaire avec forte hypercholestérolémie.

La régression de la paralysie oculaire était très nette, mais comme il persistait une diplopie gênante avec un léger ptosis et une certaine déviation, il fut entrepris le 16. VII. 58 donc trois mois après, les premiers exercices.

Comme l'angle de fusion était trop négatif et dépassait les limites de graduation du synoptophore les exercices monoculaires de provocation du regard vers le groupe musculaire paralysé c'est à dire, vers la gauche eurent lieu pendant un mois, puis les exercices binoculaires commencèrent. Aujourd'hui, ils sont arrêtés, la diplopie ayant disparu et l'amplitude de fusion allant de -12 à $+10$. Cependant le malade garde une hyperphorie gauche de 12 degrés.

DIXIEME CAS

J. A. âgé de 62 ans artéritique, azotémique et hypertendu, présente en avril 1958 une paralysie totale et incomplète du III droit par atteinte circulatoire.

Le 16.VI.58, deux mois après, 1er exercice avec angle de fusion à -15 degrés et hyperphorie de 5 degrés.

Le 5.VII.58 l'angle de fusion est de -5 degrés.

Le 10.IX.58 l'amplitude de fusion est de $+10$ à -6 degrés. L'hyperphorie ne se modifie pas.

Par la suite il faut suspendre les exercices la fatigue empêchant même toute fixation.

ONZIEME CAS

Que je dois à l'obligeance de mon ami le Docteur Camo et dont je l'en remercie.

Il s'agit d'un sujet de 25 ans qui présenta le 12.VI.58 une paralysie du VI gauche à la suite d'un bilan ophtalmologique. Ce malade était hospitalisé pour rétrécissement concentrique des champs visuels avec héméralopie évoluant de façon progressive, sans atteinte de l'acuité visuelle centrale. La suspicion d'arachnoïdite opto-chiasmatique étant fortement retenue il fut pratiqué une cisternographie dans un but de diagnostic et de traitement. Celle-ci entraîna la normalisation des champs visuels et l'amélioration de l'héméralopie mais aussi 8 jours plus tard une paralysie du VI gauche. Les exercices au synoptophore ont été entrepris le 26.VI.58 soit deux jours après avec A.O. = A. S. $+26$ degrés et fusion de $+26$ à $+16$. Ils furent terminés le 16.VII.58 avec: A. O. = A. S. = $+10$ et C. = 56 degrés. D. = -16 .

Le 2.VII.58 le Lancaster montre une récupération importante du déficit du VI gauche. Il est difficile de trouver une cause précise à cette paralysie car il se peut qu'elle soit *d'origine mécanique secondaire* à la P. L. ou *symptomatique* d'une sclérose en plaques, pour laquelle le conteste clinique manque.

INTERPRETATION

1. *De quelles paralysies s'agissait-il?*

Pour un total de II cas nous avons:

3 cas d'atteinte nucléaire certaine (cas I et 2 par polioencéphalite virale, cas 3 par troubles circulatoires).

2 cas d'ophtalmoplégie récidivante (cas 7 et cas 8).

1 cas d'atteinte du III post-traumatique (cas 6) de pathologie discutable.

5 cas où l'atteinte nucléaire est à discuter avec l'atteinte tronculaire, très vraisemblablement.

Quant au nerf oculo moteur en cause, nous présentons 8 cas de paralysie du VI et 3 cas de paralysie du III.

2. *A quel moment les exercices ont-ils été entrepris?*

Cette période s'échelonne de quelques jours à quelques mois.

1 cas a subi les exercices sitôt après l'apparition de la paralysie (cas 2).

2 cas un mois et demi après.

3 cas deux mois après.

2 cas un mois après.

1 cas trois mois après.

1 cas quatre mois après.

1 cas cinq mois après.

Et pour ces mêmes cas:

5 n'avaient pas régressé (cas I, 2, 3, 5, 8).

5 étaient en voie de régression (cas 4, 6, 7, 9, 10).

1 fut entrepris peu de jours après.

Il convient d'ajouter que les traitements médicamenteux faits sur ces malades avant d'entreprendre les exercices avaient été sans résultats.

3. *De quels exercices s'agissait-il?*

Le terme d'exercices orthoptiques est peut-être impropre et abusif et mieux vaudrait lui substituer celui de mécano-thérapie oculaire; quoiqu'il en soit ces exercices comprennent deux temps: le premier temps est celui de la lutte contre la déviation, donc de la paralysie, par des exercices de vision binoculaire au synoptophore en entraînement progressivement et par intermitence le regard dans le champ d'action du muscle au groupe musculaire paralysé. Cet exercice à l'avantage de maintenir le réflexe de fusion et a lieu 3 fois par semaine. Nous demandons

aussi au malade de lutter contre la diplopie en supprimant si possible tout obturateur, si non d'obturer l'oeil non paralysé. De plus, nous lui conseillons de provoquer le regard vers le champ d'action du muscle paralysé en suivant son doigt par exemple et ceci trois fois par jour, chez lui.

Quand l'angle de déviation oculaire dépasse les possibilités d'établir la fusion au synoptophore nous faisons suivre un test maculaire par l'oeil paralysé jusqu'à ce que l'exercice binoculaire puisse avoir lieu.

Une fois l'angle de déviation réduit et annulé nous faisons les exercices de convergence et divergence, le deuxième temps d'amplitude de fusion dure moins que le premier. Il dure en effet de deux à trois semaines alors que le premier selon l'atteinte et l'importance de la paralysie peuvent durer de 1 à 3 mois.

4. *Y-a-t-il un effet et quels ont été les effets de ces exercices?*

Nous avons présenté précédemment les clichés de ces malades et quelques test de Lancaster; voici la courbe que nous avons établi pour chacun de ceux-là.

Chaque courbe correspond à l'évolution de l'angle de déviation oculaire échelonnée sur la durée du traitement.

Nous constatons d'abord un plateau correspondant à la résistance paralytique aux exercices, puis une chute indiquant plus de souplesse neuro-musculaire ou un clocher correspondant à une contraction de l'antagoniste ou du synergique contro-latéral puis une descente en lysis progressive ou en chute. En somme trois phases principales le plateau (résistance) le clocher (la contraction réactionnelle) la chute (qui signe la diminution de la contracture et la reprise de fonctions du muscle paralysé). Il semble que plus ces exercices sont entrepris tôt, plus la durée des exercices est réduite.

Nous avons noté l'importance de la fatigue et de l'état général sur l'évolution de cette courbe et de l'angle de déviation; la fatigue et la déficience de l'état général annule les effets des exercices durant quelques séances, il convient alors de les suspendre le temps qu'il est nécessaire. Mais une fois la normalisation oculomotrice obtenue elle est durable car depuis 1955, nous pouvons constater qu'elle s'est maintenue. Par contre dans le cas de paralysie d'un muscle d'action complexe nous remarquons la persistance de l'Hyperforie.

5. *Comment interpreter ces effets, autrement dit quelle est l'action de ces exercices*

Etant donné le tracé des courbes, la réduction de l'angle de déviation, le bien être éprouvé par le malade, il est incontestable qu'une amélioration a été obtenue.

En rétablissant la vision binoculaire et en la maintenant dans le champ d'action du muscle paralysé il semble que nous agissions autant sur le muscle paralysé en le

réduquant que sur le muscle contracturé en l'assouplissant, si nous en croyons nos graphiques de Lancaster.

Nous savons tous qu'il existe de nombreuses paralysies oculo-motrices qui disparaissent d'elle-même sans aucune thérapeutique. Elles peuvent se classer en deux groupes, celles qui s'améliorent en l'espace de 3 ou 4 jours et celles qui mettent plusieurs mois voire un an. Evidemment il est impossible de faire la comparaison évolutive sur le même sujet, mais nous avons pu remarquer que certaines paralysies du VI d'étiologie méconnue comme celle observé pour les cas 2,4,7 mettent presque 1 an à se normaliser, alors que deux mois d'exercices ont réduit celles dont nous avons fait état.

Nous avons présenté quelques cas pris en cas très tard et sans signes régressifs de la paralysie; trois mois plus tard les mouvements oculaires étaient normaux. S'agit-il d'une série heureuse?

De telles constatation nous font penser plutôt que cette mécanothérapie oculaire favorise la régression de certaines paralysies oculo-motrices et abrège le temps de cette évolution favorable. Nous pensons aussi qu'elle l'active et la complète et nous croyons qu'elle peut être appliquée à des cas semblables à ceux que nous avons pu vous présenter, c'est à dire où l'évolution régressive paraît ne pas se faire, se fait lentement, incomplètement et ou l'étiologie demeure parfois méconnue. Elle permettra aussi de ne plus attendre ce délai d'un an jusqu'ici nécessaire pour se prononcer en faveur de l'acte chirurgical. Elle pose cependant un problème de déterminisme que je laisse en suspens puisque en agissant sur le muscle nous arrivons à faire récupérer le déficit nerveux.

Perpignan, France

RESIDUAL ASTIGMATISM WITH CONTACT LENSES

BY

NEAL J. BAILEY, O. D.

Columbus, Ohio

During the past ten years, corneal contact lenses have enjoyed a constantly accelerating rate of application and it seems probable that this popularity will not soon diminish. Comfort and wearing time with contact lenses have been aided by improved lens design and improved techniques for producing smoother and better surfaces and edges. However, even though their optical performance has been improved through the production of better surfaces, the majority of contact lens wearers do not see as well as they formerly did with spectacles because cylindrical corrections rarely are incorporated in the new corneal lenses.

Textbooks 1, 2, 3, 4, tell us that the astigmatism which can be demonstrated in the eye wearing a corneal lens seldom is of sufficient amount to interfere with the acceptance of the new visual device. This statement is, of course, quite true; if it were not, few contact lenses would be prescribed today. However, the concern formerly expressed by refractionists for the effects of small degrees of uncorrected astigmatism upon visual comfort and efficiency probably have had some basis in fact. It seems hardly reasonable to assume that uncorrected astigmatism which exists while a contact lens is worn is visually less important than the uncorrected astigmatism of the person who wears no optical correction at all. In addition, it is not uncommon to find that a contact lens wearer is unable to tolerate his lenses for various tasks because of this problem⁵.

Contact lens practitioners often insist that their patients see better with contact lenses than with spectacles but measurements of visual acuity do not bear out this claim except in a relatively small proportion of the contact lens population. Most contact lens wearers will express a decided preference visually (but not cosmetically) for the addition of a cylindrical correction over their contact lenses. However, because of corneal changes produced by the contact lenses ("spectacle blur"), these same patients will tell their doctor that they see better with their contact lenses on than with their spectacles.

Residual astigmatism, i.e., that astigmatism which remains uncorrected while a contact lens is being worn, most commonly approaches one-half a diopter and rarely exceeds one diopter, although a few cases of one and half to three diopters have been seen by the author. In our office, all contact wearers are refracted a number of times while their lenses are being worn and it has been found that at least seventy-five per cent of eyes have residual astigmatism of one-half diopter or more. Characteristically, this uncorrected error of refraction takes the form of a minus cylinder axis at, or rather near, ninety degrees.

Though residual astigmatism in scleral type contact lenses often can be traced to the fitting characteristics of the lens, it is unlikely that such is the case with corneal lenses. The author has attempted to alter the uncorrected astigmatism by varying the lens size from eight and one-half millimeters to twelve millimeters and by varying the "fit" of the lens from apical clearance to as much as five diopters flat. A markedly thin lens will sometimes produce a variable astigmatic error due to a vertical bending of the lens but its effect is unpredictable and the extremely thin lens often cannot be worn with physical comfort. It seems probable, then, that residual astigmatism in corneal lenses is rarely the result of a poorly fitted lens.

Three anatomical sites which might contribute to this residual error can be considered: 1) the cornea, 2) the crystalline lens, and 3) the retina.

When corneal contact lenses are fitted, it is generally assumed² that the corneal astigmatism as measured with the ophthalmometer is completely neutralized by the lacrimal lens which is found between the front surface of the cornea and the back surface of the contact lens. However, this assumption is based upon a generalization which may not be true. The ophthalmometer is calibrated for an index of 1.3375 while the cornea has a somewhat higher index for about 1.376. Hence, the ophthalmometer really is measuring only $.3375/.376$ or about nine-tenths of the corneal astigmatism.

If the cornea is a "plano" sheet of tissue and hence acts as a "bi-toricens" of plano power, it is probable that the remaining ten percent of the corneal astigmatism is removed by the back surface of the cornea. However, if the cornea is not a "plano" sheet but instead has plus or minus power which differs in its two principal meridians, the astigmatism indicated by the ophthalmometer will not be fully corrected by the lacrimal lens.

In addition, since the bundle of rays from the point of fixation passes through the cornea along a path which makes an oblique angle⁶ with the optic axis of the eye, the cornea would tend to introduce more plus power along its horizontal than its vertical meridian.

The crystalline lens of the eye also may provide an astigmatic component via two sources: 1) the crystalline lens may not be a spherical lens but instead may have the form of a spherocylinder; 2) the lens may be oriented so that the chief ray of light passes through the lens at an oblique angle to the optic axis of the lens. In either event, the crystalline lens would have an astigmatic focus.

A third, but somewhat improbable, source of astigmatism would exist if an irregularity in the macular area of the retina were such as to require an astigmatic image for clearest vision.

Of the three potential sources of residual astigmatism, the cornea probably exerts the greatest influence. If it is assumed that the incident light makes an angle of about five degrees with the optic axis of the eye, one might predict that about one-half to three-quarters of a diopter of astigmatism should be presented with a spherical cornea whose radius of curvature were approximately eight millimeters. The correcting cylinder would have its minus cylinder axis at, or very near, the vertical meridian.

It is instructive in this connection to point out that, in an analysis of seven aphakic eyes wearing spherical corneal contact lenses, five eyes showed residual astigmatism between one-half and three-quarters of a diopter, and two eyes had between one-quarter and one-half diopter. In all seven cases, the correcting minus cylinder axis was within ten degrees of the vertical meridian.

Since the vast majority of non-aphakic patients also require about this same amount and type of astigmatic correction over their contact lenses, it seems probable that the "obliquity factor" at the cornea is the most common and most important site of responsibility for residual astigmatism when spherical contact lenses are worn. However, whether or not agreement can be reached as to the cause or causes of residual astigmatism, a reasonable solution to the problem of astigmatic corrections in corneal lenses must be sought.

It is possible to produce a corrective cylinder effect by grinding cylinder power on either the back surface or the front surface of a contact lens or, indeed, the lens might be made bitoric but of spherical power as read in the lensometer. Let us examine each of these possible solutions in turn.

Nissel⁵ has pointed out the mechanical and optical advantages and disadvantages of the inside surface cylinder. He points out that: 1) the machinery necessary to grind inside cylinders is simpler than for outside cylinders, and 2) a much larger margin of error is permissible since the lacrimal fluid will reduce the apparent error. However, as he also states, a cylinder of nearly three times the desired correcting effect is required because of this same lacrimal fluid neutralizing effect.

An otuside surface cylinder, since its full effect as read in the lensometer would be apparent must, be made far more accurately, hence difficulties would be greater. However, since only the residual error of refraction need be incorporated, the cylinder required would be markedly less.

The bi-toric lens would present extreme difficulties in its fabrication if the lens were to be ground. However, if it were made by "warping or bending a spherical corneal lens, it might provide a very useful temporary correction, at least. Its cylindrical effect upon the contact lens-eye system would be impressed upon the lacrimal lens, hence its correcting effect could be read with reasonable precision on the ophthalmometer or radiuscope. Naturally, its power as read with the lensometer would be spherical for all clinical purposes.

If one of the above lenses can be produced, our next problem would involve a technique whereby the rotation of the lens could be stopped or at least minimized to a reasonable degree. The author and a number of others^{5, 6, 7, 8, 9, 10} have found methods which will reduce rotation of a corneal contact lens so that an astigmatic correction might be feasible.

Perhaps the simplest and most generally applicable technique would be that of an unbalanced or "weighted" lens. This technique makes use of a prism in the lens of about one degree apical angle or approximately one prism diopter of power. Such a procedure would demand that an equal amount of prism be incorporated in each lens in a binocular case unless a vertical phoria correction were desired. A second type of weighting involves the implanting of a small metallic disc⁷ near one margin of the lens. The base of the prism and the metallic disc both tend to orient themselves at the lowest point of the corneal lens.

One manufacturer⁸ has stopped or minimized lens rotation by the use of a toric peripheral or secondary curve on the lens. The curvature difference between the principal meridians of the secondary curve can be varied to fit the needs of the individual cornea.

A second manufacturer⁹ has used a toric inside curve plus small facets to eliminate rotation of this corneal lens but it should be noted that this solution demands that the astigmatic correction desired and the minimum degree of toricity required to stop rotation must be in good agreement unless the facets alone are used.

Corneal lenses which are made oval¹⁰ in shape (rather than round) tend to assume a position with the major axis of the lens along the horizontal meridian of the eye. Hence, this design also might be used to reduce or remove rotational effects.

It is probable that each manufacturer and each practitioner will find reasons to argue for one of the above techniques but a weighted lens has the major advantage that it would require no particular change in our present techniques of contact lens fitting. Adjustments on such a lens could be carried out just about as they are at present.

The manufacturing costs of astigmatic contact lenses would appear at first glance to be rather high but if it is remembered that the residual astigmatism is of consequence only when it is from about one-half to one diopter in amount, and further, that its minus cylinder axis nearly always is at or very near the vertical meridian, it would seem possible to make such lenses on a stock basis just as are spherical lenses. Special lenses would be needed only when a rather extra-ordinary problem occurred.

If it might be assumed, for example, that the prismatic technique for stopping rotation were to be used and that the cylinder were to be ground on the front surface, lenses, then would be made with a plus cylinder of about one-half diopter ground with its axis at right angles to the base apex line of the prism. Only in a few special cases would spherical lenses be required and in still fewer cases would cylinders greater than one-half diopter be ordered.

Residual astigmatism is a problem which today is recognized by relatively few practitioners in the eye care field. It is however, one of the very real and, I believe, rather serious problems remaining in the contact lens field. When it is solved, we shall have made a real stride in the direction of making the contact lens a really complete visual aid.

2697 Cleveland Avenue

BIBLIOGRAPHY

1. BENNETT, A. G., *The Optics of Contact Lenses*, Second Edition, Association of Dispensing Opticians, London, 1956, pp. 18-19.
2. BIER, N., *Contact Lens Routine and Practice*, Second Edition, Butterworth Scientific Publications, London, 1957.
3. WESLEY, N. K., and JESSEN, G. N., *Contact Lens Practice*, The Professional Press, Inc., Chicago, 1953.
4. OBRIG, T. E., and SALVATORI, P. L., *Contact Lenses*, Third Edition, Obrig Laboratories, Inc., New York, 1957.
5. NISSEL, G., *Correction of Residual Astigmatism with Contact Lenses*, The Optician, Vol. 137, No 3552, pp. 407-408, May 1, 1959.
6. MOTE, H. G., and FRY, G. A., *The Relationship of the Keratometric Findings to the Total Astigmatism of the Eye*, American Journal of Optometry and Archives of the American Academy of Optometry, Vol. 16, No 11, p. 409, Nov. 1939.
7. LONG, W., Mueller-Welt Contact Lenses, Inc., Chicago, Illinois, Personal Communication.
8. International Lenses Laboratories, Detroit, Michigan.
9. Stimson Lens Laboratories, Los Angeles, California.
10. Carskadden Optical Co., Zanesville, Ohio.

ZONULOLISIS ENZIMATICA

Experiencia personal en 297 casos: resultados y conclusiones

POR

JOAQUIN BARRAQUER, M. D.

Barcelona, España

Introducción

Con nuestro descubrimiento de la acción de la alfa-quimotripsina sobre la zónula humana (zonulolisis enzimática) en junio de 1957*, se ha introducido un cambio fundamental en la cirugía del cristalino que justifica plenamente la abundante literatura aparecida sobre este tema en los últimos meses.

En este trabajo exponemos nosotros la experiencia personal con la "zonulolisis enzimática" desde enero de 1958 hasta febrero de 1959. Durante este período, fueron operados 297 globos oculares, pertenecientes a individuos de las más diversas edades.

Hemos clasificado los 297 casos objeto de este trabajo, en dos series. La *primera serie* comprende todos los casos operados desde enero a julio de 1958 con las mismas indicaciones y técnica quirúrgica, deducidas de nuestra labor experimental. La *segunda serie* comprende los casos operados a partir de septiembre de 1958, con ciertas variaciones en las indicaciones y técnica, fruto de nuestra experiencia en la primera serie y de la de otros muchos autores que fueron comunicándonos sus resultados.

Las complicaciones habidas y los resultados obtenidos se exponen en diversos cuadros resumen a continuación de los que se especifican algunos de los casos más interesantes.

Finalmente se resumen las indicaciones y técnica de elección.

* BARRAQUER, JOAQUIN, "Zonulolisis Enzimática - Contribución a la Cirugía del Cristalino", Nota previa, abril de 1958.

1ª SERIE (Enero - Julio de 1958)

Comprende 195 casos de extracción total del cristalino en pacientes de 1 a 90 años de edad ** aplicando alfa-quimotripsina por detrás del iris, a través de la pupila y de la iridectomía periférica.

En conjunto los resultados fueron francamente buenos. La extracción total del cristalino se logró fácilmente en todos los casos utilizando soluciones al 1/5.000 (en 7 casos la cápsula se rompió al final de la extracción, lográndose fácilmente totalizarla). Sin embargo, ocurrieron algunas complicaciones que se presentaban excepcionalmente en nuestras últimas estadísticas:

Retraso en la formación de la cámara anterior.

Pérdida de la cámara anterior.

Sinequias anteriores.

Hernias de iris.

Epitelización de la cámara anterior.

Estas complicaciones se detallan en el siguiente cuadro

CUADRO I

La mayoría de estas complicaciones se presentaron en pacientes mayores de 30 años, es decir, en aquellos casos que antes eran operados con mayor o menor facilidad por el método clásico intracapsular.

Así mismo, vimos aumentar el número de casos con filtración subconjuntival de la incisión, sin otra patología especial.

La coincidencia de incisión corneal pura en los tres casos de entreabertura tardía de la incisión, en el caso de epitelización de la cámara anterior, en dos de los casos de hernia de iris y en los cuatro de queratitis estriada prolongada, nos hizo pensar en la posibilidad de un efecto nocivo de la alfa-quimotripsina sobre los procesos de cicatrización de la incisión en determinados casos. En este mismo sentido podrían interpretarse los retrasos en la formación de la cámara anterior. La formación de sinequias anteriores obedecería al mismo mecanismo (filtración con oclusión de la misma por la raíz del iris) o a una irritación del iris por los lavados (sea química o mecánica).

** BARRAQUER, IGNACIO, Evolución de la Facoéresis, Vol. I, "I Curso Intern. Oftalm.", pág. 1.

BARRAQUER, JOAQUIN, Conclusiones deducidas del estudio de nuestra estadística de operados de facoéresis, año 1958, Vol. VIII, "EE. II. 00", T. 25.

BARRAQUER, JOAQUIN, y BOBERG-ANS, J., Cararact Surgery, Vol. 43, Nº 2. "British Journal Ophthalm.", págs. 69-77.

CUADRO I

1a. Serie

Enero a Julio de 1958 - 195 casos (edades: de 1 a 89 años)

Cuadro general de COMPLICACIONES

Edad	N.º casos	Ineficacia a-quimotripsina	Pérdida de vítreo	Retraso formación cámara anterior	Pérdida cárm. ant. 10-20 días	Sinequias anteriores con hipertensión	Hernia de iris	Entreabertura incisión sin hernia de iris	Queratitis estriada 10-20 días duración	Epitelización cámara anterior	Desprendimiento de retina
de 20 a 89 años	170	2	1	3	8	3	3	3	4	1	3
de 10 a 19 años	13		2			2	—————→				2
de 1 a 9 años	12		2		1						

Esta interpretación de las complicaciones citadas, nos indujo a modificar nuestra técnica con el fin de eliminarlas. Aquí entramos en la *segunda fase de nuestra experiencia clínica*, que comprende, por tanto, los pacientes de nuestra:

2ª SERIE (Septiembre 1958 a febrero 1959).

En principio, no utilizamos la zonulolisis en pacientes mayores de 60 años, excepto si sospechábamos una fragilidad capsular exagerada.

Por otra parte, al presentarse en esta serie algunas complicaciones graves en pacientes de menos de 10 años, tuvimos que limitar las indicaciones, según expondremos al final de este trabajo.

Las modificaciones y técnicas ensayadas han sido las siguientes:

Alfa-quimotripsina: con el fin de disminuir al mínimo sus posibles efectos secundarios, ensayamos nuevamente en determinados casos, la solución al 1/10.000. Con ello aumentó el porcentaje de roturas capsulares (todas las presentadas en el período de septiembre 58 a febrero 59. Cuadro N.º II b, casos C.J.M.N) y en cambio siguieron presentándose las complicaciones que tratábamos de evitar. (Cuadro II b, casos I.Q.) También se presentaron pequeñas filtraciones subconjuntivales sin patología secundaria, igual que con las soluciones al 1/5.000.

Todo esto nos llevó a la conclusión de que la solución al 1/10.000 no tiene ninguna ventaja, y que su uso disminuye el porcentaje de extracciones totales.

Inhibidores de la alfa-quimotripsina: en 6 casos empleamos el ácido B-fenilpropiónico en forma de irrigación de la cámara anterior para inactivar la quimotripsina. En uno de dichos casos se presentó así mismo un retraso en la formación de la cámara anterior, y en dos, discreta filtración subconjuntival.

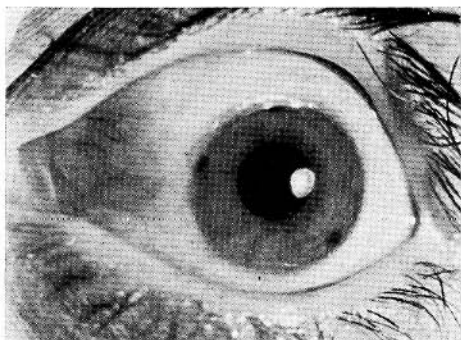


Fig. 1. Ojo con filtración subconjuntival. La profundidad de la cámara anterior y la tensión son normales.

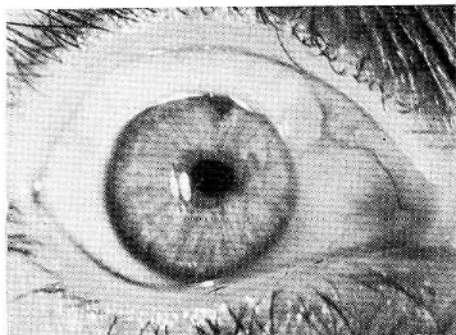


Fig. 2. Filtración subconjuntival importante que desapareció en 20 días.

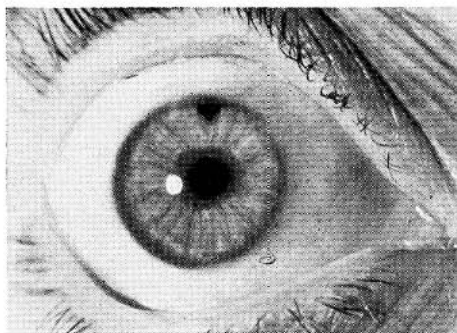


Fig. 3. El mismo ojo 20 días después. La filtración ha desaparecido.

No consideramos ventajoso su uso, máxime teniendo en cuenta que su adición representa un nuevo tiempo operatorio. Simplemente hemos continuado lavando con solución salina fisiológica para arrastrar los restos de fermento, una vez conseguida la zonulolisis.

Incisión: Con el fin de conseguir una cicatrización más activa y un cierre hermético inmediato, hemos empleado sistemáticamente en nuestra segunda serie una incisión más escleral con colgajo-conjuntival, que la recubre en toda superficie de sección.

Esta técnica nos ha proporcionado una gran seguridad, no habiéndose entabiado ninguna incisión desde que es utilizada sistemáticamente; la mejoría del trofismo de la incisión queda demostrada por la ausencia o rápida desaparición de la queratitis estriada, a la inversa de lo que ocurre algunas veces con la incisión corneal.

Extracción: En algunos casos hemos utilizado el método de SMITH, que tendría la ventaja de prescindir de la introducción de un instrumento para la extracción, disminuyendo el traumatismo operatorio (Paufique).

Seguimos considerando la ventosa como método de elección en la mayoría de los casos.

Tratamiento postoperatorio:

Medicación: Con el fin de evitar cualquier reacción inflamatoria que pudiese contribuir a las citadas complicaciones, administramos sistemáticamente pequeñas dosis de esteroides antiinflamatorios y de antihistamínicos durante los primeros días del postoperatorio.

Reposo: En cama, con vendaje binocular durante 3 días. Con vendaje monocular y reposo relativo durante 3 días más.

Hasta 20 días después de la intervención el paciente debe usar protector de plástico durante el reposo nocturno.

Para exponer los *resultados* obtenidos en esta segunda serie, hemos clasificado los 102 casos de la misma en tres grupos, según la edad de los pacientes:

Grupo 1º de 20 a 79 años: 83 casos (cuadros II, II a II b)

Grupo 2º de 10 a 19 años: 9 casos (Cuadro III).

Grupo 3º de 1 a 9 años: 10 casos (Cuadro IV).

No hemos aplicado la alfa-quimotripsina en pacientes mayores de 80 años ni menores de 1 año.

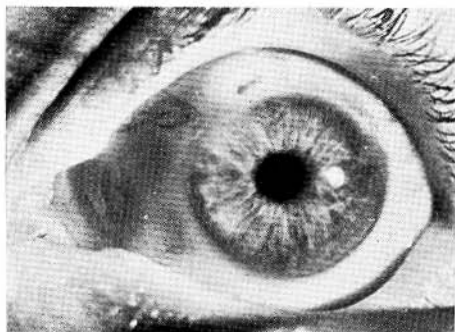


Fig. 4. Otro ojo con filtración subconjuntival persistente. El aspecto de la cicatriz es el de una trepanación de Elliot. Cámara anterior y tensión normales.

CUADRO II

2ª Serie - Grupo 1º

Grupo 1º: (83 casos) los resultados han sido uniformemente *excelentes*.

La incisión operatoria se hizo invisible en la mayoría de los casos a los 30 días de la operación. En 20 casos se presentó filtración subconjuntival, sin retraso en la formación de la cámara anterior ni pérdida de la misma. En 16 casos esta filtración fue mínima y desapareció espontáneamente en pocos días. En 4 casos, 30 días después de la operación, continuaba existiendo una ampolla de humor acuoso-conjuntival. Sin embargo, la tensión ocular era normal. Sólo en un caso (Cuadro II b, caso R) en que la incisión fue puramente corneal, se presentó queratitis estriada intensa que cedió en 15 días de tratamiento con rayos infrarrojos. La córnea estuvo ligeramente entreabierta por sus capas superficiales a nivel de la incisión sin que se observase ningún signo de filtración.

La pupila se mantuvo redonda y central en todos los casos excepto en 4 (sinequias anteriores, Cuadro II b, casos A, D, O, Q.) En 3 casos la iridectomía fue total (en 2, iridectomía previa por iridociclitis; en 1, en iridectomía operatoria por rigidez pupilar, caso P).

El *iris* no presentó alteraciones anatómicas ni funcionales excepto en 3 casos en que se presentaron diversos grados de atrofia (casos A, D, N).

El *ángulo camerular* se observó siempre normal excepto en 4 casos (A, D, O, Q), en los que se presentaron sinequias anteriores. En 3 casos de filtración subconjuntival persistente se observó una pequeña entreabertura endotelial de la incisión.

La membrana limitante anterior del humor vítreo se mantuvo íntegra en 77 casos. Se presentó rotura tardía en 3 casos. En otros 3 casos (B, D, P) la rotura fue operatoria.

De los 77 casos con membrana íntegra, en 55 ésta era retropupilar, en 6 la procidencia anterior a través de la pupila era mínima, en 9 de mediano tamaño y en 7 grande.

La tensión ocular, medida con el tonómetro de aplanación a los 20 días de la operación, fue normal en todos los casos (cifras entre 10 y 20 mm. Hg.) En los casos afectos de glaucoma (cirugía previa o simultánea), la tensión quedó controlada. En dos casos (D, G,) se presentó hipertensión persistente.

La agudeza visual osciló entre 0,2 y 1 en 72 casos, según se detalla en el cuadro II a. En 8 casos la mejoría no pasó de 0'1 por lesiones retinales antiguas.

ZONULOLISIS ENZIMATICA

En 1 caso (G), V= 0'1 (D.R. curado).

En 1 caso (D) la visión quedó reducida a percepción luminosa por D.R.T.

En 1 caso (N), amaurosis por hipema grave y "tisis bulbi".

CUADRO II

2a. Serie — Grupo I a.

(Septiembre de 1958 a Febrero de 1959) - 83 casos (edades: de 20 a 79 años)

Cuadro de EDADES Y DIAGNOSTICOS

Edad años	DIAGNOSTICO	Edad años	DIAGNOSTICO
De 20 a 29 años	Catarata congénita { madura 1 inmadura 1	De 50 a 59 años	Catarata senil { madura 1 inmadura 3
	Catarata traumática 1		intumesciente 2
	Catarata sintomática 1		Catarata miópica 15
	Alta miopía 10		Catarata y glaucoma 1
De 30 a 39 años	Catarata senil intumesciente 1	De 60 a 69 años	Catarata senil { madura 4 intumesciente 2
	Catarata sintomática 2		Catarata miópica 3
	Cristalino transparente en alta miopía 6		
	Catarata traumática 2		
De 40 a 49 años	Catarata senil { inmadura 3 intumesciente 1	De 70 a 79 años	Catarata senil madura 2
	Catarata miópica 3		Catarata y glaucoma 1
	Catarata y glaucoma 2		
	Cristalino transparente en glaucoma 1		
	Cristalino transparente en alta miopía 9		
	Cristalino subluxado 1		

CUADRO II a

Las complicaciones se han reducido al mínimo y solo han sido fatales en 2 casos (D y N). Se resumen en el cuadro II b, y las más interesantes se especifican a continuación.

CUADRO II b

A. - Retraso en la formación de la cámara anterior por bloqueo de la pupila y de la iridectomía periférica (aire en cámara anterior). Se practica nueva iridectomía periférica (inferior). Quedan sinequias periféricas que deforman la pupila. Ambliopía.

C. - Cuerpo extraño intracristaliniano. Lavado con solución de alfa-quimotropina al 1/10.000. Queda un fragmento de cápsula por detrás del iris.

CUADRO II a

2a. Serie — Grupo 1o.

Septiembre de 1958 a Febrero de 1959 - 83 casos (edades: de 20 a 79 años)

Cuadro de RESULTADOS VISUALES

	AGUDEZA VISUAL									
	De 0,01 a 0,1	0,2	0,3	0,4	0,5	0,6	0,7	0,8	0,9	1
N.º de casos (Catarata senil, traumática, sín- tomática, etc.)	5	2		1	2	5	4	2	3	7
N.º de casos (Cristalino trans- parente en alta miopía y cata- rata miópica)	6	3	9	4	13	4	7	3	1	2

D. - Alta miopía con grandes lesiones de esclerocoroidosis. Inmediatamente después de tallada la incisión, protrusión del cristalino que es expulsado con pérdida de vítreo, poco después de aplicar la quimotripsina. No formación de la cámara anterior por bloqueo de la pupila y de la iridectomía periférica por humor vítreo espeso. (aire). Hipertensión. Se practica esclerotomía, posterior e iridectomía periférica. Treinta días más tarde nuevo brote hipertensivo (extensas sinequias periféricas). Ciclodialisis. Hipotonía. Desprendimiento de retina total (D. R. T.)

E. - Pérdida incompleta de la cámara anterior de causa desconocida. Recuperación espontánea en 4 días.

F. L. - Inefectividad de la alfa-quimotripsina al 1/10.000 por presencia de vítreo flúido en cámara anterior antes de practicar el lavado.

G. - Síndrome de Marfan incompleto. Iridociclitis hipertensiva. Hipotensión. D. R. Cura con resección escleral laminar (R. E. L.)

H. - Pérdida de la cámara anterior por desprendimiento de coroides (D. C.) Cura en 8 días con tratamiento médico.

I. - Retraso en la formación de la cámara anterior por filtración subconjuntival de la incisión. Formación en 3 días al dilatar la pupila.

ZONULOLISIS ENZIMATICA

J. - Rotura capsular al pasar el cristalino a través de la pupila poco dilatada (alfa-quimotripsina 1/10.000).

K. - Desprendimiento de retina al cabo de un mes de una operación sin incidentes y con hialoides íntegra retroirídea. En curso de tratamiento con R.E.L.

N. - Grandes lesiones de coroidosis miópica. Esclerosis vascular hipertensiva. Un vaso profundo de la incisión escleral sangra intensamente, galvanocauterización. Rotura capsular (alfa-quimotripsina al 1/10.000). A las 6 horas de operación hipema total con hipertensión que simula hemorragia expulsiva. Se reabsorbe lentamente. Hipotonía. "Tisis bulbi".

CUADRO II b

2a. Serie — Grupo 1o.

(Septiembre de 1958 a Febrero de 1959) - 83 casos (edades: de 20 a 79 años)

Cuadro de COMPLICACIONES

Edad	DIAGNOSTICO	Infecti- vidad + quimo- tripsina	Rotura capsular	Rotura hialoides en me- diosis	Cristalino sublu- xado	Hipema	Retraso cámara anterior	Pérdida cámara anterior	Sinequias anterio- res	Hernia iris	Iridocic- litis	Quarantía estricta 10-20 días duración	Hiperten- sión se- cundaria	Despen- dimiento retina
De 20 a 29 años	Catarata congénita 1 (A) Catarata sintomática 1 (B) Catarata traumática 1 (C)		C	B			A		A					
De 30 a 39 años	Alta miopía 1 (D)			D			D		D				D	D
De 40 a 49 años	Catarata senil inmadura 1 (E) Catarata senil intumesciente 2 (FG) Catarata y glaucoma 1 (H) Alta miopía 1 (I)	F						E H			G		G	G
De 50 a 59 años	Catarata senil intumesciente 1 (J) Catarata miópica 4 (KLMN) Alta miopía 1 (O)	L	J MN			N								K
De 60 a 69 años	Catarata senil madura 2 (PR) Catarata senil intumesciente 1 (Q)			P	P				O			R		
									Q	Q				

Cada letra del cuadro se corresponde con la misma letra de la casuística detallada que sigue a continuación y sirve para designar un ojo de un paciente. Una misma letra repetida en varias complicaciones indica, por consiguiente, que estas tienen lugar en un mismo ojo (son consecuencia de otras ocurridas anteriormente).

O. - Sinequia anterior sin patología previa. Deformación pupilar.

P. - Pupila rígida, poco dilatada, que dificulta la extracción. Subluxación del cristalino provocada a la segunda aplicación de la ventosa. Extracción fácil con iridectomía total y asa. Visión = 0'8 n° 1.

Q. - Bloqueo de la pupila e iridectomía periférica, lo que provoca hipertensión y varias pequeñas hernias de iris subconjuntivales. Se practica iridectomía periférica inferior que restablece la comunicación entre la cámara posterior y la anterior, con reducción espontánea de las hernias de iris, quedando solamente una pequeña sinequia periférica anterior, que deforma la pupila.

Grupo 2º (9 casos). — Los resultados funcionales han sido similares a los del grupo 1º. La técnica operatoria es de realización *mucho más difícil* por existir tendencia al prolapso del humor vítreo.

Las complicaciones se resumen en el cuadro III y los resultados se detallan a continuación:

CUADRO III

A. - Catarata traumática subluxada, 10a. Vítreo previo. Aire en cámara anterior y maniobra espátula. Resultado anatómico perfecto. Atrofia antigua del II par.

B. - "Ectopia lentis" (Marfan) 14a. Vítreo previo. Aire y espátula. Pupila redonda. Visión = 0'8 Nº 1.

C. D. - Coloboma del cristalino con subluxación y alta miopía, A. O. 12 a. Ojo izquierdo, ineffectividad de la alfa-quimotripsina por vítreo fluido. Zonolulosis efectiva en ojo derecho con las precauciones y técnica especiales que se mencionan más adelante. Aire. Pupila redonda. V.D. = 0'6, nº 1; V.I. 0'5 Nº 1.

G. - Alta miopía. 16 a. Aire. Pupila redonda. V = 0'6 Nº 1.

H. - Catarata sintomática, 17 a. Aire Pupila redonda. Resultado anatómico perfecto, D. R. T. antiguo.

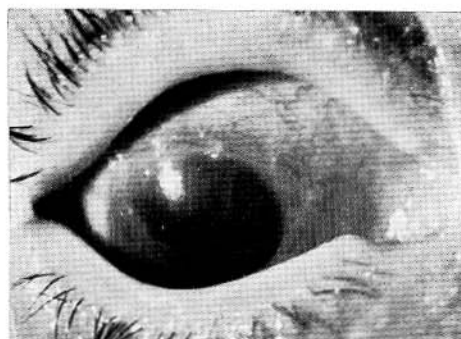


Fig. 5. Queratitis estriada (incisión sin colgajo).

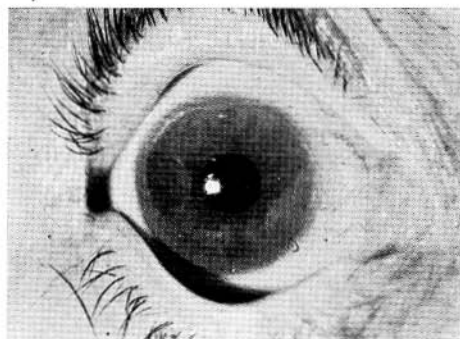


Fig. 6. Desaparición de la queratitis después del tratamiento.

I. - Alta miopía, 17 a. Pérdida de vítreo al cerrar la sutura. Iridectomía total. Aire V. = 0'4.

Grupo 3º (10 casos). — Los buenos resultados y las pocas complicaciones de nuestra primera serie (Cuadro Nº 1) nos autorizaban a emplear la alfa-quimotripsina en paciente jóvenes.

CUADRO III

2a. Serie — Grupo 2o.

Septiembre de 1958 a Febrero de 1959 - 9 casos (edades: de 10 a 19 años)

Cuadro general y de COMPLICACIONES

Edad	DIAGNOSTICO	Ineficacia a- quimotrip- sina	Pérdida de vítreo	Retraso for- mación cám. anterior	Sinequias anteriores
De 10 a 19 años	Cristalino subluxado 4 (ABCD)	D	ABCD		
	Catarata traumática 1 (F)				
	Catarata sintomática 1 (H)				
	Alta miopía 3 (EGI)		EI	E	E

Cada letra del cuadro se corresponde con la misma letra de la casuística detallada que sigue a continuación y sirve para designar un ojo de un paciente. Una misma letra repetida en varias complicaciones indica, por consiguiente, que estas tienen lugar en un mismo ojo (son consecuencia de otras ocurridas anteriormente).

Sin embargo, en la 2ª serie las *complicaciones graves* han sido la regla y, aunque en la mayoría de los casos pudieron ser solventadas, creemos que la extracción total del cristalino en este grupo debe reservarse para casos experimentales hasta tanto no se consiga una técnica que nos ponga al margen de dichas complicaciones.

Las complicaciones se resumen en el cuadro IV y los resultados se especifican a continuación.

CUADRO IV

- Catarata nuclear polar anterior 2 a., aire en cámara anterior, hipertensión por bloqueo de la pupila y de la iridectomía periférica que retrasa la formación de la cámara anterior. Se forman extensas sinequias periféricas.
- Catarata nuclear y capsular posterior 3 a., aire en cámara anterior, formación de sinequias anteriores extensas, pupila "en hamaca". Visión útil.

- c) Catarata congénita madura 3 a., zonulolisis difícil pero efectiva al repetir los lavados. Aire en cámara anterior. Bloqueo de la pupila y de la iridectomía periférica, con retraso de la formación de la cámara anterior. Hernia de iris. Resección y esclerotomía posterior. Pupila "en hamaca". Visión útil.
- d) Catarata nuclear y polar anterior 3 a. Aire en cámara anterior. Bloqueo de la pupila y de la iridectomía periférica, con retraso de la formación de la cámara anterior y hernia de iris. Glaucoma secundario. Se reseca la hernia. Queda pupila "en hamaca" y estafilomas en la zona de la incisión. Visión mala.
- e) Catarata córticonuclear monocular (ambliopía) 5 a., pérdida de vítreo, iridectomía total, aire. Entreabertura de la incisión que precisa nueva sutura. Glaucoma secundario. Visión mala.
- f) Catarata capsular anterior 7 a. Aire. Retraso formación de la cámara anterior con hernia de iris. Resección, esclerotomía posterior e iridectomía periférica inferior. Pupila "en hamaca", V. = 0'4, N° 1.
- g) Cristalino subluxado con coloboma 7 a., prolapso de vítreo, previo a la extracción. Zonulolisis efectiva, aire, pupila en hamaca. V. = 0'2 N° 3.
- h) Catarata córticonuclear, microcórnea 9 a. Cristalino es aspirado por la ventosa. Aire. Retraso formación de la cámara anterior por bloqueo pupila e iridectomía periférica. Precisa practicar iridectomía periférica inferior. Pupila redonda. V. = 0'2 N° 3.

CUADRO IV

2a. Serie — Grupo 3o.

Septiembre de 1958 a Enero de 1959 - 10 casos (edades: de 1 a 9 años)

Cuadro general de COMPLICACIONES

Edad	DIAGNOSTICO	Pérdida de vítreo	Retraso formación cámara anterior	Sinequias anteriores	Hernia de iris	Glaucoma secundario
De 1 a 9 años	Catarata congénita madura 2 (ce)	e	ce		c	e
	Catarata congénita inmadura 7 (ijadfhb)	ij	adfh	abij	df	d
	Cristalino subluxado 1 (g)	g				

Cada letra del cuadro se corresponde con la misma letra de la casuística detallada que sigue a continuación y sirve para designar un ojo de un paciente. Una misma letra repetida en varias complicaciones indica, por consiguiente, que estas tienen lugar en un mismo ojo (son consecuencia de otras ocurridas anteriormente).

- i) Catarata capsular posterior monocular (ambliopía) 9 a. Pérdida de vítreo. Aire y espátula. Sinequias periféricas extensas con atrofia de iris. Pupila desviada, V = 0'1 N° 8.
- j) Lenticono posterior, monocular (ambliopía) 9ª. Pérdida de vítreo. Aire. Espátula. Sinequias periféricas con atrofia de iris. Pupila redondeada. V = 0'03 N° 9.

La experiencia adquirida con la "zonulolisis enzimática" en el transcurso de los últimos catorce meses y que acabamos de resumir, nos ha permitido precisar las indicaciones e instituir la técnica operatoria de elección:

A. *Indicaciones:*

a) *Según el diagnóstico:*

- Cataratas de todos los tipos.
- Cristalinos transparentes (alta miopía, glaucoma por cierre angular o "angle closure glaucoma" de los anglosajones).

b) *Según la edad:*

- En pacientes mayores de 20 años y hasta los 60, es aconsejable el uso sistemático de la zonulolisis enzimática.
- En pacientes mayores de 60 años puede prescindirse de ella, estando indicada en las cataratas intumescientes, en la alta miopía y siempre que se sospeche una mayor resistencia zonular que capsular.
- En adolescentes de 10 a 20 años, un cirujano experto puede obtener muy buenos resultados, aunque hay que hacer resaltar que la intervención resulta difícil.
- En pacientes menores de 10 años, por razones técnicas no superadas todavía y sin relación con la zonulolisis, la intervención debe ser reservada para casos experimentales.

B. *Técnica:*

a) Preparación de la *solución de-alfa-quimotripsina*

- Concentración al 1/5.000. Las concentraciones más bajas muchas veces resultan ineficaces.
- Temperatura ambiente o próxima a 37°. La actividad del fermento aumenta con la temperatura de la solución. En cambio, su estabilidad disminuye, es decir, pierde actividad más pronto. El procedimiento más cómodo y eficaz consiste en utilizarla a la temperatura ambiente.
- La solución debe ser reciente, pues se inactiva a las pocas horas.
- La jeringa y la cánula deben esterilizarse al calor seco, pues los residuos de alcohol o de detergentes que podrían contener al esterilizarlas con estos agentes, inactivarían al fermento.

- b) *Anestesia general* potencializada, con aquinesia general mediante curare.
- c) *Pupila dilatada* (8 mm.) con neosinefrina al 10% (1 gota 2 horas y 1 gota 1 hora antes de la operación). Aconsejamos aquí dilatar la pupila (contrariamente a nuestras publicaciones anteriores) pues los lavados camerulares frecuentemente tienen tendencia a contraerla.
- d) *Incisión corneoescleroconjuntival*

Es muy importante que ésta se efectúe de forma que permita luego un cierre *hermético, inmediato y seguro* de la cámara anterior. Para conseguirlo, la incisión debe practicarse de forma que sus bordes sean regulares (mejor coaptación), tengan boquillas vasculares (cicatrización más rápida y segura) y que una vez suturada quede recubierta por un delantal conjuntival (cierre en dos planos).

La topografía de la incisión obliga a practicar una cuidadosa hemostasia de su borde escleral. El método más seguro, más rápido y menos traumatizante, consiste en irrigar la incisión con suero fisiológico para así poder localizar exactamente las bocas sangrantes y coagularlas con precisión con la ayuda de un galvanocauterio fino.

Debe ser amplia (180 a 200^o) para facilitar las maniobras quirúrgicas intraoculares y reducir al mínimo el traumatismo sobre el endotelio corneal, que de esta forma sufre menos rozamientos. Esta amplitud lleva emparejada la necesidad de un cierre perfecto, tal y como acabamos de exponer.

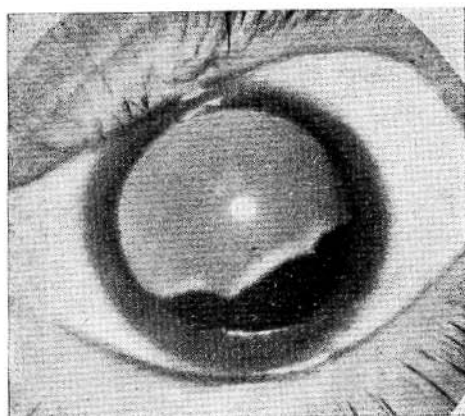


Fig. 7. Subluxación y coloboma del cristalino con alta miopía.

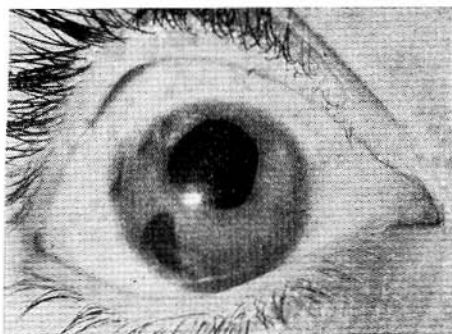
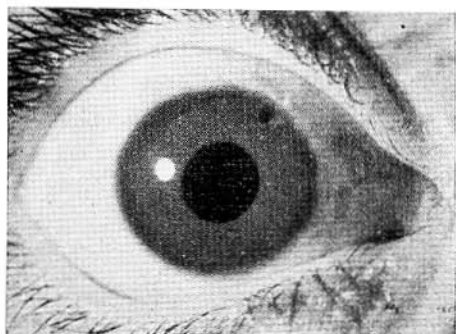


Fig. 8. Resultado después de la extracción.

Fig. 9. Pupila desviada por hipertensión y sinequias anteriores. Nueva iridectomía periférica (inferior).

e) *Iridectomía periférica*, pequeña y basal.

f) *Un punto de sutura córneoesccleral* a las 12 horas.

g) *Lavado con solución de alfa-quimotripsina al 1/5.000.*

- Mediante una jeringa de precisión, cuyo émbolo se desliza muy suavemente.
- Una cánula de plata de bordes muy romos y pulidos (una cánula corriente puede lesionar más fácilmente el iris o el cristalino).
- Para asegurar la precisión de los movimientos de la cánula en el interior del globo ocular, debe cogerse la jeringa con la mano derecha

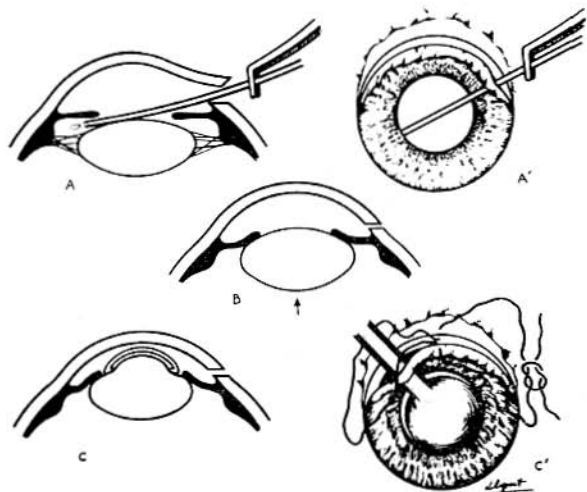


Fig. 10. Técnica del lavado con alfa-quimotripsina (A y A'). En B se demuestra que el cristalino, una vez liberado, se vuelve más esférico y tiende a prolap-sarse. Aplicación de la ventosa (C y C').

- mientras se sujeta por su base con una pinza mantenida en la mano izquierda.
- La cánula debe penetrar en la cámara posterior entre el iris y el cristalino, pasando a través de la pupila para irrigar directamente la zónula. Es conveniente deprimir ligeramente el cristalino con la cánula para no traumatizar la capa pigmentaria del iris y evitar así la liberación de su pigmento. Esta depresión debe ser muy suave con el objeto de no lesionar la cápsula ni luxar el cristalino.
 - Se inyecta suavemente, en los cuatro cuadrantes, un total de 2 cm³ de la solución.
 - A continuación se lava la conjuntiva y los bordes de la incisión con suero fisiológico mientras se mantiene cerrada la cámara anterior con la ayuda de una pinza.
 - Finalmente se lava la cámara anterior con solución salina fisiológica, suero Ringer, solución Hank, penicilina, etc., para arrastrar los restos de fermento una vez que éste ha cumplido su cometido (2-4 minutos).

h) *Extracción del cristalino:*

- Preferentemente con ventosa.
- Sin ejercer presión contra la cápsula anterior (peligro de luxación del cristalino).
- Con maniobra de versión, que facilita la separación de la cristaloides posterior de la membrana hialoidea.
- A “cielo abierto” para comprobar dicha separación, la integridad y silencio del humor vítreo y disminuir al mínimo los rozamientos contra el endotelio corneal.
- Si durante la versión comprobamos la existencia de una adherencia



Fig. 11. Un caso de catarata traumática con cuerpo extraño intracristalino.

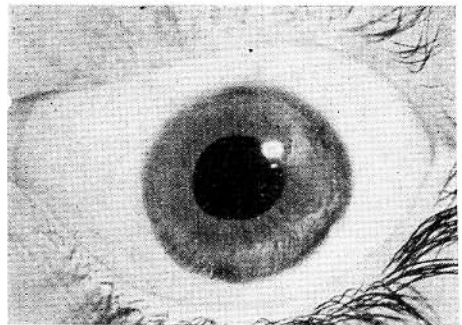


Fig. 12. Extracción perfecta mediante el método de Smith en un paciente de 16 años.

cápsulo-hialoidea, ésta puede ser liberada con la cara interna de la córnea, haciendo contrapresión con la pinza desde el exterior. Lo mismo debe hacerse si el vítreo tiene tendencia al prolapso.

- i) Lavado con solución de acetilcolina al 1/10.000 para contraer la pupila.
- j) *Sutura* corneoescleral *múltiple*, subconjuntival, que nos garantiza el cierre hermético y seguro de la incisión. La seda virgen (natural, teñida con azul de metileno) permite la colocación de 7-14 puntos corneoesclerales sin producir reacción inflamatoria. Los puntos quedan recubiertos por conjuntiva, con lo que el cierre de la incisión es más hermético.
- k) Fijación del colgajo conjuntival con solución de plasma-trombina; en su defecto pueden colocarse dos puntos conjuntivo-conjuntivales.
- l) La cámara anterior se deja llena de solución salina fisiológica, y excepcionalmente, de aire estéril.
- m) Inyección subconjuntival de cortisona (10 mgr.) y aplicación de pomada de pilocarpina.

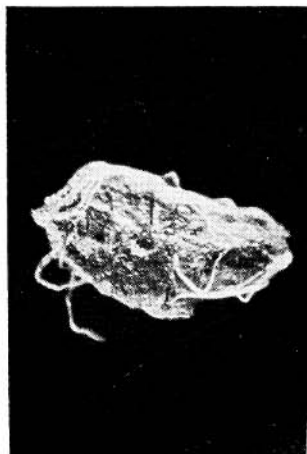
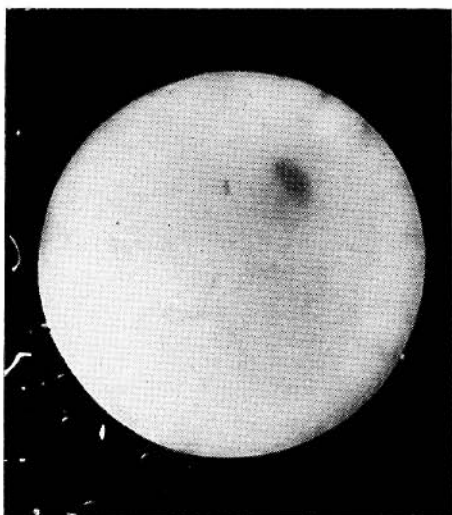


Fig. 13. Cristalino íntegro con cuerpo extraño junto a su cápsula posterior (13 horas).
A la derecha el cuerpo extraño, una vez extraído para ser examinado.

CONCLUSIONES

Del estudio de nuestros 297 casos de extracción intracapsular del cristalino mediante "zonulolisis enzimática" y de las comunicaciones recibidas de otros autores se deduce que:

- 1º La alfa-quimotripsina en solución acuosa al 1/5.000 es altamente eficaz para lisar las fibras zonulares o su unión al cristalino.
- 2º No se presentan efectos secundarios de importancia si se tienen en cuenta las precauciones anteriormente mencionadas.
- 3º En casos de humor vítreo degenerado, para obtener un zonulolisis efectiva, es fundamental evitar el contacto del mismo con la zónula para permitir la actuación de la alfa-quimotripsina, sobre ella. En estos casos es muy útil la colocación del anillo de Flieringa, que impide el colapso del globo. La irrigación con alfa-quimotripsina debe practicarse lo más cerca posible de la zónula.
- 4º El mayor porcentaje de filtraciones subconjuntivales, generalmente pasajeras y sin patología secundaria, hacen sospechar un cierto retraso en la cicatrización del endotelio corneal en los casos operados con alfa-quimotripsina. Esta contingencia carece de importancia y deja de presentarse si se siguen las normas de técnica que hemos preconizado.
- 5º Es aconsejable prescindir de la inyección de aire en la cámara anterior pues puede producir un bloqueo de la iridectomía periférica y de la pupila, con sus consecuencias. Solo debe inyectarse en los casos de pérdida de vítreo en combinación con la sección de prolapso y su reducción mediante la espátula.
- 6º En los casos de catarata traumática o en los que se sospeche una cápsula cristaliniana muy frágil, es de elección la maniobra de Smith para lograr la extracción intracapsular.
- 7º Aunque carecemos de experiencia con las suturas de catgut, y a causa de un posible efecto lítico de la alfa-quimotripsina sobre el mismo * creemos que su uso no es aconsejable hasta tanto los experimentos en curso no aclaren este extremo.
- 8º Nuestra amplia experiencia personal (297 casos) nos permite afirmar que las complicaciones citadas por algunos autores (queratitis estriada intensa, liberación del pigmento iridiano, rotura de la membrana limitante del humor vítreo, glaucoma secundario, etc.) no están en relación directa con la alfa-quimotripsina, y que se evitan fácilmente con una técnica operatoria depurada.

Muntaner, 314

* G. B. KARA, Comunicación al Congreso de la O. S. U. K., Londres, 1959.

RESULTADOS DE LA QUERATOPLASTIA EN EL QUERATOCONO

POR

JOSE I. BARRAQUER M., M. D., SALOMON REINOSO A., M. D.

Bogotá, Colombia

En este trabajo nos proponemos exponer los resultados obtenidos en 95 casos de queratocono intervenidos por queratoplastia, todos ellos en un mismo centro, en condiciones semejantes.

La técnica quirúrgica empleada puede resumirse así:

a) *Queratoplastia Penetrante. Injerto cilíndrico.*

Definición con trépano profundizando hasta abertura de la cámara anterior. Resección completada con tijera curva o cuchillote acodado. Injerto obtenido exclusivamente con el trépano. Fijación con sutura borde a borde múltiple, aproximadamente 16 puntos. En los últimos casos se empleó una sutura continua según describimos en Arch. of Ophthal. 56, pág. 526.

Injerto Escalonado: Seguimos exactamente la técnica descrita por uno de nosotros en Corneal Grafts de B. W. Rycroft-Butterworth, London 1955, pág. 98.

b) *Queratoplastia Laminar:* Delimitación con trépano, incisión de 0.3 milímetros de profundidad. Disección con espátula piriforme. Colocación de un injerto de las mismas características y obtenido por la misma técnica. Fijación por sutura borde a borde como en las queratoplastias penetrantes.

En los grandes queratoconos fue preciso practicar una paracentesis de la cámara anterior antes de fijar el injerto, para no efectuar tracciones demasiado intensas para coaptar la herida.

Material dador: En todos los casos se empleó córnea conservada en nevera indiferentemente en cámara húmeda o en aceite de parafina a una temperatura entre +2 y +4 grados centígrados. En todos los casos se empleó material aparentemente en buenas condiciones de viabilidad.

Curso Post-operatorio: El curso post-operatorio fue prácticamente ambulatorio en todos los casos. El paciente abandonó la clínica al tercer día, por término medio, acudiendo al consultorio para las curaciones sucesivas. El ojo no intervenido no se ocluyó y cuando se hizo, fue por espacio de 24 horas.

CUADROS: En los siguientes cuadros están clasificados los 95 casos de la siguiente forma:

Cuadro Primero: Queratoplastia penetrante en hombres.

Cuadro Segundo: Queratoplastia penetrante en mujeres.

Cuadro Tercero: Queratoplastias laminares en hombres.

Cuadro Cuarto: Queratoplastias Laminares en mujeres.

Cuadro Quinto: Queratoplastias laminares clasificadas según el diámetro del injerto.

Cuadro Sexto: Queratoplastias penetrantes clasificadas según el diámetro del injerto.

Cuadro Séptimo: Casos en que se prescribió lente de contacto.

Cuadro Octavo: Porcentajes de visión pre-operatoria.

Cuadro Noveno: Porcentajes de agudezas post-operatorias.

Cuadro Décimo: Tiempo de observación.

Del examen de los cuadros anteriores se deduce:

1. Que la queratoplastia en el queratocono avanzado permite obtener un 78.9% de mejorías funcionales.
2. Que el promedio de agudeza visual después de la intervención es de 0.60.
3. Que el promedio de ametropia esférica es de -3.19 , si bien un tanto por ciento de los casos tiene tendencia a miopizarse en el curso de los años.
4. Que el promedio de ametropia astigmática es de -4.50 dioptrías.
5. Que las grandes queratoplastias permiten una mejor reconstrucción de la córnea con ametropías menores.
6. Que en queratoplastias penetrantes, si bien pueden obtenerse éxitos aislados, no es aconsejable emplear injertos mayores de 7 milímetros de diámetro.
7. Que la queratoplastia laminar permite obtener cuando no hay opacidad de los planos profundos resultados visuales tan buenos (0.55) de agudeza visual como

QUERATOPLASTIA EN QUERATOCONO

Cuadro Nº 1

QUERATOPLASTIAS PENETRANTES EN HOMBRES

Caso	Historia	Ojo	Edad	Visión Previa	Dimensión m. m.	Transparencia Obtenida	Refracción	Visión Final
1	509	D	18	0.1	7.0	P.	+ 1.00 Esf (-4.00 x 20)	0.7
2	509	I	20	0.2	7.0	P.	- 5.00 (-5.50 x 55)	0.5
3	522	D	24	0.04	7.0	O.		P. L
4	522	D	26	P.L	7.0	O.		P. L
5	569	D	35	0.05	7.0	P.	- 3.50 Esf (-6.00 x 20)	0.8
6	569	D	37	0.05	7.0	P.	- 1.50 Esf (-6.50 x 165)	0.8
7	1530	D	22	0.2	6.0	P.	- 3.00 (-3.00 x 180)	0.5
8	1634	D	19	0.1	6.2	P.	+ 3.50 (-2.00 x 180)	0.8
9	1770	D	31	0.05	6.0	P.	N Esf (-14.00 x 20)	0.7
10	1789	I	12	P.L	6.0	N.	- 5.00 (-2.00 x 25)	0.3
11	1892	I	16	0.2	6.2	P.	N. Esf. (-2.75 x 70)	0.5
12	3103	D	20	0.4	7.0	P.	+ 1.00 (-8.50 x 5)	0.5
13	3103	I	20	0.05	7.0	P.	+ 1.50 (-8.00 x 10)	0.6
14	3128	D	38	0.05	7.0	P.	- 7.25 (-8.00 x 152)	1.00
15	3136	D	34	P.L	7.0	P.	- 2.50 (-1.00 x 140)	1.00
16	3136	I	34	O.4	7.0	P.	N Esf (-8.00 x 35)	0.7
17	3164	D	22	0.5	7.0	P.	- 13.00 (-5.00 x 180)	0.3
18	3503	I	21	0.05	7.0	O.		P. L
19	3549	D	18	0.1	7.0	O.		P. L
20	3549	D	19	P.L	7.0	P.	- 4.00 (-3.00 x 170)	0.7
21	3684	I	22	P.L	7.0	P.	- 11.00 (-0.75 x 30)	0.5
22	3711	I	19	0.1	7.0	P.	- 1.50 (-0.75 x 45)	1.00
23	3788	I	23	0.1	6.0	P.	- 2.50 (-8.00 x 35)	0.2
24	3802	D	19	0.3	7.0	P.	- 2.50 (-8.00 x 40)	0.2
25	3920	I	25	0.05	7.0	P.	N Esf (-3.50 x 20)	0.7
26	1932	I	48	0.05	7.0	P.	- 15.50 (-2.00 x 105)	0.4
27	1932	D	50	0.05	7.0	O.		P. L
28	1932	D	51	P.L	6.2	P.	- 17.50 (-1.25 x 90)	0.2
29	5266	D	28	0.01	6.5	P.	- 0.75 (-2.50 x 160)	0.5
30	5447	I	22	0.02	6.5	P.	- 3.25 (-3.50 x 5)	0.4
31	5447	D	23	0.02	7.00	P.	- 1.00 (-8.00 x 5)	1.00
32	5486	D	24	0.1	6.2	P.	- 4.75 (-6.00 x 27)	0.6

* D: Derecho
I: Izquierdo

** P: Perfecta
N: Nebuloso
O: Opaco

Cuadro Nº 2

QUERATOPLASTIAS PENETRANTES EN MUJERES

Caso	Historia	Edad	Visión Previa	Dimensión m. m.	Transparencia Obtenida	Refracción	Visión Final
1	0021	21	0.3	6.0	P.	- 5.50 (-5.00 x 15)	0.8
2	0021	22	0.2	6.0	P.	- 5.25 (-4.00 x 15)	1.00
3	0043	50	PL	7.0	P.	Catarata Senil	PL
4	1016	20	PL	6.2	P.	- 5.00 (-5.00 x 135)	0.6
5	1016	21	0.2	6.5	P.	- 6.00 (-8.00 x 15)	0.5
6	1029	25	0.05	6.2	P.	- 11.00 (-4.00 x 40)	0.7
7	1027	14	PL	7.0	O.		PL
8	1027	15	PL	7.0	O.		PL
9	1027	18	PL	7.0	O.		PL
10	1096	30	0.7	5.5	P.	- 9.50 (-3.50 x 75)	1.00
11	1116	24	0.6	5.5	P.	- 2.00 (-3.00 x 100)	1.00
12	1129	15	PL	6.2	N.	+ 3.50 (-2.00 x 160)	0.5
13	1185	22	0.1	6.0	N.	- 8.00 (-4.00 x 105)	0.7
14	1272	33	0.05	6.0	N.	N Esf. (-1.00 x 50)	0.5
15	1316	26	0.1	6.1	P.	- 0.75 (-5.00 x 105)	0.8
16	1441	19	0.4	6.2	P.	- 0.25 (-0.50 x 170)	0.7
17	2028	36	PL	7.0	P.	- 2.00 (-5.00 x 5)	0.7
18	2028	38	PL	6.0	N.		0.05
19	2029	19	0.01	7.0	P.	- 5.50 Esf (-2.25 x 70)	0.5
20	2029	19	0.05	7.0	P.	- 12.50 Esf (-6.00 x 140)	0.5
21	2033	45	PL	6.5	P.	- 5.50 (-5.00 x 90)	0.5
22	2128	22	0.1	7.0	P.	+ 1.50 (-8.00 x 175)	0.5
23	2128	23	0.2	7.0	P.	- 2.75 (-6.00 x 180)	0.7
24	2300	20	0.1	8.0	P.	- 1.00 (-3.00 x 165)	0.7
25	2300	22	0.2	7.0	P.		0.5
26	2442	20	0.2	7.0	N.	N Esf (-1.25 x 135)	0.8
27	2442	20	0.6	7.0	P.	- 1.00 (-6.00 x 22)	0.5
28	2590	18	0.3	6.0	P.	N Esf (-3.75 x 165)	1.00
29	2668	24	0.3	7.0	O.		0.02
30	2672	18	0.1	7.0	P.	+ 2.00 (-8.50 x 115)	0.8
31	2771	24	0.02	6.0	P.	N Esf (-7.50 x 167)	1.00
32	2771	26	0.05	6.1	P.	- 6.00 (-1.25 x 70)	1.00
33	2811	55	PL	6	O.		PL
34	2850	24	0.10	7	N.	- 3.00 (-3.50 x 30)	0.5
35	2868	25	PL	7	O.		PL
36	2941	59	PL	7	O.		PL
37	2941	61	PL	7	O.		PL
38	4146	18	0.10	7	P.	- 2.00 (-3.00 x 180)	0.3
39	4146	18	0.10	6.2	N.		0.05
40	4291	36	PL	6.5	P.	- 4.00 (-7.50 x 15)	0.5
41	4407	36	PL	6.2	P.	- 11.00 (-3.50 x 60)	0.3
42	4437	22	0.2	6.2	P.	- 1.25 (-5.00 x 50)	0.6
43	4480	50	0.1	6	P.	- 4.00 (-4.00 x 5)	0.7
44	4499	18	0.05	6.5	P.	- 2.00 (-4.75 x 8)	0.5
45	4578	30	0.05	6	P.	+ 2.50 (-8.00 x 25)	0.7
46	4662	24	0.02	6	P.	- 1.00 (-3.50 x 150)	0.7
47	4682	18	0.05	6	P.	- 5.75 (-6.00 x 170)	0.5
48	4682	19	0.05	6	P.	- 3.50 (-2.75 x 5)	1.00
49	4897	30	0.05	6.2	P.	- 18.50 (-3.50 x 150)	0.3
50	4222	13	0.01	6.5	P.	+ 1.50 (-10.00 x 180)	0.5
51	2868	25	PL	6	O.		PL

QUERATOPLASTIA EN QUERATOCONO

Cuadro Nº 3

QUERATOPLASTIAS LAMINARES EN MUJERES

Caso	Historia	Edad	Visión Previa	Dimensión m. m.	Transparencia Obtenida	Refracción	Visión Final
1	1517	43	PL	8.00	P.	- 1.00 (-4.00 x 55)	1.00
2	0525	28	0.1	11.00	P.	- 5.00 (-2.00 x 25)	0.3
3	8196	16	0.4	9.00	P.	+ 4.50 (-6.50 x 180)	0.7
4	8322	20	0.05	8.00	P.	- 1.00 (-6.50 x 10)	0.4

Cuadro Nº 4

QUERATOPLASTIAS LAMINARES EN HOMBRES

Caso	Historia	Edad	Visión Previa	Dimensión m. m.	Transparencia Obtenida	Refracción	Visión Final
1	1038	32	0.05	11.00	N.	- 4.50 Esf	0.4
2	2002	40	0.05	11.00	N.		0.05
3	2019	25	PL	11.00	P.	- 1.50 (-1.50 x 160)	0.3
4	4407	35	PL	10.00	Velo en plano de unión		PL
5	6070	18	0.3	8.00	P.	- 0.75 (-1.50 x 40)	1.00
6	6005	19	0.3	8.00	P.	+ 4.00 (-4.00 x 145)	0.3
7	6491	28	0.01	8.00	P.	N Esf (-3.00 x 10)	0.8
8	1258	52	0.2	8.1	N.		0.3

Cuadro Nº 5

QUERATOPLASTIAS LAMINARES CLASIFICADAS SEGUN
EL DIAMETRO DEL INJERTO

Laminar m.m.	Transparentes Nº	Nebulosos Nº	Opacos %	Esférico Nº	Cilindro %	Visión Pmdio.	Visión Pmdio.	Visión Pmdio.	Visión Pmdio.	Visión Pmdio.
						Preoperat.	Final	Final	Final	Final
11.00	4	2— 50	%	2— 50	%	—	- 2.75	- 0.8	0.05	0.26
10.00	1	—		1—100	%	—	—	—	PL	PL
9.00	1	1—100	%	—		—	+ 4.50	- 6.50	0.4	0.7
8.1	1	—		1—100	%	—	- 2.00	- 4.00	0.2	0.3
8.00	5	5—100	%	—		—	+ 0.27	- 3.80	0.13	0.76
Promedio total										
	12	8— 66.6%		4— 33.3%		—	- 3.02	0.156	0.40	0.22

Cuadro N° 6

QUERATOPLASTIAS PENETRANTES CLASIFICADAS SEGUN EL DIAMETRO DEL INJERTO

Penetrante Dimensión m.m.	Transparentes			Nebulosos		Opacos		Con reacción		Sin reacción		Esférico Pmdio.	Cilindro Pmdio.	Visión Preop. Pmdio.	Visión Final Pmdio.	Visión Ganada Pmdio.
	Nº	Nº	%	Nº	%	Nº	%	Nº	%	Nº	%					
8.00	1	1	100%	0	0%	0	0%	1	100%	0	0%	-1.00	-3.00	0.1	0.7	0.6
7.00	42	28	66.66%	2	4.76%	12	29%	32	76.19%	10	23.80%	-2.54	-3.12	0.10	0.42	0.32
6.5	8	8	100%	0		0		5	62.50%	3	37.50%	-2.51	-5.62	0.073	0.55	0.48
6.2	13	11	84.61%	2	15.30%	0		9	69.23%	4	30.76%	-5.23	-3.19	0.1	0.48	0.38
6.1	2	2	100%			0		1	50%	1	50%	-3.00	-4.50	0.15	0.9	0.75
6.0	15	9	60%	4	26.66%	2	13.33%	7	46.66%	8	53.33%	-2.33	-4.41	0.06	0.54	0.48
5.5	2	2	100%					0		2	100%	-5.75	-3.25	0.65	1.00	0.35
Promedio	83	61	73.49%	8	9.63%	14	16.86%	55	66.26%	28	33.73%	-3.19	-3.87	0.175	0.656	0.481

QUERATOPLASTIA EN QUERATOCONO

Cuadro N° 7

CASOS EN QUE SE PRESCRIBIO LENTE DE CONTACTO

Caso	Tipo Injerto	Refracción	Visión	Visión	Observaciones
			Sin c.c.	Con lente contacto	
3.711	Penetrante	— 1.50 Esf (-0.75 x 45)	0.4	1.00	Comenzó a usarlos al 3er. año de intervenido.
5.957	Penetrante	— 11.00 Esf (-5.50 x 115)	0.3	0.4	Comenzó a usarlos al 6º mes de la intervención.
5.486	Penetrante	— 4.75 Esf (-6.00 x 27)	0.1	0.6	En el 2º año de intervenido comenzó a usarlos.
8.196	Laminar	+ 4.50 Esf (-6.50 x 180)	0.2	0.7	Comenzó a usarlos al 10º mes de la intervención.
1.185	Penetrante	— 8.00 Esf (-4.00 x 105)	0.1	0.7	Comenzó a usarlos al 5º año de la intervención.
2.590	Penetrante	N. Esf (-3.75 x 165)	0.4	1.00	En el 2º año de la intervención comenzó a usarlos
4.578	Penetrante	+ 2.50 Esf (-8.00 x 25)	0.2	0.7	Comenzó a usarlos al 14º mes de la intervención.
1.316	Penetrante	— 0.75 Esf (-5.00 x 105)	0.4	0.8	Al 4º año de la intervención los usó.
1.096	Penetrante	— 9.50 Esf (-3.50 x 75)	0.1	1.00	Comenzó a usarlos al 4º año de la intervención.

Cuadro N° 8

PORCENTAJES DE VISION PREOPERATORIA

Visión Previa	Número de casos	%
P. L — 0.02	31	32.6
0.05 — 0.1	40	42.1
0.2 — 0.4	20	21.1
0.5 — 0.7	4	4.2
Total	95	100

Cuadro N° 9

PORCENTAJES DE AGUDEZAS POST - OPERATORIAS

<i>Visión</i>	<i>Número de casos</i>	<i>%</i>
1.00 — 0.8	21	22.2
0.7 — 0.4	42	44.3
0.3 — 0.2	14	14.7
Menos de 0.2	18	18.9 %
Total	95	100 %

Cuadro N° 10

TIEMPO DE OBSERVACION

<i>Período de Observación</i>	<i>Tipo Injerto</i>	<i>Número de Casos</i>	<i>%</i>
5—5½ años	Laminar	4	4.2 %
	Penetrante	22	23.2 %
3—4 años	Laminar	1	1.05%
	Penetrante	16	16.8 %
4—5 años	Laminar	1	1.05%
	Penetrante	16	16.8 %
2—3 años	Laminar	2	2.1 %
	Penetrante	20	21.1 %
1—2 años	Laminar	4	4.2 %
	Penetrante	9	9.48%

QUERATOPLASTIA EN QUERATOCONO

los obtenidos con injertos de todo el espesor (0.62) con un riesgo quirúrgico menor 8.3% contra 19.2% en las penetrantes.

8. La dimensión más aconsejable es la de 8 milímetros.

9. Que existe una mayor incidencia de la afección en el sexo femenino (62%) que en el masculino (38%).

10. Que la edad promedio de los pacientes intervenidos fue de 27 años y que se obtuvieron injertos con transparencia perfecta en el 72% de los casos.

11. Que el promedio de agudeza visual incluyendo los casos con resultados desfavorables fue de 0.47.

12. Durante el curso post-operatorio se presentó un caso de glaucoma agudo, 2 casos de sinequia anterior parcial, un caso de hernia de iris.

Estos casos obtuvieron respectivamente una agudeza visual final de 1.00, 0.05, y percepción luminosa.

Apartado Aéreo Nº 11056

EPITELIZACION DE INJERTOS LAMINARES TALLADOS EN ESTADO DE CONGELACION PROFUNDA

POR

JOSE I. BARRAQUER M., M. D.

Bogotá, Colombia

A excepción de los casos de autoqueratoplastia en que el epitelio del injerto persiste, en las homoplastias corneales con tejido fresco o conservado, aunque el injerto puede conservar su epitelio, éste se descama y regenera en los primeros días del curso post-operatorio. Su regeneración tiene lugar a expensas del epitelio de la córnea receptora.

En los injertos congelados la baja temperatura produce la pérdida de la adhesión del epitelio (Herman y Hickman), el cual se desprende durante las maniobras de preparación del injerto e intervención, de forma que cuando la operación termina el injerto está prácticamente desprovisto de epitelio.

En ambos casos la regeneración epitelial se produce a partir de la cornea receptora y el epitelio comienza a recubrir el injerto en aquellos puntos en que la coaptación y afrontamiento de la Bowman es mejor.

Una pequeña entreabertura o desnivel en la coaptación de esta membrana retrasa el avance del epitelio en esta zona, ya que debe rellenarla. El epitelio llega antes, a la zona de coaptación deficiente, a través del injerto.

La técnica quirúrgica empleada para la fijación del injerto tiene también influencia en la rapidez con que se inicia la epitelización.

El uso de membranas protectoras o de lentes de contacto retrasa la marcha del epitelio sobre el injerto, aunque ello no tiene consecuencias para el curso post-operatorio ulterior.

La sutura borde a borde es el método que permite una epitelización más precoz, iniciándose ésta en los espacios libres entre dos puntos de sutura, pues estos constituyen un obstáculo para la marcha del epitelio.

En este trabajo estudiamos la epitelización del injerto en 11 casos de queratoplastia laminar anterior practicados con injertos tallados en estado de congelación profunda, según la técnica descrita en Arch. Soc. Amer. Ofal. Optom. Vol. I pag. 237.

En 10 casos el injerto fue fijado borde a borde, con 4 o 5 puntos de seda virgen, la cornea fue conservada junto con el globo en aceite de parafina, a una temperatura entre +2 y +4 grados centígrados durante un tiempo inferior a ocho días.

Únicamente en el séptimo caso se empleó una cornea glicerizada que nos fue gentilmente enviada por el Dr. John Harry King, Jr. En este caso el injerto fue hidratado en solución salina por espacio de 15 minutos, y congelado y tallado al torno en la forma habitual. Se fijó con un lente de contacto e hilos de contención.

En casi todos los casos a (excepción de los números 3-4 y 11) se instiló desde el primer día una gota de fluoresceína para comprobar al microscopio la marcha de la epitelización, ésta es perfectamente visible sin este recurso, pero el colorante facilita el examen y permite el registro fotográfico.

La epitelización del injerto comenzó a las 24 horas en tres casos (2-5-8) y al segundo día cuatro (1-6-9-10) siendo completa al tercer día en dos (2 y 5) y al cuarto en siete (1-4-6-8-9-10-11).

En el caso número 7 en que se empleó lente de contacto, el cual se retiró al cuarto día, la epitelización se inició 24 horas después completándose en el curso de los cinco días siguientes.

En todos los casos se emplearon injertos entre 5,9 y 7,0 milímetros de diámetro. Los detalles de cada caso y la marcha de la epitelización pueden apreciarse claramente en los cuadros adjuntos.

Del estudio de los mismos se desprende:

a) Que la epitelización de los injertos laminares practicados con cornea tallada durante la congelación, se inicia precozmente y se completa en el transcurso de 3 o 4 días.

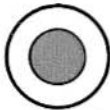

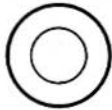

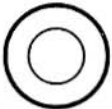

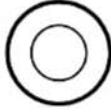


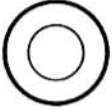
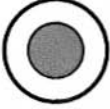


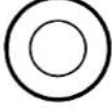

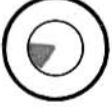





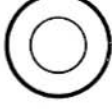
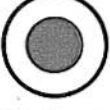

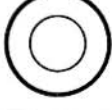

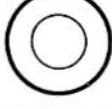
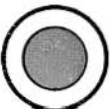



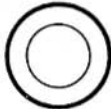
b) Que el uso de lentes de contacto para su fijación retrasa el inicio de epitelización, si bien, ésta se efectúa en la forma habitual una vez retirado el lente.

c) Que las corneas conservadas en glicerina se comportan, desde este punto de vista, como las córneas frescas aunque la observación de un solo caso es insuficiente.

CONCLUSION

La epitelización de los injertos laminares tallados en estado de congelación profunda se efectúa a partir del receptor en los primeros días del curso post-operatorio, cuando se emplea sutura borde a borde, y no difiere de la epitelización de los injertos no congelados y tallados por los métodos clásicos.

ESQUEMAS FLUORESCENCIA DE LA EPITELIZACION

	24 HORAS	DIA 2	DIA 3	DIA 4	DIA 5
CASO: 1					
CASO: 2					
CASO: 4					
CASO: 5					
CASO: 6					
CASO: 8					
CASO: 9					
CASO: 10					
CASO: 11					
	DIA 4	DIA 5	DIA 6	DIA 7	DIA 9
CASO: 7					

IDENTIFICATION OF ROD AND CONE RECEPTOR MECHANISMS IN ATYPICAL CONGENITAL ACHROMATOPSIA

BY

H. RICHARD BLACKWELL, Ph. D.,
O. MORTENSON BLACKWELL, A. B.

Columbus, Ohio

Introduction

In recent years, there has been a renewed interest in congenital achromatopsia, in part because of the discovery of what have been designated "atypical cases". (Ref. 1, 2, 3). A few of these atypical cases exhibit normal or supranormal visual acuity and yet reveal a total absence of chromatic discrimination. However, most of the cases of atypical achromatopsia exhibit greatly reduced visual acuity, but demonstrate some remnant of chromatic discrimination. Both classes of patients show evidence of an hereditary transmittance of the abnormality. In contrast to both classes of atypical cases, cases of atypical achromatopsia reveal both a total absence of chromatic discrimination and greatly reduced visual acuity.

In view of obvious differences between these two broad categories of atypical congenital achromatopsia, the present authors suggested recently (Ref. 4) that patients with achromatopsia be described by compound terms, the first describing the number of cone receptor systems found to be operative, the second describing the type of chromatic discriminations which the patient was capable of making. Thus, for example, there may be tri-cone monochromacy in which red, green, and blue cones are present but the patient still has a monochromatic functional system. Or, there may be di-cone monochromacy in which two cone systems are present, or mono-cone monochromacy in which only one cone system is in operation.

The suggested use of this compound nomenclature implies, of course, that sources of evidence other than chromatic discriminations can be found which will identify the number and spectral characteristics of the cone systems present. The literature suggests that luminosity (Ref. 5), the acuity function of luminance

(Ref. 6), and the effect of chromatic adaptation on acuity (Ref. 7) provide useful indices of the number of cone mechanisms in operation, independent of the patient's ability to make chromatic discriminations.

We have initiated a study of atypical achromatopsia with the objective of identifying the cone mechanisms in operation as well as the chromatic discriminations of which the patients are capable. The results to be reported here are concerned with three cases of atypical congenital achromatopsia with reduced visual acuity and some chromatic discriminations. Further data on these and similar patients will be reported in more detail subsequently.

The present patients were referred to us by Dr. Harold F. Falls of the Department of Ophthalmology, the University of Michigan, and our measurements were made while we were working in the Vision Research Laboratories of that institution. Analysis of the data has been made possible in part by a grant from The Knights Templar Fund at the University of Michigan, in part by support from Dr. William H. Havener, Chairman of the Department of Ophthalmology at The Ohio State University, and in the largest part by generous support from The Ohio Lions Eye Research Foundation.

Experimental Data

A. Medical Findings

The three patients were brothers; the quantitative data are so nearly identical for the three that in most instances it will be sufficient to present average data. The ages of the three patients at the various times at which we studied them varied within the following limits: 16 to 19, 20 to 23, and 23 to 26. The visual abnormality was known to be congenital, having been reported in other male members of the family. There was no evidence of change in the abnormality observed in the brothers since birth.

The pedigree obtained by the University of Michigan Heredity Clinic consists of forty related persons. The three affected brothers had one sister who was tested and found to be normal. The father was tested and found to be normal. All his relatives were reported to be normal. The mother was normal except for foveal dark adaptation which showed some evidence of rod function. One of the mother's three brothers was reported by the patients to have the same defect as they both with regard to visual acuity and color vision. The maternal grandfather of the patient's mother was also reported individuals were known. Thus the inheritance of the patients' color vision defect appears to follow the classical sex-linked pattern.

Ophthalmological examinations by Dr. Falls gave the following results. The patients had no chromatic discriminations under normal testing conditions but their visual acuity was correctable to about 20/60. There was an absence of gross nystagmus, no photophobia, and no evidence of retinal degeneration. There was no evidence of suppression or of eccentric fixation. The only fundus abnormality was a faint "stippling" of the choroidal pigment in the macular region, combined with a slight barring of the peripheral choroidal vasculature.

We assessed the patients' chromatic discriminations with the Farnsworth-Munsell 100-hue test, administered under a Macbeth Illuminant C easel lamp. Numerous errors were made throughout the spectrum, with the fewest errors at R and YYG. The patients showed somewhat better discrimination than typical achromats, who generally show a pattern of errors at and beyond the extremes of the graph. Measurements on a Nagel-type anomaloscope indicated that the patients were dichromatic or monochromatic, with the brightness even more depressed in the red than is found in protanopia.

B. Psychophysiological Measurements

Special equipment was constructed to enable us to measure luminosity and chromatic discriminations, dark adaptation, and visual acuity at various luminance levels.

1. Luminosity

Visual luminosity, or apparent brightness of various parts of the visible spectrum, was measured in a spectral comparator based upon a Hilger prism monochromator (Ref. 8). Radiometric measurements were made of the output of the device at various wavelengths, and cut-off filters were used to eliminate the spectral stray light which would otherwise have invalidated our measurements. This device was used first (Ref. 9) to measure luminosity functions on normal observers, and an optimal technique was developed. A standard source of 402 millimicrons was usually used and each subject was required to match other wavelengths of the spectrum in turn to this standard. Matches were always verified after adaptation to a white light of matched luminance, to minimize the possibility of chromatic adaptation. For normal subjects, it was found desirable to reduce color differences between the standard and comparison fields by use of a series of standards spaced across the spectrum. For the present patients, initial use of this procedure demonstrated that the patients had no difficulty matching all wavelengths against a single standard. Subsequent tests required the patients to match all wavelengths against the 402 millimicron standard. It was shown that the measurements obtained with the two methods were equivalent and they have been used interchangeably through the present report.

In our spectral comparator, the photometric comparator is of the concentric ring variety. In our device, the diameter of the inner field, which contained the standard, subtended 30 minutes. The diameter of the outer field, containing the spectral comparison, subtended 1 degree. Thus, the entire field will fall within the supposedly rod-free fovea centralis if the subject fixates the precise center of the comparator as he is instructed to do. The device incorporates an artificial pupil so that it is possible to compute the retinal illuminance produced by the standard when set at various radiance levels. A special lens is used (Ref. 10) to reduce difficulties due to the chromatic aberration of the human eye.

During the luminosity measurements, it was a simple matter to ask the patients if they could obtain a perfect match between various spectral stimuli and the 402 millimicron standard. Insofar as they were able to obtain a perfect match, it could be concluded that the patients were without chromatic discrimination.

Luminosity measurements were first made at the maximum intensity of the 402 millimicron standard. Utilizing the 1951 photopic luminosity values proposed by Judd (Ref. 11), the retinal illuminance was computed as 1.76 trolands for normal subjects. The data for the three patients are shown in Figure 1, together with average data on 10 normal subjects obtained under the same conditions.

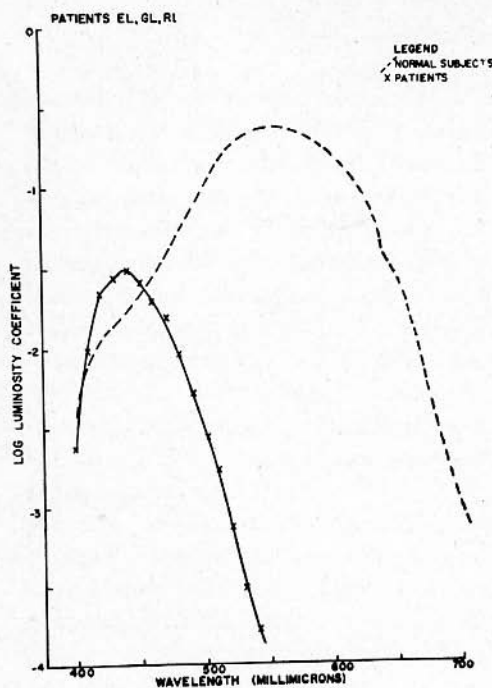


Fig. 2. Luminosity data for the patients and for normal subjects at a retinal illuminance of 1.76 trolands.

The luminosity coefficients are defined by the relation:

$$\text{Luminosity Coefficient} = \frac{N_{\hat{\lambda}}}{N_{\lambda}} V_{\hat{\lambda}}$$

where

$N_{\hat{\lambda}}$ = radiance of 402 millimicron standard;

N_{λ} = radiance of comparison wavelength; and

$V_{\hat{\lambda}}$ = CIE photopic luminosity coefficient
at 402 millimicrons.

By this definition, each luminosity coefficient should agree with the standard photopic luminosity value if the luminosity function is normal. The data obtained on our 10 normal subjects agree in general with the standard values, exhibiting a peak luminosity at about 555 millimicrons, and substantial luminosity between the limits of 400 and 700 millimicrons. In contrast, the data on our patients show a very narrow luminosity function with a peak at 440 millimicrons. The extent of difference between our patients and the normal subjects is perhaps concealed by the use of a logarithmic scale of luminosity coefficients. In fact, the luminosity function for our patients is less than one-thousandth normal at 540 millimicrons, and indefinitely less at longer wavelengths!

The patients were able to obtain perfect matches between all comparison stimuli and the 402 millimicron standard. Thus, we may conclude that they were functionally monochromatic under these conditions. For what it is worth, it should be reported that the patients all referred to the color of the photometric field as "blue". One of them stated that the field looked "like a blue sweater in a black room". Because of the narrowness of this luminosity function and the great resemblance of this curve to the z function in the C. I. E. colorimetric system, we have concluded tentatively that only one cone receptor system is present and that it may be designated the blue cone system. It was on this basis that these patients were originally designated blue monocone monochromats (Ref. 4).

Measurements were next made under identical conditions except that the intensity of the 402 millimicron standard was reduced so that it provided only 0.0754 trolands. The data obtained on the three patients under these conditions are presented in Figure 2. Data obtained on the 10 normal subjects at the lower luminance level are presented for comparison. Note that the luminosity function is now considerably broader than before, with the peak value at about 520 millimicrons. Thus, our patients exhibited a massive "anti-Purkinje" shift, the luminosity shifting to longer wavelengths as the luminance is reduced. It is still

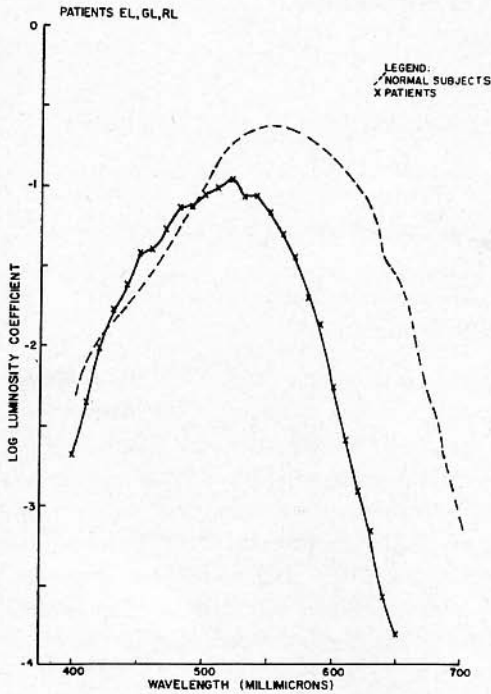


Fig. 2. Luminosity data for the patients and for normal subjects at a retinal illuminance of 0.0754 trolands.

apparent, however, that the luminosity function is far from normal. The patients were able to obtain perfect matches between all spectral comparison stimuli and the 402 millimicron standard. In this case, they reported the photometric field as "colorless". Thus, again they may be considered monochromatic.

The luminosity curve obviously bears some resemblance to the scotopic luminosity curve, which is known to be produced by the action of rod receptors. This resemblance creates at once an interesting possibility. It is to be remembered that our patients were instructed to make these luminosity settings with foveal fixation and that they believed they were following these instructions. If this is the case, and assuming the patients did not exhibit an unaccustomed nystagmus, the rod-like luminosity curve we have obtained was produced by receptors located in the fovea centralis.

A quantitative comparison of these data and the standard 1951 CIE scotopic luminosity function (Ref. 11) is presented in Figure 3. This comparison can only be made in relative terms so that all we can judge is the similarity of shape of the two luminosity curves. It is apparent that there is good agreement at wavelengths longer than 550 millimicrons but that there is a considerable depart-

ATYPICAL CONGENITAL ACHROMATOPSIA

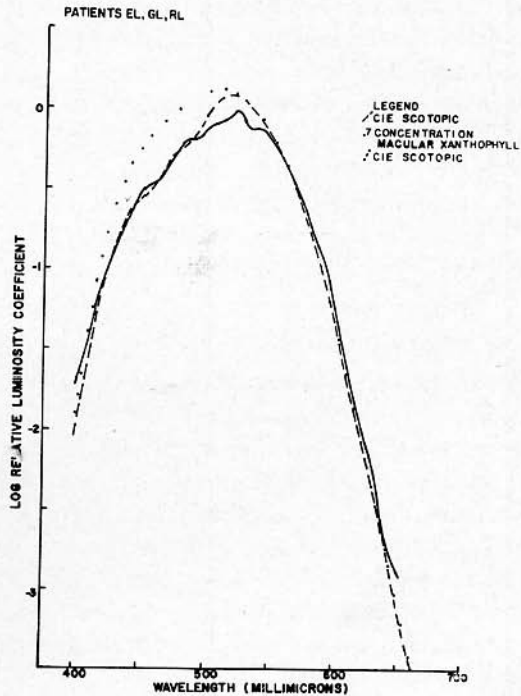


Fig. 3. Luminosity data for the patients at a retinal illuminance of 0.0754 trolands in comparison with theoretical curves.

ure between the curves in the range from 420 to 540 millimicrons. Initially, we postulated that the relative loss in luminosity exhibited by the patients at the short wave end of the spectrum reflected an inhibitory effect of blue cones.

While discussing these data with W. S. Stiles of the National Physical Laboratory, Teddington, Great Britain, an alternative explanation occurred to us. We begin by noting that Wald (Ref. 12) has reported the existence of an inert yellow pigment which exists in relatively high concentration in the macular retina, and which reduces the sensitivity of macular receptors in the short wave end of the spectrum. This pigment has an absorption spectrum which closely resembles crystalline leaf xanthophyll. It exists also in the peripheral retina, in concentrations no greater than one-fifteenth those found in the macular retina.

If we assume that this xanthophyll exists in the macular retinæ of our patients, then the luminosity function of the underlying receptors will be altered at the short end of the spectrum by absorption due to macular xanthophyll. We have postulated that rods exist in the fovea centralis of these patients, and that their absorption spectrum was indeed modified by the presence of macular xanthophyll.

It is necessary to construct theoretical scotopic luminosity curves modified by various concentrations of macular xanthophyll, since the concentration can easily vary among patients, and is known to vary with retinal location. The dashed curve in figure 3 represents modification of the CIE scotopic luminosity function by a concentration of macular xanthophyll 70% as great as that reported by Wald. We believe the agreement between this modified luminosity function and our luminosity data is quite good. Thus, we feel that we have probably established the existence of a foveal rod luminosity function, to be distinguished from the usual scotopic curve, which may well be considered a peripheral rod luminosity function.

It is known that the standard scotopic luminosity function closely resembles the absorption spectrum of rhodopsin. This is not surprising, since the concentration of macular xanthophyll is known to be low at the retinal locations used to determine the scotopic luminosity function. The fact that the concentration of macular xanthophyll presumably decreases gradually from a maximum at the fovea would seem to imply that there should be various scotopic luminosity curves for different retinal locations, modified by different concentrations of macular xanthophyll. If this is reasonable, then the precise shape of the scotopic luminosity curve can perhaps be used to determine to some extent the retinal location used by the patients in making psychophysiological measurements. We shall have occasion to make use of this notion in subsequent analysis of our data.

After discovering the presence of the blue cone and foveal rod receptor systems, each producing monochromatic vision at one luminance level, we measured luminosity curves at various luminance levels between the two values originally studied. We generally obtained what must be regarded as mixed luminosity curves, and we found a dichromatic color system in each case.

The solid curve in Figure 4 represents an example of the mixed curves. The data are for patient EL at a retinal illuminance level of 0.508 trolands. (It is necessary to present individual data in this case, since the mixed curves obtained by the three patients at a given luminance level differ to such an extent that averaging would not be advisable.) When these mixed curves were obtained, the patients were able to make perfect matches between the spectral lights and the 402 millimicron standard only from about 400-450 millimicrons. Otherwise, the spectral light was reported as chromatically different from the standard. In general, the patients reported a "neutral zone" in the region from about 450 to 510 millimicrons, in which the spectral light was reported as white or cream colored. Spectral lights beyond 510 millimicrons were invariably called "yellow". This result was obtained both when the patients were matching these wavelengths with the 402 millimicron standard and when they observed unknown wavelengths

ATYPICAL CONGENITAL ACHROMATOPSIA

singly and without reference to other color stimuli. Thus, at the luminance levels at which mixed luminosity curves were found, the patients clearly exhibited a dichromatic color system. It seems reasonable to suppose that the dichromatic color system was based upon blue cone and foveal rod receptor systems, since the dichromacy occurred at luminance levels at which both systems might reasonably be expected to be operative.

The constructions in Figure 4 are intended to demonstrate the reasonableness of this postulation. The blue cone curve shown in Figure 1 and the theoretical foveal rod curve shown in Figure 3 have been plotted in comparison with the mixed luminosity curve. These curves have been arbitrarily adjusted for good fit with the mixed luminosity curve, with the result shown in Figure 4. It is apparent that the mixed curve has sufficient width to be composed of blue cone and foveal rod mechanisms as postulated. It is also apparent that the form of the mixed curve conforms reasonably well to the blue cone and foveal rod curves at the ends of the spectrum where only one receptor system would be expected to be operative. The appearance of the mixed curve in the region from about 450 to 510 millimicrons could be interpreted as due to some kind of facilitation of the blue cone and foveal rod systems upon each other. The mixed luminosity

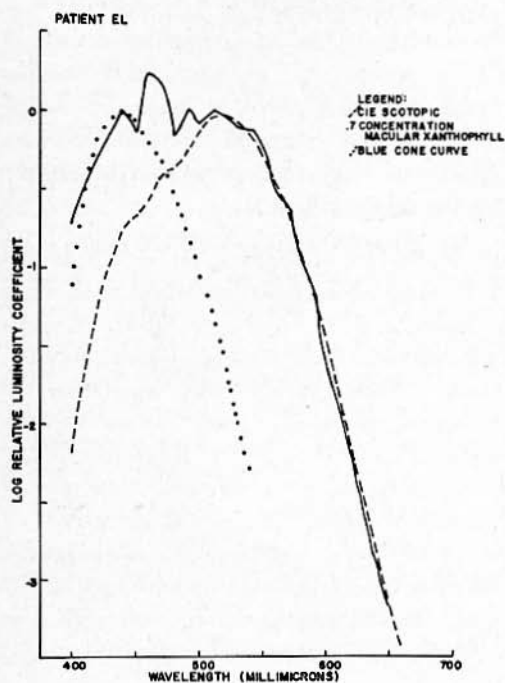


Fig. 4. Luminosity data for one patient at a retinal illuminance of 0.508 trolands in comparison with theoretical curves.

curves obtained on these patients almost invariably reveal this evidence for facilitation in the spectral region where the two receptor systems are presumably of about equal sensitivity. We cannot consider this point proved, of course, but the data can perhaps be interpreted in this manner.

We believe the luminosity data have revealed the presence of blue cone and foveal rod receptor systems in the foveal or near-foveal retinae of our patients, and have suggested that chromatic discriminations can be made whenever both these systems have about equal sensitivity. The implications of these results for color vision theory are of particular interest and we are continuing efforts to develop a theoretical model by means of which such a color discriminative system can operate.

2. Dark adaptation measurements

Foveal and peripheral dark adaptation were measured in a recording dark adaptometer constructed along the general lines of the instrument described by McLaughlin (Ref. 13). Basically, the device consists of a pre-adapting field of controllable luminance and duration, and a flashing test of continuously variable intensity which the subject maintains at a threshold level during dark adaptation by adjustment of a knob control linked to a circular neutral photometric wedge. For the present studies, the test light subtended 15 minutes of arc, and was presented intermittently during the entire test period in the following sequence: 2.4 seconds on, 1.2 seconds off, 2.4 seconds on, etc. Viewing was binocular and natural pupils were utilized. The experimental sequence consisted of a 900 second initial period of dark adaptation to wash out effects of unknown previous adaptation, followed by a period of 500 second light adaptation to a luminance of 1280 foot-lamberts, followed by the dark adaptation test.

For tests of "foveal" adaptation, the test target was presented in the center of a diamond configuration composed of four white fixation lights. Each light subtended 4.6 minutes of arc, and the distance from the center of the configuration to the center of each light was 48 minutes. The test subject controlled the intensity of the fixation lights from time to time during dark adaptation by adjustment of a rheostat so that the lights remained clearly visible without becoming bright enough to have a deleterious effect upon the threshold of the test light. The results obtained by the patients with a white test stimulus are presented in Figure 5. Here we have plotted the logarithm of the luminance of the test stimulus required for it to be at threshold level during adaptation to darkness. Data on thirteen normal subjects are present for comparison. (The data are not precisely comparable, since the light adaptation luminance was 1155 foot-lamberts rather than 1280. Ancillary experiments suggest, however,

that this difference in the pre-adaptation luminance does not affect the dark adaptation curve appreciably). It is obvious that the patients exhibit an initial deficiency in adaptation but that after some 12 minutes in the dark, they have greater sensitivity than normal subjects. The initial deficiency in sensitivity is quite large, the threshold being as much as 50 times greater than normal. The dark adaptation curve obtained on the patients appears to have the change in slope usually attributed to a transition from cone to rod receptors (Ref. 14).

In order to identify the photoreceptor systems responsible for adaptation at different times in the dark, we utilized what might be called "spectrophysioanalysis. The basic idea is that we can identify receptor mechanisms responsible for visual function from data obtained with stimuli of different spectral quality, upon the basis of the known action spectra of the receptor systems. This idea has utilized previously (Ref. 15, 16, 17, 18) but deserves considerably more attention. In the present instance, we repeated our foveal dark adaptation measurement after insertion of a Wratten N^o 47B colored filter in the test stimulus beam. This filter has a peak transmittance at about 430 millimicrons and a half width of about ± 20 millimicrons. Now, we can compute the effective optical density of the N^o 47B filter, assuming a given photoreceptor sensitivity function, and test the extent to which this computed density brings the dark adapta-

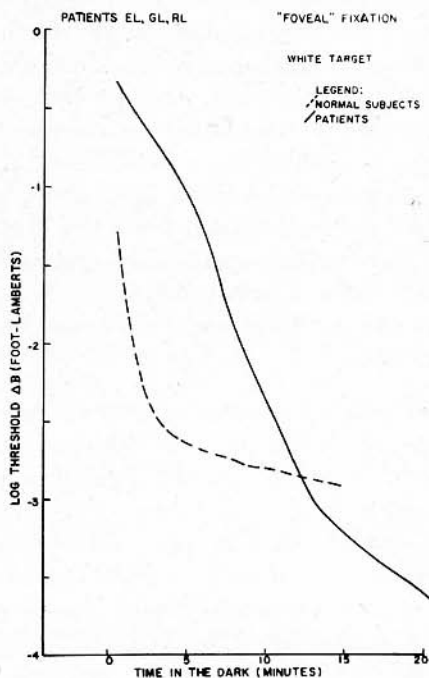


Fig. 5. "Foveal" dark adaptation data for the patients and for normal subjects with

tion function obtained with blue light into agreement with the original dark adaptation function obtained with white light. This analysis necessitates accurate radiometric data on the energy distribution of the light source used in the test stimulus. Measurements of the radiant energy emitted at various wavelengths by the test target system were made by our associate B.S. Pritchard, utilizing a double grating monochromator previously calibrated against a standard lamp of known color temperature. It is also necessary to take account of the fact that the so-called neutral wedge used to adjust the test light to threshold, and other neutral filters used to reduce the test light to near threshold, are spectrally selective and hence change their calibration when the blue filter is inserted. We measured the spectral transmittance of the various neutral materials with the double grating monochromator and computed the densities of these materials when the incident light was modified by insertion of the blue filter. The density changes were quite small but appropriate allowances have been made in the data.

Initially, we tested the assumption that either the ordinary scotopic or photopic receptor systems might have been involved. The density of the blue filter was found to be 1.45 for the standard scotopic function and 2.59 for the Judd 1951 photopic function. The test of the assumption that the entire foveal dark adaptation function is provided by a receptor system having the sensitivity of the CIE scotopic function consists of computing the values of threshold AB on the assumption that the effective density of the blue filter is 1.145, and comparing the dark adaptation data for the blue and white test lights after making this correction. This procedure was followed, with the results shown in Figure 6. The dashed curve represents the data obtained with the test light, assuming that the density of the blue filter was indeed 1.45. The solid curve represents the data obtained with the white test light, as shown previously in Figure 5. We consider the data for the blue and white test stimuli after seven minutes in the dark to be in excellent agreement. This presumably means that the receptor system responsible for dark adaptation after seven minutes has the wavelength sensitivity of the CIE scotopic function. Presumably, this identifies the receptors as peripheral rods.

During the first seven minutes of dark adaptation, the blue light curve falls considerably below the white light curve. This presumably means that a receptor system more sensitive to blue light than peripheral rods is at work. It naturally occurred to us that the blue cone mechanism, previously identified in the luminosity data obtained at high luminance, was responsible for this portion of the dark adaptation curve. In order to make a quantitative check of this assumption, we computed the density of the blue filter for blue cone receptors, utilizing the average luminosity curve presented in Figure 1. The density was found to be

ATYPICAL CONGENITAL ACHROMATOPSIA

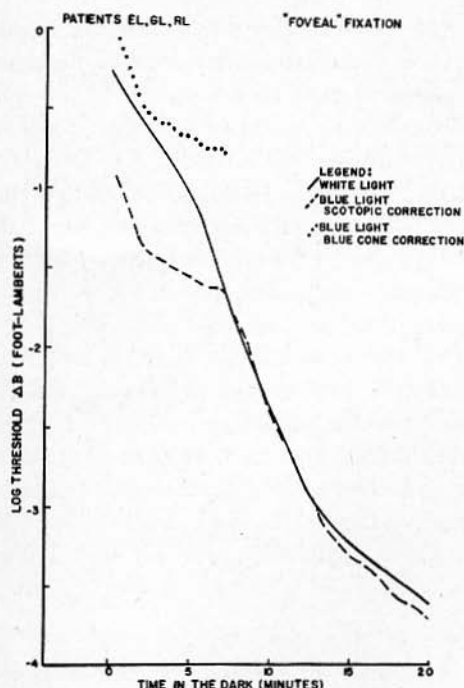


Fig. 6. Comparisons of "foveal" adaptation data obtained by the patients with white and blue test lights.

0.53. We must also remember that the neutral filters used during the blue light dark adaptation test have a different effective density for blue cone receptors than for peripheral rod receptors. It is possible to allow for this effect by computation. Allowances for this effect, and for the blue cone density of the blue filter, have been made in the blue light adaptation data obtained during the first seven minutes. The corrected data are plotted in Figure 6 as closed circles. The blue light data now fall somewhat above the white light data, indicating that the receptors responsible for this portion of the dark adaptation process are somewhat less blue-sensitive than the blue cones. It is perhaps most reasonable to suppose that both blue cones and rods are involved in the adaptation process during the first seven minutes in the dark. The distance of the white light curve from the dotted and the dashed blue light data presumably reveals the relative contribution of the rods and blue cones at various moments. In general, the blue cones have the largest relative effect during the first three minutes, as might be expected. From these data it seems safe to assume that "foveal" dark adaptation represents the joint action of blue cones and rods, with the blue cones predominating during the first three minutes, and the rods contributing with increasing significance, until after seven minutes the function appears entirely dependent upon rods.

There is one small lack of consistency in our analysis which has not as yet been pointed out. The luminosity data shown in Figure 3 were used to demonstrate that foveal rod photoreceptors do not have the CIE scotopic function, but rather a modification of this function due to the absorption of macular xanthophyll. It would seem reasonable that we should use the modified CIE scotopic function in computing the effective density of the blue filter used to adjust the blue light data of Figure 6, rather than the CIE scotopic function which we used, since the patients were supposed to be using foveal fixation. The effective density of the blue filter for the CIE scotopic function modified by .7 concentration of macular xanthophyll is 1.67. This means that the blue light data should all be lowered in Figure 6 by .22 log units, if we assume that foveal rods are involved. It is apparent that this adjustment will markedly worsen the agreement between the blue and white light data obtained after seven minutes in the dark. It appears that this identifies the receptors responsible for the later part of dark adaptation as peripheral rather than foveal rods. Or more precisely, it appears that the rods responsible for adaptation following seven minutes in the dark are not overlaid with any appreciable amount of macular xanthophyll. This probably means that the patients shifted their fixation toward the peripheral retina during the latter phases of dark adaptation.

Dark adaptation measurements were also made by the patients, utilizing peripheral fixation. A single fixation point was provided which was separated by 15 degrees from the center of the test light. The fixation light was located to the right of the test light along the horizontal meridian. With binocular fixation, the test light fell on the blind spot in the left eye, and upon the temporal retina of the right eye. This procedure has the advantage that the patients use binocular fixation, which we have found more stable in the dark than monocular fixation, without having the disadvantage of our using a binocular peripheral test. (It is not uncommon for the two peripheral retinæ to vary considerably in sensitivity, which produces a rather ambiguous test result. Our foveal test was of course binocular, but we have not found there to be important sensitivity differences between the two foveal retinæ). The subjects continually adjusted the peripheral stimulus to threshold, carefully looking at the fixation light. They also adjusted the intensity of the fixation light from time to time to maintain it always clearly visible without allowing it to become bright enough to produce a deleterious effect upon the visibility of the test light.

The dark adaptation curve obtained with white light is presented as the solid line in Figure 7. For comparison, data on 13 normal subjects are presented as the dashed line. Unfortunately the data are not entirely comparable since the pre-adaptation luminance was 1078 foot-lamberts for the normal subjects and 1280

ATYPICAL CONGENITAL ACHROMATOPSIA

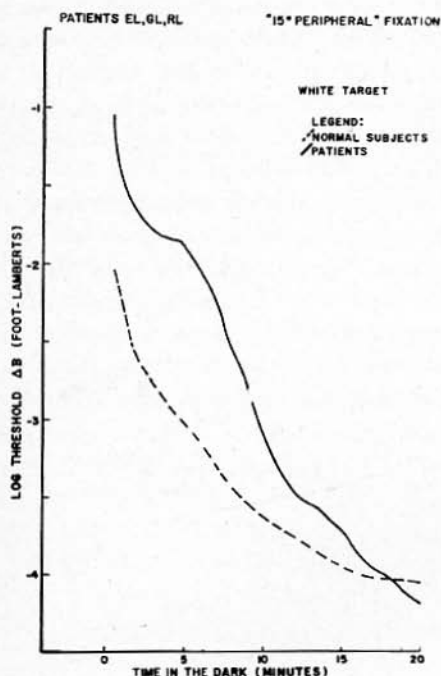


Fig. 7. 15° peripheral dark adaptation data for the patients and for normal subjects with a white test light.

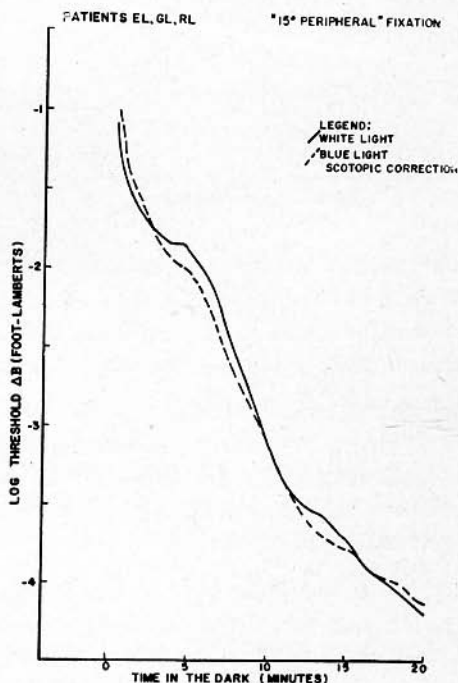
for the patients. Ancillary experiments suggested that this difference in pre-adaptation luminance should have only a negligible effect on the data. It should be noted that peripheral dark adaptation data vary so much from individual to individual that the patients' dark adaptation curve may be within normal limits.

The change in slope evident in the patients' data suggests the transition from one to a second receptor mechanism. In fact, various authors have concluded on more basis than the appearance of this curve that rod and cone photoreceptor systems are functioning during the dark adaptation process. As we shall see, this conclusion is not correct.

To identify the receptors operating during dark adaptation, we measured adaptation with a blue test stimulus, as before. The blue light data were initially corrected by the CIE scotopic function on the assumption that peripheral rods were involved, and compared with the white light data. The results are shown in Figure 8, where the corrected blue light data are represented by the dashed line. We consider the agreement between the two curves to be quite good over their entire course. This means that the receptors responsible for the entire course of peripheral dark adaptation have the wavelength sensitivity of peripheral rods.

The two curves in Figure 8 agree in exhibiting two segments with a change in slope after some five minutes in the dark. We cannot interpret this as due to receptor systems of different wavelength sensitivity, since the curves agree throughout. We must speculate that two receptor systems seem to be operative, with the same wavelength sensitivity, but with a different rate of dark adaptation. There may be two photosensitive pigments with different regenerative dynamics. Or, the two receptor systems may differ in terms of their neural connections. Receptors functioning primarily through polysynaptic neural networks may adapt at a different rate from receptors functioning primarily through directly coupled neural networks. Further research may well be expected to provide a basis for identifying the essential difference between the systems responsible for the two segments of these curves. It is interesting to note, of course, that there is very little evidence of two similar segments in the peripheral dark adaptation functions of the normal subjects.

Fig. 8. Comparison of 15° peripheral dark adaptation data obtained by the patients with white and blue test lights.



We can reject the idea that the receptors responsible for the peripheral dark adaptation curves of the patients are rods whose sensitivity has been modified by macular xanthophyll. As before, if we assume what we have called foveal rods to be responsible for the adaptation curves, we must adjust the blue light data

downward by an additional .22 log units, which will clearly worsen the agreement between the blue and white light data. This result conforms to what might reasonably expect, since there should not be any macular xanthophyll at a location 15 degrees from the fovea.

3. Visual acuity measurements.

Visual acuity was measured at different luminance levels, utilizing a letter chart and projected illumination, which could be varied in level by means of neutral filters. The charts utilized involved letters developed by Sloan (Ref. 20). The subjects hand-held a 2 mm. artificial pupil over one eye, the other eye being covered with a translucent patch. He walked toward and away from the chart, until a given criterion letter was considered to be just resolvable. The letter selected was K. By selection of a suitable size letter on the Sloan Chart, the subjects were usually prevented from having to approach to less than 5 feet from the chart. Three measurements were made at a given illumination level, then the level was changed and three new settings were made. Measurements were usually made at luminance levels which were gradually reduced from highest to lowest, then a second set of measurements were made at luminance at luminance levels gradually increased from lowest to highest. Luminance levels were studied in 0.5 log unit steps. Values of retinal illuminance were computed on the basis of the measured luminance of the test chart and the area of the artificial pupil.

Data for the patients are presented in Figure 9 for white light illumination. Each patient had been refracted in the Department of Ophthalmology and wore the indicated ophthalmic correction. No corrections exceeded three diopters. For comparison, data on two normal subjects are also presented. These data exhibit the change in slope usually attributed to the operation of rod and cone mechanisms at different luminances. Note that whereas the patients and the normals have essentially equal acuity at illuminance levels below 0.1 trolands, the acuity of the patients falls far below normal at higher luminances. The maximum acuity achieved by the patients was 20/63, whereas the normal subjects reached an acuity of 20/16. The change in slope of the acuity function of the patients, which occurs at about 20 trolands, is particularly interesting.

Acuity was measured also with blue light, in order to explore the significance of the duplex nature of the acuity function and to identify the receptors responsible for the two segments. The Wratten N^o 47B filter was either placed in the projector or hand-held by the subject. Measurements were first made to investigate whether or not a change in ophthalmic correction should be made to compensate for possible effects of ocular chromatic aberration. Perhaps because of the reduced acuity of these patients, it was not possible to show any improvement

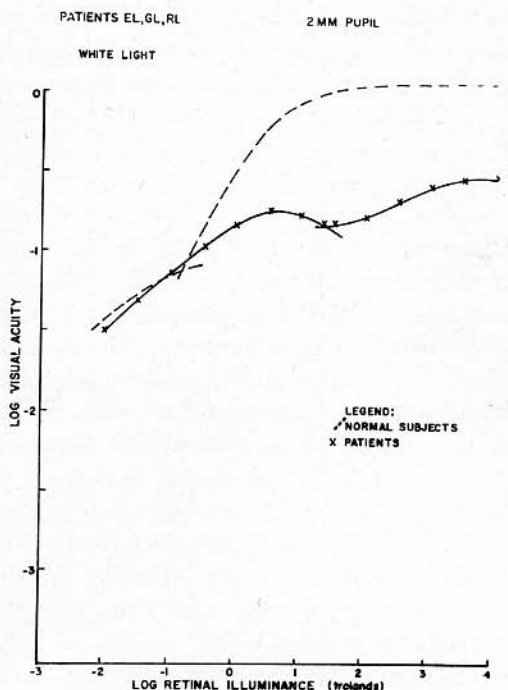


Fig. 9. The luminance function of acuity for the patients and for normal subjects with a white test light.

in the acuity function measured with blue light when changes in the ophthalmic correction were used. Hence, the patients' usual ophthalmic corrections were worn.

The acuity data obtained with blue light are plotted in Figure 10, together with the data obtained with white light. It was initially assumed that peripheral rods were responsible for the acuity data overall. Accordingly, the blue light acuity data were adjusted along the retinal illuminance scale to correspond to a density of 1.37 for the blue filter. (This density was computed on the basis of the CIE scotopic function. It differs from the value of 1.45 reported in connection with the dark adaptation data because of a difference in the radiant emission spectrum of the light sources used in the two cases).

It is apparent from Figure 10 that there is excellent agreement between the white light and the corrected blue light data up a level of 10 trolands. Above that level, the blue light data fall systematically above the white light curve.

The blue light data taken alone show evidence of a small change in slope at about the 10 trolands level. Accordingly, it was assumed that the data for illuminances below 10 trolands are produced by one receptor system, those above 10

ATYPICAL CONGENITAL ACHROMATOPSIA

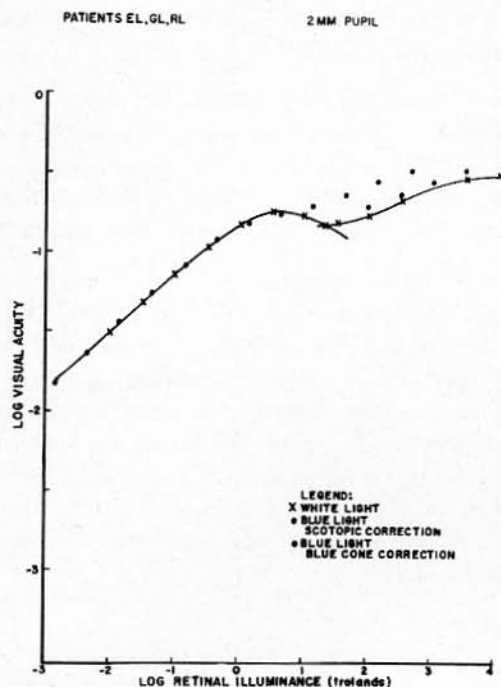


Fig. 10. Comparisons of the luminance function of acuity for the patients with white and blue test lights.

trolands level being produced by a second system. The agreement between the white and corrected blue light data below 10 trolands identifies the receptors system responsible for this portion of the acuity function as peripheral rods.

We next supposed that the upper section of the acuity function was produced by blue cones. This assumption was tested by adjusting the blue light acuity data at levels above 10 trolands to correspond to a density of the N^o 47B filter equal to 0.53, computed by utilizing the blue cone luminosity function shown in Figure 1. The points so adjusted are shown in Figure 10 to agree better with the white light data than before. This result probably justifies our assumption that blue cones are responsible for the upper segment of the acuity curve, although agreement is in this case somewhat less convincing than in the other instances reported here.

The fact that the corrected blue light data points represent higher acuity than the white light data may be interpreted as follows. Rods may well normally "inhibit" blue cones, at illuminance levels at which the two receptor systems possess nearly equal sensitivity. The presence of this inhibition can explain the deep cusp found in the white light acuity data. Use of the blue filter minimizes the

role of peripheral rods with respect to blue cones. With blue light, the acuity in the region at which the blue cones are operative is therefore enhanced.

This explanation gains credence through an additional fact. The patients reported that the use of the blue filter removed "snow" from the acuity task. This "neural noise" could be produced by rods stimulated to the point of satiation. Another generally confirmatory fact is that the acuity function obtained in blue light fails to show the deep cusp exhibited so clearly in the acuity data obtained with white light.

As in the case of "foveal" dark adaptation, we find that we cannot accept the view that acuity at low luminances is due to what we have called foveal rods. If the blue light data are corrected on the assumption that the scotopic luminosity curve is modified by the presence of macular xanthophyll, they must be moved to the left by .22 log units which greatly worsens the agreement between the white and corrected blue light data. Thus, we must conclude that our patients performed their acuity task at low luminance with rod receptors unaffected by macular xanthophyll. This presumably means that the patients used eccentric fixation during the acuity measurements at low luminance.

SUMMARY AND CONCLUSIONS

Our investigations have led to the identification of a blue cone receptor mechanism never before found completely in isolation. As will be shown in a forthcoming publication, (Ref. 19) this mechanism closely resembles the hypothetical blue mechanism inferred from color mixture data and the blue mechanism isolated functionally by selected conditions of observation. This finding certainly emphasizes the value of psychophysiological study of clinical patients for the development of visual theory.

We have also isolated what we have called foveal rods, and shown that the action spectrum of this receptor system is modified by the absorption spectrum of macular xanthophyll. We have also shown that the action spectrum of various visual functions can be used to identify to some extent the fixation utilized, since the action spectra of foveal and peripheral rods are sufficiently different for differentiation to be made between them.

We have shown that chromatic discriminations can be made with a dichromatic color system based upon blue cones and foveal rods. This demonstration that chromatic discriminations are not restricted to cone photoreceptors is also important to color vision theory.

We have shown that blue cones and foveal rods can summate their luminosity contributions, at least under conditions in which the contributions made by the two systems are of approximately equal magnitude.

We have shown that "foveal" dark adaptation in the patients studied consists initially of contributions of blue cones and rods, but that peripheral rods are responsible for adaptation after seven minutes. In peripheral dark adaptation, there are two sets of peripheral rod receptors with different time courses of adaptation.

In acuity at low luminances, our patients used peripheral rods. At high luminances, acuity is mediated by blue cones. At intermediate luminances, acuity is mediated by blue cones and rods, and the rods appear to reduce the acuity which otherwise be possible with the blue cones, perhaps by creating "neural noise".

The complex way in which the patients presumably sometimes used foveal and sometimes peripheral rods, in spite of our best efforts to control their fixation, suggests that future efforts should be made to record the precise locus of ocular fixation during various psychophysiological measurements on patients of this type.

Columbus, 8, Ohio

REFERENCES

1. FRANCOIS, J., and G. VERRIEST, Contribution A L'etude Des Dyschromatopsies Congénitales A Symptomes Intermédiaires Entre Ceux Des Systèmes Dichromatiques Classiques Et Ceux De L'Achromatopsie Typique: Observation Personelle Et Revue De La Littérature. *Annales D'Oculistique*, **192**, 81-120 (1959).
2. SLOAN, L. L., Congenital Achromatopsia: A Report of 19 Cases. *J. Opt. Soc. Amer.*, **44**, 117-128 (1954).
3. JAEGER, W., Typen Der Inkompletten Achromatopsie. *Ber. dtch. ophthal. Ges.*, **58**, 44-47 (1953).
4. BLACKWELL, H. R., and O. M. BLACKWELL, "Blue Mono-cone Monochromacy": A New Color Vision Defect (Abstract). *J. Opt. Soc. Amer.*, **47**, 338 (1957).
5. HSIA, Y., and C. H. GRAHAM, Spectral Luminosity Curves for Protanopic, Deuteranopic, and Normal Subjects. *Proc. Nat. Acad. Sciences*, **43**, 1011-1019 (1957).
6. HSIA, Y., S. HECHT, and S. SHLAER, The Acuity of Dichromats. Pp. 1. Proceedings of a Symposium on the Present Status of Fundamental Research in Vision. Office of Naval Research, U. S. Navy (1950).
7. BRINDLEY, G. S., The effects on color vision of adaptation to very bright lights. *J. Physiol. (London)*, **122**, 332-350 (1953).
8. PRITCHARD, B. S., The Design and Calibration of a Spectral Comparator for Measuring Visual Luminosity Functions. University of Michigan, Engineering Research Institute Report 2144-336-T, Pp. 35 (1958).
9. BLACKWELL, H. R., and O. M. BLACKWELL, Luminosity Functions Obtained with Different Methods and Different Viewing Conditions. University of Michigan Research Institute Report 2144-344-T, Pp. 17 (1959).

10. VAN HEEL, A. C. S., Correcting the Spherical and Chromatic Aberration of the Eye. *J. opt. Soc. Amer.*, **36**, 237-239 (1946).
11. Proceedings of the 12th Session, International Commission on Illumination, Stockholm, 1951. Volume I.
12. WALD, G., Human Vision and the Spectrum. *Science*, **101**, 653-658 (1945).
13. MacLAUGHLIN, S. C., Jr., An Automatic Recording Visual Adaptometer. *J. Opt. Soc. Amer.*, **44**, 312-314 (1954).
14. HECHT, S., Rods, Cones, and the Chemical Basis of Vision. *Physiol. Rev.*, **17**, 239-290 (1937).
15. HECHT, S., S. SHLAER, E. L. SMITH, C. HAIG, and J. C. PESKIN, The Visual functions of the Complete Color-blind. *J. Gen. Physiol.*, **31**, 459-475 (1948).
16. BROWN, J. L., Review of the Cone-to-Rod Ratio As a Specification for Lighting Systems. *Illumin. Eng. N. Y.*, **51**, 577-584 (1956).
17. SLOAN, L. L., Photopic Receptors of the Typical Achromat (Abstract). *J. Opt. Soc. Amer.*, **47**, 1052 (1957).
18. BLACKWELL, H. R., and O. M. BLACKWELL, "Blue Mono-Cone Monochromacy": Comparisons with Rod Monochromacy (Abstract). *J. Opt. Soc. Amer.*, **49**, 499 (1959).
19. BLACKWELL, H. R., and O. M. BLACKWELL, The Blue Cone System Isolated in Atypical Congenital Achromatopsia (submitted to the *J. Opt. Soc. Amer.*).
20. SLOAN, L. L., W. M. ROWLAND, and A. ALTMAN, Comparison of three Types of Test Target for the Measurement of Visual Acuity. *Quart. Rev. Ophthalmol.* **8**, 4-16 (1952).

CHIAZZA DI BITOT SENZA AVITAMINOSI

PER

CARONES A. V., M. D.

Italia

Il problema della comparsa della chiazza di Bitot non accompagnata da una avitaminosi è stato oggetto di numerosi indagini in questi ultimi anni e parecchi sono stati i casi studiati e descritti specialmente nelle regioni tropicali e sub-tropicali (Palmer ad Assam, Sie Boen Lian a Java, Aycroyd e Col. in India, Nichols e Col. a Ceylon, Wilson in Egitto) ma anche se pure in minor numero in Europa (Fronimopoulos e Cofinas, Bouzas in Grecia, Gorduren e Orgen in Turchia, terrien e Blum in Francia).

Circa due anni fa si è presentato alla nostra osservazione un caso che presentava l'aspetto clinico della chiazza di Bitot non accompagnata da alcun altro segno di avitaminosi nè oculare nè generale.

Descriviamo brevemente questo caso e gli esami effettuati.

M. Guido —anni 35— coniugato—.

All'età di 9 anni in seguito ad incidente automobilistico subì un grave trauma all'occhio S. che dovette essere enucleato. L'occhio D. è sempre stato bene fino al Dicembre del 1955 quando venne visitato da uno di noi per la prima volta per un arrossamento delle congiuntive ed un annebbiamento visivo dovuto, a detta del paziente, ad una intossicazione da pesce guasto. L'esame obbiettivo mise in evidenza una blefarite squamosa ed una lieve congiuntivite di tipo allergico. Il Visus era di 10/10 s. c. di lenti. Il paziente è stato rivisto nel Luglio 1956 per una piccola causticazione sul margine palpebrale inferiore D. In questa occasione venne constatata per la prima volta la presenza di una chiazza di xerosi congiuntivale in corrispondenza della pinguecola nasale. Da notarsi che nella primavera di quello stesso anno aveva soggiornato per due mesi in una Clinica di Davos per tubercolosi renale e che nel 1950 aveva soggiornato a Davos per un periodo assai più lungo per una forma di tubercolosi polmonare.

La chiazza di xerosi si presentava in corrispondenza della rima palpebrale del lato nasale come una formazione bianca di forma grossolanamente triangolo-ova-

lare con la base verso il limbus e con una superficie spugnoso-saponosa (Fig. 1). Null'altro si rilevava a carico del bulbo oculare che si presentava normale in tutto e per tutto compreso il campo visivo ed il senso luminoso. I comuni esami di laboratorio non misero in evidenza alcunchè di anormale ed anche il tasso di vitamina A nel sangue risultò nei limiti del fisiologico (2-3 UI. per CC. di sangue). Test di Schirmer per la secrezione lacrimale nei limiti del normale (30 mm.).

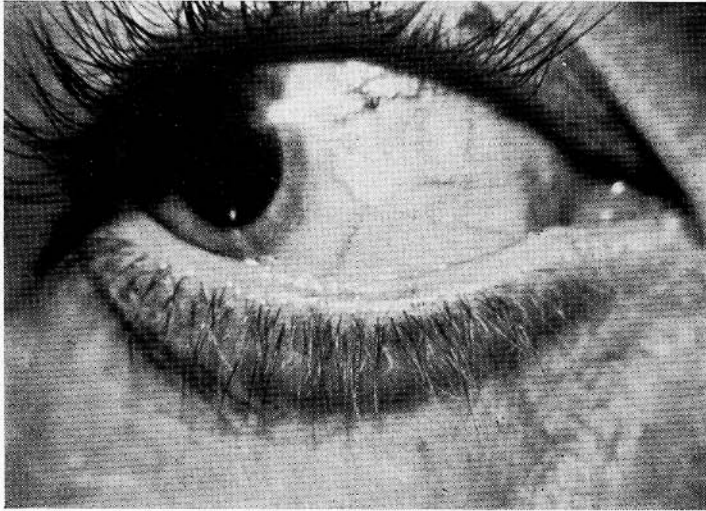


Fig. 1

Un intenso trattamento a base di Vit. A per via generale e locale non portò ad alcuna modificazione della chiazza di xerosi che ancora nel Maggio 1957 risultava invariata. Pure invariato era lo stato blefaro-congiuntiviteico con abbondante fuoruscita di secrezione dalle ghiandole dei margini palpebrali alla compressione. Il paziente si lamentava di un notevole fastidio e di sensazione di corpo estraneo nell'occhio.

Un successivo trattamento a base di Antibiotici associati a Cortisone ed Epatoprotettori (Litrison) non diede alcun vantaggio e nel mese di Novembre dello stesso anno il paziente si ripresentò assai preoccupato per l'aumento della chiazza, alla quale attribuiva un notevole peggioramento della sintomatologia subbiettiva. Si amentava infatti di dolore e fotofobia alla fissazione proungata (guida dell'auto, lettura) nonchè di diminuzione visiva. L'esame obbiettivo rilevò soltanto un notevole aumento delle dimensioni della chiazza di xerosi mentre invariato rimaneva il quadro congiuntivale.

In quest'occasione si è proceduto al raschiamento con un spatola della massa xerotica che si è lasciata facilmente asportare. Ma malgrado un ulteriore intenso ciclo di cure a base di Vit. A per via locale e generale ed estratti epatici con complesso B per via generale due settimane dopo la chiazza di xerosi si era riformata esattamente come prima.

Il paziente è stato successivamente rivisto nel Marzo 1958 sempre con la stessa sintomatologia locale alla quale si è aggiunto dal lato generale un eritema cutaneo. Soffriva anche di rinite da fieno che combatteva con giovamento con un trattamento a base di Cortico-steroidi per via generale, che tollerava male, e con Antistaminici di sintesi.

In quest'occasione venne fatta una ulteriore serie d'indagini radiologiche e di laboratorio, nonchè i test di funzionalità epatica, che risultarono tutti negativi.

Una intensa cura a base di Cortisone ad alta concentrazione, questa volta praticata seriamente (il P. si cura di norma saltuariamente ed irregolarmente) ha migliorato le condizioni locali e subiettive ma la chiazza di xerosi persisteva nella sua tendenza ad aumentare.

Questo continuo progresso delle dimensioni della zona di xerosi preoccupava talmente il paziente da creargli un vero e proprio stato ansioso aggravando la sintomatologia subiettiva locale. Si ritenne pertanto opportuno procedere all'asportazione chirurgica che venne eseguita nel Settembre dello stesso anno. La perdita di sostanza venne compensata con una plastica congiuntivale per scorrimento previa leggera diatermocoagulazione superficiale della sclera in corrispondenza della chiazza e ancoraggio sclero congiuntivale della sutura. Alla prima medicazione venne notato che la congiuntiva si era retratta per cedimento dei punti a causa di una necrosi della sclera, che presentava una perdita di sostanza più o meno ovalare a margini netti che lasciava esposta la corioide sottostante. Ripetuta la plastica allo scopo di ricoprire le perdite di sostanza, si ripeté il medesimo fatto per cui si attese la guarigione spontanea che avvenne in una diecina di giorni senza lasciare alcun segno (Fig. N° 2). Da allora non vi è stato più accenno a riformarsi della chiazza e tutta la sintomatologia subiettiva accusata dal paziente è scomparsa immediatamente confermando a nostro parere la natura psichica dei disturbi.

L'esame Istologico del tessuto asportato diede il seguente reperto: l'epitelio congiuntivale è pavimentoso stratificato, a tratti ispessito e fornito di uno strato corneo, talora sensibilmente alto, al di sotto del quale si può osservare un evidente strato granuloso. Negli strati inferiori si riscontrano modesti fenomeni di acantolisi ed infiltrazione fra le cellule epiteliali, di minuti elementi parvicellulari. Il connettivo sotto epiteliale è costituito da fasci di fibre collagene, ora compatte,

ora lasse, imbibite, le quali, negli strati superficiali, sono spesso in preda a fenomeni regressivi (omogeneizzazione basofila). Nelle vicinanze del rivestimento epiteliale specie laddove si sono osservati fenomeni di corneificazione, si trovano numerosi e cospicui focolai d'infiltrazione costituiti da elementi prevalentemente parvicellulari. I vasi di tipo capillare, sono molto dilatati e ripieni di corpuscoli ematici. A 10 mesi dall'asportazione della chiazza, la congiuntiva, si presenta, in corrispondenza della preesistente formazione del tutto normale ed il soggetto non accusa alcun disturbo.

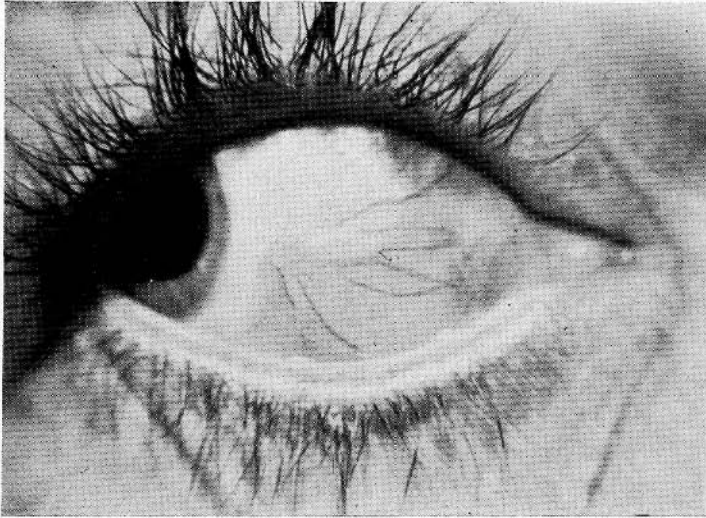


Fig. 2

Si pone ora il problema dell'insorgenza della chiazza di Bitot quando non si può rilevare nè emeralopia, nè lesioni congiuntivali o corneali.

Ricordiamo che cronologicamente le lesioni congiuntivo-corneali nella xerofthalmia legata a deficienze di Vitamina A, possono sintetizzarsi Sec. Appelmans in: 1º, Prexerosi; 2º, Xerosi congiuntivale; 3º, Xerosi corneali; 4º, Cheratomalcia. La chiazza di Bitot appare tardivamente, generalmente al II - III stadio, e va distinta dalla xerosi oltre che per l'aspetto, per le caratteristiche diverse nel comportamento in rapporto all'avitaminosi. La xerosi è un fenomeno reversibile, mentre la chiazza di Bitot è irreversibile. La osservazione di guarigione delle chiazza di Bitot con la terapia a base di Vitamina A sono presumibilmente dovute ad inesatta interpretazione di lesioni congiuntivali xerotiche che non raggiungono lo stadio delle chiazze di Bitot.

La chiazza di Bitot non è una semplice xerosi, ma ha delle caratteristiche della leucoplasia, cioè di una vera e propria neoformazione come confermato dagli esami istologici: pertanto la chiazza di Bitot rimane anche quando sono scomparsi gli altri sintomi di avitaminosi A.

Considerata sotto questo punto di vista, la chiazza di Bitot può logicamente presentarsi non accompagnata da avitaminosi ed essere cioè l'esponente di una avitaminosi pregressa. Nel caso da noi descritto, però, malgrado gli episodi specifici, le condizioni sociali del soggetto ed il tipo di vita e di alimentazione da lui tenuto, dovrebbero escludere nel modo più assoluto che ci siano state nel passato alterazioni oculari anche modeste, imputabili ad una carenza vitaminica.

Se da un lato è possibile che si possa essere verificata una insufficiente presenza di Vitamina A a causa di una insufficienza epatica o deficienza metabolica e, sotto questo punto de vista un normale apporto alimentare di vitamina non avrebbe impedito l'insorgere della lesione, ci sembra impossibile che tutti gli altri sintomi di avitaminosi possano essere passati del tutto inosservati al soggetto nè si giustifica lo spontaneo ripristino del tasso vitaminico (come confermato dagli esami da noi effettuati per la ricerca della vitamina nel sangue in un soggetto che non è mai venuto meno al suo stato di benessere fisico neppure in tempi remoti.

Il fatto che a la chiazza di Bitot insorga esclusivamente nel settore della congiuntiva lasciato scoperto dalla rima palpebrale, dà importanza ai fattori aria e luce.

Tali fattori, come predisponenti, se non provocanti la lesione, sono confermati dalla osservazione di un caso in cui, presentandosi sulla palpebra superiore un coloboma che lasciava scoperto una regione congiuntivale appunto nella zona superiore, si ebbe la comparsa di una chiazza di Bitot proprio in tale sede. La stessa maggiore incidenza della chiazza di Bitot in regioni tropicali e subtropicali, se da un lato può essere spiegata come zona di più frequente avitaminosi, da un altro può essere imputato anche ad una più facile e frequente esposizione alla luce di quelle popolazioni e le numerose descrizioni della comparsa di casi non legati ad una avitaminosi ne sarebbe una conferma.

Il soggetto da noi preso in esame, mentre da un punto di vista generale non presenta e non ha presentato alcuna deficienza alimentare o metabolica tale da giustificare avitaminosi presente o pregressa, ha sempre avuto abitudini di vita tali da rimanere a lungo esposto ai raggi solari, praticando attivamente sports che lo hanno sempre costretto ad una vita all'aperto.

Ci domandiamo pertanto se non si debba fare una netta distinzione fra la chiazza di Bitot classica e queste forme che se clinicamente ed istologicamente sono del tutto simili a tale affezione non hanno però la base eziologica comune.

O anche, e meglio, se la chiazza di Bitot non sia uno stato degenerativo che può insorgere sia su congiuntive in stato di sofferenza xerotica, sia primitivamente per cause non ben determinate, in cui gioca un ruolo importante la luce solare, lesioni del genere delle leucoplasie, o riferibile forse a disturbi metabolici locali, a cui può influire uno stato carenziale generale o locale di vitamina A. Un certo interesse riveste nel nostro caso il trattamento chirurgico. L'asportazione della congiuntiva xerotica sembra aver determinato l'eliminazione definitiva della lesione che a 10 mesi di distanza non ha mostrato alcun accenno a recidivare. Viene così confermato il risultato già ottenuto con lo stesso mezzo da Gorduren nel suo caso personale.

Il periodo trascorso sia per il nostro caso che per quello di Gorduren è ancora troppo breve per trarne delle conclusioni definitive ma sembra tuttavia che l'operazione chirurgica rappresenti effettivamente allo stato attuale l'unica terapia che abbia successo nel trattamento della chiazza di Bitot non accompagnata da avitaminosi.

BIBLIOGRAFIA

1. AYKROYD, Brit. Med. Jour. 2. 1908, 1837.
2. APPELMANS M. M., "Xérosis et héméralopie chez l'adulte", Bull. Soc. Belg. Opht. 1937, n. 74; pp. 109-119.
3. APPELMANS M. M., LEBAS B., MISSOTTEN L., "Des manifestations oculaires d'Avitaminose A". Bull. Soc. Belg. Opht. 1955, n. 113, pp. 327-355.
4. BIETTI G., "Le vitamine in oftalmologia", Cappello, Bologna, 1940.
5. BITOT, Citato da Nida.
6. BOUZAS M. A., "Il significato delle chiazze di Bitot". Bull. Soc. Franc. Opht. 1959, n. 3, pp. 185-189.
7. CORNET E., "Les xérosis en clinique oculaire, son nouveau traitement local". Ann. d'Ocul., 171, 484, 1934.
8. FRONIMOPOULOS J., KOFINAS H., "Un caso di degenerazione cheratinica della congiuntiva". Bull. Soc. Hellen. d'Opht., 23. 196, 1955.
9. GORDOREN S., ORGEN C., "Taches de Bitot sans rapport avec une avitaminose". Excerpta Medica 18^o Congr. Intern. Ophth. sept. 1958. Bruxelles.
10. METIVIER V., "Bitot's spots in Trinidad". Am. J. Ophth. 24, 1029, 1941.
11. NICHOLS L., Nimalasuriga - Lancet 13, 506, 1939.
12. NIDA, "Syndrome de Bitot et avitaminose". Soc. Opht. Paris, 19 Mars 1927.
13. PALMER, Citato da Metivier.
14. PICARD J., "Xérosis de la conjonctive". Bull. Soc. Opht. Paris, 561, 1927.
15. RIDEL, "Conjunctival xerosis". Trans. Ophthal. Soc. Kingdom, 295, 1933.
16. SIE BOEN LIAN, V. Grafés. Arch. f. Opht. 139, 17, 1938.
17. SATHE S. M., "Angular conjunctivitis with associated dermatitis". Journ. of the All India Ophth. Soc. Bombay, avril 1955, 3, n. 1, pp. 1-9.
18. SPYRATOS, "Héméralopie et altérations oculaires par carences en gréce pendant les années 1941-1945". Annales d'Oculist. 1949, vol. 182, pp. 672-685.
19. TERRIEN F., BLUM J., "Sur un cas de xérosis conjonctival". Soc. Opht. Paris, 342, 1931.
20. TERRIEN F., HALBRON P., "Sur un cas de xérosis corneo-conjonctival". Soc. Opht. Paris 14 Mai 1938.
21. VENCO, "Una sindrome tipica di xeroftalmia da avitaminosi". Arch. Ottal. 8 Août 1930.
22. WILSON, Citato da Metivier.
23. YOURISH N. B., "Significance of Bitot's spots". Amer. J. Ophth. 36, 109, 1953.

THE WOLFE SCLERAL TONOMETER: AN EVALUATION

BY

JOHN H. CARTER M., O. D.

Bloomington, U. S. A.

Introduction:

It is probable that the selection of the cornea as the popular site of tonometer application was based upon its obvious accessibility and upon the relative thinness of its epithelial coat. Further, it may have been assumed that the cornea provided more favorable physical characteristics than the sclera.

The cornea as a site for tonometer application is not, however, without its disadvantages. Certain pathological states affecting the anterior refracting surface may make tension measurement difficult, unreliable, or hazardous. Minor corneal injuries are not infrequent. Apprehension may bring about spasm of the palpebral portion of the orbicularis muscle causing pressure to be exerted against the eyeball thereby giving rise to spurious estimates as to the magnitude of the original pressure. Further, the associated use of topical anesthesia may make the corneal epithelium especially susceptible to damage.

Scleral tonometry using the Wolfe instrument has recently aroused considerable interest in certain circles and several excellent reports^(1,2,3) of the results of clinical study have appeared in the literature.

It has been the purpose of the present investigation to analyze the physical and mechanical characteristics of the Wolfe instrument as well its performance on pressure controlled animal eyes.

The Wolfe Scleral Tonometer appears to be a variant of the French Bailliart Tonometer (see Figure 1). The instrument presently available, the Wolfe Model "C", is manufactured by the Chicago Dial Indicator Company under the auspices of the Department of Clinical Research of the Illinois College of Optometry.

Hirsch⁽²⁾ has reported that the average pressure determined by the Wolfe instrument is similar to that obtained by the use of the Schiötz and further that the ranges of the two instruments appear to be very nearly identical.

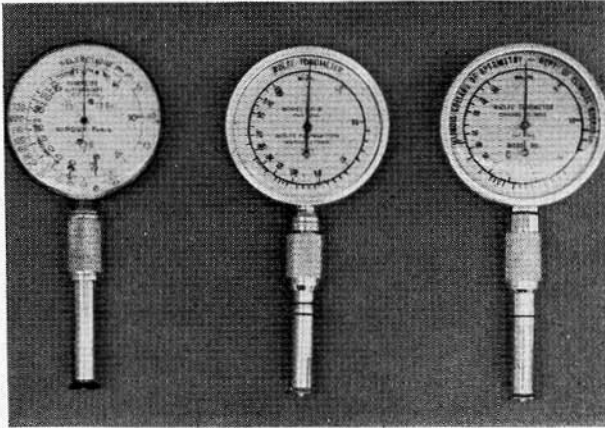


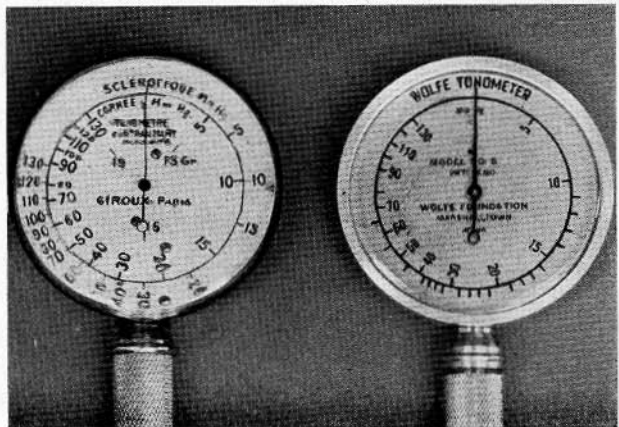
Fig. 1. The Bailliant Tonometer (left) together with the Wolfe Model B (center) and the Wolfe Model C (right).

In spite of this similarity, scleral tonometry using the Wolfe instrument appears to differ markedly from corneal tonometry in at least two respects. First, it has been reported that the initial reading taken with the Wolfe instrument is frequently spurious thus necessitating that readings be taken in multiple and the first reading be discarded. Second, a bimodality appears to be characteristic of the Wolfe distribution curve with the secondary peak occurring somewhere between twenty-five and thirty millimeters of mercury.

PHYSICAL AND MECHANICAL CHARACTERISTICS OF THE WOLFE TONOMETER:

Apparently the dial of the Wolfe Scleral Tonometer has been taken from the Bailliant instrument (Figure 2). Note that the French instrument shows a scleral

Fig. 2. The faces of the Bailliant and the Wolfe tonometers. Note the identity, even to irregular spacing, between the scale of the Wolfe instrument and the Bailliant corneal scale.



SCLERAL TONOMETER

and a corneal scale and it is the corneal rather than the scleral scale which has been used in the Wolfe Scleral Tonometer. The scale is not linear and has some irregularities as is shown in Figure 3, in which figure the Wolfe scale reading in millimeters of mercury is plotted against the needle deviation in arc degrees.

To check whether or not the irregularity of the Wolfe dial scale was in compensation for a non-linear gear mechanism (Figure 4), the depth of scleral indentation recorded in millimeters by means of specially constructed micrometer (Figure 5), was plotted against the angle of needle rotation (Figure 6). Seventy millimeters of mercury on the scale was taken as the point of zero indentation. It is apparent that the mechanism does not deviate by any significant amount from linearity except for scleral indentations of less than approximately 0.2 mms. (four Schiötz units).

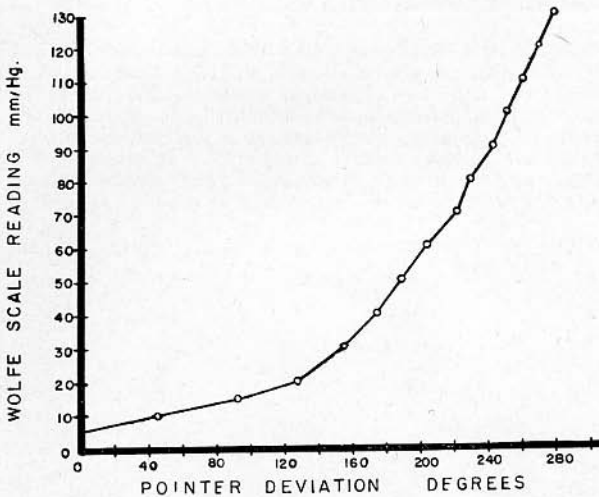


Fig. 3. The Wolfe scale reading in millimeters of mercury plotted against the angular value of the needle rotation in degrees of arc.

A calibration curve identical in form to that of the Schiötz instrument would scarcely be expected since, in a spring-loaded instrument, the plunger load is a function of the indicator position. Nevertheless, it was decided to plot the depth of scleral indentation (Schiötz units) against the pressure designation of the Wolfe instrument (mms. Hg.) (Figure 7). Since the curve is not like the Schiötz calibration curve, it is apparent that the original scale was not taken from the Schiötz.

The effects upon the clinical estimate of the intra-ocular pressure of an unusually high ocular rigidity are minimized when a tonometric device is used to measure intra-ocular pressures above its critical pressure. (4^a) The critical pressure of a Wolfe Model "C" Tonometer was calculated and found to be in excess

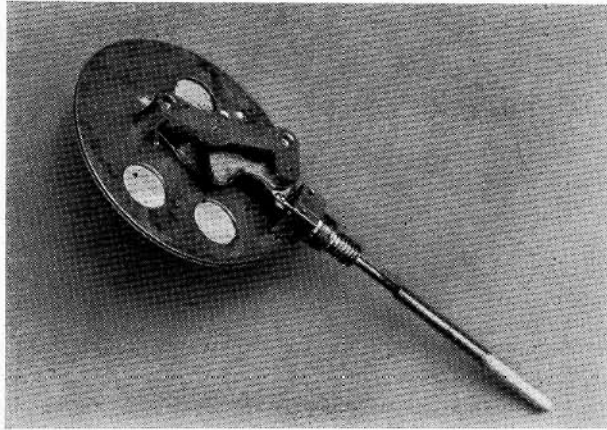


Fig. 4. The mechanism of the Wolfe Model C tonometer. The shaft within the stem of the instrument is attached rigidly to the plunger at one end and with one component of the gear mechanism at the other end. The upper end of the shaft describes an arc as the plunger is activated. This motion is permitted by a loose fit of the plunger the tonometer footplate. Obviously, plunger motion is not purely vertical but a slight rocking action of the plunger end is permitted to occur. A spring within the instrument provides the plunger load although the weight of the plunger and shaft must contribute to the load when the instrument is used in a vertical position.

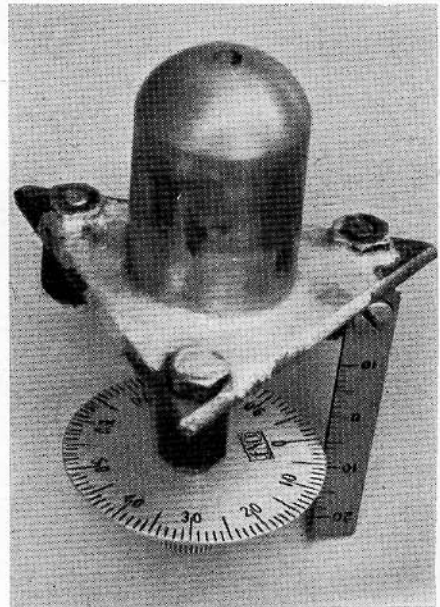


Fig. 5. A micrometer constructed for the purpose of determining the magnification characteristics of the Wolfe Tonometer. The central zone of the upper portion of the micrometer has a fifteen millimeter radius of curvature. A hole drilled in the center permits entry of the tonometer plunger where it contacts an internal shaft which in turn communicates with the screw mechanism of a modified spherometer. Plunger movement can be measured to an accuracy of 0.01 millimeter.

SCLERAL TONOMETER

of sixty nine millimeters of mercury due to the extreme amount of pressure with which this instrument is applied to the eye.

Even though the range of the dial of the Wolfe implies the greatest range of readings found in any tonometric device, being calibrated from 5 to 130 mm. Hg., seventy millimeters of mercury must be considered to be the upper functional limit of the instrument because the plunger end is flush with the footplate at this level and hence no longer indents the sclera.

The Wolfe is designed to permit measurement of the intra-ocular pressure with the patient either seated or reclining. Increased tension on the spring with in the handle partially compensates for the lack of gravit, when the instrument is used in the horizontal position.

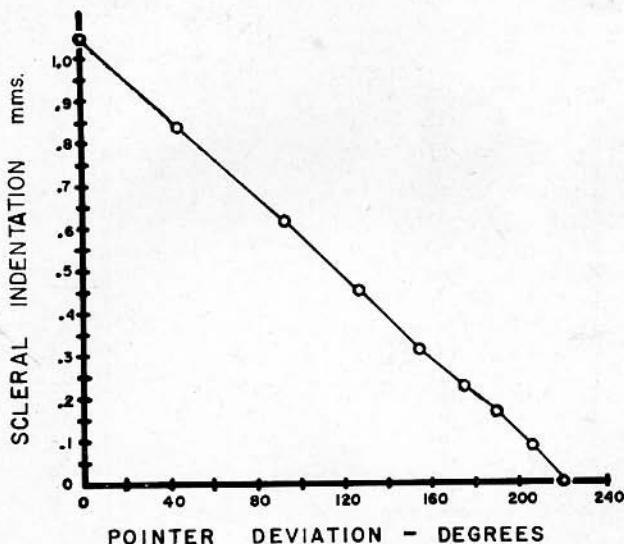


Fig. 6. The distance in millimeters by which the plunger end extends through the footplate plotted against the corresponding angular movement of the indicator expressed in degrees of arc.

Unlike the case of the Schiötz instrument in which the plunger load assumes essentially a constant value irrespective of the indicator position, the plunger load of the Wolfe and Bailliart instruments varies as a function of the Scale reading. The Bailliart dial has been provided with three check-points which may be used to periodically test the precision of the plunger spring tension. A knurled nut within the instrument stem permits alteration of the spring tension should this be required.

Although the Wolfe Model "B" is provided with a mechanism for spring tension adjustment similar to that found in the Bailliart, no scale check points are provided. In the Wolfe Model "C" no provision of adjustment of spring tension nor check points on the dial are found.

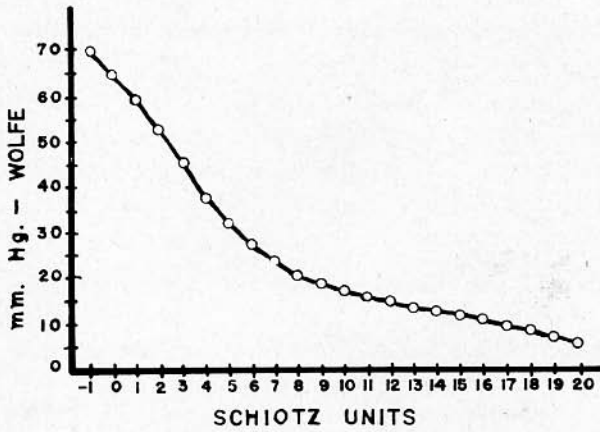


Fig. 7. The Wolfe scale reading in millimeters of mercury plotted against the plunger extension in Schiötz units as measured by micrometer. One Schiötz unit is equivalent to 0.05 millimeter.

Fig. 8. The Wolfe scale reading in millimeters of mercury plotted against the manometric reading in centimeters of saline. Data was obtain using pig eyes and an open stopcock technique.

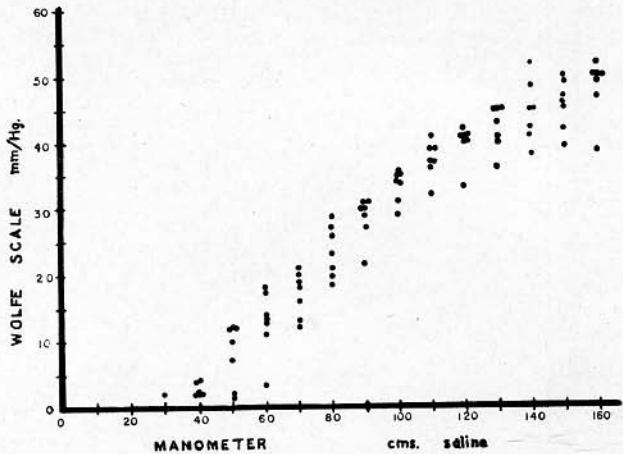


Table I illustrates several differences between the Wolfe and Bailliart instruments.

TABLE I
Several Differences Between the Wolfe and the Bailliart Tonmeters

	<i>Bailliart</i>	<i>Wolfe "B"</i>	<i>Wolfe "C"</i>
<i>Calibration Check Points</i>	YES	NO	NO
<i>Non-Adjustable Hair Spring in Head</i>	NO	YES	YES
<i>Adjustable Compression Spring in Shaft</i>	YES	YES	NO
<i>Scleral Tension Scale</i>	YES	YES*	YES*
<i>Corneal Tension Scale</i>	YES	NO	NO

* Identical to the corneal scale of the Bailliart Instrument.

PRECISION OF STANDARDIZATION OF THE WOLFE TONOMETER:

It is advantageous for all tonometers of one type to be standardized so that the reading taken upon an eye of given tension with one instrument will be identical to the reading which would have been obtained had a different instrument been used. Kronfeld has demonstrated that one satisfactory mode of tonometer standardization consists of establishing fixed values for each of the essential variables of a tonometric instrument. ^(4b) An instrument will give readings consistent with other tonometric instruments of like type only if the physical characteristics are identical.

As only one Wolfe "B" was at hand for study and as many variations between the "B" and "C" instruments were apparent, it was felt that there would be little to gain by reporting the physical characteristics of the solitary "B" model.

The physical characteristics of three Wolfe "C" instruments, all in apparently good condition, were measured. All of these instruments had been in active use in the Indiana University Optometry Clinic. None had been claimed by the manufacturer to be a standard instrument.

Table II shows the measured values for each of the three Wolfe instruments under test. Manufacturer's specifications were included when available.

TABLE II
Physical Characteristics — Wolfe "C" Tonometer*

Characteristic:	Mode of Measurement:	Finding:
1. Overall weight (Handle included)	Measured with a triple beam balance with a sensitivity of 0.01 gram.	Wolfe N ^o 1: 53.15 gm. N ^o 2: 52.91 gm. N ^o 3: 52.81 gm. **Standard: 42.5 gm.
2. Instrument weight less plunger weight plus spring tension when handle is depressed to red line	Plunger removed from instrument. Instrument supported by means of a laboratory jack attached to its handle. Instrument lowered foot first onto the pan of a triple beam balance. Height of laboratory jack and position of balance weights altered until end pointer of balance read zero at the same time that the instrument handle was depressed to the red line on the sleeve.	Wolfe N ^o 1: 64.5 gm. N ^o 2: 63.1 gm. N ^o 3: 62.4 gm.
3. Instrument weight less plunger weight plus spring tension when handle is depressed to black line	Measurement made as in 2 above except that the black rather than the red sleeve indication was utilized.	Wolfe N ^o 1: 70.7 gm. N ^o 2: 69.8 gm. N ^o 3: 70.6 gm.

* All Measurements made at a temperature of twenty seven degrees centigrade.

** It has been assumed that the manufacturer's specification for "overall weight" includes the weight of the instrument handle. If it does not, the magnitude of the discrepancy between specified weight and measured weight may be considerably reduced. The writer was unable to remove the instrument handle and hence handle weight is not known.

Characteristic:	Mode of Measurement:	Finding:
4. <i>Available spring tension to compensate for effective loss of instrument weight when instrument is used with patient in a seated position</i>	Item N ^o 3 above less item N ^o 2 above.	Wolfe N ^o 1: 6.2 gm. N ^o 2: 6.7 gm. N ^o 3: 8.2 gm.
5. <i>Plunger weight</i>	Plunger removed from tonometer and weighed on triple beam balance within an accuracy of 0.01 gm.	Wolfe N ^o 1: 1.90 gm. N ^o 2: 1.87 gm. N ^o 3: 1.89 gm.
6. <i>Plunger load</i>		

By "plunger load" is meant the spring tension acting against the plunger at a given indicator position. Naturally, the plunger load increases as the plunger is pushed into the instrument stem.

A small wooden block with a minute through its center projecting upward was placed upon the tray of the gravity balance and weighed. This weight was recorded and subtracted from each gross weight yielding the net values tabulated below.

The tonometer was carefully calibrated so as to read 70 mm. Hg. when placed upon its own test block.

The complete tonometer was supported by a laboratory jack by means of a clamp attached to its handle. The tonometer was lowered carefully so that its plunger rested upon the head of the nail.

The height of the laboratory jack was altered as were the positions of the balance weights until the balance indicator rested at zero at the same time that the tonometer needle rested at the indicated value.

These measurements were very difficult to make because of instrumental friction and in many cases a method of limits had to be used to determine the point of balance. Readings were made in multiple and averaged.

Indicator Value (mm. Hg.)	Plunger Load Tonometer N ^o 1 (gram.)	Plunger Load Tonometer N ^o 2 (gram.)	Plunger Load Tonometer N ^o 3 (gram.)	Plunger Load "Standard Instrument" (gram.)
5	12.8	12.0	12.9	
15	15.6	14.7	16.0	
30	17.9	17.0	19.4	
60	19.8	19.0	22.1	
70	21.8	19.4	22.7	12.5

7. Diameter of Footplate — Diameter of Plunger

Measurements were made within an accuracy of 0.1 mm. by means of a vernier caliper. Data for the "standard" instrument was obtained by converting the manufacturer's specifications from inch measure to metric.

Footplate Diameter (millimeters)	Plunger Diameter (millimeters)
Wolfe N ^o 1: 9.8	3.2
Wolfe N ^o 2: 9.8	3.2
Wolfe N ^o 3: 9.9	3.1
Standard: 10.109 to 10.160	3.175 ± 0.0254

8. Radius of curvature of Footplate — Radius of curvature of Plunger end

Measurements were made to an accuracy of one half millimeter by utilizing a series of templates graduated in half millimeter steps.

Radius - Footplate	Radius - Plunger
Wolfe N ^o 1: 15	15
Wolfe N ^o 2: 15	15
Wolfe N ^o 3: 15	15
Standard: 15	15

*COMPARISON OF THE THREE WOLFE INSTRUMENTS BY MEANS
OF A TAMBOUR:*

A tambour has at times been used for the purpose of comparing readings obtained under identical physical conditions by several different tonometric devices (4c). Identity of readings on the tambour tends to suggest instrumental similarity. Identity of readings on the tambour tends to suggest instrumental similarity. It does not, however, insure that identity of readings would be obtained if the tonometers were compared upon living human eyes with fixed tension. But, as human eyes with fixed tension are not readily available and as the use of the tambour yields results more satisfactory than those which would be obtained using human eyes with normal outflow characteristics, a tambour study was made. The bulb of sphygmomanometer was connected to a chamber covered by a thin plastics membrane, and to a pressure gauge reading in pounds per square inch. The reading of the pressure gauge did not hold any absolute relationship to the tonometer scale reading but merely indicated a pressure level at which the tonometric devices were being compared. The system was filled with air and testing was done at ascending pressure levels.

The tonometer under test was raised and lowered upon the tambour by means of a laboratory jack which eliminated the difficulty associated with repeated placement of a hand-held instrument upon a fixed area of the tambour.

TABLE III

*Tonometer Readings Obtained at Various Tambour Inflation Levels
with Each of Three Wolfe "C" Tonometers:*

<i>Tambour Inflation</i>	<i>Tonometer N^o 1</i>	<i>Tonometer N^o 2</i>	<i>Tonometer N^o 3</i>
A	11.7 mm. Hg.	11.7 mm. Hg.	10.2 mm. Hg.
B	21.8 mm. Hg.	19.3 mm. Hg.	19.7 mm. Hg.
C	31.8 mm. Hg.	31.2 mm. Hg.	31.5 mm. Hg.
D	37.9 mm. Hg.	37.8 mm. Hg.	38.6 mm. Hg.
E	44.7 mm. Hg.	46.3 mm. Hg.	47.3 mm. Hg.

Table III shows the reading obtained at various pressure levels with each of the three Model "C" instruments. Measurements were made at each of five levels of tambour inflation labeled A, B, C, D, and E. Each reading was made in triplicate and averaged. Only the mean reading at each pressure level for each instrument is indicated in the table.

This table would seem to demonstrate that the three Wolfe instruments under test yield readings which are in reasonable agreement when applied in succession to a tambour maintained at a fixed level of pressure.

MANOMETRIC INVESTIGATION OF THE WOLFE CALIBRATION CURVE:

Closed stopcock calibration studies (^{4d}) of a tonometric device are not entirely satisfactory because of the excessive scatter obtained when the tonometric readings are plotted against the manometric readings. The scatter is related to variations in the degree of ocular rigidity of the eyes under test. It is unfortunate that closed stopcock data are not well adapted to simple curve fitting because the closed stopcock situation more closely parallels the clinical condition than does the open stopcock situation.

Open stopcock data, (^{4e}) on the other hand, are extremely useful in that scatter is reduced by the exclusion of rigidity effects. Open and closed stopcock curves have been assumed to be identical in contour but a vertical translation of the open stopcock curve upon its axes must be accomplished before it become representative of the clinical situation*.

A burette employed as a water reservoir was attached by means of pressure tubing to a manometer and to a twenty gauge needle inserted into the anterior chamber of a pig eye at the limbus. The system was filled with physiological saline and air was excluded. By the operation of a sphygomanometer bulb and burette valve the manometric pressure could be brought to any desired level. A Wolfe tonometer was clamped to a lab jack and lowered onto the bulbar conjunctiva of the pig eye so that the edge of the tonometer foot fell one half to one millimeter from the limbus. No eye was used in which the scleral radius exceeded fourteen millimeters as measured by means of a template. An area of sclera was utilized that was judged by inspection to be quite regular. Insertions of the extra-ocular muscles were avoided.

Figure 8 is a scatterplot in which the tonometer reading in millimeters of mercury (Wolfe) is plotted against the manometric determination of the actual internal pressure of the eye under open stopcock conditions. If the calibration curve of the eye Wolfe instrument were entirely accurate, a linear relationship should have been found between the tonometric pressure in millimeters of mercury and the manometric pressure in centimeters of water. * *

* Identity of contour between the open and closed stopcock curves was assumed by Schiötz in his calibration studies. Such an assumption leads only to a very slight inaccuracy in the interpretation of clinical data. In more recent calibration studies, Friedenwald modified his curve by the utilization of a factor which takes into consideration the "normal" coefficient of ocular rigidity and the volume of fluid displaced by the tonometer plunger. The latter value is, of course, a function of the indicator position.

** In plotting open stopcock manometric values directly against the corresponding Wolfe scale values, the assumption was made that the open and closed stopcock curves deviated from each other by a constant value throughout their entire extent. This is an assumption which had been made by Schiötz in his calibration studies but is not entirely true. It is sufficiently precise for our present purpose, however.

SUMMARY AND CONCLUSION:

Scleral tonometry using the Wolfe instrument has recently aroused considerable interest. It has been shown that the Wolfe Scleral Tonometer constitutes a variant of the Bailliart corneal scleral instrument. The scleral escale of the Wolfe was found to be identical to the corneal scale of the Bailliart and to be quite irregular. The irregularity of the dial scale cannot be explained on the basis of the mechanical characteristics of the gear mechanism as this was established as being essentially linear over the major portion of its range. Further, it was demonstrated that the dial scale was not taken from the Schiötz calibration curve.

The effects of ocular rigidity upon the clinical estimate of the intra-ocular pressure are the critical pressure minimized when of the Tonometer is lower than the intra-ocular pressure. The critical pressure of a Wolfe Model "C" Tonometer was calculated and found to be in excess of sixty nine millimeters of mercury.

Although the dial of the Wolfe instrument implies a range from five to one hundred thirty millimeters of mercury, seventy millimeters of mercury constitutes the upper functional limit of the instrument.

While the Wolfe Tonometer is constructed in such a manner as to be usable with the patient either erect or reclining, it has been established that the instrument exerts a greater force upon the eye when held vertical rather than horizontal.

Whereas the plunger load assumes a constant value in a gravity type instrument, it varies as a function of indicator position in a spring loaded device such as the Bailliart or the Wolfe Tonometer. Although means are provided in the Bailliart Tonometer for adjusting plunger load, no means for insuring load are available in the case of the Wolfe instrument.

The physical characteristics of three available Wolfe instruments were measured and found not to be identical.

When the readings of three Wolfe instruments were compared on a tambour maintained at various pressure levels, reasonable consistency was noted. Although this is suggestive of instrumental similarity, it does not insure that consistency of readings would have been obtained had they been taken using human eyes with fixed tension.

An open stopcock manometric calibration study was undertaken using the Wolfe Tonometer to make pressure measurements on pig eyes maintained at known levels of pressure. When the tonometric findings were prottel against manometrics findings, the relation ship appeared not to be linear.

The author is greatly indebted to Dr. M. J. Allen of the Division of Optometry, Indiana University, for his invaluable assistance in the execution of this study. He is also indebted to Dr. Henry Hofstetter, Director, Division of Optometry, Indiana University, for reading the manuscript and contributing many helpful suggestions.

Bloomington, Indiana

BIBLIOGRAPHY

1. WICK, RALPH E., Experiences with Scleral Tonometry, American Journal of Optometry and Archives of the American Academy of Optometry, 33 (3) : 118-122, 1956.
2. HIRSCH, MONROE J., An Evaluation of Scleral Tonometry. Ist Reliability, Validity, and Clinical Applicability, American Journal of Optometry and Archives of the American Academy of Optometry, 32 (8) : 391-403, 1955.
3. TALCOTT, RICHARD E., A Comparison of Corneal and Scleral Tonometry, American Journal of Optometry and Archives of the American Academy of Optometry, 35 (1) : 31-36, 1958.
4. American Academy of Ophthalmology and Otolaryngology, Standardization of Tonometers - Decennial Report by the Committee on Standardization of Tonometers, American Academy of Ophthalmology and Otolaryngology, 1954.
 - 4a. Pages 119-121.
 - 4b. Pages 33-43.
 - 4c. Pages 6-7.
 - 4d. Pages 112-151.
 - 4e. Pages 100-112.

HOME VISUAL TRAINING SUGGESTIONS

BY

F. J. DAY, O. D.

Birmingham, U. S. A.

In the course of rendering modern visual care it is most necessary to give some type of visual training to a large percentage of patients in order to achieve a reasonable improvement in visual efficiency. There are a great many of these patients who should have training in the office with instruments designed primarily for that purpose, however many patients can not come to the office at regular intervals, three times a week, without inconvenience, or they feel that they can not afford it. Due to this unfortunate circumstance there are many patients who can only do home training, but this helps greatly if done regularly.

In all cases it is a primary concern to improve basic visual skills so there will be no antagonisms present to retard action of the extrinsic muscles under skeletal neural control, or hesitance in accommodative facility under visceral neural control, and both systems must synchronize with each other.

To improve the skeletal functions it is necessary to learn proper control of the muscles by practicing the use of them. This is easily done by a variety of pursuit and fixation movements of the eyes. In all cases it is best to improve these functions monocularly first, then binocular training is taken only after the monocular functions have greatly improved. This monocular work is done with both eyes open but one eye occluded either with a plastic occluder clipped over one lens of the patients glasses or with a deep cup patch occluder tied around the head that will allow free movement of the covered eye. Head is held straight forward and only the eyes are moved. Some examples of pursuit training are listed below.

1. Follow with the uncovered eye the eraser of a pencil moved slowly in a large circular direction by some other person.

2. Place a small fixation target at one end of a yard stick, bright colored paper glued to the stick, so that it protrudes above the edge of the stick about 1/4 inch like a gun sight. The stick is held by the patient so that the plain end

is steady against his cheek below the eye being trained and the stick is moved so that the fixation target rotates in a large circle and the patient looks down the edge of the stick following the target.

3. Cut out a cardboard circle about 2 feet in diameter and punch a large nail, 60 penny common, through the center of it, but leave the hole small enough so the nail head will not come through the cardboard. Place a large thread spool on the nail, or similar pulley wheel with a small center hole just slightly larger than the nail. Attach a long heavy string to the cardboard circle close to the peripheral and wrap the string around the spool or pulley. Then if the nail is held in one hand and the string pulled with the other hand, it makes the circle go around. The patient stands in front of the circle on the nailhead side and follows a bright colored fixation target on the peripheral edge of the circle while some other person at home pulls the string to move the rotating device.

4. A 2 or 3 inch diameter solid sponge rubber ball with a string punched through it can be suspended by the string from overhead. The ball should hang 2 or 3 feet above the floor and the patient can lay on the floor on his back directly under the ball and follow the ball as it swings in a circular movement.

5. The patient follows the chalk as he draws a large circle on a chalkboard over and over in the same circle.

Give 50 to 75 rotations clockwise and the same number counter clockwise in any above procedure.

Some examples of fixation movements are as follows:

1. Some person holds two different colored pencils in front of the patient about 20 to 24 inches apart and 18 to 20 inches from the patients eye, and calls out the colors to patient at about 1 second intervals, for example red - green - red - green, etc. The pencils are held first to give horizontal fixation on the 180 line, then vertical on the 90 line, then the two diagonals on each side of the first directions.

2. On a large cardboard circle 20 to 24 inches in diameter, place numbers around the periphery in such a position that two numbers are un the 180 line, two numbers on the 90 line, and two for each of the two diagonal directions. Someone then calls the numbers out to the patient and he looks from one number to the other as they are called to him. This is done with the circle of numbers directly in front of the patient about 20 to 24 inches from his eyes.

3. The patient stands at one end of a fairly large room and some other person calls out different objects in the room to him, such as chair, clock, lamp, vase, etc. The patient looks from one object to the other for 3 to 5 minutes with each eye. This brings in the size and distance factor as well as learning sacaddic movements.

4. A dot is placed on a chalkboard by someone and the patient is instructed to place his chalk upon this dot. The other person then places another dot on the board in any position and the patient is instructed to connect the dots with a line. He is now instructed to hold his chalk on the second dot so he will be ready to move immediately to the third dot which will next be put on the board. This can continue until the board is full of dots and lines in every conceivable direction.

Of equal importance besides rotations and fixations is accommodative ability both as to amount of it and speed and flexibility of function. Some examples of home training for these is as follows,

1. Have a printer to print some letter or number charts on plain white paper 9 x 11 inches so that there are one line each of 20/100 and 20/60 size letters, and at least two lines each of 20/40, 20/30, and 20/20 letters. Keep a supply of these in your office and give one to each patient you desire to have this training. With one eye occluded have the patient look at one word of small print in a book or paper and pull the print as close to his eye as he can to barely make out each letter in the word, then look at one letter or number on the smallest line he can make out on the distance chart you have furnished him, placed approximately 20 feet away. The patient reads the book or paper in this manner, one word at near and one letter at distance, etc., going over and over the letters on the distance chart while he continues through the subject matter at the near point. It is necessary to hesitate slightly at both distances to begin with in order to give the eyes time for maximum response, and sometimes it is necessary for the patient to blink his eyes to achieve satisfactory clarity at one point or another, but as the ability improves the print can be held closer to the eye and patient can change focus faster from near to far etc. Have the patient read 10 to 15 minutes with each eye in this manner.

2. This procedure requires the same set up as described in No 1. above, except that the patient holds a large table spoon or any long handle occluder in one hand and the small print in his other hand. Instead of doing the accommodative work with one eye constantly occluded, he alternates the occlusion so that he reads a word of print with one eye and a letter on the distant chart with the other eye, alternating eyes each time he changes from near to far etc. Do this so that each eye will have 10 to 15 minutes work at both distance and near.

3. Provide yourself with several all metal, large eye size, frames and have your optical laboratory put flat lenses in each of these frames so that you have a +.50 sphere in one eye and -.50 sphere in the other eye of one frame, and do the same thing with other frames to give you + and -1.00, + and -1.50, + and -2.00, & + and -2.50. Then attach a wood handle about 10 inches long

X R V F Z Q E

W N Y E D R S

W N K R N S G O

V S L M W K B C E

A Y Y C D E T F R

W Y Q R A T B N

W A Z C D S E F V

Q U E J Z Y N A C L M O N

to each frame at or across the bridge so the handle is in the same plane as the lenses and at a right angle to a line between the center of the two lenses. With one eye occluded the patient holds small print at his usual reading distance or placed on a table, and with one hand holds a pair of the + and — glasses by the handle. He reads one line of print looking through the + lens, then turns the handle and reads the next line looking through the — lens, etc. Start with a weak set of lenses and progress to the stronger ones as the patients ability improves. These + and — glasses are loaned to the patient one pair at a time and exchanged for a stronger pair as he progresses. Read 10 to 15 minutes with each eye.

Every patient who needs visual training for improvement of ductions, accommodative functions, suppression of vision, amblyopia of any type, strabismus, or other problem, needs to have work in the above mentioned functions of monocular pursuit, monocular fixations and monocular accommodative rock. Each eye individually must perform its functions better before the two eyes can work together as a team with maximum efficiency. There are a few exceptions to this rule but they are unusual and I will not discuss them here. After the monocular functions are greatly improved, then all of the training procedures can be done binocularly, however as the monocular work improves, in most cases the binocular skills automatically improve simultaneously.

There are some patients who should have additional specific training directed at one particular aspect of their problem. This is given as an additional procedure at first with the basic work, and later done by itself to strengthen the weak abilities. A few examples of specific training are as follows.

1. For cases of suppression with amblyopia, or any case with uncorrectable poor vision, either monocular or binocular, and having no apparent pathological reason, this procedure will help many times. The patient places on the wall a letter chart, described in N^o 1 of accommodative rock examples, in a completely dark room. He positions a light in such a way as to be close to the chart and shaded so that the light is directed on the chart but does not shine in the patient's face who is standing in front of the chart and as far away as he can to just be able to see some of the letters in the lines of larger size. The light wire is plugged into a 25¢ christmas tree light winker which is then plugged into the electrical outlet. These little light winkers are not designed for long or heavy duty use and the speed of the flash is not regulated precisely, but after the winker warms up in 10 to 15 seconds with a 60 to 100 watt light bulb, most of the winkers give about 1 to 2 seconds of light and 3/4 to 1 second of dark. The patient stares at the chart while the light goes on and off, trying to see the letters progressively better and gradually move farther from the chart and try to read smaller and smaller letters. This can be done with a reduced snellen chart at near also.

2. For any case that is suppressing, either monocular or alternating, or circumstance where patient needs work to improve simultaneous vision, this procedure helps. Have the patient stand in a corner of a room about 2 feet from each wall and face one of the walls. He then places a small bright colored picture at eye level on the wall not being faced and out from the corner as far as he is standing. Patient then holds a small round mirror 4 or 6 inches in diameter or oval shaped 3 x 5 against his upper cheek and on the same side of his nose as the small picture is on and as close to the nose as possible. The mirror is held at an angle so that the patient sees the picture in the mirror with the eye on that side and sees the design of the wall straight in front of him with the other eye. When the mirror is slowly angled back and forth the patient will see the picture move back and forth across the wall in front of him as long as he has simultaneous vision. If a large piece of paper with a different color and design from the wall with the picture on it is placed on the wall that the patient is facing and at eye level, this is helpful too. Due to the different images seen with each eye this stimulates simultaneous vision and if a suppression occurs, blinking by the patient helps to restore and maintain vision in both eyes. Watching the picture move back and forth also gives pursuit training. Patient should work a while with this set up, then reverse the set up so he faces the other wall and watches the picture with the eye that did see the wall in front of him.

3. Another procedure to help build simultaneous vision is done with bright red and green cellophane. The patient wears red and green anaglyphs, which are glasses with a red lens in one eye and a green lens in the other, or if he normally wears an Rx, then place a transparent red plastic clip over on his glasses before one eye and a green clip over before the other eye. Then a large piece of cardboard is secured and a hole is cut in it the same size and shape as the patient's television picture. The patient is also provided with some red cellophane and some green cellophane. This cellophane is attached to the cardboard covering the hole so that half of the hole is red and the other half is green. The cardboard is then hung over the front of the television set with string so that the hole is directly in front of the picture. The patient now watches the television picture and if he has simultaneous vision he will see the entire picture, however if the suppresses vision in one eye he will only see half of the picture and the other half will be solid black. This is accomplished by virtue of the green clip over before one eye will completely black out the half picture covered with red cellophane and the red clip over blacks out the green side of the picture. Be sure the cellophane is dark enough to black out the television picture completely when viewed through the clip overs or anaglyphs. Sometimes it is necessary to use a double thickness of cellophane over one side or the other of the picture to completely neutralize the color in the clip overs. These clip overs and cellophane are furnished to the

patient by the doctor. I use a dark red and dark green Sylvania Cellophane manufactured by Metal Goods Corporation in St. Louis 15, Missouri, U. S. A. and red and green clip overs manufactured by Watchemoket Optical Company in Providence Rhode Island, U. S. A. who also make opaque clip over occluders used often in monocular training. Substitute colored clip overs can be made by cutting a hole in an opaque clip over and attaching red cellophane over the hole of one occluder and another one has green cellophane. The patient sits at a close distance to the television picture and gradually moves away, maintaining the entire picture visible which gives simultaneous vision, or he starts at a great distance and gradually moves closer, depending on the visual problem and the coordination mechanism of the two eyes. If a suppression occurs, then the patient blinks his eyes to help overcome this or if that does not help then he occludes the non suppressing eye momentarily to force the other eye to see and in this way try to trick the suppressing eye into seeing all the time. The red and green clip overs are alternated in front of the eyes every 5 to 15 minutes to give further stimulation and prevent color fatigue. This same procedure can be used at the near point with the red and green cellophane being placed over a page of print in a book or paper instead of the television picture and a strong light is directed on the book or paper while the patient reads.

4. A method of giving fusion training is to have the patient hold a bright colored pencil or stick directly in front of his nose and while looking at the pencil be sure to see only one pencil and be conscious of seeing two distant targets 10 to 20 feet away. Then look at the distant target and be sure of seeing only one while maintaining consciousness of two pencils at the near point. The distant target can be any relatively small object of contrasting color to the background. The pencil must be held so the end of the patients nose, the pencil, and the distant target are all in a straight line. The pencil is held as close to the patients nose as he can maintain fusion when looking at it, whether the distance is at arms length or 2 inches from the nose, and as he looks back and forth from the pencil to the distant target, etc., trying to move the pencil gradually closer to the nose or farther away from the nose depending on the starting point of the pencil. Patient should gradually build up to where he can do 150 round trips at any position of the pencil. It is necessary for patient to always be sure there are two of the objects not being looked at to insure that simultaneous vision is present while doing this work. This procedure is called physiological diplopia training.

5. Another method of giving convergent fusion training is with a fusion card. This fusion card is a piece of good grade white cardboard $2\frac{1}{2}$ x $5\frac{1}{2}$ inches with 3 blue dots on one side of it and 3 red dots placed exactly opposite to the blue ones on the other side of the card. There should be a large red and large blue

round dot 20 millimeters in diameter placed opposite to each other in the center of the cardboard about 5 to 10 millimeters from the end of the card, and a set of small dots, 10 millimeters in diameter, placed in the center and about 25 millimeters from the opposite end of the card from where the large dots are. Then evenly spaced between the large and small dots should be placed a set of dots 15 millimeters in diameter. This gives three blue dots of different size and about 30 millimeters apart on one side and three red dots on the other side of the card. The patient holds the card between his thumb and forefinger on the bottom edge with the end of card touching the end of his nose and it should have about the same amount of light on each side of it. The card should be angled downward slightly on the distant end and the small dots should be closest to the nose with the large dots on the distant end. When the patient looks at the large dot, he sees a blue one with one eye and a red one with the other eye, which he should be able to fuse together giving only dot having a lavender appearance, and he should also see the other 2 blue dots with one eye and the 2 red dots with the other eye to give proof of simultaneous vision at all times, and produce a 5 dot situation. The patient looks at the large dot and is conscious of seeing the other 4 dots, then looks at any distant object directly in front of him, then back to the big dot, etc. If he can not fuse the 2 large dots, then the card can be moved out from the nose gradually until he can fuse them. The card does not work very well if the distant end is more than 10 inches from the nose. As the patients fusion ability improves then have him gradually move the card closer to the nose. He should build himself to where he can do 150 return trips, then he can work on the middle dot the same way, then finally he may work on the small dot. These fusion cards can be made yourself using colored art paper to cut out the dots and paste them on the white cardboard, or they can be ordered at a very nominal cost from Allbee & Son Company, Waterloo, Iowa, get Form 122.

There are a large number of other home training procedures in use, some commonly prescribed by most practitioners and others are pet procedures used by only a few. There is not time or space here to describe or discuss a large number of other procedures I know and use or have read of, some used for basic training and others for specific purposes. I am sure some of you have special training devices and procedures you already use that produce excellent results which I have not mentioned and probably never heard of, however there may be a few men that will derive some helpful information from this paper. I hope it will provoke some stimulating thought in all who read this.

Birmingham 6, Alabama

THE MEASUREMENT AND CORRECTION OF HYPERPHORIA

BY

WILLIAM D. DELLANDE, O. D.

Columbia, U. S. A.

I. One of the important considerations in a complete refraction routine is the measurement of hyperphoria. This paper shall describe a clinical method of examination for and measurement of hyperphoria. Discussion of correction will be confined to the consideration of the use of prismatic effect incorporated into ophthalmic lenses. No differentiation of etiology of the vertical phoria will be attempted. The categories of hyperphoria considered will be limited to the static and parietic classes (1). It should be understood, therefore, that no attempt at complete coverage of hyperphoria and its various means of correction is intended.

II. *Method of Measurement*

The sequence in the total refracting routine, of the measurement of hyperphoria is of significance for properly evaluating the status of the patients binocular functioning. In general the earlier the presence of a hyperphoria is uncovered the greater will be the validity of the other measures of refraction that follow.

A thorough measure of hyperphoria throughout the binocular motor field usually is not routinely done on all patients, so the indications of a thorough examination of the hyperphoria will now be considered.

There are a few cues that indicate the necessity for a complete study. They follow:

1. Presence of prismatic effect in the currently worn spectacles is due either to intent of the previous examiner or to improper vertical decentration of the lenses. In either event a complete vertical phoria investigation is in order to confirm the need for the prismatic effect.

(1) Sir W. Stewart Duke-Elder, *The Neurology of Vision* (St. Louis, The C. V. Mosby Company, 1949) Text-Book of Ophthalmology Volume IV, pp. 3970-3971.

2. The nature of the case history may directly indicate symptoms of vertical phoria or tropia or may suggest the possibility of their presence. Case history is not characteristically an indication of this defect, however. Specific complaint of vertical diplopia is not anticipated in the categories of vertical deviations under discussion.
3. In the author's refractive routine, ocular motility is measured before the retinoscopy is performed. Careful observation of the Patient's ocular excursions will sometimes indicate the existence of faulty performance of a vertically acting muscle or muscles by overtly observable vertical deviations by one eye from the line of the target used in testing the rotations. The author uses a muscle light as the corneal reflection it produces is of assistance in making this observation. The observation of a vertical phoria in any direction of movement of the extraocular muscles is greatly facilitated by the use of the cover test. The existence of a lateral phoria by itself or accompanying a vertical phoria in any direction of movement of the extraocular muscles is greatly facilitated by the use of the cover test. The existence of a lateral phoria by itself or accompanying a vertical deviation will, of course, render the objective observation more complicated. In any event, even the suspicion of a pure vertical deviation should lead to testing for the error by more definitive methods.
4. Even if failure to uncover a vertical deviation by objective observation occurs, as in the case of small static errors (present throughout the motor field) the measurement of the vertical phoria in the primary position of gaze farther along in the refracting routine through the refraction or trial frame, will indicate that verification should be pursued. The normal routine of refraction may then be interrupted to allow for the thorough investigation of the phoria, or if binocularity is intact and the routine can be completed, the testing can be done following refraction.

Now we shall proceed to consider the means of making a thorough investigation of a vertical phoria. The patient should be seated with his body and head comfortable erect and with the eyes positioned level. The Maddox rod either of red or white glass is used. Light source at 20 feet is a white square or circle of light. A muscle light is used for testing at the near range.

The distance test is made with the Maddox rod before one eye, the other eye viewing the target. When the horizontal line created by the Maddox rod is not vertically bisecting the light target, prism in various gradation are successively held with the rod, the base-apex line of the prism perfectly vertical (base in the indicated direction) until the horizontal line is made to appear to pass through the center of the light target. The cover test should then be performed to ascer-

tain if the line continues to bisect the target. If it does not, it is suspected that the patient has been utilizing some degree of fusion of the similar targets. The Maddox rod should be held before the other eye as the amount of hyperphoria may be variable in a parietic case depending on whether the deviation is primary or secondary.²

An advantage of the distance measure is that in hyperphoria subtended angle of separation of the line from the light source is greater at distance than near point and therefore affords a critical measurement of the defect. The distance hyperphoria may be measured throughout the field of motor action by having the position his head as desired.

The near measurement shall be considered only by two significant means. The *first* is with the Maddox rod and a muscle light. Again the line is made, by use of the proper prism, to bisect the light. Again the line is made, by use of the proper prism, to bisect the light bulb. The cover test again guards against fusion attempts. In addition to the measurement in the primary visual position the muscle light is held so that the patient is looking in turn in each of the six positions of maxim action of the extra-ocular muscles, and in the estimated habitual reading position and if needed in the peculiar positions required of the eyes in carrying out the occupational routine.

The Maddox rod must be held so as to be level at all times and so that the line of sight strikes the rod perpendicular to its plane. If these cautions are not observed the horizontal line created by the rod will be curved rather than perfectly straight.

The amount of prism for each direction of gaze is recorded. The findings should be confirmed by repeating the measurements with the Maddox rod before the other eye. The prism base will then be reversed. The degree of hyperphoria may vary depending on primary or secondary deviations.³

The *second* method of testing the vertical phoria is with a ruby glass held before one eye. Where diplopia exists in a given position of gaze it may be eliminated by the proper prism. In the event this cannot be accomplished due to a horizontal phoria the red and white images of the muscle light can be made level. The ruby glass test will *not* correspond exactly with the Maddox rod test and in the case of small errors may not produce any diplopia even though the Maddox test gave rise to a phoria measurement. Also, suppression may prevent the response of

(2) Richard G. Scobee, *The Oculo-rotary Muscles*. Second Edition (St. Louis, The C. V. Mosby, Company, 1952) p. 192.

(3) Richard G. Scobee, *The Oculo-rotary Muscles*. Second Edition (St. Louis, The C. V. Mosby, Company, 1952) p. 92.

diplopia of the similarly shaped images of the ruby glass test. For these reasons, the author prefers the Maddox test as a more provocative measure of hyperphoria. The ruby lens test however, is an important adjunct in considering whether prism or how much prism should be prescribed. It is valuable by indicating in which positions fusion of the red and white lights occurs. Where fusion is easily maintained throughout the motor field prism prescription may be contraindicated.

III. *Advisability of Correction*

As was pointed out in the introduction, considerations is given only to stable and parietic types of hyperphoria.

1. *Stable Hyperphoria*

Where the defect is approximately consistent throughout the motor field and of one prism diopter or more, prism should be considered. There are several factors to evaluate:

a) Patients Complaint and Symptoms:

If these are attributable to the phoria, correction should be made. Symptoms may vary from general discomfort of eyes to reports of occasional diplopia or blurring of vision. Other complaints are fatigue with close work and headaches with use of eyes. Greater ease of reading with one eye is diagnostically significant. Compensatory head postures, placing one eye higher than the other are sometimes seen in substantial hyperphorias.

b) Presence of Suppression:

As the degree of suppression increases, symptoms usually decrease. Judgment should be used before prescribing for a symptom free hyperphoric patient.

c) Associated Lateral Phoria:

Vertical are more difficult to overcome by fusional adjustments than lateral phorias. The presence of a lateral phoria which by itself might not be detrimental to fusion and binocularity can become unmanageable when compounded by the presence of hyperphoria. In a case of this type the correction of the vertical phoria would aid the patient by correcting one barrier to fusion, leaving only the management of the lateral phoria up to the patient.

d) Visual Acuity:

In a case where permanent visual acuity of the two eyes is sufficiently different to hamper bi-macular coordination, the presence of small vertical errors may be justifiably overlooked.

e) Age:

If the patient is of an age, either young or old, such that the subjective responses are questionable, hyperphoria measurement based on these responses

should be considered of dubious merit. Objective cover tests are of more value. Correction should be based on objective tests if a subjective and objective measurements are different.

f) Occupation:

Generally, the greater the demand for binocular vision of the particular occupation, more seriously should the examiner consider the use of vertical prism.

g) Limit of Correction:

Should the error be extremely gross, for example, over 10 prism diopters, the advisability of correction is questionable. Ordinarily there will be no symptoms, and the decision to prescribe for the error would have to be based on convincing factors.

2. *Paretic Hyperphoria* (hyperphoria variable throughout motor field)

We shall consider under this category patients who manifest at least one-half prism diopter in the field of minimum hyperphoria. The same things evaluated before correcting stable hyperphoria should be considered in this category. But in alleviating hyperphoria of a paretic nature, the correction will be effective maximally in some regions of the motor field and minimally in the regions where the defect is most pronounced. The minimal hyperphoria measured with the habitually fixating eye regarding the muscle light is generally the limiting factor in paretic cases. Exceptions will be considered in the next section.

IV. *Prescribing the Prism*

1. Stable Hyperphoria

Hyperphoria of one prism diopter or less characteristically does not elicit subjective complaints from the patient. Notwithstanding this, if the patient is in need of spectacles for constant wear and the ruby glass test, produces diplopia in significant positions, there are at least two conditions where incorporation of prism in the correction is justified. The first is the occupation as discussed previously. One-half of the error can be corrected to guard against binocular interference and possible future discomfort. The second condition is that of associated lateral phoria. Binocularity is susceptible to stress from lateral phoria and the incorporation of prism into the spectacles will assist the patient to combat weakening of binocularity with accompanying suppression.

In patients with one prism diopter of vertical error or more who show diplopia in the ruby lens test in significant positions of gaze, the correction of the error can be assumed to be of beneficial effect when glasses are to be

worn constantly. Exceptions for use of prism are for patients who prefer not to wear glasses constantly. Also some factors listed in considering advisability of correction, such as gross difference in visual acuity of the two eyes, would make the prescribing of prism ineffectual.

Where prism is used, the power may vary between full correction to within one-half to one diopter of the error depending on pertinent circumstances as discussed earlier.

2. Paretic Hyperphoria

Since the hyperphoria or phoria-tropia varies in the motor field, this fact dictates the amount of prism to be used. The minimum measurement of hyperphoria must be considered first.

- a) If the least error prevails in the primary position of gaze with habitually fixating eye viewing the muscle light, the correction should not exceed that amount of error. This assertion can be seen to apply to a hyperphoria present maximally in one lateral field of movement, as well as one that reverses hyperphoric eyes from one lateral field to the other. In the latter instance probably no correction would result, which indeed should be the proper disposition where the hyperphoria is right looking to one side and left in directing the eyes to the opposite side.
- b) If the minimal hyperphoria exists with the eyes directed to one extreme lateral position, the evaluation of amount of prism to be prescribed must be based on data concerning the patient's visual habits and the spectacle relationship to the patient's eyes.

A. First, in relation to visual habits, the extent of the lateral conjugate movements utilized in the patient's visual routines must be ascertained. The comparison of the difference in lateral conjugate sweep of a bookkeeper and a watch repairman illustrates the problem. If the lateral movement is wide, the maximum prism prescribable is limited by the hyperphoric measurement at the terminus of the lateral excursion. For example, the hyperphoria is zero with eyes looking somewhat to the right of center and a right hyperphoria develops as the eyes move horizontally toward and then into the left field. Further, at the primary position the right hyperphoria is two prism diopters and increases to a maximum of four in extreme levoversion. If the patient in his occupation utilized a lateral excursion extending 15° to each side of the primary position, the right hyperphoria should be measured with eyes looking 15° to the right side. The right hyperphoria at that point

might be one prism diopter and that would be the limit of the prism prescribable under existing occupational usage of the approximate limit of lateral version before head movement reflexly occurs)⁴.

- B. Secondly, the spectacle measurement is a limiting factor. In the example above if the binocular field of vision through the spectacles exists 25° to the right side, then very conservatively the hyperphoria measurable at this position would be prescribable. Bifocals are obtainable in widely varying dimensions and the limitation of lateral versions imposed by the segment is another consideration. A practical dictum to follow is to limit the prismatic power to the least hyperphoric measurement 15° from the primary position. This would probably be the motility limit but the prism power should preferably be slightly less than the total correcting prism at this 15° position.
- C. If a negligible hyperphoria exists in the primary position of gaze, but hyperphoria is present in the reading position (eyes directed downward), the refractive power of the lenses should be compared before prism in the reading fields is considered. If the resultant power in the vertical meridian is different for the two lenses, the prismatic effect resulting from looking below the optical centers of the lenses (reading position) must be calculated to determine if the effect corrects or exaggerates the hyperphoria. Often the error is nullified to some degree relieving the necessity to prescribe a prismatic correction in the lower part of the lens (slab-off or bifocal prism). If the error is increased by prismatic effect of looking down through the lenses, then prismatic power sufficient to correct the muscle error plus the spectacle error would require consideration.

Under item a) it was stated that the correction should not exceed the error present in the primary position of gaze. If this error is zero or of negligible quantity, but a substantial hyperphoria exists to one side, nothing can be done with prism. However, the patient should be advised to avoid looking to the side of the maximum error or to turn the head so as to avoid the eye position which is apt to place stress on the binocular mechanism.

3. Prescribing the Prism

Prismatic effect may be achieved in several ways, some of which are applicable only in special circumstances.

(4) Richard G. Scobee, *The Oculo-rotary Muscles*, Second Edition (St. Louis, The C. V. Mosby Company, 1952) p. 92.

a) Prism incorporated into the lens.

This is the most common form of prism used for vertical hyperphoria. The direction of the base of the prism may be specified by the examiner, commonly the base is placed up before the hyperphoric eye. The prismatic power may be shared between the two eyes in large hyperphorias.

b) Vertical lens decentration

In cases of strong lense correction and small vertical imbalance, the desired amount of prismatic effect may be achieved by decentering the ophthalmic lenses along the vertical meridians. The decentering effect in prism diopters multiplied by the amount of decentration in centimeters.

c) Prism in segment

In presbyopes when the amount of hyperphoria in the reading position differs from that in the primary position, this discrepancy may be corrected by prescribing prismatic power in the segments.

d) Slab-off prism

In single vision lenses, the same kind of adjustment achieved in (c) may be accomplished by the method of slab-off prismatic power. This method produces a base up effect in the lower part of the lens which is ground off.

e) Complicated vertical prisms

Lateral and vertical prismatic power may be incorporated into the same lens.

V Summary.

The scope of this paper has been limited to the common types of hyperphoria that are frequently encountered in practice.

Method of measurement of hyperphoria is of great importance if prismatic correction is to be considered. The amount of error in the significant directions of gaze, the reading position, and the habitually usable fields of mobility, should be known.

The presence of a hyperphoria does not always demand its correction. Many factors such as those considered under section III. must be weighed. The single most important factor contraindicating demand for prismatic correction alone is presence of suppression.

The correction of vertical deviations sets the stage for more precise and efficient binocular and bi-macular visual functioning.

Professional Building

PERSONAL EXPERIENCES WITH THE USE OF ALPHA-CHYMOTRYPSIN

BY

R. M. FASANELLA, M. D.

New Haven, U. S. A.

Above a picture of the world in the Air Travel Bureau in Barcelona, one can find the following words: "At first it was thought this was the end of the world. Now it has been found only to be the starting point." (This is a free English Translation, of course).

It seems rather fitting that Barcelona should be the starting point for another exploration. This time, some centuries later, the Barraquers started their pioneer work on the use of Alpha-Chymotrypsin in cataract surgery. As you know, Alpha-Chymotrypsin is a proteolytic enzyme obtained from mammalian pancreas.

You may recall that others used the trypsin in the eye in an attempt to dissolve blood and vitreous opacities. Some abandoned its use because the lens was noted to be dislocated.

It was Joaquin Barraquer a who was keen enough to foresee the potential good of what seemed at the time to be a major complication. One day, after an attempt to dissolve a large vitreous opacity with trypsin, he noted that the lens had become dislocated. At this point, instead of abaddoning the use of the drug as harmful, Dr. Barraquer set up a series of animal experiments to determine a safe concentration of Alpha-Chymotrypsin for chemical zonulysis. This substance, when injected into the anterior chamber of the human eye, seemed to have a selective lytic action on the zonule and, in the proper concentration, respected the remaining intraocular structures. This new adjunct seemed to open a new surgical field substituting a mechanical zonulotomy technique, employed up until this time, by a chemical one.

Ideally, in the beginning it was felt that:

1. Intracapsular extractions would be possible at any age.
2. Extracapsulars would be unnecessary and in consequence, the inherent complications of an extracapsular would be lessened, such as;
 - a. secondary cataract
 - b. iridocapsular synechias, and

c. accidents occurring during and after intracapsular extractions would be minimized or eliminated such as;

- a. tearing of the capsule
- b. iridocyclitis secondary to excessive traction
- c. loss of vitreous humor secondary to excessive traction or pressure and
- d. retinal detachment due to excessive traction or pressure.

As in all new work, sometimes the ideal cannot be achieved at first or at any time.

It is the purpose of my paper to review briefly the first fifty cases of cataract extraction, using Alpha-Chymotrypsin, that I have personally performed and analyzed.

I shall also comment on other difficult cases done at New Haven Hospital that I have personally observed.

My objective in this study was to try to determine the indications for the use of Alpha-Chymotrypsin, its contraindications, the pitfalls, complications in its use and the management of these complications. In a sense, this study might be compared to the work of an engineer on studies of metal tolerances.

I shall also try to point out in my comments how and where I think Alpha-Chymotrypsin works, what studies are now being done, and what may be the future trends in its use.

It must be pointed out that my thoughts and ideas have been flexible and changable as I have talked and written to colleagues. I am deeply indebted to the Barraquers whose clinic I visited and with whom I have interchanged many letters as problems presented.

I would like to express my gratitude also to Drs. Richard Troutman, (3) Robert Bedrissian, (4) Morgan Raiford (5) and Hervery Thorpe (6) along with many others for their thoughts and interchange of ideas.

The following plates summarize my personal experiences in the first fifty clinical cases in which I used Alpha-Chymotrypsin.

SUMMARY OF USE OF ALPHA - CHYMOTRYPSIN
IN 50 CLINICAL CASES

AGE GROUPS	RACE AND SEX
80—89=90	Race — All White
70—79=13	Sex — Males 23
60—69=15	Females 27
50—59= 5	(Oldest — 88)
40—49= 5	(Youngest — 11)
10—39= 3	

Fig. 1

ALPHA - CHYMOTRYPSIN

CATARACT TYPES

ADULT

<i>Senile</i>	<i>Traumatic</i>
Mature — 27	Mature — 2
Immature — 17	Immature — 1
CONGENITAL	
Mature — 1	
Immature — 2	

Fig. 2

COMPLICATIONS — PREOPERATIVE

SYSTEMIC (SEVERE)	LOCAL
Allergy — 4	Glaucoma — 4 (2 secondary)
Hypertension — 6	Corneal Scars — 3
Diabetes — 7	Myopia (high) — 3

Fig. 3

ANESTHESIA

GENERAL	7
LOCAL	43
LOCAL AND INTRAVENOUS	7
(ANESTHESIA INADEQUATE IN 5 CASES)	

Fig. 4

ALPHA - CHYMOTRYPSIN (ALL PEVYA) (3 BATCHES)

DILUTION	AMOUNT
1/5000 in 46 cases	1 cc. to 5 cc. (Average 1 to 2 cc.)
1/2500 in 4 cases	

TIME IN SOLUTION BEFORE USING — 1 to 3 hours

TIME IN ANTERIOR CHAMBER BEFORE LAVAGE — Average 3 minutes

ADDITIONAL ALPHA - CHYMOTRYPSIN — 4 cases

Fig. 5

*OPERATIVE TECHNIQUE**INSTRUMENTS*

Keratome and Scissors	47
Graefe Knife and Scissors	2
Scratch (2 keratomes were no good)	1

SUTURES

Chromic (2 was least number-average 5)	45
Silk (8 was largest number-average 5)	5

FLAP

Limbal in each case

EXTRACTION

Intracapsular (2 started as extra-)	48
Extracapsular	2

Fig. 6

POST - OPERATIVE MEDICATION

Pilocarpine	17
Atropine	13
Nothing	19
Unlisted	1

Fig. 8

*COMPLICATIONS**OPERATIVE*

Loss of Formed Vitreous	1
Presentation of Formed Vitreous With no Loss (Vitreous Lens Adhesion)	8
Pigment Loss	8

POST OPERATIVE (IMMEDIATE)

Hyphemia	5
Absent Shallow Anterior Chamber	4
Iris Prolapse	2

POST - OPERATIVE (LATE)

Wound Disruption (5 weeks post-op)	1
Detachments (1 month post-op)	1

Fig. 9

FACILITY OF CATARACT EXTRACTION

Easier	31
Not Easier	12
Questionable	7

Fig. 10*

* In many cases I had to use the erisophake to slow down and brake the delivery of the lens which presented rapidly. In a sense one is handling a "greased pig" or operating on more dislocated lenses. Fortunately, however, at the time most vitreous faces are intact.

OPERATIVE TECHNIQUE - PRESENT

-
1. Corneo-scleral incision with fornix based flap.
 2. Post placed sutures.
 3. Alpha-Chymotrypsin (1/5000)
(Injected below usually).
 4. Wait 3 minutes.
 5. Iridectomy or iridotomy.
 6. Erisophake filled with saline.
 7. New washing (optional).
-

Fig. 11

SUMMARY AND SOME CONCLUSIONS OF MY OWN FIFTY CASES

Clinically, in these fifty cases performed by me, I have lost *formed* vitreous once. This one seemed related to lens vitreous adhesions and poor anesthesia. In addition, in this case, despite the seven sutures, I had an iris prolapse.

WOUND SEPARATION

I have had one wound separation in a child of ten years of age. This child was diagnosed as having subnormal development as well as retrolental fibroplasia and nystagmus. The patient who was living in an Institution at the time, fought her eye medications and it is possible that trauma, rather than the Alpha-Chymotrypsin, caused the wound to become disrupted. The wound separation occurred approximately five weeks post-surgery. Discounting this case of late wound separation, I have had two iris prolapses, both of which were noted and repaired one day postoperatively. One of these cases had only two chromic sutures. In this patient, I used only two sutures because the patient was unusually cooperative and because the corneal incision and closure seemed so perfect.

In a letter from Joaquin Barraquer, he told me that he had a recent series in which he had wound separation at approximately the two-week period. Therefore, he was washing out the Alpha-Chymotrypsin more carefully and using more of the 7-0 silk sutures.

PIGMENTATION (EXCESSIVE)

On injection of the Alpha-Chymotrypsin with one particular bath of the Barraquer solution, I noted more pigmentation resembling that seen in operating on diabetics.

I noted less when I irrigated in only one site under the iris rather than four sites as I had done to this point. After I received a fresh bath of Alpha-Chymotrypsin, I noted even less pigmentation. No excessive pigmentation was noted in the four cases in which Alpha-Chymotrypsin was diluted to 1/2500. Therefore, I concluded that the mechanical processes an/or the standarization of the Alpha-Chymotrypsin might have been at fault. Clinically speaking again, I have seen no real iris atrophy as I would expect with loss of pigment. In this respect, I wondered how much of a part the irrigation and irritation of normal saline may have had the chemical contact and recontact of the iris by the needle used in inserting the Alpha-Chymotrypsin may play a part in this loss of pigment. These contacts therefore should be kept to a minimum.

RETINAL SEPARATION

To date there has been only one case of postoperative retinal separation in a patient in whom I used Alpha-Chymotrypsin. This patient had a mature cataract with a controlled closed angle glaucoma. I performed an uncomplicated intracapsular extraction with a broad basilar iridectomy. In this case there seemed to be no relationship to the use of Alpha-Chymotrypsin. There was no loss of vitreous or vitreous presentation. There seems to be a disagreement as to whether Alpha-Chymotrypsin will cause or lessen detachments.

I have looked for damage to the vitreous face, ciliary body and retina but have not been able to see any to date. Indirectly, I learned from Dr. Maumenee (7) that in the rabbit injected with Alpha-Chymotrypsin which has not had the solution washed out, some may have worked its way back to the retina damaging it.

ALLERGIC REACTIONS

I feel that I have to watch round pupils after the use of Alpha-Chymotrypsin somewhat more than those in which I have not used Alpha-Chymotrypsin. The

pupils seem more sluggish, but this could possibly be related to the trauma of the injection of the Alpha-Chymotrypsin and washing it out. Those eyes in which I used Alpha-Chymotrypsin seemed to stay a little redder than those without.

Personally, I have noted no allergic type reaction in the eyes where I used Alpha-Chymotrypsin a second time or in eyes showing allergies to other drugs.

For example I had a thirty-two year old white male with marked local eye allergies, preoperatively but did not show any increased eye allergy following the use of Alpha-Chymotrypsin.

EFFECT ON SUTURE MATERIAL

Alpha-Chymotrypsin has no effects in chromic suture material.

EFFECT ON CORNEA

Alpha-Chymotrypsin did not reactivate an interstitial keratitis with active "ghost vessels" not did it reactivate several scarred corneas. Although I used Alpha-Chymotrypsin on the eyes with preoperative corneal scarring, I avoided its use in four eyes that I had performed corneal transplants. In all four, intracapsulars were obtained *without* the use of Alpha-Chymotrypsin. The corneas remained clear.

EFFECT ON EXTRACAPSULARS

In two extracapsulars, which started as intracapsulars, it was possible to wash out the capsule almost in one piece. It was felt that Alpha-Chymotrypsin may have dissolved the zonules thus making the remaining capsule wash out somewhat easier.

SOME POSSIBLE INDICATIONS

At present, I personally do not feel that Alpha-Chymotrypsin should be used in patients under twenty years of age or in those patients sixty years or over. Alpha-Chymotrypsin has been of definite help to me in the twenty to forty age group. In the younger age group, the greater danger of vitreous loss is a deterring factor. This may partially be explained by the higher frequency of lens vitreous adhesions. In most patients, after sixty, unless the cataract is predominantly nuclear, I feel that Alpha-Chymotrypsin is of no benefit and may even cause a fragile capsule to rupture more easily because of the extra manipulation. However, Thorpe felt just the opposite in regard to mature cataracts. He felt there was a greater chance to obtain more intracapsulars with Alpha-Chymotrypsin.

In the intumescent cataract, I personally feel that the capsule and zonules are so weak that Alpha-Chymotrypsin is indicated in most cases of anterior and posterior polar, complicated (posterior cortical) and nuclear cataracts.

I personally favor the use of Alpha-Chymotrypsin in high myopes and those with potential or previous detachments. Many will disagree with me on this, I am sure.

For those who prefer sliding, selective insertion of Alpha-Chymotrypsin at the 12 o'clock position with the head inclined slightly backward so that the Alpha-Chymotrypsin can reach this area and stay in this area may facilitate the extraction. The head, of course, should be level during the actual extraction.

Selective sliding or tumbling may be facilitated by the selective injection of Alpha-Chymotrypsin in other areas.

In one case in which the selective sliding should have worked, it failed.

Alpha-Chymotrypsin should be fresh and not inactivated by heat such as probably occurred in one case in which a substitute nurse placed the enzyme in a hot autoclave unbeknown to me. It did not work in this case of course.

DIFFICULT CASES I PERSONALLY OBSERVED

Except for the first case, I shall describe briefly only the more unusual cases.

Alpha-Chymotrypsin was first used in New Haven Hospital in May, 1958 on a twenty-six years old male patient. This Alpha-Chymotrypsin was sent to me by the Barraquers. The operation was witnessed by many, all of whom felt that it was one of the most exciting events in many years in the field of ophthalmology. The operator feared that the lens might dislocate posteriorly; actually, the Bell erisophake was applied only once and with a tumbling maneuver the lens was delivered without incidence. The Alpha-Chymotrypsin was instilled thru the iridectomies as was originally recommended by the Barraquers. At that time it had been feared that the Alpha-Chymotrypsin might create poor wound healing or might destroy chromic sutures used in cataract surgery. Since then, however, our own studies with chromic sutures and Alpha-Chymotrypsin show no changes of the suture material. This was confirmed by experimental work of others.

The second case which I observed was that of a thirty year old white male with a congenitally dislocated lens. The patient has asthma and a high myopia. My colleague felt that this case was not a Marfans but that the remaining zonules would be resistant. Therefore, he felt that Alpha-Chymotrypsin might aid in the extraction of the lens. Alpha-Chymotrypsin did not seem to help. Vitreous was present and some was lost. More work with Alpha-Chymotrypsin in ectopia lentis may be indicated.

ATOPIC DERMATITIS

In a patient twenty years of age with atopic dermatitis and a mature lens, Alpha-Chymotrypsin did not help. Vitreous was lost and a flat chamber followed. Technical problems unrelated to the Alpha-Chymotrypsin were probably responsible. Fortunately, however, the final postoperative vision with correction was 20/30+4.

AGE GROUP 0 TO TEN

In a four-year old operated on by an excellent surgeon with a good assistant, the Alpha-Chymotrypsin was repeated and the zonules had to be stripped manually and vitreous was lost. Possible causes of failure here were vitreous lens adhesions and lack of freshness of the Alpha-Chymotrypsin (four hours or more old).

In a five-years old with a clinical diagnosis of mongolian idiocy, Alpha-Chymotrypsin seemed most helpful in performing an intracapsular extraction combined with a total iridectomy. However, when the second eye was operated on by the same team of surgeons, vitreous was lost. A vitreous lens adhesion seemed responsible in the case of the second eye. It is difficult to explain why in one eye there seemed to be a vitreous adhesion while in the other there was apparently none.

FINAL DISCUSSION AND CONCLUSIONS

The work of Bedrossian suggest that the enzyme softens or weakens the zonule without actually dissolving it.

Dr. Leon Stone of Yale, in a personal discussion, of the mechanism of the trypsins felt that the effect of these enzymes in correct concentration might be on the intercellular cement substance without dissolving the cells themselves. Over-simplifying this conception, we might consider the effect of a chemical agent attacking the cement holding bricks together but not destroying the bricks themselves. Barraquer and Bedrossian separately reported no microscopic changes in other structures of the eye. Bedrossian, however, concluded that although no microscopic changes were seen in other structures, they might be affected as in the zonules and still show no pathologic changes.

I could not find any details on the chemistry of the zonules. Examining the intracapsular lenses under the biomicroscope postoperatively, I noted no attachment of the zonules to the lens. This suggested to me that perhaps the weakest point might be at the insertion of the zonules to the lens proper. Franceschetti (8) feels this is the site of action. As yet, our pathologist has been

unable to help me on this point. As you may know, there are often detachments of the zonular lamella secondary to technical difficulties in preparing slides of the capsule and zonules.

Following the suggestion of Thorpe, I examined as many eyes as I could with the gonioscope and I felt that the break may have occurred in this place. However, in a few cases the break seemed to occur at the ciliary processes. Others have reported that in normal intracapsular extractions, usually the break is at the mid-point of the zonules. Some feel the mid-point is also where Alpha-Chymotrypsin works.

Bedrossian in careful studies on animal eyes concluded that corneal wounds are not affected when they are bathed in 1:5000 solution of the enzyme for thirty minutes nor was wound strength diminished after fourteen days (9).

However, when I told him about the excess pigmentation I was encountering and the one delayed wound rupture I had, Joaquin Barraquer wrote the following in December, 1958:

"I have not observed any loss of iris pigment and I should think it might indeed be a mechanical problem, just as you yourself suppose.

I have seen more cases of late loss of the anterior chamber lately, usually, ten or twelve days after operation. This might be due to a delay in cicatrization, but I am not sure about it and I am now trying to find its cause.

In so far as congenital cataracts are concerned, I had excellent results in my first series of April, May and June of 1958. There were round pupils and showed no greater difficulties than in older people.

My second series as you saw yourself presented difficulties with vitreous herniation. The third series in November was made with total iridectomies and the vitreous showed a greater tendency to prolapse. I do not understand this difference between my first case and the others. Perhaps it is due to a difference in the enzyme."

Dr. Barraquer concluded in his letter, "For the time being, I advise the use of the enzyme only in patients from *ten* to *fifty* or *sixty* years of age. In other words, I would not advise its use in small children or in old people."

There has been a loss of vitreous in 100% of the cases of operations on children under the age of ten using Alpha-Chymotrypsin. Dr. Paufigue (10) reported he had three cases, Dr. Franceschetti (11) two cases and Dr. Boyd (12) two cases. I do not know many cases Dr. Cogan has had of this type but he described operating on a child under the age of ten using Alpha-Chymotrypsin as "extreme suffering." (13)

BARRAQUER'S CONCLUSIONS

March-1959

In March, 1959, Dr. Barraquer wrote me the following: "As far as cicatrization of the incision is concerned, I think that Alpha-Chymotrypsin actually does produce a bit of delay (a certain subconjunctival filtration is seen more frequently). This delay is of no clinical consequence if the suturing is done carefully. The incision should be closed in two planes (with limbus based flaps or with fornix based flaps) and all the stitches should be covered by conjunctiva. It is advisable to use natural silk. It is essential to wash the incision well with saline solution after having applied Alpha-Chymotrypsin in the posterior chamber."

Dr. Barraquer continued, "with regards to the use of the enzyme in young patients, my experiences lead me to the following conclusions:

1. Alpha-Chymotrypsin produces a lysis of the zonules which is similar to that in older people.

2. The problem in the young is due to the tendency of vitreous to prolapse probably because the sclera is more elastic and compresses the vitreous humor when the anterior chamber is opened.

3. Zonulolysis should *not* be employed in children under ten years of age, except in experimental cases and in specialized centers in order to study attempt to overcome this complication. This, of course, only in blind eyes (i.e. in unilateral cataracts with irreversible amblyopia).

4. In the age group between ten and twenty, a skilled surgeon may employ the enzyme provided he has a good anesthesia and hypotony.

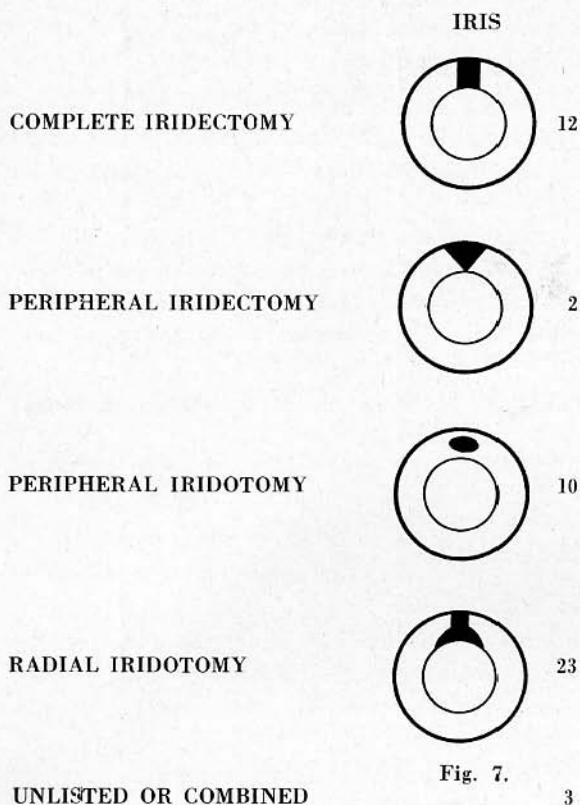
5. From the age of twenty onwards, I have not had any more difficulties than with senile cataracts except on rare occasions (some myopes).

6. The Alpha-Chymotrypsin must be freshly prepared in individual vials.

At present, I prefer to dilate the pupil with Neo-synephrine to about 8 mm., for sometimes washing the enzyme behind the iris contracts the pupil further. I am still using a 1:5000 solution. Higher dilutions have often proved ineffective. I think the suction cup is most ideal".

FUTURE TRENDS AND STUDIES

As you know, the Rockefellers have given many millions of dollars to charity and to investigative research. It is said that the best \$ 5,000 they ever gave was that given to Dr. Fleming who discovered penicillin. As you know, research and discovery of many other antibiotics blossomed from this "accidental discovery" by a man who had eyes to see. Today there are many new antibiotics. Likewise, there are many other enzyme preparations like Alpha-Chymotrypsin that remain



to be investigated. At our institution some of this work has already begun. The exact chemistry of the zonules, the lens capsule and the exact mode of action of drugs like Alpha-Chymotrypsin is still to be solved. We are all indebted to the Barraquers for all they have done toward the goal of making cataract surgery safer.

One day, it is hoped, "we will need speak no longer of management of eye surgery, but only of avoidance of pitfalls."

842 Howard Ave.

BIBLIOGRAPHY

1. VON SALLMAN, L., In discussion by Troutman, R. C.: "Alpha-Chymotrypsin", Manhattan Eye Ear and Throat Hospital, Eye Staff Conference, December 15, 1958.
2. BARRAQUER, J., Personal Communication.
3. TROUTMAN, R. C., Personal Communication.
4. BEDROSSIAN, R. B., Personal Communication.
5. RAIFORD, MORGAN, Personal Communication.
6. THORPE, HARVEY, Personal Communication.
7. MAUMENEE, A. E., Personal Communication.
8. FRANCESCHETTI, A., From: "Enzymatic Zonulolysis", *Highlights of Ophthalmology*, Vol. 3, n. 1, 1959.

LES ACIDES AMINES LIBRES DE L'HUMEUR AQUEUSE ET DU LIQUIDE INTERSTITIEL DU STROMA CORNEEN

PAR

J. FRANCOIS, M. D., M. RABAEY, M. D., N. RECOULES, M. D.

Gand, Belgique

Les données de la littérature concernant l'examen chromatographique sur papier et l'identification des acides aminés libres dans l'humeur aqueuse de l'homme ou de l'animal sont encore assez rares.

Le premier travail est celui de Blaha, Wewalka et Zwiauer (1953), qui comparent, chez le lapin, les acides aminés du serum sanguin à ceux de l'humeur aqueuse. Ils ne trouvent aucune différence significative, ni en ce qui concerne le nombre, ni en ce qui concerne la concentration des acides aminés, et concluent que ceux-ci parviennent dans la chambre antérieure par diffusion, plutôt que par sécrétion.

En utilisant 50 à 150 mm³ d'humeur aqueuse non traitée, ils trouvent que cette dernière contient normalement les acides aminés suivants: glycine, serine, alpha-alanine, leucine, acide glutamique, valine, lysine, arginine, tyrosine, proline et glutamine. En utilisant des plus grandes quantités d'humeur aqueuse (400 mm³), ils trouvent en outre: acide aspartique, acide alpha et gamma-aminobutyrique, thréonine, hydroxy-proline et phenylalanine.

L'hydrolyse acide de l'humeur aqueuse ne produit aucun changement important; le nombre et la concentration des acides aminés restent les mêmes; il y a cependant une augmentation de l'acide glutamique par transformation de son amide, la glutamine. Les auteurs concluent à l'absence, dans l'humeur aqueuse, de peptides à petites molécules.

Wunderly et Cagianut (1954), ainsi que Cagianut (1957) arrivent à des conclusions différents chez l'homme. Normalement, les acides aminés sont moins nombreux et moins concentrés dans l'humeur aqueuse que dans le serum sanguin; la même observation a d'ailleurs été faite pour le liquide céphalo-rachidien.

L'humeur aqueuse normale, déprotéinée par l'éthanol, contient surtout des acides aminés aliphatiques à poids moléculaire faible: c'est l'alanine qu'on trouve toujours en plus grande quantité; la leucine, la lysine, parfois même la méthionine et la théonine peuvent être identifiées, mais en faible concentration. Dans les cas pathologiques (irido-cyclites), on constate une augmentation générale des acides aminés et l'apparition d'acide glutamique, d'histidine et de valine.

Steiger (1957) étudie, chez l'homme, les acides aminés obtenus après hydrolyse des fractions protéiniques de l'humeur aqueuse, du cristallin et du serum sanguin. Il trouve des différences importantes: l'acide glutamique, l'arginine et l'alanine sont plus abondants dans l'humeur aqueuse, alors que la lysine est plus concentrée dans le serum.

Comme les résultats des différents auteurs ne sont pas entièrement concordants, nous avons cru intéressant de reprendre l'étude des acides aminés libres par la chromatographie sur papier non seulement dans le serum sanguin et l'humeur aqueuse, mais également dans le liquide interstitiel du stroma cornéen.

Technique

Nos différentes matières premières proviennent du boeuf.

Le *serum sanguin* est préalablement déprotéiné par l'acide trichloracétique à 20%.

L'humeur aqueuse est examinée:

- a) directement sans traitement préalable;
- b) après avoir été déprotéinée par l'acide trichloracétique à 20% ou par ultrafiltration.

Comme la concentration des acides aminés est moindre dans l'humeur aqueuse que dans le serum, il faut, pour obtenir un chromatogramme convenable, utiliser une plus grande quantité d'humeur aqueuse. Dans ces conditions, les sels en présence ont une action perturbatrice, qui trouble le déplacement des acides aminés. Aussi est-il nécessaire d'éliminer les sels à l'aide d'un échangeur d'ions. Dans ce but, nous utilisons l'amberlite I.R. 120 H. Les acides aminés sont ensuite entraînés par une solution ammoniacale 2 N et soigneusement isolés. Après évaporation à chaleur, le résidu étant dissous dans l'éthanol à 80%.

Les acides aminés du *liquide interstitiel du stroma cornéen* ne peuvent pas être séparés, sans traitement préalable à cause de la présence de sels. Mais, étant donné la quantité minimale de matière première, nous ne pouvons pas recourir à un échangeur d'ions. Force nous est d'utiliser une micro-technique pour l'élimination des sels: après avoir amené sur le papier 100 ul de liquide interstitiel de la cornée,

l'élution est faite à l'aide de méthyl-éthyl-cétone-5%. HCL⁰ N. L'extrait est évaporé et le résidu dissous dans 100 ul d'eau bidistillée.

Il faut à ce sujet remarquer qu'il n'est pas impossible que les acides aminés basiques soient parfois retenues sur l'échangeur d'ions utilisé, ce qui pourrait par exemple expliquer l'absence de lysine et d'arginine dans le liquide interstitiel de la cornée.

Por la chromatographie *en deux dimensions* (méthode ascendante), nous utilisons les quantités suivantes de liquide: por le serum 20 ul, 30 ul et 50 ul, pour l'humeur aqueuse 40 ul et 50 ul, por le liquide interstitiel de la cornée, 100 ul.

Les deux dimensions sont réalisées soit avec le mélange butanol-acide acétique et le phénol saturé d'eau, soit avec le butanol-pyridine et le système phénol-éthanol-amoniaque.

La méthionine est identifiée par la peroxydation oxydative.

L'emplacement des acides aminés est révélé par la réaction colorée à la ninhydrine.

Résultats

La concentration des acides aminés est relativement moindre dans l'humeur aqueuse (Fig. 1, 2 et 3) que dans le serum sanguin (fig. 4): alors que 30 ul de

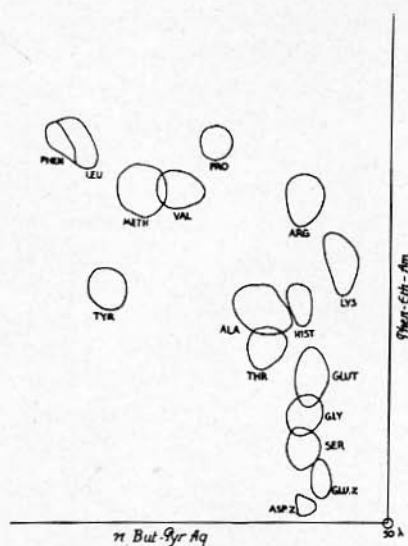


Fig. 1. Humeur aqueuse de boeuf (50 ul). Chromatographie en deux dimensions, sans traitement préalable.

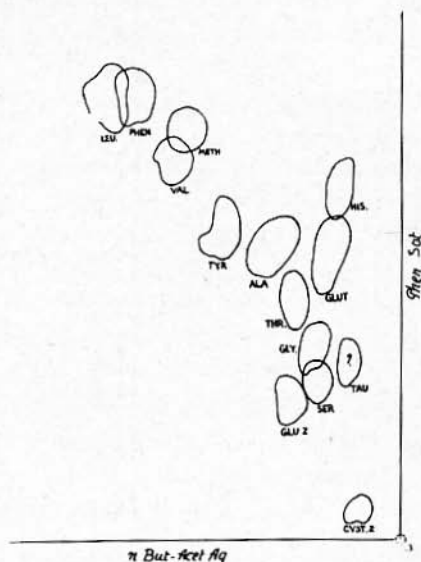


Fig. 2. Humeur aqueuse de boeuf (50 ul). Chromatographie en deux dimensions, sans traitement préalable.

serum suffisent pour obtenir un chromatogramme convenable, il faut 50 ul d'humeur aqueuse pour obtenir le même résultat.

Nous avons pu identifier un grand nombre d'acides aminés (18): acide aspartique, acide glutamique, sérine, glycine, thréonine, glutamine, tyrosine, alanine, lysine, histidine, arginine, proline, méthionine, valine, leucine, isoleucine, phénylalanine, peut-être histidine.

Le tableau suivant permet de comparer la concentration des différents acides aminés dans le serum, l'humeur aqueuse et le liquide interstitiel de la cornée.

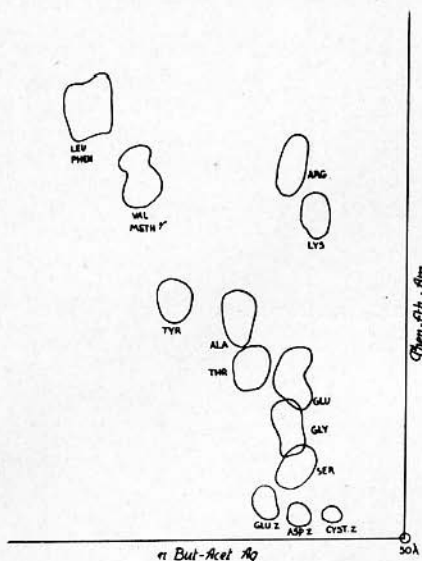


Fig. 3. Humeur aqueuse de boeuf (30 ul). Chromatographie en deux dimensions après traitement à l'acide trichloracétique.

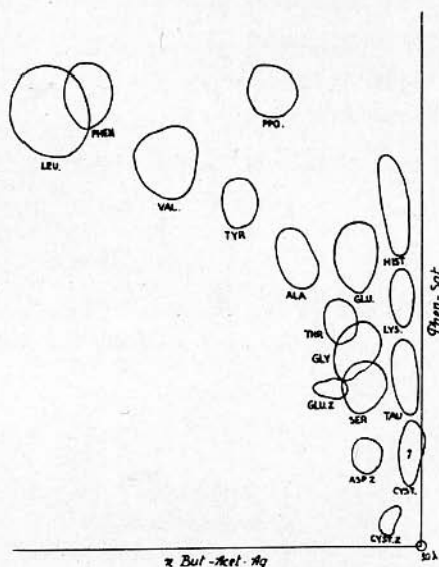


Fig. 4. Serum sanguin de boeuf (30 ul). Chromatographie en deux dimensions après traitement à l'acide trichloracétique et élimination des sels.

Dans l'humeur aqueuse (Fig. 1, 2 et 3) nous trouvons:

1. En forte ou très forte concentration: alanine et glutamine, méthionine, valine, leucine et iso-leucine, ce qui correspond en grande partie aux observations de Wunderly et Cagianut (1954) chez l'homme.
2. En concentration modérée: phénylalanine sérine et glycine.
3. En faible concentration: acide aspartique, acide glutamique, thréonine, tyrosine, lysine, histidine, arginine, proline, peut-être acide cystéique et taurine.

Dans le liquide intestitiel de la cornée, (Fig. 5), on trouve:

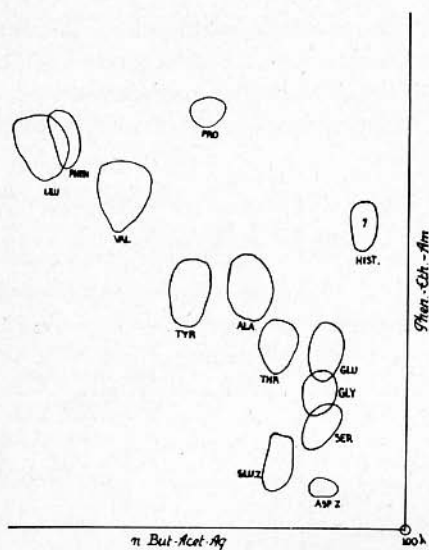


Fig. 5. Liquide interstitiel du stroma cornéen (100 μ l). Chromatographie en deux dimensions après éfution à l'acide de méthyl-éthyl-cetone 5%. HCL-6N.

1. En très forte concentration: alanine.
2. En forte concentration: glutamine et acide glutamique, dont la concentration respective varie, sans raison connue, d'un chromatogramme à l'autre, valine et leucine.
3. En concentration modérée: sérine et glycine.
4. En faible concentration: acide aspartique, thréonine, tyrosine, proline, phénylalanine et peut-être histidine.

De ces faits, nous pouvons conclure:

1. Chez le boeuf la concentration des acides aminés libres est plus petite dans l'humeur aqueuse et surtout dans le liquide interstitiel de la cornée que dans le sérum sanguin.

2. L'acide glutamique se trouve en beaucoup plus forte concentration dans le liquide interstitiel de la cornée que dans l'humeur aqueuse ou le serum. Il faut rappeler à ce propos que l'acide glutamique est un constituant du glutathion, qui participe aux phénomènes d'oxydo-réduction, et que, se trouvant au carrefour du métabolisme azoté, il occupe une position importante dans les échanges intermédiaires.

3. La méthionine se trouve en forte concentration dans l'humeur aqueuse, alors qu'elle est absente dans le liquide interstitiel de la cornée et le serum. Rappelons à ce sujet que la méthionine, le plus important des acides aminés soufrés, joue un

rôle physiologique considérable dans le métabolisme intermédiaire grâce à la trans-méthylation. Elle constitue, en effet, avec la choline, la principale source de groupements méthyles mobiles: c'est ainsi qu'elle est à la base de la transméthylation de la glycoyamine en créatine et de la diméthylcolamine en choline. Elle participe en même temps à la synthèse de la cystéine.

4. Quant aux autres acides aminés nous n'avons pas trouvé de différences significatives entre l'humeur aqueuse, le liquide interstitiel de la cornée et le sérum sanguin.

<i>Acides aminés</i>	<i>Sérum sanguin</i>	<i>Humeur aqueuse</i>	<i>Liquide interstitiel de la cornée</i>
Cystine	**	—	—
acide cystéique	—	*	—
acide aspartique	**	*	**
acide glutamique	**	**	****
taurine	**	** (?)	—
sérine	**	**	**
glycine	****	**	**
thréonine	**	**	**
glutamine	****	****	****
tyrosine	**	**	**
alanine	****	****	****
lysine	**	**	—
histidine	**	**	** (?)
arginine	—	**	**
proline	**	**	**
méthionine	—	****	—
valine	****	****	****
leucine	****	****	****
phénylalanine	**	**	**
iso-leucine	—	****	—

* = concentration très faible.
 ** = faible
 *** = modérée
 **** = forte
 ***** = très forte

Resumé

1. Chez le boeuf, la concentration des acides aminés libres est plus petite dans l'humeur aqueuse et surtout dans le liquide interstitiel de la cornée que dans le sérum sanguin.

2. L'acide glutamique se trouve en beaucoup plus forte concentration dans le liquide interstitiel de la cornée que dans l'humeur aqueuse ou le sérum.

3) La méthionine se trouve en forte concentration dans l'humeur aqueuse, alors qu'elle est absente dans le liquide interstitiel de la cornée et le sérum.

4. Quant aux autres acides aminés, il n'y a pas de différences significatives entre l'humeur aqueuse, le liquide interstitiel de la cornée et le serum sanguin.

Summary

1. In the ox, concentration of the free amino acids is less in aqueous humour and especially in interstitial liquid of the cornea, than in serum.

2. Glutamic acid is in much higher concentration in interstitial liquid of the cornea than in aqueous humour or serum.

3. Methionine is in high concentration in aqueous humour, but absent in interstitial liquid of the cornea and in serum.

4. With regard to the other amino acids, there are no significant differences between aqueous humour, interstitial liquid of the cornea and serum.

Clinique Ophthalmologique Universitaire

BIBLIOGRAPHIE

BLAHA H., WEWALKA F. et ZWIAUER A., Untersuchungen ueber die freien Aminosäuren im Kammerwasser des Kaninchens. *Klin. Mbl. Augenh.*, **123**, 552-555, 1953.

BLOCK R. J., R. LE STRANGE et G. ZWEIG, Paper chromatography (A Laboratory Manual). Ed. Academic Press, New-York, 1952.

CAGIANUT B., Die freien Aminosäuren im menschlichen Kammerwasser. *Documenta Ophthalm.*, **11**, 242-251, 1957.

CRAMER Fr., Papierchromatographie. Ed. Verlag Chemie, Gmbh., Weinheim|Bergstr., 1953.

LEDERER E. et LEDERER M., Chromatography. Ed. Elsevier, Amsterdam, 1957.

SMITH I., Chromatographic Techniques (Clinical and Biochemical Applications). Ed. William Heinemann, Londres.

STEIGER R., Die Aminosäuren der Kammerwasser proteine. *Documenta Ophthalm.*, **11**, 237-241, 1957.

WUNDERLY C. et CAGIANUT B., Free Amino-acids in the human aqueous humour. *Brit. J. Ophth.*, **38**, 357-363, 1954.

OPTOMETRIC VISION CARE OF THE PARTIALLY SEEING CHILD

BY

ALDEN N. HAFFNER, O. D.

New York, U. S. A.

For the past 40 years or so, society has become increasingly aware of its obligation to the partially seeing child. One recent estimate indicates that there are about 50,000 such children in the United States.

It is part of the American tradition in education that these children be given every possible opportunity to develop their abilities to enable them to make a maximum contribution to society, and to draw from it the richest possible life.

The methods utilized in achieving this goal for the partially seeing are in the process of change. When it was first realized that the visual handicap impeded learning in the normal classroom environment, special visual environments were created. These included the special classes for the partially sighted, utilizing large print books, large writing and typing, and the like. At best, visual care consisted of the use of a distance correction — to be used for all purposes — and whatever medical aid could be found to help.

However, more recently, it has been recognized that there are many desirable aspects to minimizing the time spent in the special classroom, provided the partially sighted child can adjust to the demands of the normal classroom. The more recent developments in the care and education of the partially sighted lean in this direction.

Visually too, there is much more that can be done to aid the child in meeting a normal environment successfully, than the traditional care previously indicated. This type of care is the optometric vision care that we are to discuss here today. It consists of three phases:

The first — an understanding of the child, his problems, and his way of meeting these problems.

The second — improvement of the situation by visual care NOW — and

The third — continued supervision of the visual situation to insure the ability to meet the continually increasing visual demands of maturation in our culture.

The improvement in visual care has been made possible through the development by optometrists, of many new types of magnification devices, known as telescopic and microscopic spectacles. These spectacles were developed originally, at the German School for Optometrists at Jena, and were used to aid the partially sighted. They were cumbersome and not especially attractive. About 25 years ago, Dr. William Feinbloom, an optometrist, developed an improved telescope which was easier to use while walking about, than those already available at that time. These are now obsolete — but they aroused the original interest in subnormal vision aids in the United States. Since then and especially in the past 10 years, optometrists have continually improved the efficiency and cosmetic acceptability of these devices.

Today, in general visual care, optometrists are stressing the importance of vision at near, and the concept of special glasses of bifocals for reading in certain school situations, is obtaining wider acceptance. This use of special near work glasses applies with even greater emphasis to the partially seeing. These students often attempt to hold material closer than the average to that the object appears larger. However, this close working distance, tends to place additional demand on the focus mechanism, so that sustained close work becomes either uncomfortable or reduced to a minimum. With adequate visual care, clear and comfortable vision can be achieved at the reading distance.

In addition, we should consider the concept of training and developing the facility of seeing. There is much that can be accomplished along the lines of developing visual functions in the partially seeing.

Through training, focus and fusion ability can be improved to that clear single binocular vision is obtained at a reading distance of say 8 inches. If a device is used, control of head movement, eye movement and book position must be learned. In some instances where perception is erroneous, so that E's and A's and the like are confused, this type of education is helpful.

The use of enlarged print results in a shorter eye span for each fixation, and consequently a larger number of fixations must be summated to create the perception of a phrase or sentence. Methods of application of optical aids to specific tasks are better learned under guidance.

And lastly, there are among the partially sighted those who do not wish to use the vision they have — and who should be taught or tutored for this reason.

Changes in these factors make programmed vision care rather necessary for the maximum educability of the developing partially seeing child.

What then should be done to provide visual care? The answer is obvious where the cooperation of the optometrist and educator works to the advantage of a youngster in their care. But what should the practitioner do with a partially sighted child?

He can first determine the patient's present status. A questionnaire for the non-achieving child has been covered very amply in a booklet prepared by Dr. Bing's American Optometric Association Committee, and applies very well in the present problem. Interviews with the parents alone, and with the child alone, may be necessary. The past history is helpful. Where does the child have a problem? When does he utilize vision, when not? What is his educational placement? How does he manage to see the board, to read his assignments at what distance? What are his play activities, his social side? This information is not intended merely to fill up space on a form in a filing cabinet. It has great significance — and its purpose is to permit the optometrist to visualize the daily life of the child as completely as possible. However, we stress problems of acuity, of field of vision and the like. Previous care recommendations are also bits of useful information.

Also a part of the visual status is an investigation of the patient's vision. The refractive state, the acuity, an idea of the cause of lowered acuity, the field of vision. It may be advisable to investigate the status of accommodation and of convergence. Of course, we should note the presence of active pathological processes and see that they are cared for.

After a complete examination, we must decide on a course of action. What can be done for the patient? What would be a tentative visual solution in view of his total situation? This solution should be tried and its effects noted. Is it used effectively on school books. Can the patient adapt to its limitations? Does he need education — does he need a partial correction for temporary use at first. Must he be guided on the basis of office control — or can he be given an aid to take home to try?

On the basis of several visits the effectiveness of the tentative solution can be observed and modified until the optometrist is ready to embark on a relatively long range program. The necessary visual aids are then prescribed and training or motivational program set up. The child should be under constant care by means of short progress study visits to insure maximum utilization of vision and visual aids. This type of program tends to eliminate loss of control of the situation, which does occur occasionally, and permits changes in prescribed aids or advice as needed.

As the ability of the child to utilize normal materials increases, his dependence on the resources room or sight conservation class will diminish. But still the child should remain under care in that he always has a future appointment — preferably but not necessarily specific.

Instruction may be necessary for methods of meeting specific visual situations. Personality and viewpoints often change, so that conferences can be necessary. Also as the child matures, especially in the lower grades, the size of type in normal school books gradually decreases, and the white space decreases as well, so that optical aids may be needed.

A last word on what type of optical aids may be needed. Although there is no strict relationship between Snellen acuity and legibility of running type because of other visual factors, the Snellen acuity can be used as a first approximation. For acuities down to 20/100 as an upper limit, high adds that permit comfortable reading at distances varying from 13 inches to 4 inches may be useful. From 20/80 down, either microscopic or telescopic spectacles may be used. A microscope is an efficiently designed magnifier for near, as its name implies.

11 West 36th Street

THE CLINICAL IMPORTANCE OF THE PERMEABILITY OF THE BLOOD-AQUEOUS BARRIER

BY

ALFRED HUBER, M. D.

Zurich, Switzerland

The blood-aqueous barrier, the mechanism controlling the passage of fluid and solved substances from the blood to the aqueous humour, represents only a special form of the barriers which exist all over the organism between the blood and the tissue-spaces. One common factor of importance forming these barriers throughout the body are the *capillary walls* consisting of endothelial cells, placed edge to edge to form a mosaic, the opposing edges being made tight by an intercellular cement substance which contains potential spaces acting somewhat after the manner of pores. Most anteriorly on the anterior surface of the iris the blood-aqueous barrier consists essentially of the uveal capillary walls alone. More posteriorly the barrier is composed of the walls of the uveal capillaries plus the twin-layered ectodermal prolongation of the retina, the *ciliary epithelium* which is interposed between aqueous humour and the capillary walls of the ciliary body. It is however important to recapitulate the evidence that *the capillary walls form the essential over-all blood aqueous barrier* and that the twin-layered epithelium interposed in one segment of the globe exerts a modifying influence only. In the ciliary body where the capillaries are extremely developed the surface of their walls amounts to about 670 mm². First of all some remarks concerning the qualities of the capillary wall. In the rest of the body small molecules pass through the intercellular spaces of the capillary walls - sodium, potassium, chloride, nitrate and urea almost as easily as water. Even such large molecules as inulin escape through the glomeruli of the renal circulation. This is a physical passage by diffusion through relatively inactive intercellular spaces. In the eye however we are dealing with entirely different conditions. The general delay in passage of all molecules in traversing the ocular capillary walls, the equality of the rate of passage of molecules of very different sizes, and the inequality of the rate of passage of those of comparable size, a difference depending essentially on their chemical composition,

all these considerations seem to confirm that in the eye *the transference from the blood into the anterior chamber takes place through the cell bodies of the capillary walls and not through intercellular spaces*. The general basis of the formation of the aqueous humour according to modern concepts seems to be a *diffussional interchange* with the blood through the endothelial cells of the capillary walls, an exchange at the same time constituting the background of the metabolic interchange. There is consequently an essential difference from the tissuefluids elsewhere, for in their case the diffussional interchange takes place through the intercellular spaces, which explains their relative richness in protein (50 per cent in the liver and the intestines in contrast to 0.02 per cent in the aqueous humour). What is the real reason of such a cardinally different behaviour of the blood-aqueous barrier? Probably it is concerned with the maintenance of an optically efficient organ. This necessitates optical clarity in its media and an internal tension sufficiently high to maintain the globe as a relatively rigid optical system. The peculiar impermeability of the ocular capillaries can be regarded as a teleological adaptation to maintain the ocular media optically clear and homogeneous. Thus the *process of transference from the blood into the anterior chamber*, taking place through cell bodies and not through intercellular spaces is essentially something *more subtle and discriminating than ordinary diffusion and presumably purposive*. However studies of the most varied nature in other organs of the body have consistently failed to produce any evidence that the capillary walls act otherwise than as a simple filter. They may *block* substances, but they have not so far been detected in the act of secreting any substance. Therefore it is on the one side easily to be understood that practically all the constituents of the intraocular fluid are in deficit when compared with their concentration in the plasma. On the other hand there exist three important exceptions not to be overlooked and neglected: *hyaluronie acid, ascorbic acid and salts*. Hyaluronic acid is undoubtedly a local secretory product, and the excess of ascorbic acid and salts implies that in their transfer from the blood to the anterior chamber of the eye energy must be expended, that is, they must be *secreted*. *On the fundamental process of diffussional exchange is therefore superimposed some secretory activity*, modifying the concentration of certain substances, which are essential for the peculiar physiology of the eye. The evidence from chemical analysis and auto-radiography indicates that the site of such a secretion is the ciliary region, and especially the *double layer of epithelium*, which allows the formation of differences in electrical potential (oxidative and reductive processes according to Friedenwald) so that some degree of unidirectional permeability and the capacity for maintaining concentration gradients exist. We have already mentioned that transparency of the aqueous humour as an optical medium is and must be maintained by excluding

substances which could make it opaque. This necessity on the other hand involves a significant deficiency of substances as for instance sugar, urea, amino-acids and proteins. This has to be made up. By a secretion of salts, the function of which is to maintain osmotic flow and equilibrium through the tissues, this is made up and more than made up, so that beside the capillary pressure there is enough excess of osmotic pressure to maintain the globe as relatively rigid optical system. It is not certain, whether other substances are involved in this secretory mechanism of the ciliary epithelium, but so far as our knowledge goes at present, definite proof is lacking. *In its essentials the mechanism of the aqueous production and thus the action of the blood-aqueous barrier is a wonderfully working combination of diffusional exchange in the main and secretion as a superimposed process.*

The title of our paper promises indications with regard to the *clinical importance of this blood-aqueous barrier*, the physico-chemical and physiological properties of which have been just summarized in a few remarks. Undoubtedly there is in daily clinical work the necessity of controlling or even measuring the permeability of this barrier. Why? Because numerous pathological conditions above all of the anterior segment of the eyeball are accompanied or even directly or indirectly caused by disturbances of the permeability of the blood-aqueous barrier. Understanding of its function under normal and pathological conditions represents a key to better etiologic and diagnostic understanding, but also to better therapeutical management of diseases of the anterior uveal tract, as the following statements will confirm.

But how may the ophthalmologist having not a big laboratory at his disposal acquire a bedside knowledge of this barrier? Let us call to mind *Ehrlich's* pioneer experiment in 1881, who for the first time could observe the appearance of the green dye *fluorescein* in the anterior chamber of the rabbit after having injected it subcutaneously a short time before. Fluorescein consequently has been used on animals and men for about sixty five years without furnishing practical results of clinical value. In 1945 we elaborated together with Amsler a standardised technique suitable for measuring this permeability for clinical purposes with the aid of the slitlamp and called this method "*fluorescein test*".

The principle of this test is the following: an intravenous injection of 2 cc. of a 10 per cent solution of sodium fluorescein is administered and stop watch set in motion. At a variable period from one to six minutes thereafter, the green colour of the dye becomes apparent in the pupil, in other words the green fluorescence generated by the very bright beams of the slit-lamp. The increasing fluorescence of the aqueous is measured at suitable intervals. The intensity of fluorescence has

to be inversely proportional to the quantity of light required to make it visible, quantitative measurements are made by continuously increasing the resistance in the slit-lamp circuit and thus reducing the light in the anterior chambre until the colour is just not visible. Repeated readings are made (first each minute, then every two, then every five) from a amperemeter and a curve constructed therefrom giving the relationship in time-amperes, in other words, timefluorescein concentration over a period of thirty minutes.

Having performed more than three thousand such fluorescein tests in the past fifteen years we can only confirm its absolute harmlessness and certify that fluorescein administered in the quantity and concentration as mentioned already proves to be completely indifferent from the pharmacological point of view.

In the normal eyes of healthy individuals it is found that there is a *normal curve of fluorescein-permeation* which varies within narrow limits, showing a slow and regular increase in the passage of the dye across the blood-aqueous barrier during the period of observation. In these normal cases the excretion of fluorescein is almost the same in both eyes, and the day-to-day variations lie within the so called *normal band* which has been established by examination of two hundred healthy eyes. The dye remains in the chamber at the level reached at the end of the observation time of thirty minutes for a few hours to fall thereafter slowly and gradually, disappearing completely from the anterior chamber after fifteen to twentyfour hours.

In pathological conditions the permeation of fluorescein is always increased, though the degree of this increase is variable. Also the type of curve is different: here we distinguish two forms of pathological curve, the one resembling normal curves, but situated above the normal band and indicating according to our experience only slight and reversible damage to the blood-aqueous barrier, the other characterized by a steep initial phase changing into a high flat plateau manifesting an intense and most frequently irreversible lesion of the barrier.

The moment has come when some remarks must be added concerning the *meaning of the fluorescein transport through the blood-aqueous barrier*. An escape of intravenously injected fluorescein occurs throughout the capillaries of all the tissues of the body; but there, where the permeability of the endothelial wall is relatively much higher, the escape is so free and rapid that in general terms the permeability to fluorescein measures not capillary permeability but the rate of blood flow, that is, it gives an indication of the quantity of fluorescein available to escape from the circulation rather than the difficulty it experiences in doing so. We have already mentioned that the capillaries of the eye are distinguished from those elsewhere by their relatively great impermeability. Only a small proportion of the fluorescein in the blood traverses

their wall and it does so slowly. In fact we could prove that *in the normal eye the concentration of the dye in the aqueous averages only about 1/100 of that in the blood.* In abnormal conditions however, this amount is increased, but without corresponding increase of the freely diffusible rate of fluorescein (not adsorbed to the plasma proteins) in the blood. *Hence the increase of the fluorescein-permeation into the anterior chamber reflects a dilatation and above all an increased permeability of the ocular capillaries,* in other terms, a breakdown of the normally high impermeability of the blood-aqueous barrier. Thus the fluorescein test may be considered really as permeability test of this barrier, realizable by means of a set of simple instruments and representing so to say a bedside procedure.

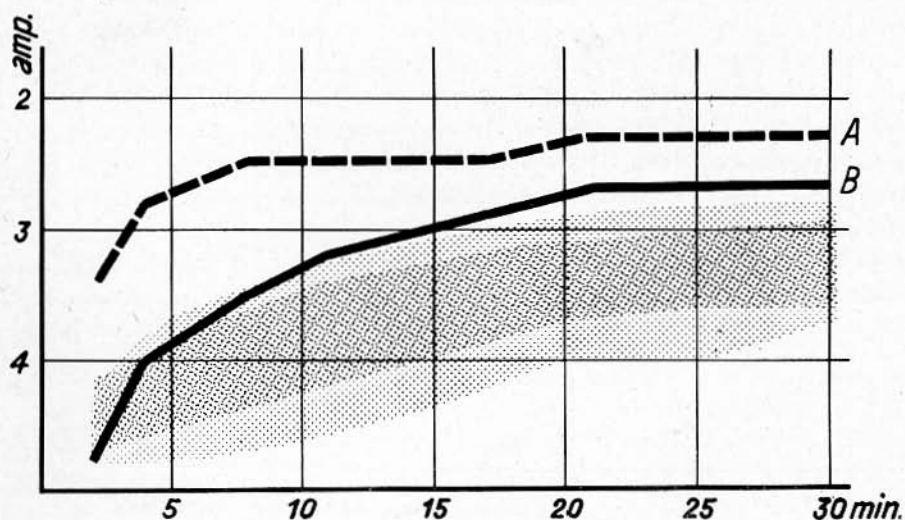


Fig. 1. *Anterior uveitis:*
 A: increased fluorescein-permeability of the affected eye
 B: normal permeability of the healthy eye

The changes in permeability which occur in pathological conditions are a considerable importance. The prototype of increased permeability is to be observed in acute or chronic *iridocyclitis*, where the curves rise steeply to an abnormal height indicating a comparatively intense breakdown in the barrier. Repeated fluorescein test enable us to follow the evolution of the permeability in the course of the disease as influenced by time and especially also by treatment. In the very early stages of anterior uveitis, when typical clinical signs are still missing, the fluorescein permeability may already prove to be increased and thus help to confirm the diagnosis, a fact, which is of great importance for instance in the detection of early signs of sympathetic ophthalmia. In hetero-

chronic cyclitis the permeation of the dye into the anterior chamber is especially exaggerated. In hypertensive uveitis there is a distinct increase of permeability too, but no relationship to intraocular pressure.

In cases of *keratitis* a positive fluorescein test indicates existence and degree of a simultaneous uveal affection, whereas in cases of *cataract* it may prove their iridocyclitic origin and help confirm their secondary complicated character. After intraocular operations practically always a slight increase of permeability remains for ever as a sign of by all means unimportant barrier lesion.

In primary simple *glaucoma* there is also a tendency to increased permeability for fluorescein. The increase may be sudden and irregular and is typical of a high or decreasing phase of intraocular pressure. This is an indication that a fundamental feature of such cases may be an alteration in capillary function. A very high permeability increase in a case of exaggerated intraocular pressure speaks in favour of a congestive glaucoma or even rather of hypertensive uveitis. At any rate such facts tend to blur the classic distinction between simple and congestive glaucoma, at least what concerns the vascular types.

As an interesting demonstration of the unity of the entire intraocular circulation it must be mentioned that an increase of fluorescein permeability occurs also in diseases of the posterior segment, not only in *chorioiditis*, but also in affections of the retina such as *venous thrombosis*, *periphebitis retinae* and *retinal detachment*. In the differential diagnosis between *glioma* and *pseudo-glioma* a negative fluorescein test points to the former, a positive one to the latter possibility.

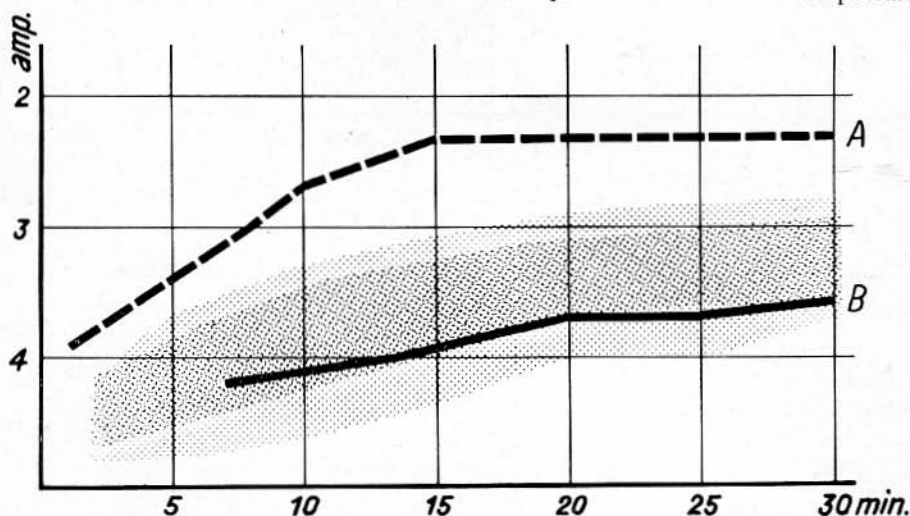


Fig. 2. *Contusion*:

A: increased permeability of the injured eye
B: normal permeability of the healthy eye

Injuries to the globe are also of significance in respect of alteration of the blood-aqueous barrier. A rapid rise to an increased height follows *contusions* or a *penetrating wound*. In contusion even of the severe type the increase of fluorescein permeability is limited strictly to the affected eye, whereas in perforating injuries we may make the very important observation that the fellow eye, although apparently normal from the clinical point of view, shows a similar however moderately increased permeation of the dye, the curve in this case following faithfully the fluctuations found in the injured eye. We call this phenomenon "*sympathetic dyschoria*" and consider it as a very early stage of sympathetic uveal irritation, which in the majority of the cases is reversible, but under unfavourable circumstances might develop into real sympathetic ophthalmia.

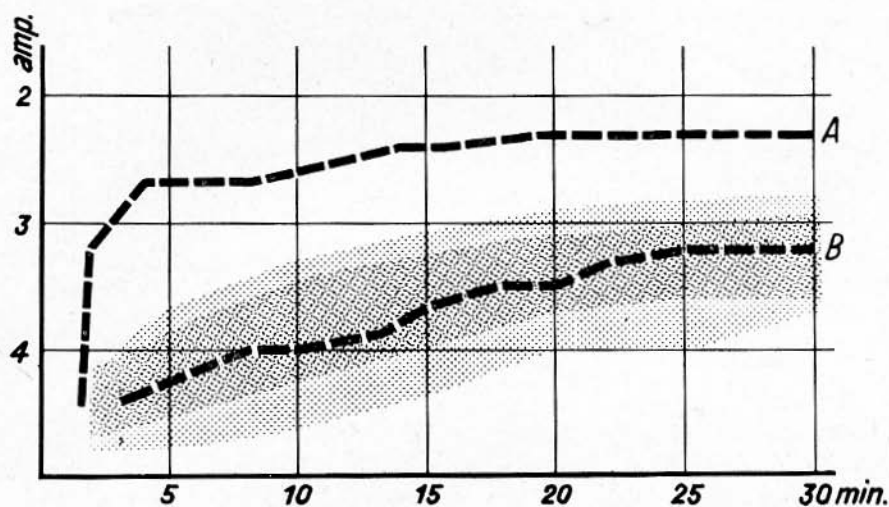


Fig. 3. *Perforating injury:*

- A: increased permeability of the injured eye
- B: normal permeability of the healthy eye

The phenomenon of the "*sympathetic dyschoria*" although up to now were not able to observe its transition into fully developed sympathetic ophthalmia, nevertheless dictates our course of action: the healthy eye repeatedly showing normal permeability of the blood-aqueous barrier we rather feel reassured and continue conservative treatment of the injured eye; but when it manifests a sudden rise in the curve or curves that are clearly and persistently high above the normal level, we tend to remove the perforated eye. For increased permeability, as we have seen, may be a preclinical sign of incipient anterior uveitis; sympathetic ophthalmia however being a special form of anterior uveitis it is obvious to suppose the beginning of a similar process in the exaggerated fluores-

cein permeation of the healthy eye. It cannot be only a consensual phenomenon for different reasons: it only occurs in 20 per cent of the cases, it is restricted to perforating injuries and is never to be observed in a healthy eye where the contralateral side shows increased permeability due to simple iridocyclitis, keratitis, etc.

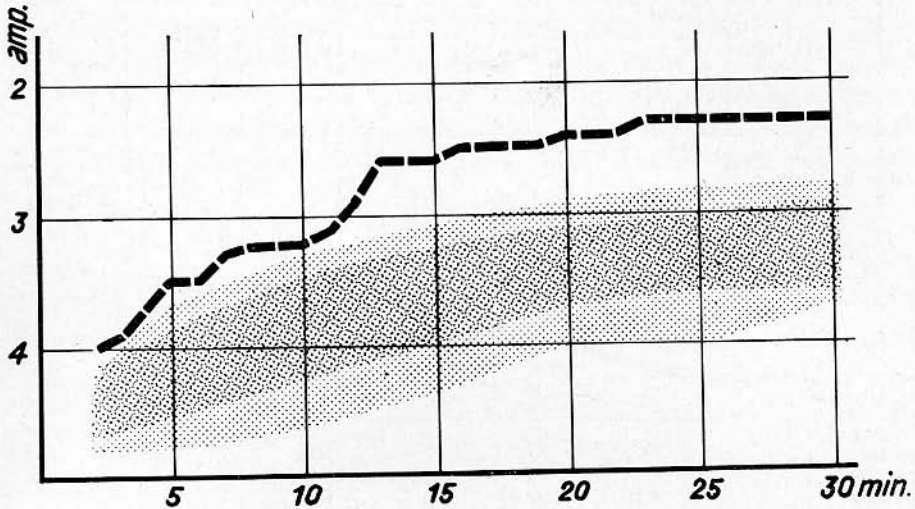


Fig. 4. Increased fluorescein-permeability of the blood-aqueous-barrier in case of *endogenous exzema*

Of great clinical importance is the fact that *general diseases* also alter the permeability of the blood-aqueous barrier as if this barrier like a very sensitive amperemeter was intercalated into the general permeability processes all over the whole organism. Thus the fluorescein test becomes a valuable method for evaluating the permeability degree of the capillaries not only of the iris and the ciliary body, but also of the body throughout. In about fifty per cent of the cases of *diabetes*, there is a slightly heightened ciliary permeability, even when no signs of retinopathy are present. In Kimmelstiel-Wilson's syndrome when renal insufficiency is added to the diabetic diathesis the increase of fluorescein permeation becomes greatly accentuated. A similar increase is seen in *malignant hypertension* whereas the benignant stage of essential hypertension does not manifest any change of permeability of the blood-aqueous barrier. Here the fluorescein test may help to indicate in a rather early stage the tendency for transition from benignant to malignant hypertension the knowledge of which is so important for our colleagues of the interbal medicine. Even when no intraocular complications exist, however, a similar breakdown in the barrier

BLOOD - AQUEOUS BARRIER

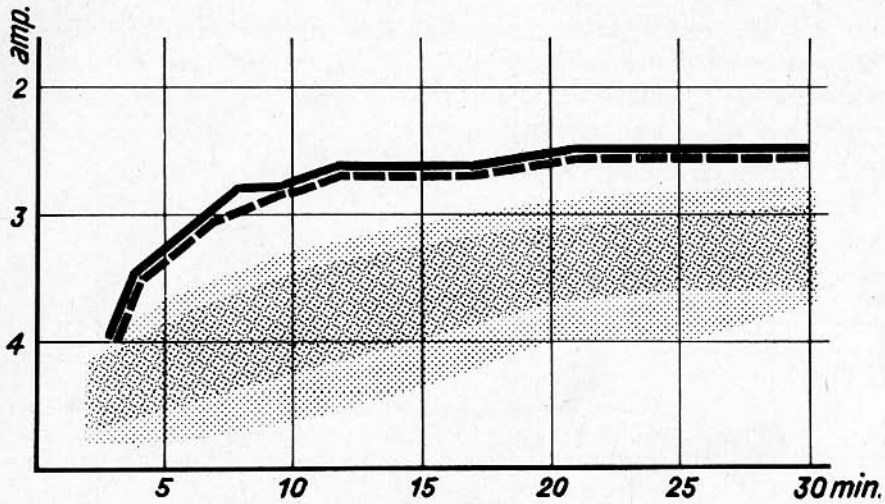


Fig. 5. Diabetic retinopathy: increased permeability on both eyes.

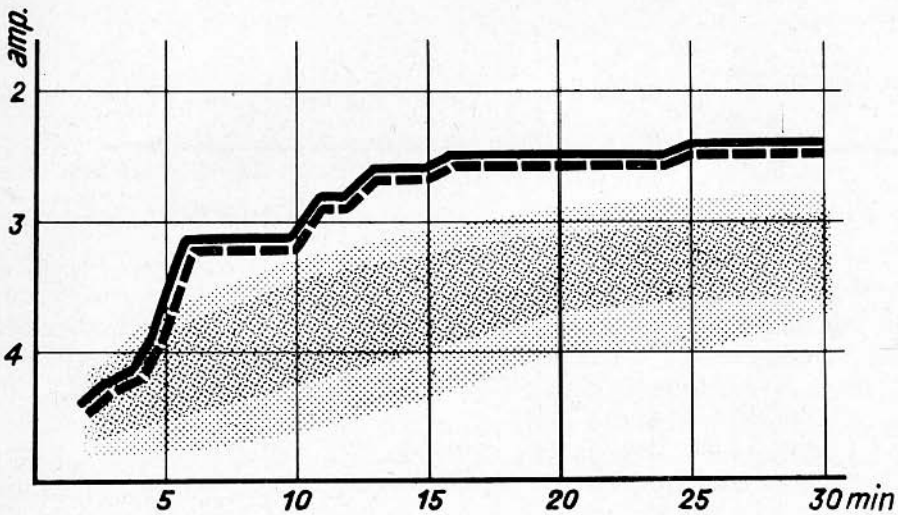


Fig. 6. Malignant hypertension: distinctly abnormal fluorescein-permeability of the blood-aqueous-barrier

frequently occurs in *metabolic diseases*, such as obesity, hyperthyroidism or Addison's, in *oedematous states* whether renal, lymphatic, angioneuritic or premenstrual, in *allergic conditions* such as eczema or bronchial asthma, or in *acute general infections* such as polyarthritis rheumatism or epidemic hepatitis. These facts confirm again the intimate relations existing between the organism as a

whole and that transparent organ with which we are concerned and in which we can observe the effects of vegetative disturbances, elsewhere in the body deeply hidden in opaque tissue, as if they were exhibited in a shop window!

Finally the fluorescein test lends itself to various *pharmacodynamic experiments*, as drugs of different types administered locally or generally may vary the degree of the permeability of the blood-aqueous barrier. *Pilocarpine* and

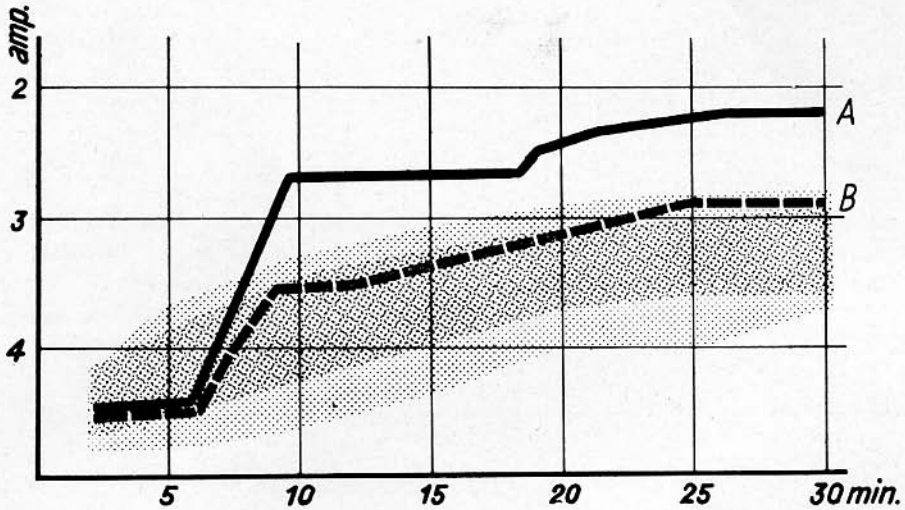


Fig. 7. Effect of *pilocarpine* 2% in for mof drops: distinct increase of permeability (A) in contrast to non treated eye (B)

eserine cause a heightening of the curve, indicating an increased capillary blood-flow and especially an exaggerated permeability. *Atropine* has no marked effect. *Adrenalin* and *laevoglucosan* lower it and may bring down the abnormally high excretion curve of an anterior uveitis. Subconjunctival injection of *saline* will raise the curve if concentrations over 3 per cent are used. *Cortisone* in form of drops or subconjunctival injections has a significant effect on the permeability of the ciliary vessels in the sense of definitely reducing it, above all if it has been already heightened by an inflammatory process. A similar, but far less intensive decreasing effect can be obtained by intravenous injection of *calcium*, in strong doses (20 to 40 cc of a 20 per cent solution) or of a combination of calcium with antihistamine, which may often lower the abnormally high permeability curves of iridocyclitis, chronic glaucoma and concussed or perforated eyes. Its action is almost immediate, but does not last for more than a few hours. Calcium on the other hand has no effect at all on the permeability of normal healthy eyes which proves that a certain normal permeation must be

BLOOD - AQUEOUS BARRIER

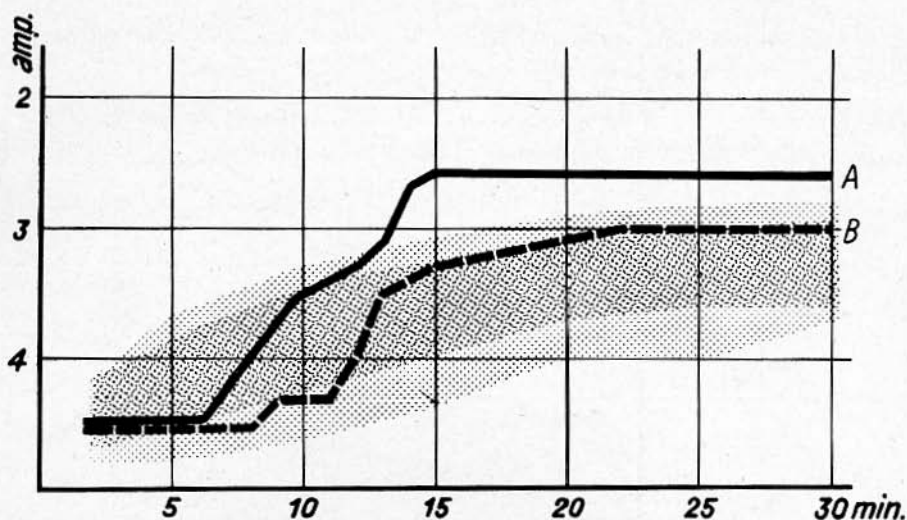


Fig. 8. Increase of permeability after injection (subconjunctival) of sodium chloride 3% (A). B: control eye

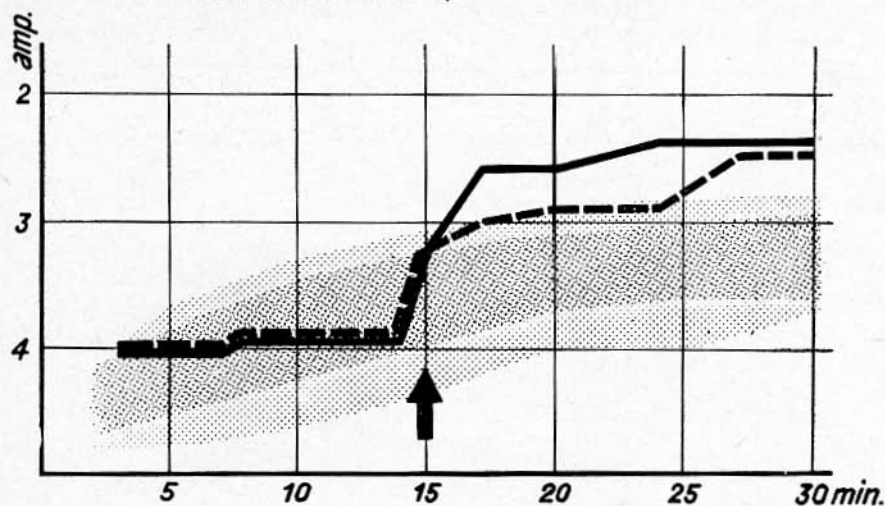


Fig. 9. Effect of Cortisone (subconjunctival injection) on the permeability of the blood-aqueous-barrier
A: before Cortison. B: after application of Cortisone

maintained in any case in order to guarantee a minimal metabolic interchange between blood and aqueous humour. An exactly opposite action is achieved by *histamine* which may suddenly open an even normally functioning barrier to high degrees of increased permeability. The same can be said about *hyaluronidase*

which locally administered produces as a rule a distinct increase of fluorescein permeation into the anterior chamber.

The fluorescein test of the permeability of the blood-aqueous barrier represents an artificial procedure which uses a rather small molecule, i.e. the fluores-

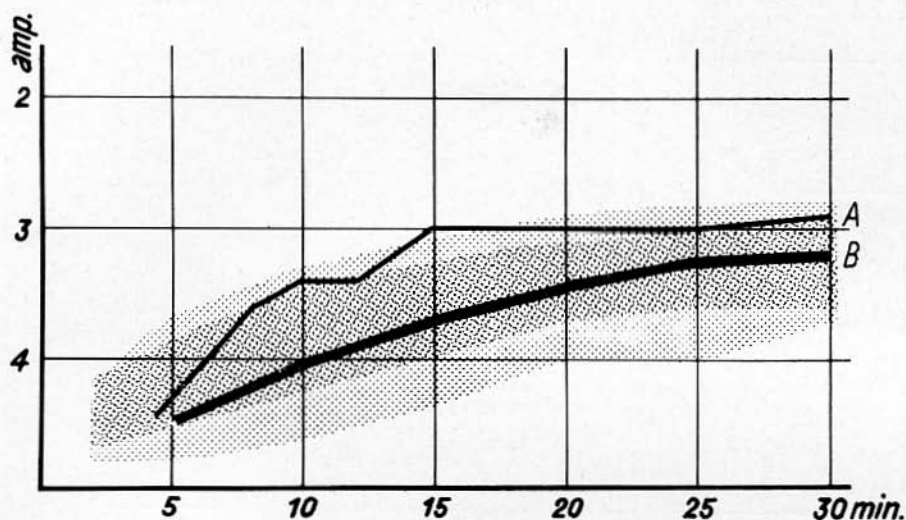


Fig. 10. Calcium diminishes the fluorescein-permeability of the blood-aqueous-barrier.

A: before. B: after intravenous injection of 20 ccm 10% Calcium

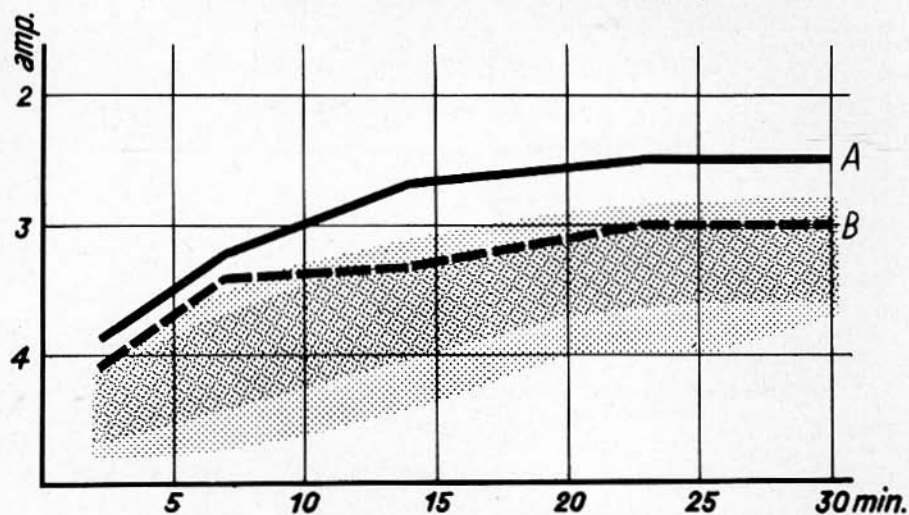


Fig. 11. Effect of histamine (subcutaneous injection) on permeability of blood-aqueous-barrier

cein molecule, for detecting disturbances of the permeation faculty of the capillary walls. This has the great advantage of being a rather accurate method able to find out even early and minute permeability disorders. Nature however has given us in addition in the eye so to say a natural permeability test, which we all know under name of the *tyndall phenomenon*. Increase of the permeability of the blood-aqueous barrier in a certain advanced stage leads to permeation of even big molecules as proteins into the aqueous: they produce in the light beam of the slit lamp by means of diffraction the tyndall phenomenon the intensity of which is proportional to their concentration and can be measured by help of a special tyndallimeter. Values of tyndall intensity plotted at varying dates on a diagram furnish a so called *tyndallogram* which gives valuable information about the fluctuations of the protein content of the aqueous and at the same time of the blood-aqueous permeability for proteins in the course of a certain disease.

Fluorescein test and tyndallogram, they both represent clinical possibilities of measuring and evaluating the permeability of the blood-aqueous barrier the importance of which in daily clinical and practical work - as has been demonstrated - cannot be denied.

Stadelhoferstr. 42

REFERENCES

- AMSLER, M. (1946). Bull. Mém. Soc. franç. d'Ophthal., 59, 304.
 AMSLER, M. (1948). Trans. Ophthal. Soc. U. K. (Bowman Lecture), 68, 45.
 AMSLER, M., and HUBER A. (1946). Ophthalmologica, 111, 155.
 AMSLER, M., and HUBER A. (1947). Bull. Mém. Soc. franç. d'Ophthal., 60, 359.
 AMSLER, M., and HUBER A. (1949). Graefes Arch. f. Ophthal., 149, 578.
 AMSLER, M., VERREY, F., and HUBER A. (1947). Schweiz. med. Wochenschr., 77, 1321.
 DAVSON, H., and QUILLIAM, J. P. (1947). Brit. J. Ophthal., 31, 717.
 GOLDMANN, H. (1949). Ophthalmologica, 117, 240.
 HUBER, A. (1947). Ibid., 114, 217.
 HUBER, A., and WALSH, J. (1949). Dermatologica, 98, 242.
 MORONE, G., and NIZETIC, B. (1949). Boll. d'Ocul., 28, 403.
 STOCKER, F. W. (1947). Arch. Ophthal., 37, 583.

ALGUNAS CONSIDERACIONES SOBRE VISION SUBNORMAL

POR

HERNANDO HENAO R., O. D., CARLOS TELLEZ D., O. D.

Bogotá, Colombia

Podemos considerar como caso de visión sub-normal, aquel que teniendo en uso la mejor corrección con lentes comunes, no alcanza suficiente habilidad visual para desempeñar trabajos industriales o de hogar, para los cuales la visión es esencial. Dichos casos, por lo general, presentan reducciones considerables en el campo visual, y su agudeza es inferior a 20/200. También hemos considerado como tales, casos con agudeza de 20/80 o 20/70, y que por presentar muy pequeña eficiencia visual, también se han beneficiado con aditamentos especiales.

El caso de visión sub-normal se analiza siempre teniendo en cuenta la eficiencia visual presente, no la agudeza en sí; un paciente puede presentar un bajo nivel de agudeza en términos de medida, pero su habilidad visual puede ser superior a lo esperado dada su baja agudeza. Así, por ejemplo, una mesa puede ser interpretada a una distancia de 5 metros como un bulto borroso, pero siempre ser considerada como un obstáculo. El paciente no necesita conocer perfectamente su forma, para definir ciertas características y su situación.

DESARROLLO HISTORICO

Hace más de 200 años se concibió por primera vez la idea de ayudar a personas de baja percepción visual, aprovechando la magnificación producida por sistemas ópticos compuestos como por Telescopios y Microscopios; sin embargo estos intentos no cristalizaron hasta el siglo XX, en el cual se logró resolver los múltiples problemas que presentaba su adaptación correcta. Fue Moritz Von Rohr, quien aplicó por primera vez el Telescopio de Galileo, construido y usado en forma de anteojos. En un principio estos Telescopios tan solo fueron prescritos en míopes altos, lográndose por lo general un considerable cambio en la agudeza visual, no solo por la magnificación inducida, sino también porque

se incorporaba la corrección refractiva del paciente dentro del mismo sistema focal. El primer Telescopio ensayado tenía un poder de 1.8x.

En 1920 una compañía de los Estados Unidos, diseñó un Telescopio de mayor poder (2,2x) el cual benefició otros casos, pero siempre el campo visual era considerablemente reducido. En el año de 1952 el Dr. William Feinbloom presentó su Telescopio denominado "de imagen clara" con un poder 2,2x, permitiendo un campo visual de 22° y siendo el más liviano presentado hasta entonces en su clase, ya que fue fabricado de material plástico.

Hacia la misma época la Kolmorgan Optical, presentó uno de características muy parecidas al de William Feinbloom con poder de 1,7x y el cual proporcionaba un campo visual relativamente amplio. Diversos Telescopios se han construido en los últimos años, por laboratorios alemanes, franceses, ingleses, japoneses y canadienses, con poderes que varían entre 1.5x y 3.5x. En nuestra práctica solo hemos considerado la prescripción de los dos nombrados anteriormente.

AGUDEZA VISUAL

El sistema universalmente usado para medir agudeza visual fue ideado por Snellen, quien siguiendo las sugerencias de Donders, en 1862 construyó los primeros optotipos los cuales consisten en figuras o letras formadas de pequeños cuadrados que subtienden un ángulo de un minuto a una distancia determinada. Utilizó esta medida siguiendo la teoría del ángulo mínimo; esta teoría está basada en la relación espacial de dos conos retinianos, que al estar separados por uno intermedio, forman un ángulo de un minuto en el punto nodal. Se usa la relación de dos conos separados, ya que la discriminación no sería correcta si el impulso llegase a dos conos vecinos. Las letras y figuras formadas por estos pequeños cuadrados, subtienden un ángulo total de 5 minutos en el mismo punto nodal del ojo, a la distancia establecida de 6 metros o 20 pies, (infinito óptico) pues se considera que en esta posición de mirada, no hay prácticamente acomodación en acción. Un individuo tiene 100/100 de visión cuando puede reconocer a la distancia de 6 metros, un objeto cuyo tamaño subtiende el ángulo de 5 minutos en el punto nodal. Si el tamaño del objeto se varía, necesariamente se modifica el ángulo formado, fraccionándose proporcionalmente las medidas de agudeza visual.

La magnificación producida por los aditamentos de visión sub-normal, modifican este ángulo conservando una distancia fija; teóricamente podemos calcular el aumento de agudeza que se puede alcanzar en un paciente, pero normalmente, hay otros factores que tienen influencia en ella, tales como el tamaño de la pupila, iluminación, color del optotipo y su contraste, etc.

CAUSAS PRINCIPALES DE REDUCCION DE AGUDEZA VISUAL

Existen muchas clasificaciones de las causas, del deterioro de la agudeza visual pero podemos resumir las más frecuentes en 5 grupos generales: 1ª Defectos congénitos; 2ª Ambliopía-Ex-Anopsia; 3ª Traumatismos; 4ª Degeneraciones seniles y 5ª Secuelas de patología ocular.

ADITAMENTOS DE VISION SUB-NORMAL

Son considerados como aditamentos de visión sub-normal: 1º Telescopios; 2º Microscopios; 3º Lentes de Contacto; 4º Magnificadores de alto poder; 5º Anteojos con Estenopeicos múltiples, y 6º Ganchos para ptosis.

OPTICA DE LOS TELESCOPIOS Y MICROSCOPIOS

Todos los Telescopios usados en visión sub-normal, están contruídos bajo el principio del Telescopio de Galileo; constan de un objetivo (lente de poder positivo) y de un ocular (lente de poder negativo). El sistema focal produce magnificación cuando el ocular se coloca exactamente en el segundo plano principal del objetivo, es decir, la distancia entre las dos lentes es determinada por su poder dióptrico el cual es igual a la suma algebraica de sus distancias focales, se obtiene una imagen derecha (directa).

El Microscopio consta de dos lentes positivos altos, plano-convexos, separados entre sí de acuerdo con sus poderes dióptricos; estos pueden fabricarse con magnificaciones que varían entre 2x y 22x. Debido a que las lentes usadas producen una distancia focal muy corta, los microscopios solo pueden utilizarse para visión próxima.

La alta magnificación que producen y corta distancia focal impiden su uso binocular, siendo tan solo posible su adaptación monocular. Dentro del sistema óptico del microscopio solo se incorpora la corrección cilíndrica en aquellos casos en que el astigmatismo es superior a 5.00 dioptrías, los errores de refracción esféricos se compensan con pequeños cambios en distancia.

MAGNIFICACION

La Magnificación se mide, tomando como base de comparación el tamaño normal de la imagen; un adimento que aumente el tamaño del objeto al doble, se dice que tiene un poder de 2x ó 100/100. De la misma manera un Telescopio de 1.7x magnifica la imagen en 70%.

Cuando se prescriben adiciones para visión próxima, para ser sobre-puestas, a los Telescopios de distancia, se debe tener en cuenta que las lentes esféricas positivas producen magnificación cuando su poder es superior a 4.00 dioptrías; en estos

casos la magnificación producida por el telescopio se suma a la producida por la adición. Es importante tener en cuenta en la prescripción final que debe ordenarse la mínima magnificación que proporcione la mejor agudeza visual.

PRESCRIPCION

La experiencia en nuestros casos nos ha demostrado que un 65% de pacientes con visión sub-normal pueden ser ayudados con el uso de aditamentos especiales; este porcentaje se refiere a personas que usan frecuentemente tales aditamentos y logran superar las dificultades que estos ocasionan. El porcentaje de pacientes que logran un aumento en agudeza visual es ciertamente muy superior al ya anotado, pero este cambio en agudeza no constituye una norma fija en la conducta a seguir por el profesional. La edad del paciente no es un factor de mucha importancia, ya que en nuestras historias clínicas contamos con pacientes entre los 15 y los 88 años de edad. El cambio mínimo conseguido en agudeza visual de lejos, es de un 5%, y el máximo de un 60%. En general el Telescopio de distancia con su adición para visión próxima, parece ser la combinación más aceptada. Recientemente se ha experimentado el Telescopio de distancia y el Microscopio de cerca dentro de la misma unidad con notorio éxito, este aditamento se denomina Tri-Optico y de él nos ocuparemos brevemente más adelante.

En el uso de Telescopios, la dificultad más frecuente radica en la reducción del campo visual, y en la rapidez del desplazamiento de las imágenes; las actividades estacionarias (cine, televisión, etc.) son indicadas al paciente en los primeros días, a fin de conseguir un entrenamiento que lo adapte a su vida ordinaria, ya que ciertos problemas de localización espacial se presentan mientras el paciente aprende a reconocer su nuevo panorama visual. Aunque algunas personas se han adaptado al uso casi permanente de los Telescopios, en otras su aplicación se limita a los momentos de incapacidad visual.

En visión próxima, la reducción del campo visual es mayor ya que generalmente estos casos son adaptados monocularmente; si se desea visión binocular y esta es posible, cuando se prescriben adiciones de 4 a 6.00 dioptrías, debe tenerse en cuenta la incorporación de prismas base interna, a fin de controlar fenómenos diplópicos propios. Si se precisan adiciones mayores, los fenómenos diplópicos no pueden ser tratados con prismas, siendo la mejor conducta seguir, la inhibición de un ojo y proseguir con el caso monocularmente. Es por esta razón que los Microscopios no pueden ser usados binocularmente.

Cuando se emplean las combinaciones de Telescopios con adiciones de cerca, el paciente tiene que ser advertido cuidadosamente acerca de su uso, explicándole que la distancia de uso será muy pequeña y fija; esto se debe a que un pequeño cambio en la distancia produce un completo emborronamiento por la alta

magnificación. Los Microscopios son aditamentos indispensables, cuando la percepción visual es muy baja; generalmente el cambio en cuanto a visión se refiere es radical, lo que predispone favorablemente al paciente para superar las dificultades que se crean con la corta distancia de lectura del Microscopio.

SELECCION Y CONDUCTA DE PACIENTES

Los principales aspectos que deben tenerse en cuenta son los siguientes:

1. Condición Patológica. La atención del paciente puede estar dirigida al origen de su problema visual, no teniendo mayor interés en lograr un cambio en visión, sin eliminar la causa de su defecto. En otros casos existe el temor de que el uso de tales aditamentos produzca algún empeoramiento de su condición patológica aun cuando esta se halle controlada. Una explicación adecuada debe recibir el paciente a este respecto.

2. Estado Habitual. El paciente debe estar convencido de que su habilidad visual es muy deficiente, para que pueda apreciar un cambio favorable en visión. Cuando la mala visión es congénita o adquirida a muy temprana edad, seguramente ha adquirido cierta destreza a base de entrenamiento, o en otros casos con educación especial, sintiéndose satisfecho con su estado actual; por consiguiente, perseguir un cambio completo en su organización, aprovechando la recuperación visual, es algo casi imposible de lograr, sin que exista el deseo absoluto por parte del paciente. Este cambio tan radical crea profundas alteraciones en el individuo.

3. Existiendo una actitud favorable por parte del paciente, y cuando la prescripción de los aditamentos es indicada, conviene observar la facilidad que muestra el individuo con el manejo de estos; en algunos casos la ortóptica como complemento es indispensable, pero si aún las dificultades son muy marcadas, el pronóstico es reservado.

AVANCES RECIENTES EN VISION SUB-NORMAL

Hasta hace poco los aditamentos descritos se usaban como unidades sencillas, limitando su uso a una actividad determinada; últimamente han empezado a utilizarse diferentes combinaciones de los mismos que amplifican el campo de acción de los pacientes.

El *Bióptico* consiste en la combinación de un telescopio y un microscopio dentro de la misma unidad, siendo colocado el primero en la parte superior para distancia y el segundo en el plano inferior, facilitando la visión próxima.

El *Trióptico* consiste en la combinación de un telescopio y un microscopio dentro de la misma unidad, separados en la zona intermedia por una lente de correc-

ción regular del error de refracción, habilitando al paciente para el uso de los aditamentos magnificadores únicamente en las circunstancias requeridas.

El *Telecom* presentado por el Dr. William Filderman, O. D., sigue los mismos principios ópticos del telescopio ya descritos y utiliza un lente de contacto en el plano corneal como ocular y el objeto colocado en forma de anteojos en un plano determinado. Este nuevo sistema presenta grandes ventajas en cuanto al tamaño del campo visual y distorsión se refiere.

Calle 58 N° 13-08

BIBLIOGRAFIA

JIM H. DAY O. D. Refracción en Casos de Visión Subnormal. Arch. de la Soc. Am. de Oft. y Opt. I. 108.

EMSLEY. Visual Optics. 1950. Halton Press.

BORISH. Clinical Refraction. 1949. Profesional Press.

HENRY J. HOFF O. D. Application of Telescopic and Microscopic Spectacles in Subnormal Vision. Part. I. Washington D. C.

ENDOTHELIAL STUDIES OF CORNEAS PRESERVED IN VARIOUS MEDIA

BY

H. M. KATZIN, M. D., M. MARTINEZ, M. D.

New York, U. S. A.

The refinement of surgical methods for keratoplasty in conjunction with the better understanding of its physiopathology has made corneal transplantation more popular in recent years, with a resulting increase in the need for fresh donor material. The supply of this material fluctuates and corneal preservation is one method by which we have tried to compensate for the discrepancy between the increasing demand and the inconstant supply.

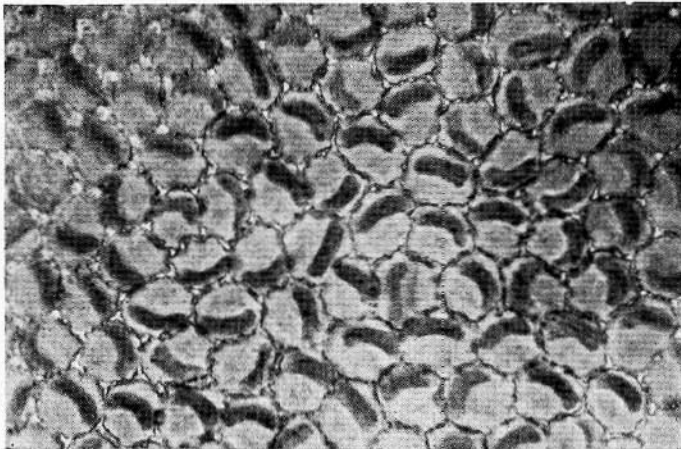


Fig. 1. Normal cat endothelium, alizarin red stain, phase contrast microscope, 320 X.

* From the laboratory of The Eye-Bank for Sight Restoration, Inc., Manhattan Eye, Ear and Throat Hospital, aided by a grant from The Knights Templar Eye Foundation.

Several methods for corneal preservation have been developed, studied and tested: dehydration, preservation in tissue culture media, cold temperatures, mineral oil, water soluble plastics, glycerine, glycerine-dehydration, sugars, homologous serum or whole blood, etc. Stocker and his co-workers¹ and researchers

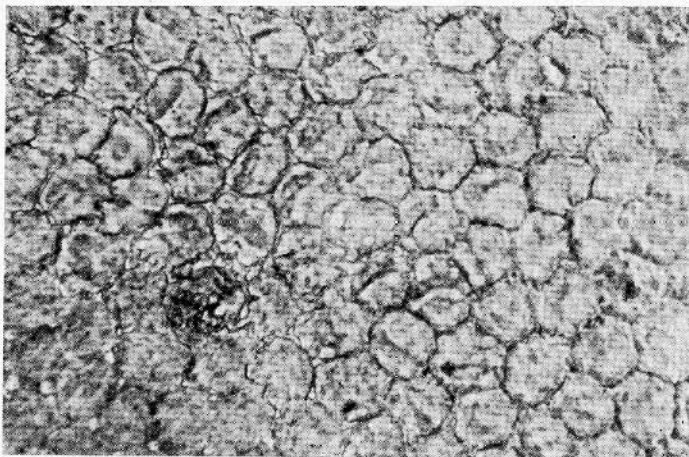


Fig. 2. Typical picture of (*) endothelial damage. Moderate cell shrinkage, mild contraction of the cytoplasm. Alizarin red stain, phase contrast microscope, 320 X.

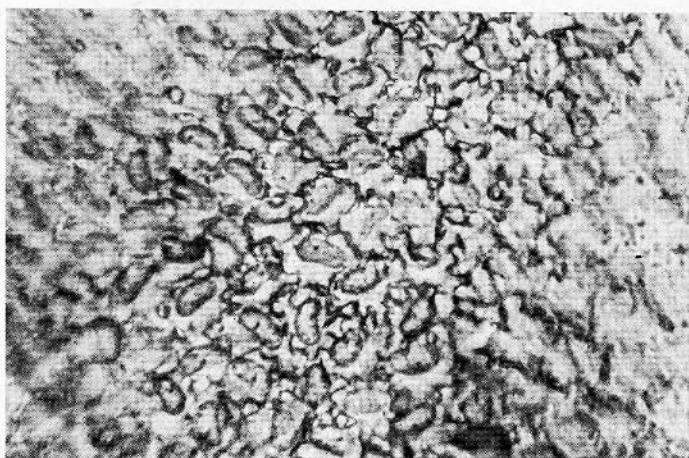


Fig. 3. Typical (***) endothelial changes. Cornea immersed for one hour in dextrose-saline at room temperature. Marked cell distortion due to dehydration and moderate increase in size of nuclei. Alizarin red stain, phase contrast microscope, 320 X.

PRESERVED CORNEAS

at the University of Toronto² have tested a number of these techniques of preservation by trying to grow the preserved corneal tissues by tissue culture methods.

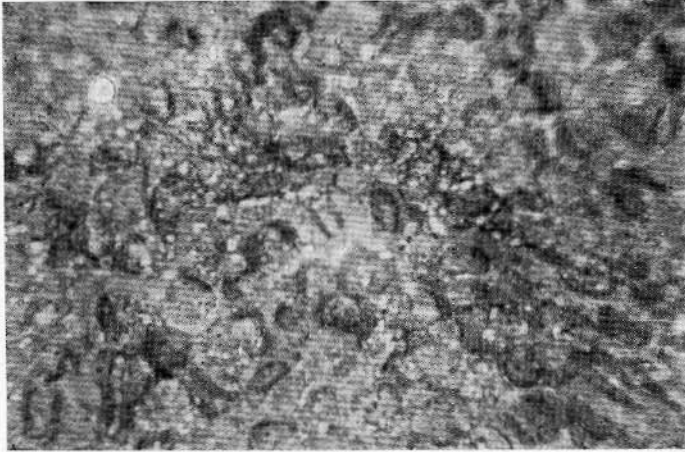


Fig. 4. Typical (***) endothelial changes. Cornea preserved three days in 5% glycerine-saline at 4°C. Moderate increase in size of nuclei, marked formation of vacuoli, rupture of the membranes in several cells. Alizarin red stain, phase contrast microscope, 320 X.

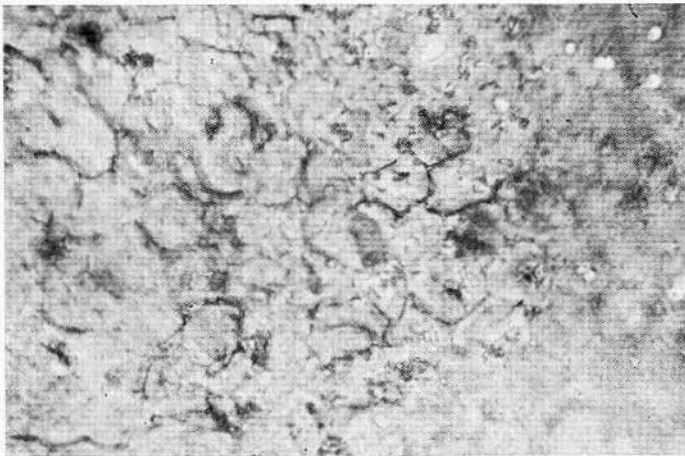


Fig. 5. Typical (****) endothelial changes. Cornea preserved four days in glycerine 5% at -10°C. Almost complete disorganization of cell structure. Alizarin red stain, phase contrast microscope, 320 X.

Other investigators have evaluated the preservation methods clinically in experimental animals. Whatever means is used to preserve the tissues, it is our impression that corneas preserved for more than several days may be satisfactory for partial thickness grafts only.

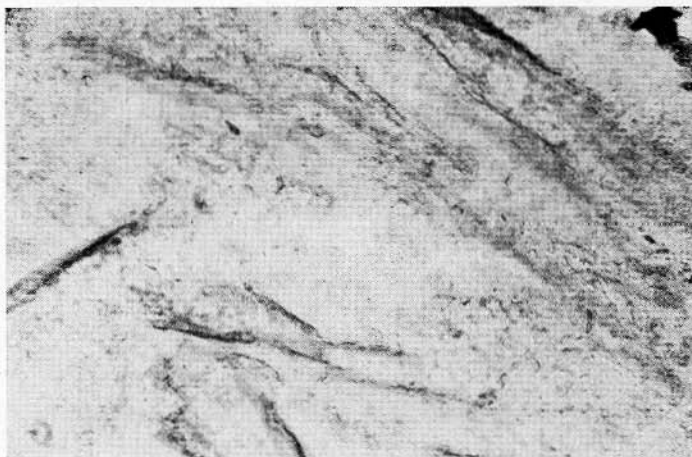


Fig. 6. Typical (*****) changes. Cornea preserved in 15% glycerine three days at 4°C. Complete cellular disorganization, endothelial detachment in several places. Alizarin red stain, phase contrast microscope, 80 X.

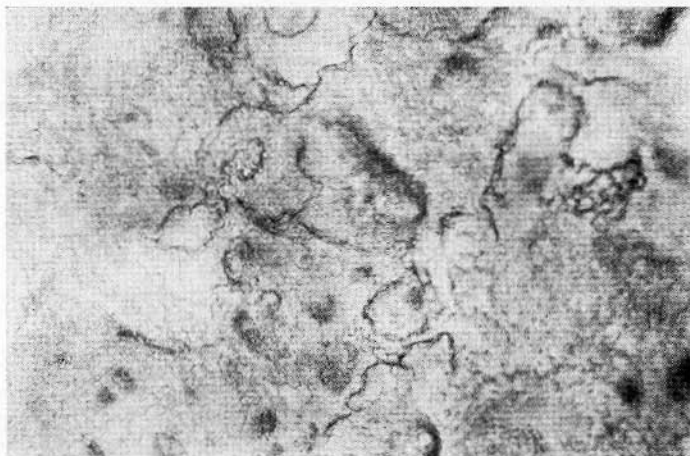


Fig. 7. Endothelial changes after immersion in 95% glycerine at -10°C. Complete cell destruction. Alizarin red stain, phase contrast microscope, 80 X.

PRESERVED CORNEAS

The success of clinical tests in the case of lamellar transplants has raised the question as to whether full viability of all layers of the cornea is really necessary. Some experience seems to indicate that it is not.

In addition to tissue culture and actual transplantation of preserved corneas, oxygen consumption, vital staining, lipogenesis, and the uptake of isotopes are among the more popular testing techniques, and while none of them give us the information needed to predict accurately the clinical result, they do give us some idea of the comparative chance of success.

The problem of full-thickness transplantation with preserved material, however, remains an important one and is at present unresolved. Most attempts at fullthickness grafts with corneas preserved for a prolonged time result, sooner or later, in the opacification of the tissue and failure to restore useful vision. The reason for this failure are complex and not well understood, but the viability and functional integrity of the endothelium and the role of Descemet's membrane appear to be of primary importance.

We have conducted a study on the morphological changes taking place in the endothelium during the preservation process.

METHODS

Our endothelial studies have been made on cat corneas obtained from the American Society for the Prevention of Cruelty to Animals, where the cats are sacrificed by sudden decompression and vacuum in a hermetic chamber. After that the

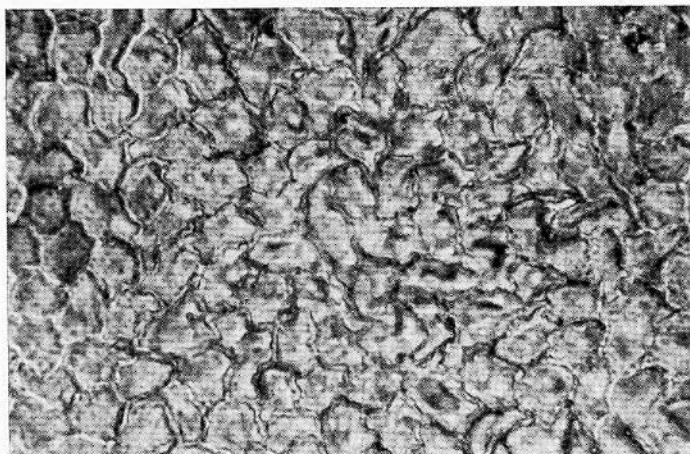


Fig. 8. Endothelial changes after three days preservation in mineral oil at -10°C . Mild cell distortion. Mild cytoplasmic changes. Alizarin red stain phase contrast microscope, 320 X.

animal is decapitated and the head is brought immediately to our research laboratory. The eyes are nucleated and the whole cornea is removed, cutting 1 mm. behind the limbus with razor blade and curved scissors, taking extreme care to avoid mechanical trauma to the endothelium. Immediately after removal the cornea is placed in the preservative medium or solution. The preserved corneas are removed at varying intervals for study, and rehydrated, when necessary, in normal saline or Hank's solution at pH 7.2.

The preserved corneas are evaluated as to transparency, pliability, rehydration properties and the condition of the endothelium. This report will concern itself chiefly with studies of the endothelium using flat preparation, vital staining, and phase contrast microscopy. This technique reveals the intercellular cement substance and shows up the structure of the endothelium exceptionally well. Extreme care is taken during the microscopic examination to avoid pressure on the tissue or drying of the preparation since this would induce profound changes in the delicate cells.

Table I shows the degree of change found in the endothelium of corneas examined after various intervals of time.

Corneas were stored in 5, 15, 50 and 95% glycerine at room temperature, 4° C and —10° C, for varying lengths of time. We also used solutions of the same concentrations of dextrose, fructose and sorbitol at the same temperatures.

The results of our experiments indicate that the endothelium suffers profound morphological changes with storage in glycerin-saline in all of the various concentrations, temperatures or time intervals are increased. They range from simple cellular shrinkage due to dehydration to the complete rupture and disorganization of the cellular elements.

Of the various conditions of concentrations and temperatures, the low concentrations of dehydrating substances at room temperature produce the most rapid deterioration of the endothelium. With higher concentrations, up to 50%, the changes are delayed somewhat but they take place nevertheless, and they are not reversible upon rehydration. Similarly, if the lower concentrations are used in conjunction with a temperature of 4° C the changes are delayed, but by the tenth day there is complete disorganization of the cellular elements.

At temperatures of —10° C parallel results were noted. Rapid changes occur in low concentrations and slower changes with higher concentrations, but here there is the added factor of trauma produced by ice crystal formation. In short, every possible combination of temperature and dehydration will, by the tenth day, produce irreversible morphological changes in the endothelium.

PRESERVED CORNEAS

We believe that neither sub-zero temperatures nor partial dehydration will prevent autolysis and pyknosis. Corneas preserved at low temperatures in concentrations of 15% glycerine invariably show detachment of the endothelium by the third day. The fact that the cellular elements in these tissues stain deeply with 1% alizarin red, while fresh tissues take the stain lightly or not at all, would seem to

EXTENT OF ENDOTHELIAL CHANGE IN PRESERVED CORNEAS

15 MIN.

%	GLYCERINE			DEXTROSE			FRUCTOSE			SORBITOL		
	+20°C	+4°C	-10°C	+20°C	+4°C	-10°C	+20°C	+4°C	-10°C	+20°C	+4°C	-10°C
5	+		+	+	+	+	+	+	+	+	+	+
15	+		++	+	+	++	++	++	++	+	+	++
50	+++		+++			+++			+++			+++
95	+++		++++			▼			+++			▼
1 HOUR												
5	+			++	+		+++	++	+	++	++	+
15	++			++	++		+++	++	++	+++	++	++
50	+++				+++		+++	+++	+++	+++	+++	+++
95					▼		▼	▼	▼	▼	▼	▼
3 DAYS												
5	+++	+++	+++	+++			+++	+++	++	+++	+++	++
15	+++	+++	++	+++			+++	+++	+++	+++	+++	+++
50	+++	▼	+++	+++			+++	▼	▼	▼	▼	▼
95	+++	▼	▼	▼			▼	▼	▼	▼	▼	▼
10 DAYS												
5			+++	+++	+++	+++	+++	+++	+++	+++	+++	+++
15			+++	▼	▼	▼	▼	▼	▼	▼	▼	+++
50			+++	▼	▼	▼	▼	▼	▼	▼	▼	▼
95			▼	▼	▼	▼	▼	▼	▼	▼	▼	▼
15 DAYS												
5			+++									
15			+++									
50			▼									
95			▼									

- + MILD CELL SHRINKAGE.
- ++ CELL SHRINKAGE, CYTOPLASMIC DISTORTION, MILD INCREASE SIZE OF NUCLEI.
- +++ MARKED CELL DISTORTION, INCREASE SIZE OF NUCLEI.
- ++++ CELL MEMBRANE RUPTURE, PYKNOSIS OF CELL ELEMENTS.
- +++++ COMPLETE DISORGANIZATION OF CELL STRUCTURE.

indicate that some degenerative change has taken place. We believe that the mobilization, diffusion and adsorption of cellular enzymes play an important part in this problem, causing partial digestion of the corneal structures. This process is, of course, irreversible.

We undertook a study of corneas preserved in various sugars in the belief that sugars might be less toxic than glycerine, but subsequent chemical and histological studies did not bear this out, and experimental transplants in cats were unsuccessful.

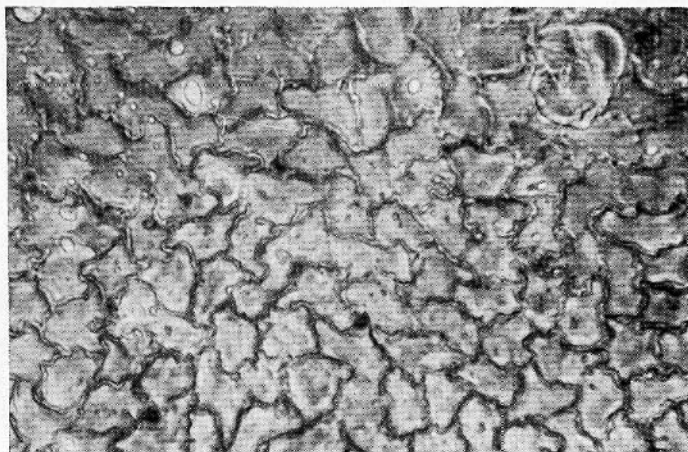


Fig. 9. Endothelial changes after 10 days of preservation in mineral oil at 40° C. Mild distortion of cellular elements. Alizarin red stain, phase contrast microscope, 320 X.

SUMMARY

Studies were made of the endothelium of corneas preserved by various methods for varying lengths of time.

According to our criterion based on the morphological appearance of the endothelium when stained with alizarin red and examined under the phase contrast microscope, all of the corneas preserved for more than three days exhibited degenerative changes such as disorganization of the cellular elements and detachment of the endothelium.

The results of our studies lead us to believe that none of the methods we used satisfactorily arrests the action of autolytic processes that cause irreversible changes in the endothelium.

210 East 64th Street

REFERENCES

1. STOCKER, F. W., EIRING, A., GEORGIADÉ, R., and GEORGIADÉ, N., Evaluation of viability of preserved rabbit corneas by tissue culture procedures. *Am. J. Ophth.* 47: 772, 1959.
2. COCKERAM, A. M., BASU, P. K., and ORMSBY, H. L., The effect of glycerine, dehydration, merthiolate, and zephiran on the viability of cornea. *Am. J. Ophth.* 47: 308, 1959.

SOME OBSERVATIONS RELATING TO THE KINEMATICS OF THE EYE

BY

HENRY A. KNOLL, M. D.

New York, U. S. A.

The kinematics of the monocular eye positions have been known for well over a hundred years. Donders's and Listing's Laws are discussed in most texts relating to the physiology of the eye. Helmholtz¹, Burmester², Lamb³ and very recently Westheimer⁴ have provided us with various mathematical treatments of the known relationships.

It is the purpose of this paper to describe some experiments designed to test the stability of the kinematic relationships and to suggest a theory which relates the geometry of the eye, the eye, muscles and the known facts of the kinematics of the eye.

Experiments were performed to answer the following four questions:

1. Does the primary direction vary with tilt of the head about the base line (the line connecting the centers of rotation of the two eyes)?
2. Does the primary direction vary for various fixation distances?
3. Does the primary direction vary with rolling of the head (inclination toward one shoulder)?
4. Can the primary direction be moved?

The after image method of locating the primary direction was used. The position of the primary point was approximated and a vertical strip of black paper was mounted at this point. With the head held in a fixed position the induced after image was observed as the point of regard swept (jumped) along a plumb line. The strip was adjusted laterally until the after image remained superposed on the plumb line throughout its length. A similar procedure was used for a horizontal strip and a horizontal line. A specially constructed yoke permitted one to tilt the head about the base line and to hold it fixed at any desired setting. Tilting

the head $+5^\circ$, $+10^\circ$, $+15^\circ$ and using fixation distances of 1 meter, 50 cm., and 33 cm., the primary direction of two right eyes (my own and my assistant's) remained fixed with respect to our heads. These experiments answered questions one and two in the affirmative. This result indicates that the tonic impulses set up in the central nervous system by tilting the head and by accommodation did not affect the primary direction.

Helmholtz answered the third question in the affirmative and I have verified this in my own right eye. This adds additional evidence to the non-interference of tonic impulses and introduces additional evidence that changing the muscle relationship does not change the primary point. When rolling the head, the human eye will undergo a partial compensatory cyclorotation (about 5 degrees in my own eyes) which will alter the relationship of the muscle planes and the coordinates of the head. In spite of this the primary point remains fixed respect to the head.

Now for the final question. The primary point can be located reasonably well by just pointing to it. It is the point which is subjectively "straight ahead". If a pair of prisms, their bases to the right, are placed before the eyes, objects located straight ahead lies slightly to the left of body straight ahead. On several days I have located my primary direction and then worn a pair of six diopter prisms, base right for eight hours. Upon removing the prisms and again measuring the location of my primary direction, I find that it has moved to the left. By the next morning the primary direction is back in its original location.

It is of interest to report that the primary directions of my right and left eyes diverge by an amount very nearly equal to my distance phoria.

To summarize these experiments: the primary point seems to be quite rigidly fixed with respect to the head even when certain neuromuscular relationships are altered. The primary point can be moved, however, by artificially producing a new relationship between the bodily perceived world and the visually perceived world. The adjustment and re-adjustment seem to take place rather quickly.

The question now arises are the kinematics of the eye established entirely by some process of conditioning? The results of the final experiment would suggest that this is quite possible. However, the physical relationships of the muscle planes of the recti muscles to the eye do suggest that a close approximation to the facts are "built into" the system and only minor adjustments are necessary.

That this is true was suggested to this author by a recent analysis of eye positions given by Boeder⁵. Boeder used a stereographic projection of the meridians of the eye to point out certain relationships in the kinematics of the eye. It occu-

rred to me that if the recti muscles were stereographically, so to speak, connected to the eye and the orbit, the kinematic laws of eye position would be obeyed automatically. Such an eye would have four thread like muscles inserted at the anterior pole of the globe. The muscles would hug the exterior surface of the eye and have a common origin in the orbit at a point tangent to the posterior pole of the eye when the eye was fixed in its primary direction. These four muscles, acting on a perfectly spherical eye with no other attachments, would position the eye in perfect accord with the described facts. No obliques would be needed, except to introduce cyclomovements in response to body orientations. In fact our own eye is not very far removed from this basic design. The insertions have been moved back to allow a clear view through the cornea, and, of course, nerves and blood vessels have been attached. For limited eye movements, approximately twenty degrees from the primary direction, these complications will not introduce serious errors. The origin has been moved back into the orbit and displaced twenty-two and one half degrees nasally. This is enough change to destroy the perfect kinematic positioning and throw the primary direction twenty-two and one half degrees temporally, but we still have those obliques make any corrections that may be needed. Also the recti muscles have finite width, a factor which permits each to introduce some rotary movement about the line of sight.

The visual primary direction and the proprioceptive primary direction (such as even a blind person must possess) are constantly being unified in each individual as he moves about in his environment and handles objects. It is suggested here that these two are closely related by the particular relationships which exist between the anatomical connections between the head and the eye- the approximate stereographic attachment of the recti muscles. The errors introduced by the lack of perfect stereographic connection must be corrected by a process of continuous conditioning, mainly by the action of the obliques.

New York, N. Y.

REFERENCES

1. HELMHOLTZ, H. VON, *Physiological Optics* (English translation) Menasha, Wis., Banta, 1925, Vol. 3, 71-107.
2. BURMESTER, L., *Kinematische Aufklarung der Bewegung des Auges*, Munchen Akad. d. Wiss., math - phys. Kl., 171-202, 1918-1919.
3. LAMB, H., *The Kinematics of the Eye*, *Phil. Mag.*, 6th series, **38**, 685-695, 1919.
4. WESTHEIMER, J. G., *Kinematics of the Eye*, *Jour. Opt. Soc. Am.*, **47**, 967-974, 1957.
5. BOEDER, P., *An Analysis of the General Type of Unioocular Rotations*, *A. M. A. Archives of Ophthal.* **57**, 200-206, 1957.

L'IRIDECTOMIE TARSORAPHIE

PAR

J. LEGRAND, M. D.

Nantes, France

La pathologie oculaire s'est considérablement modifiée depuis quinze ans et l'utilisation des antibiotiques et des corticostéroïdes a changé l'évolution et le pronostic de beaucoup de maladies. Certains traitements qui s'imposaient n'ont plus aujourd'hui leur utilité, et ne sont plus employés, d'autres, ont vu leur intérêt supplanté par des thérapeutiques plus simples et plus efficaces qui les ont remplacés.

Sans méconnaître la primauté des traitements modernes, il serait regrettable qu'on oublie des techniques qui, bien que d'indications plus rares, sont dans certains cas précis supérieures à toute autre thérapeutique et permettent d'obtenir la guérison là où les remèdes classiques ont échoué.

Parmi ces affections rebelles aux traitements locaux ou généraux et dont le caractère traînant et récidivant font le désespoir de l'ophtalmologiste, les segmentites antérieures doivent être placées au premier rang, et parmi les nombreux traitements qu'elles ont suscités, l'iridectomie associée à la tarsoraphie est celui qui m'a donné les meilleurs résultats.

Les segmentites antérieures sont réalisées toutes les fois qu'une kératite évolue vers la chronicité et qu'aux destructions cornéennes sont associées des lésions iriennes et ciliaires. La clinique nous montre, au cours des kératites les plus diverses, la fréquence de la participation du corps ciliaire: les exsudats, les synéchies, les précipités de descemète.

Nous connaissons le spasme du sphincter irien au cours des lésions de la cornée et l'apaisement qu'apporte à sa douloureuse contraction l'instillation d'une goutte d'atropine.

L'histopathologie a mis en évidence l'infiltration leucocytaire et la congestion de l'iris dans l'ulcère à hypopion, les zones de nécrose, les nodules inflammatoires de l'iris dans une kératite zonateuse, l'hyphéma de certaines kératites herpétiques.

TRANTAS a montré dans ces études gonioscopiques la fréquence au cours des kératites parenchymateuses tuberculeuses des lésions de l'angle iridocornéen et des goniosynéchies.

Une thérapeutique rationnelle doit donc tendre dans une segmentite antérieure à cicatrifier l'ulcère cornéen et à mettre l'iris et le corps ciliaire au repos.

C'est le but poursuivi dans les traitements innombrables qu'ont inspiré les kératites et si suivant l'étiologie la thérapeutique générale varie, les traitements locaux sont à peu près tous dans la même ligne: destruction du microbe ou du virus pathogène par des antibiotiques ou des antiseptiques ou des cautérisations ignées ou chimiques de l'ulcère et adjonction d'un mydriatique.

Parfois cette classique méthode suffit pour amener la sédation des phénomènes mais fréquemment aussi elle échoue. C'est que si la réaction irienne commande l'atropine, la défense cornéenne exige pour la vitalité de ses tissus une tension oculaire basse peu compatible avec l'emploi des mydriatiques.

On sait d'ailleurs le rôle favorable de l'hypotonie dans l'évolution des kératites et il suffit d'avoir vu l'éclaircissement d'une cornée ulcérée sous l'effet des paracentèses pour s'en convaincre. On a d'ailleurs beaucoup utilisé l'ésérine dans le traitement des kératites et avec succès mais elle ne calme pas les douleurs du malade, elle favorise la formation de synéchies postérieures, elles-mêmes génératrices du glaucome secondaire.

A cette difficulté dans le choix des myotiques ou des mydriatiques viennent s'ajouter les échecs que nous réservent les tentatives de cicatrisation. Les antibiotiques sont bien bactéricides mais restent sans action sur les virus.

Les troubles trophiques ne sont point améliorés, en règle générale, par les corticostéroïdes. Et il est trop fréquent de voir des ulcérations cornéennes dont le caractère infectieux paraît disparu mais qui restent irritées, torpides, à fluorescéine ++. L'altération de la trophicité entretient la lésion.

Quelle thérapeutique adopter dans ces formes subaigues décourageantes par leur tendance à la chronicité?

C'est pour pallier à ces écueils que j'ai été amené à associer deux thérapeutiques qui répondent chacune aux objections que j'ai présentées: *l'iridectomie qui tranche le dilemme atropine, ésérine, et la tarsoraphie qui constitue le pansement idéal de la cornée.*

Bien entendu, il n'y a lieu de l'envisager que si les traitements médicaux ont échoué ou si l'amaïnissement de la cornée par la kératite entraîne une ectasie avec menace de perforation qui commande une thérapeutique d'urgence, et il est certain que j'ai beaucoup moins souvent qu'autrefois motif à l'utiliser.

La technique que j'utilise consiste, après anesthésie locale et injection rétro bulbaire et palpébrale de novocaïne, à faire une iridectomie totale à la pique à midi avec distrète tunellisation conjonctivale pour faciliter la cicatrisation de la plaie. Ensuite, une tarsoraphie médiane de 10 à 15 millimètres est pratiquée, nécessitant deux ou trois points de suture à la soie huilée. Il est tout à fait indispensable dans ce temps opératoire de ne découper aux ciseaux qu'une très mince languette du bord postérieur de la tranche palpébrale. En particulier, il faut éviter que la tranche de section n'entame le sol ciliaire car il entrainerait des déviations secondaires des cils qui sont une des complications qui ont été le plus invoquées par les adversaires de la tarsoraphie. Si l'avivement ne découvre pas les bulbes ciliaires, il n'y a pas lieu de redouter l'apparition d'un trichiasis.

La tarsoraphie faite, instillation d'atropine, pansement binoculaire pendant 2 ou 3 jours. L'œdème palpébral post opératoire disparaît rapidement et on peut alors par les deux extrémités de la fente palpébrale surveiller la cornée et l'évolution de la maladie.

Les suites opératoires sont tout à fait démonstratives. Le malade qui se plaignait de céphalées, de photophobie, de larmoiement, est immédiatement calmé et peut souvent, très rapidement, reprendre un travail qu'il avait abandonné depuis longtemps.

Quand, au bout de quelques semaines, l'œil est cicatrisé et la fluorescéine négative, la tarsoraphie est ouverte d'un coup de ciseaux et le bilan des lésions cornéennes alors pratiqué permet d'envisager ou le statu quo ou, pour l'avenir, une kératoplastie.

J'ai pratiqué cette opération une centaine de fois et pour des cas très dissemblables: ulcères infectieux de la cornée rebelles aux traitements médicaux, segmentite antérieure tuberculeuse, kératites herpétiques, kératites disciformes, kératites zonateuses, etc. Je n'ai eu qu'à me louer des résultats obtenus.

Jamais, en particulier, les complications, que me faisaient initialement redouter une iridectomie faite à chaud, ne se sont produites; et le soulagement immédiat des douleurs du malade et la cicatrisation le plus souvent durable des ulcères ont été à peu près constamment obtenus.

Je pense que le mode d'action est sensiblement identique dans ces divers cas: l'iridectomie lève le spasme irien puisqu'elle coupe le sphincter. Elle garantit des accidents glaucomateux et permet l'usage de l'atropine qui met le muscle ciliaire au repos. Elle entraîne une hypotonie qui favorise la cicatrisation.

La blépharorrhaphie par le repos qu'elle apporte à l'œil, par la mise à l'abri des irritations extérieures (lumière, vent, poussière) par les conditions d'humidité et de chaleur où elle met la cornée, contribue certainement à sa défense.

En outre, dans les kératocèles, compression élastique et permanente de la sangle palpébrale sur l'oeil qu'elle contient et qu'elle moule agit mécaniquement contre les perforations en doublant une cornée amincie et lui permettant de se cicatriser, de se consolider en bonne position et d'éviter la formation d'un staphylome.

En définitive, les deux opérations combinées mettent l'oeil au repos absolu, ce qui constitue un élément primordial de guérison.

11 Rue Copernic.

LA IRIDECTOMIA PERIFERICA FILTRANTE

POR

JORGE MALBRAN, M. D., ENRIQUE MALBRAN, M. D.

Buenos Aires, Argentina

En anteriores oportunidades, (1, 2, 3, 4 y 5) nos hemos ocupado de cuales son los fundamentos de orden patogénico y clínico que rigen nuestra conducta en la selección de la cirugía del glaucoma primario. Volvemos a llamar la atención, sobre la necesidad de recurrir a una clasificación clínica en base a los datos obtenidos por medio de la gonioscopia realizada durante la hipertensión, la tonometría, la tonografía y eventualmente el resultado de las pruebas de provocación.

Deliberadamente no incluimos aquí otros elementos de diagnóstico a considerar en la clínica del glaucoma, como el estado de la agudeza visual, concomitancia de catarata, reducciones periféricas o amenaza del punto de fijación en el campo visual, etc., que pueden modificar en determinados casos la conducta y el momento quirúrgico. En posteriores oportunidades hemos de ocuparnos de estos puntos recientemente mencionados y en particular lo referente al campo visual y cirugía del glaucoma.

En algunas de las antedichas publicaciones, (4 y 5) hemos descrito la técnica quirúrgica de la iridectomía periférica filtrante comentando así mismo los resultados obtenidos en nuestros primeros 50 casos operados. Como en la actualidad, nuestra experiencia con este tipo de operación se ha acrecentado con el agregado de 56 nuevos casos, creemos oportuno hacer nuevas consideraciones al respecto. Aunque este tipo de intervención por nosotros propuesto ha sido también practicado en algunos casos de glaucoma secundarios post-uveíticos, uveítis hipertensivas, glaucomas absolutos, etc., con promisorios resultados, solo comentaremos aquí lo observado en los glaucomas llamados primitivos.

A nuestro modo de ver la iridectomía periférica filtrante tiene su indicación en las siguientes oportunidades:

- a) Glaucomas crónicos de ángulo cerrado.

- b) Glaucomas de patogenia mixta en los cuales existe una componente mecánica favorecida por el ángulo estrecho y un glaucoma de ángulo abierto que mantiene elevada la tensión y el C tonográfico por debajo de 0.12 mm³/min/mm Hg. en forma permanente.
- c) Glaucomas de ángulo estrecho pero abierto, por lo tanto sin componente mecánica.
- d) Glaucomas de ángulo amplio y abierto. Anteriormente abordábamos esta variedad de glaucoma con una ciclodiálisis superior e inversa o por medio de una operación filtrante clásica (Elliot o iridencleisis), pero en la actualidad dado los buenos resultados obtenidos por la iridectomía periférica filtrante preferimos recurrir a ésta.
- e) En cualquier variedad de los glaucomas arriba mencionados que no respondieron a una operación antiglaucomatosa anterior, incluida una iridectomía periférica filtrante previa. Aquí hemos cambiado de parecer, puesto que antes aconsejábamos la ciclodiálisis como segunda operación para los fracasos operatorios previos.

Pre-operatorio

Preferimos operar en miosis, sin fenómenos congestivos y con la tensión normalizada dentro de lo posible.

La miosis es muy útil, pues al estar el iris extendido permite la realización de una iridectomía bien periférica y facilita también después la pronta reducción del iris. Con este fin instilamos eserina 3 hs. antes de la operación.

El tratar de evitar los fenómenos congestivos para el momento de la operación es una condición tácita de buena cirugía de modo que no necesita comentarios. Con este motivo la administración de esteroides y/o irgapyrine según el caso es realizada en los días previos a la operación.

Preferimos no operar en hipertensión pues muchas de las complicaciones operatorias o post-operatorias como las hemorragias en el segmento anterior y las modificaciones del campo visual son la consecuencia de la descompresión brusca en el momento operatorio aún cuando con una técnica apropiada pueda hacerse la descompresión suavemente.

La disminución tensional para la operación es conseguida en base a:

- a) Acetazolamida en dosis adecuadas desde varios días antes. Como un mínimo administramos 250 mgs. 2 veces por día.
- b) Premedicación con fenergán, atropina, demerol.
- c) Anestesia general potencializada o anestesia local por medio de una inyección retrobulbar de 1,5 cms³. de xilocaína al 2% con hialuronidasa y aquinesia del orbicular.

Operación

- 1) Lazo de fijación en el recto superior.
- 2) Punción en bisel de la cámara anterior con un trozo de hoja de afeitar para impedir su vaciamiento. Esta maniobra tiene como objeto disponer de una vía de abordaje pre-realizada para así evitar al final de la intervención, maniobras peligrosas en caso de ser necesario profundizar la cámara anterior.
- 3) Inyección subconjuntival de xilocaína al 2% con un fin anestésico y sobre todo para formar un buen plano de clivaje en el colgajo conjuntival que al estar edematizado suele conservarse mejor.
- 4) Incisión conjuntival con tijeras a 12 mm. del limbo esclero corneal.
- 5) Disección del colgajo conjuntival hacia el limbo, con tijera y pica o trozo de hoja de afeitar.
- 6) Luego de coagular los vasos superficiales con un electrocauterio de Hildreth, gancho de estrabismo o varilla de vidrio calentada, se procede a realizar una incisión perpendicular de la esclera (ab externo) con un trozo de hoja de afeitar, a una distancia de 1,5 mm. del borde anterior del limbo. Esta incisión de 4 a 6 mm. de largo interesa en su primera etapa aproximadamente los 2/3 externos de la pared escleral.
- 7) Ambos labios de la incisión son diatermizados con un electrodo de punta, lo que produce su retracción entreabriéndose la herida. La misma operación vuelve a repetirse en la cara interna y ángulo de la brecha, lo que permite observar un nuevo ensanchamiento de la misma. A 2 mm. del borde posterior, se realizan una serie de coagulaciones superficiales en 3 bandas separadas entre sí a 2 mm. de distancia y con la concavidad dirigida hacia el limbo. Esto tiene por objeto producir una retracción a distancia del labio posterior, lo que contribuye a aumentar aún más la abertura de la brecha escleral.
- 8) Con la punta de la hoja de afeitar dirigida hacia arriba (por transfixión) se completa el 1/3 restante de la incisión escleral. Generalmente en este momento el iris se prolapsa espontáneamente. Si esto no ocurre aconsejamos comprimir suavemente ambos labios de la incisión con instrumentos romos hasta obtener la salida del iris. De esta manera no es necesario introducir instrumentos en la cámara anterior.
- 9) Iridectomía periférica.
- 10) El colgajo conjuntival es reclinado hacia atrás e inmediatamente se practica un suave masaje de la región limbar con un lápiz de algodón o instrumento romo para redondear la pupila.

- 11) La incisión conjuntival se cierra con puntos separados de seda virgen.
- 12) Si la cámara anterior se ha vaciado, se profundiza la misma con una inyección de aire a través de la incisión en bisel previamente realizada (ver 2).
- 13) Vendaje binocular por 24 horas.

Post-operatorio

El ojo no operado se descubre al día siguiente. El ojo operado se mantiene cubierto por 4 o 5 días. Se instila diariamente un colirio de hidrocortisona con antibióticos y midriáticos.

La cicatriz filtrante puede aparecer desde los primeros días pero en varios casos la hemos visto aparecer tardíamente lo que permite mantener una conducta conservadora con tratamiento médico. Como curiosidad, queremos señalar a este respecto, que uno de nuestro casos (S. M. ojo único), que figuró como fracaso en nuestra primera estadística, desarrolló una cicatriz filtrante un año después de la operación con tensión de 20 mm. al tonómetro de aplanación.

Corresponde agregar que cuando la cicatriz filtrante dentro del mes no se ha desarrollado, resulta conveniente aconsejar al enfermo practique un masaje ocular varias veces por día con el fin de obtener esta cicatriz.

Si la pupila no se redondea al final de la operación, rara vez ello se consigue en el post-operatorio, razón por la cual aconsejamos realizar la maniobra descrita en la técnica quirúrgica para redondear la pupila. Aún cuando en alguno de los casos en que la pupila quedó deformada, la inclusión de una porción del iris en la herida hizo las veces de iridencleisis, normalizando la tensión debemos recalcar que un buen número de nuestros fracasos se debió a este inconveniente.

Resultados obtenidos

A continuación transcribimos los resultados de nuestra primera estadística ya comentada.

I Serie

RESULTADO EN LOS 50 PRIMEROS CASOS

Nº de casos	Clasificación	Buenos resultados	Mejoría parcial	Fracasos
21	Crónico de ángulo cerrado	16	3	2
14	Patogenia mixta	12	1	1
12	Angulo estrecho y abierto	10	2	0
3	Angulo amplio y abierto	3	0	0
50		41 (82%)	6 (12%)	3 (6%)

Como ya aclaramos más arriba, en dicha estadística figura como fracaso un caso de glaucoma crónico de ángulo cerrado (S. M.) pero que en la actualidad presenta una tensión de 20 mm. al tonómetro de aplanación, ya que desarrolló tardíamente una cicatriz filtrante (1 año después).

II Serie

RESULTADO EN 56 CASOS SUBSIGUIENTES

Nº de casos	Clasificación	Buenos resultados	Mejoría parcial	Fracasos
30	Crónico de ángulo cerrado	28	1	1
8	Patogenia mixta	8	0	0
5	Angulo estrecho y abierto	2	1	2
13	Angulo amplio y abierto	11	1	1
56		49 (88%)	3 (5%)	4 (7%)

Tiempo transcurrido

Oscila entre 3 años y 2 meses para los primeros casos operados y 2 meses para los últimos casos incluidos en la nueva serie.

Buenos resultados en ambas series

Sobre un total de 196 casos operados:

Buen resultado en 90 casos (85%). Solo en 9 ojos (10%) fueron necesarios los mióticos para normalizar la tensión post-operatoria.

Comentario

Dentro de los glaucomas crónicos de ángulo cerrado están incluidos 12 glaucomas agudos que no habían mejorado con tratamiento médico o que el episodio llevaba más de 48 horas de evolución. En todos ellos se obtuvo una normalización de la tensión.

En 8 oportunidades sobre 7 ojos la iridectomía periférica filtrante se realizó en glaucomas acompañados de exfoliación capsular. Salvo uno de los ojos de un paciente (M. Z.) en que fracasó la primera operación, no así la segunda iridectomía filtrante practicada en el mismo ojo, en todos los demás el resultado tensional fue bueno.

En ninguno de los casos operados pudimos observar el desarrollo de un hi-phema.

Prácticamente en ningún caso hemos observado fenómenos de iritis o ciclitis post-operatoria (la llamada quiet-iritis por Kalt) tan frecuentes en las operaciones fistulizantes (Elliot). La mayoría de los casos con pupila redonda, libre y sin miosis espontánea o provocada por sinequias posteriores lo que deja al cristalino en mejores condiciones fisiológicas.

Aparte de los casos comentados en la estadística anterior, en la nueva serie de 56 casos, tuvimos una cámara anterior plana en 10 casos que apareció del segundo al cuarto día post-operatorio. En todos ellos, la cámara anterior se

profundizó con tratamiento médico, ya sea mióticos o midriáticos según el estado de la pupila, azetozolamida y reposo. En esta nueva serie no encontramos ninguna complicación grave atribuible a este inconveniente. Es necesario recalcar que este trastorno fue, en todos los casos, post-operatorio, pues como insistimos en el detalle de la técnica quirúrgica, cuando la cámara anterior se aplana durante la cirugía, invariablemente ella es reformada con aire a través de la punción previamente realizada.

Conclusiones

La iridectomía periférica filtrante es una operación simple de realizar, con la cual se obtiene un porcentaje de buenos resultados tensionales semejante a otras operaciones antiglaucomatosas técnicamente más difíciles o expuestas a complicaciones más graves.

A nuestro modo de ver esta técnica tiene las siguientes principales ventajas:

- a) Técnica quirúrgica simple.
- b) Menor operación comparada con otros procedimientos de resultados tensionales semejantes.
- c) Profundiza la cámara anterior y evita más que otras operaciones el desarrollo de goniosinequias aún cuando esto a veces también pueda ocurrir. Este detalle resulta particularmente interesante en los glaucomas mecánicos.
- d) En buen número de casos puede evitarse durante la cirugía el colapso de la cámara anterior. Cuando esto igualmente ocurre, ella es invariablemente reformada al final de la operación a través de la incisión previamente realizada.
- e) En general, se obtiene una pupila redonda, lo que altera menos la arquitectura y dinámica intraocular.
- f) Luego de un fracaso operatorio, la iridectomía periférica puede ser nuevamente realizada sin mayores inconvenientes y en general con buenos resultados según nuestra experiencia. Si otra técnica quirúrgica resultase más conveniente para una reoperación, una iridectomía filtrante previa compromete menos su realización que otras operaciones antiglaucomatosas como por ejemplo la iridencleisis.
- g) Permite una descomposición progresiva durante el acto operatorio, lo que previene las complicaciones hemorrágicas en el segmento anterior y las alteraciones que de ello resultan.

Resumen

Los autores recuerdan trabajos anteriores en donde se comentan los fundamentos etiopatogénicos y clínicos de la indicación operatoria en el glaucoma pri-

mario. Se describe nuevamente la técnica quirúrgica de la "iridectomía filtrante" y finalmente se analizan los buenos resultados obtenidos (85%) en 106 casos operados hasta el momento.

Parera 94

BIBLIOGRAFIA

1. MALBRAN, J., y MALBRAN, E., Consideraciones sobre el tratamiento quirúrgico del glaucoma. Arch. Soc. Oftal. Buenos Aires, 32: 295, Nov. 1957.
2. MALBRAN, E., Sobre el tratamiento quirúrgico del glaucoma primario. Relato oficial al tema de mesa redonda II Jornadas Rioplatenses de Oftalmología, Punta del Este, R. O. del Uruguay, Dic. 1957.
3. MALBRAN, J., y MALBRAN, E., Consideraciones sobre la orientación quirúrgica del glaucoma primario. Arch. Soc. O. Hispano-Americana, 18 (Nº 5) 427-450, 1958.
4. MALBRAN, E., Glaucoma Surgery (A motion picture). Transactions of the American Academy of Ophthalmology and Otolaryngology. Vol. 2: Nº 4, pag. 630. July-August 1958.
5. MALBRAN, J., and MALBRAN, E., Surgical management of primary glaucoma: a description of a new technique. Am. J. Ophth. 47: Nº 1, 34-41, January 1959.

BEHANDLUNG VON TUMOREN DURCH LICHTKOAGULATION

G. MEYER - SCHWICKERATH, M. D.

Bonn, Germany

I. Wir begegnen in der Augenheilkunde den verschiedensten benignen und malignen Tumoren. Für den Betroffenen sind sie aber alle bösartig, wenn sie das Sehvermögen bedrohen. Besonders tragisch ist die Lage, wenn beide Augen oder das einzig sehfähige Auge betroffen sind. In Jedem Falle ist jedoch das ideale therapeutische Ziel, den Tumor zu zerstören und gleichzeitig das bedrohte Sehvermögen wenigstens teilweise zu retten.

II. Die Enukleation verzichtet auf dieses Ziel, dafür bietet sie in den meisten Fällen die Sicherheit, daß die Geschwulst radikal entfernt wird. Sie ist deshalb die Therapie der Wahl bei malignen Tumoren, wenn keine Chance besteht, die Geschwulst unter Erhalt einer brauchbaren Funktion zu zerstören.

Mit *Röntgenstrahlen* und anderen ionisierenden Strahlen ist es unter Umständen möglich, bösartige Geschwülste zu zerstören und die Sehkraft des Auges zu retten. Leider ist diese Therapie durch die große Strahlenempfindlichkeit der Linse, die schon auf kleine Mengen mit Cataract reagiert, belastet. REESE in New York und STALLARD in London haben eine originelle Applikationstechnik ausgearbeitet, um die Strahlugsschädigung der Linse möglichst zu vermeiden.

Die *Diathermie-Koagulation* von Tumoren ist von WEVE in Utrecht in hervorragender Pionierarbeit für die verschiedensten Tumoren mit unterschiedlichem Erfolg verwendet worden.

Die *Lichtkoagulation*, welche anfänglich nur zur Therapie der Netzhautablösung gedacht war, hat sich auch bei der Behandlung verschiedener Augentumoren gut bewährt.

Mit wenigen Sätzen möchte ich die physikalischen Grundlagen der Methode erklären. Das Auge bietet auf Grund der Durchsichtigkeit der brechenden Medien die Möglichkeit, strahlende Energie ohne größere Verluste im Innern des Körpers zur Wirksamkeit zu bringen. Die "Durchsichtigkeit" betrifft aber nur einen

kleinen Teil des elektromagnetischen Spektrums, nämlich den Bereich des sichtbaren Lichtes und einen Teil des kurzwelligen Ultrarots, also die Strahlung zwischen 350 und 1000 m. u. Strahlen außerhalb dieses Bereiches sind nicht nur nutzlos für die Lichtkoagulation, sondern auch gefährlich, da sie zur Ultraviolett-schädigung der der Hornhaut und zur Ultrarotschädigung der Linse führen können. Eine Schädigung der brechenden Medien tritt aber nicht auf, wenn die Strahlung nur durchgelassen, gebrochen, zerstreut oder reflektiert wird. Elektromagnetische Strahlung wird nur am Ort der Absorption wirksam. Das heißt, daß die Lichtkoagulation nur da entsteht, wo das Licht absorbiert wird. Im normalen Auge findet die Absorption vorwiegend im Pigmentepithel und in der Aderhaut statt. Je dunkler das zu koagulierende Gewebe, bzw. der Tumor ist, um so mehr Licht wird absorbiert und um so kräftiger wird die Koagulation. Die technischen Einzelheiten des Gerätes sollen jetzt nicht besprochen werden. Man versteht sein Prinzip am einfachsten, wenn man sich einen elektrischen Augenspiegel vorstellt, dessen Lichtquelle durch kurzfristige Überlastung so hell gemacht wird, daß es an der beobachteten und beleuchteten Stelle zur Koagulation kommt. Der elektrische, optische und mechanische Aufwand für einen solchen "Augenspiegel" ist leider sehr groß. Wir haben inzwischen bei 130 Patienten Tumoren mit Lichtkoagulation behandelt und ich möchte über die dabei gewonnenen Erfahrungen berichten.

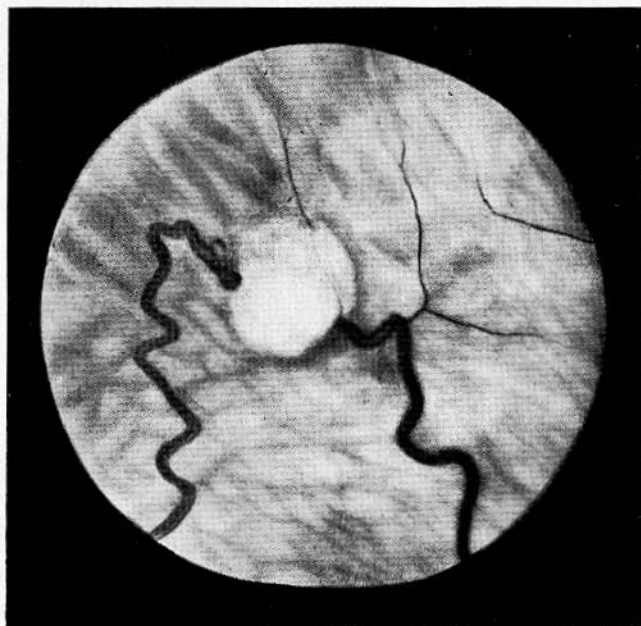


Abb. 1. Angiomatosis Retinae (von Hippel-Lindau).

III.

a) Melanoblastome (Melanosarkome) der Aderhaut sprechen sehr schlecht auf Röntgenstrahlen an. Sie sind histologisch und biologisch mit den bösartig pigmentierten Geschwülsten der Haut nicht vergleichbar. Da es sich um eine maligne Geschwulst handelt, werden diese Augen durchwegs enukleiert. Obwohl da durch die Geschwulst in der Regel radikal entfernt wird, hat die E nukleation der Melanoblastome eine schlechte Prognose: Im Verlauf von 10 Jahren sterben 60% der Patienten an Metastasen, vor allem in der Leber. Das ist umso verwunderlicher als diese "Metastasen" noch nach einer Latenzzeit von über 30 Jahren auftreten können. Wir wissen zwar aus histologische Untersuchungen, daß schon in den frühesten Stadien des Melanoblastoms vitale Zellen des Tumors in großer Zahl in die Blutbahn gelangen. Wir kennen aber nicht die Bedingungen, unter denen diese Zellen zu Metastasen werden. WEVE hat die transclerale Diathermie-Koagulation der Melanoblastome eingeführt und dabei ausgezeichnete Ergebnisse erzielt. Er hat im Verlauf von 20 Jahren über 20 Patienten behandelt, von denen bis jetzt keiner an Metastasen gestorben ist (MELCHERS). Bei den ersten experimentellen Untersuchungen über die Lichtkoagulation wurden Augen mit Melanoblastom kurz vor der E nukleation koaguliert, um die histologische Wirkung zu prüfen. Dabei fiel auf, daß das Tumorgewebe auf die Lichtkoagulation mit tiefen Nekrosen reagiert. Ermutigt durch diese Befunde und die Ergebnisse von WEVE, habe ich inzwischen 52 Melanoblastome mit Lichtkoagulation behandelt. In 17 Fällen wurde anschließend enukleiert, um die Wirkung der Lichtkoagulation und die Grenzen der Therapie im histologischen Präparat festzustellen. In den übrigen Fällen wurde das befallende Auge mehrfach mit Lichtkoagulation behandelt. In 18 Fällen erscheint die Geschwulst ophthalmoskopisch sicher zerstört. Die übrigen 19 Fälle befinden sich noch in Behandlung.

Wir haben mit dieser Behandlung Ende 1952 begonnen. Bisher ist bei keinem der nicht enukleierten Patienten eine Metastasierung bekannt geworden.

Da die Lichtkoagulation die ernährenden Gefäße um der Tumor herum verschließt, wird dieser von seiner Umgebung isoliert. Die koagulierten Tumormassen können deshalb nur sehr langsam abtransportiert werden, und bleiben Monate bis Jahre lang sichtbar. Der langsame Abtransport des toten Pigments, kann leicht neues Wachstum vortäuschen. Man kann die Lichtkoagulation auch mit der WEVE'schen Diathermie-Koagulation kombinieren, was wir insbesondere bei großen Tumoren im einzigen Auge durchgeführt haben.

b) Aderhaut-Metastasen von Brustcarcinomen sprechen im Gegensatz zu den Melanoblastomen gut auf Röntgen-Therapie und auf Hormonbehandlung an. Wir haben uns daher nur in 3 Fällen von kleinen Metastasen im zweiten Auge

zur Lichtkoagulation entschlossen. In 2 Fällen gelang es den Tumor zu zerstören. Im 3. Fall wurde die Behandlung durch Röntgenbestrahlung fortgesetzt.

c) Das Retinoblastom (Glioma retinae) ist eine der bösartigsten Geschwülste des frühesten Kindesalters. In etwa 20 % der Fälle sind beide Augen betroffen.

Obwohl wir bei anderen Tumoren gute Erfolge mit der Lichtkoagulation gemacht haben, haben wir verhältnismäßig spät erst die Koagulation von Retinoblastomen versucht. Der vorwiegende Grund für diese Zurückhaltung liegt in der Tatsache, daß die Röntgen-Therapie des Retinoblastoms durch die von REESE und STALLARD eingeführten Verbesserungen wesentliche Fortschritte gemacht hat. Trotzdem lassen sich Strahlenschädigungen der Linse und der Haut und spätere schwere intraoculare Blutungen nicht immer vermeiden.

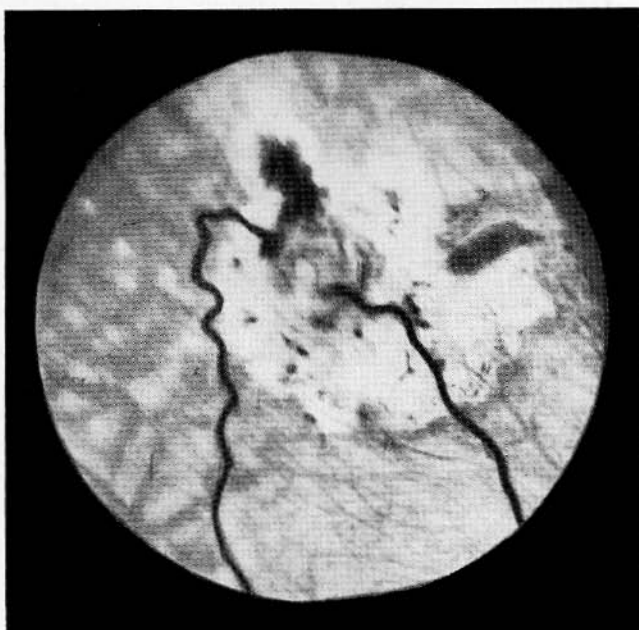


Abb. 2. Derselbe Tumor wie Abb. 1, 6 Wochen nach Lichtkoagulation.

Der erste von uns mit Lichtkoagulation behandelte Fall (1955) nahm einen so günstigen Verlauf, daß wir diese Therapie systematisch ausgearbeitet haben.

Wir haben seitdem in 16 Fällen mit doppelseitigem Gliom das 2. Auge mit Lichtkoagulation behandelt. In 3 Fällen war der Tumor so groß seine Zerstörung nicht gelang. Die Behandlung wurde mit Röntgenstrahlen fortgesetzt. In den anderen Fällen gelang es, die Tumoren durch wiederholte Lichtkoagulation

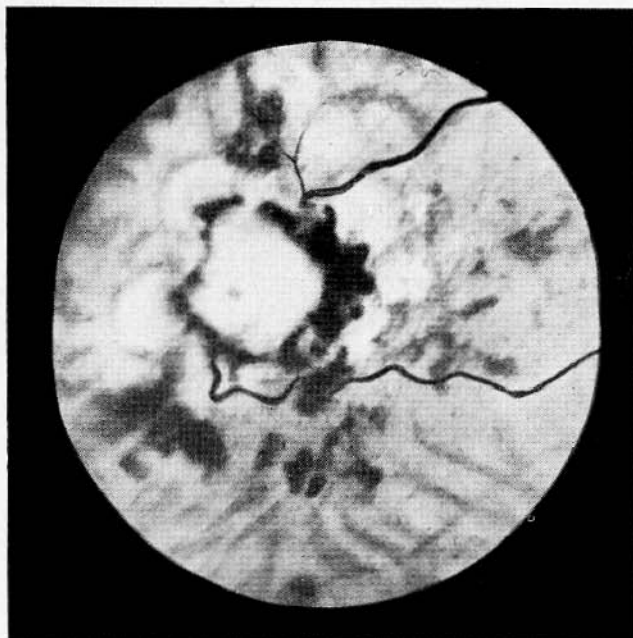


Abb. 3. 2 Tage nach der
3. Lichtkoagulation.

zu zerstören. Die Geschwulst verwandelt sich in eine glatte, pigmentierte Narbe, in der weiße, kugelige Kalkbröckel eingelagert sein können.

Die Retinoblastome neigen zu multiplem Auftreten und zu Recidiven im Zentrum oder am Rande der Koagulationsnarbe. Die Kinder müssen also sehr genau in Narkose in Abständen von 2 - 4 Wochen untersucht werden.

Die guten Erfolge, die wir bei kleinen Retinoblastomen erzielen konnten, führte zu der Forderung bei allen Kindern die bereits ein Auge an Gliom verloren haben, eine sorgfältige Überwachung des 2. Auges durchzuführen. Für diese Untersuchung haben wir ein eigenes Schema ausgearbeitet, (Meyer-Schwickerath, Helferich, Klin. Mbl.)

d) Die Angiomatosis retinae (von Hippel-Lindau) ist sicher eine der dankbarsten Indikationen für die Lichtkoagulation. Auch in Fällen von zahlreichen Tumoren in allen 4 Quadranten der Netzhaut und in Maculanähe ist eine schonende Zerstörung der Knoten durch Lichtkoagulation möglich. Wir haben in zwei Augen mit 7 bzw. 8 Tumoren eine Zerstörung der Knoten erreicht, ohne ernstliche Komplikationen zu erleben. Es hat sich gezeigt, daß man große Angiomknoten nicht zu schnell zerstören soll, da sonst Netzhautablösungen und schwere Kreislaufstörungen auftreten können.

Gerade hier zeigt sich der große Vorzug der Lichtkoagulation die gegenüber der Diathermic-Operation beliebig oft wiederholt werden kann. Es ist also eine langsame Zerstörung der großen Angiomknoten möglich. Wir haben bis jetzt 24 Patienten mit dieser fast immer auf beiden Augen auftretenden Erkrankung behandelt. Mit Ausnahme von einem riesigen Angio-Endotheliom neben der Papille gelang es, alle behandelten Tumoren zur Zurückbildung zu bringen.

Die guten Ergebnisse bei der Behandlung von Netzhautangiomen sind umso erfreulicher, als die operative Prognose der zugehörigen Kleinhirntumoren ausgesprochen günstig ist.

e) Der Angiomatosis retinae nahestehend ist die Miliaraneurysmen retinitis (Leber). In 3 Fällen haben wir nach vorsichtiger Koagulation der Aneurysmen einen Rückgang der ausgedehnten gelblich-weißen Netzhauterde gesehen.

f) In der gleichen Weise sind Haemangiome der Aderhaut und der Netzhaut der Lichtkoagulation zugänglich. Wir verfügen über je einen Fall dieser seltenen Erkrankung, die auf Lichtkoagulation gut angesprochen haben.

g) Iristumoren lassen sich ebenfalls mit Lichtkoagulation behandeln. Wir benutzen hierzu ein einfaches Zusatzgerät, welches ursprünglich für die Pupillenbildung in linsenlosen Augen entwickelt wurde. In diesem Gerät werden die Lichtstrahlen durch die Lupe zu einem Brennpunkt gesammelt, der unter Lu-

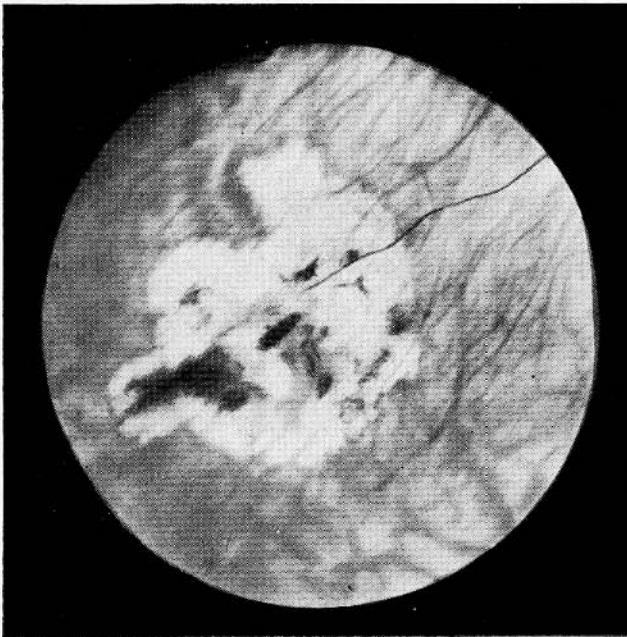


Abb. 4. 4 Wochen nach der 3. Lichtkoagulation.

penkontrolle auf die Iris gebracht wird. Um die Hornhaut vor Austrocknung und Erwärmung zu schützen, wird ein Plastikring auf den Limbus gesetzt, der mit Kochsalzlösung gefüllt wird. Durch dieses Wasser hindurch, durch die Hornhaut und durch die Vorderkammer wird der abgeschwächte Focus auf die zu koagulierende Stelle der Iris gelenkt. Bei Öffnung des Verschlusses setzt im Focus die Koagulation des Gewebes ein. Normalerweise bevorzugen wir bei abgegrenzten Iristumoren die Iridektomie, um nachher eine histologische Diagnose stellen zu können. Nur in einem Fall in einem Auge, dessen Partner praktisch erblindet war, haben wir ein kleines wachsendes Melanosarcom durch Lichtkoagulation zerstören können. Die Linse zeigte unter der Koagulationsstelle eine kleine subkapsuläre Trübung, die sich nicht veränderte.

In einem anderen Fall hatte ein Haemangiom der Iris zu zahlreichen Blutungen Anlass gegeben. Durch eine zweimalige Licht koagulation konnte das Angiom zerstört werden und die Blutungen blieben aus.

Haut-und Bindehauttumoren

Mit dem gleichen oben beschriebenen Zusatzgerät lassen sich auch Haut- und Bindehauttumoren behandeln. So haben wir ein Limbuscarcinom und ein Melanoblastom durch zweimalige Lichtkoagulation völlig zerstören können, ohne das Auge zu schädigen.

Die meisten Erfahrungen haben wir mit Xanthelasma, die nach Lichtkoagulation ohne Narben verschwinden. Der Vorzug der Lichtkoagulation liegt darin, daß die obersten durchsichtigsten Hautschichten fast unverletzt bleiben, da die Lichtabsorption im wesentlichen im Xanthelasma selbst vorsich geht. Das gleiche gilt für die Behandlung für Haemangiome der Lidhaut.

Insgesamt wurden bis August 1958 folgende Tumoren behandelt:

1. Melanosarcome	63	davon 20 enukleiert
2. Carcinommetastasen	4	
3. Retinoblastome	20	davon 3 enukleiert
4. Angiomatosis retinae	24	
5. Miliaraneurysmenretinitis (Leber).	3	
6. Tuberöse Sclerose	1	
7. Iristumoren		
a) Melanosarcom	1	
b) Angiom.	2	
8. Limbustumoren	2	
9. Hauttumoren	56	

176

Augen Klinik Venusberg.

MICROVENTOSA PARA LA EXTRACCION DE LA CATARATA

POR

JULIO MORENO, M. D.

Granada, España

Con el mayor gusto escribo estas líneas en homenaje del gran maestro Dr. Ignacio Barraquer, al que tantos vínculos me unen.

Se remontan mi devoción y recuerdos al año 1926, fecha de mi encuentro con este gran hombre, rama ya de aquel tronco señero que fue don José Barraquer y Roviralta, primera gloria de la Oftalmología española.

La acogida en aquella Clínica de la Ronda de San Pedro fue para mí verdaderamente excepcional y mi vida toda ha girado alrededor de este momento.

Las enseñanzas teóricas, el modo de hacer, el sentido de la alta clínica, todo rezumaba allí, apto ya para ser incorporado al bagaje científico y para formar un modo de ser en la vida.

Concretamente, en la operación de la catarata había que luchar en aquellos años con la quistitomía tan en boga, y era absolutamente excepcional encontrar partidarios de la extracción total. La constancia y fe, basada en la verdad, con que BARRAQUER llevaba adelante su técnica han dado ya su fruto, y hoy nadie discute la supremacía de su técnica.

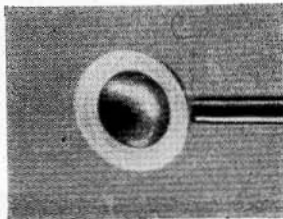


Fig. 1. Ventosa MORENO.
Detalle ampliado.

Yo he seguido fielmente la facoerisis utilizando el motor eléctrico cuya instalación es algo penosa para quien no tiene una Clínica propia para la hospitalización de sus enfermos. Con esta técnica he operado siempre y me cabe el honor de que la primera facoerisis hecha por José Ignacio BARRAQUER fuera junto a mí en mi Clínica de Granada. Luego José Ignacio ha escalado las altas cumbres de la especialidad y hace honor a tal padre y a tal abuelo.

Hasta 1946 usé el motor y crisifaco originales; pero en ese año, la lectura de un trabajo de Pérez Lforca me llevó a utilizar un modelo de ventosa manual que, modificada por mí, ha dado, desde entonces, excelentes resultados.

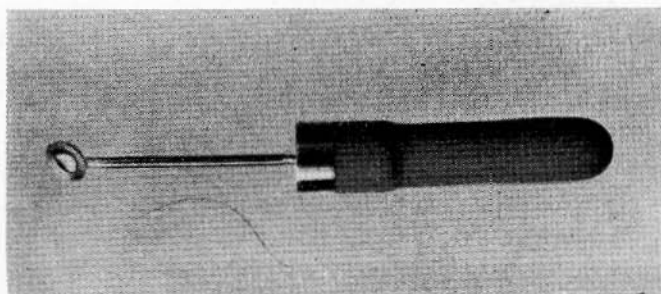


Fig. 2. Ventosa MORENO
Tamaño natural.

Mi modelo utiliza como "fuente de vacío" una tetina de goma de las que se usaban en los cuenta gotas de los colirios. Está construida en platino y su borde es plano con inclinación adecuada para adaptarse a la curva de la cara anterior del cristalino. Con esta ventosa he conseguido la extracción total en un 95% de los casos, siempre en enfermos de más de 40 años. El problema estaba en conseguir la rotura zonular en enfermos más jóvenes. Y vuelve a ser otro BARRAQUER, Joaquín, el que recientemente descubre la zonulolisis, que ya todo el mundo conoce, con la que se vence ese no pequeño escollo.

Puedo asegurar que la operación de la catarata debe a la estirpe BARRAQUER lo fundamental de la actual perfección.

Mi pequeña ventosa no puede mejorar al erisifaco, pero sí hace más fácil el empleo de la técnica, especialmente a quien no tenga instalación adecuada de Clínica.

Termino renovando mi cariño y fervorosa gratitud a esta familia y felicitando con la mayor efusión al Profesor BARRAQUER, para el que pido a Dios largos años de vida.

NUEVO SIGNO CAMPIMETRICO

POR

HECTOR M. NANO, M. D., HUMBERTO PEREZ, M. D.

Buenos Aires, Argentina

Al abordar el estudio detallado de las investigaciones de que han sido objeto pacientes con aparente dispar patología oftálmica encontramos un común denominador en la esfera ocular, papila pálida y nuevo signo campimétrico. Basados en ello hemos hallado en la tabulación estadística, elementos comunes que nos llevaron a la concepción de este trabajo y comunicar los resultados obtenidos. Este estudio ha sido realizado en pacientes diversos en forma sistemática y personal, para igualizar las condiciones de examen, y ello nos ha permitido la visión del síndrome ocular con sus signos objetivos y subjetivos.

El síndrome está integrado por signos y síntomas pesquisados mediante un tipo standard de examen ocular constituido en lo fundamental y práctico por:

- 1) La indicación primaria del examen básico fue la de todos aquellos pacientes que necesitan examen campimétrico.
- 2) Campimetría y test macular de Amsler - Visión cromática.
- 3) Historia clínica.

Los casos que así lo requieren se completan con exámenes especiales. Consideramos fundamental la historia clínica. Se practica en forma adecuada disponiendo el tiempo necesario en cada caso. Pensamos racionalmente el beneficio que otorga en la revisión de antecedentes, signo, etc., una comprensión mutua que solo se logra con tiempo y confianza adecuada. Cuantas veces nos ha sorprendido, en un balance final, el inmenso valor de la historia clínica, al lado de comprobaciones de laboratorio u otras, costosas en tiempo y dinero y a veces no del todo inocuas.

Con respecto al fondo de ojo se ha efectuado reiteradamente en todos los casos y en un porcentaje respetable es la papila pálida el signo más notable y único.

La campimetría muestra otra de las características del síndrome: el escotoma que hemos denominado *del vertical superior* y la contracción de la isóptera 1/2000.

En un trabajo anterior (ver "Dialéctica Campimétrica") habíamos insistido sobre las condiciones mínimas exigibles para que la campimetría rinda al máximo sus posibilidades; a él nos remitimos.

El escotoma del vertical superior: este signo aparece casi constante. Su descripción corresponde a la de un escotoma absoluto y/o relativo ubicado entre los 21 y 24° en la pantalla tangente de Bjerrum justo sobre el radio de 90° esto es: el vertical; su altura vertical oscila entre 3 y 4° y su medida horizontal entre 4 y 6° de manera que es aproximadamente ovalado. Se destaca con el índice 2/2000. Los diagramas campimétricos de las Figs. 1, 2, y 3 muestran casos típicos.

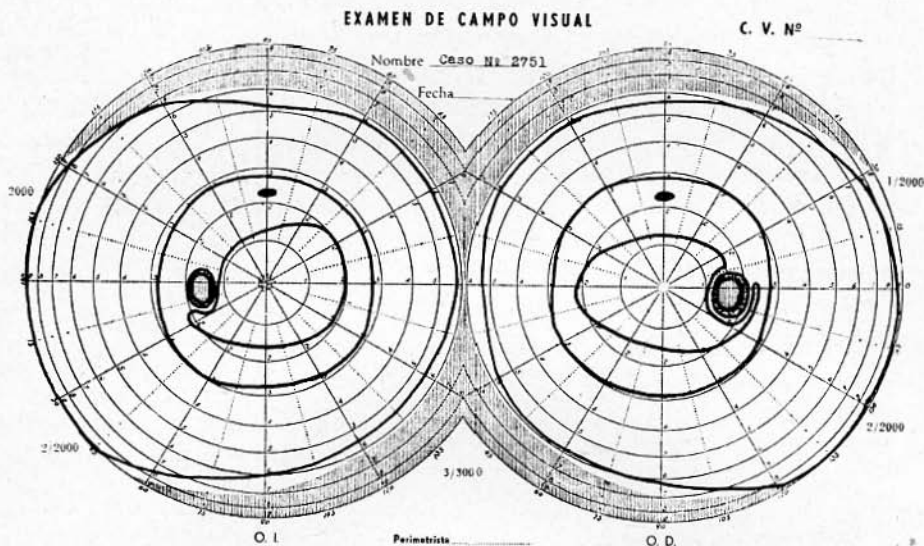


Fig. 1.

Su aspecto es muy parecido en todos ellos por lo cual publicamos solamente algunos de los diagramas obtenidos.

Su punto correspondiente en la retina, calculado trigonométricamente, está ubicado a 7, 2 mm. suponiendo que se encuentre a 22° y 8 mm., si aparece a 24°. Oftalmoscópicamente no hemos hallado alteración en esa zona ni sus proximidades. Su hallazgo puede pasar desapercibido en un examen de rutina en varias circunstancias a saber: falta de entrenamiento del paciente, pues el vertical superior puede equivocadamente ser el primer radio examinado, recordando de nuestro trabajo citado que la explicación previa y que en el transcurso se hace al enfermo no le deja entrever que es lo normal y que es lo patológico, de manera que ignoran, en lo posible, el significado de lo que observa; 2º el factor velocidad

NUEVO SIGNO CAMPIMETRICO

EXAMEN DE CAMPO VISUAL

C. V. N°

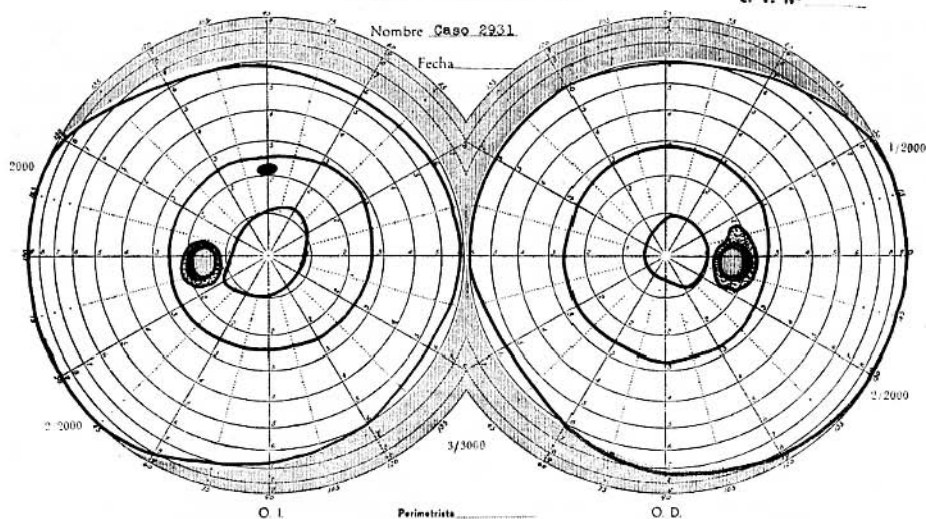


Fig. 2.

de desplazamiento del optotipo de 2 mm.; debe ser suficientemente lento como para no provocar una ambliopía de fijación o por el contrario no tan rápido que no pueda acusar esta alternativa, por razones que son obvias.

El escotoma del vertical superior se ha comprobado bilateralmente en respetable porcentaje de los casos (90%) y unilateral en los restantes. Hasta el momento hemos reunido 32 observaciones, en 1290 campimetrías o sea aproximadamente en un 3% de los diagramas obtenidos, porcentaje no despreciable.

Cuidadosos, prolongados y repetidos exámenes campimétricos en estos pacientes no nos han permitido comprobar el escotoma en otros radios, lo cual descarta la posibilidad de un artificio.

Es un signo "no espontáneo" pues hay que investigarlo, buscarlo, es absolutamente objetivo.

En la bibliografía a nuestro alcance que hemos revisado en la posibilidad de nuestros medios no hemos encontrado la descripción ni la mención de este pequeño escotoma, que más allá de lo que pueden aclarar estas líneas se comprendería fácilmente con el estudio de los diagramas campimétricos que acompañan este trabajo.

Esta característica que sin ser común es relativamente frecuente, no lo hemos encontrado mencionado en los textos ni revisando los diagramas campimétricos de los tratados de C.V. más conocidos.

En una comunicación verbal el doctor Roberto Sampalessi, nos ha mencionado que ha comprobado la existencia del escotoma con características ligeramente diferentes en cuanto a tamaño, en un caso de glaucoma. La contracción de 1/2000; esta contracción comienza primero en el sector superior, luego hace una exclusión papilar y por fin se contrae casi concéntricamente. Esto explica por qué solo el índice de 2 mms. detecta el escotoma. La contracción de la isóptera es un elemento constante y otro signo del síndrome.

No se observan otras alteraciones campimétricas cuando el cuadro es simple, no complicado, o sea en estado de pureza que es lo común. Hemos desechado para su consideración de conjunto aquellos casos que presentaban otras alteraciones de fondo de ojo que podrán inducir a error de interpretación.

La isóptera 3/3000 es siempre normal.

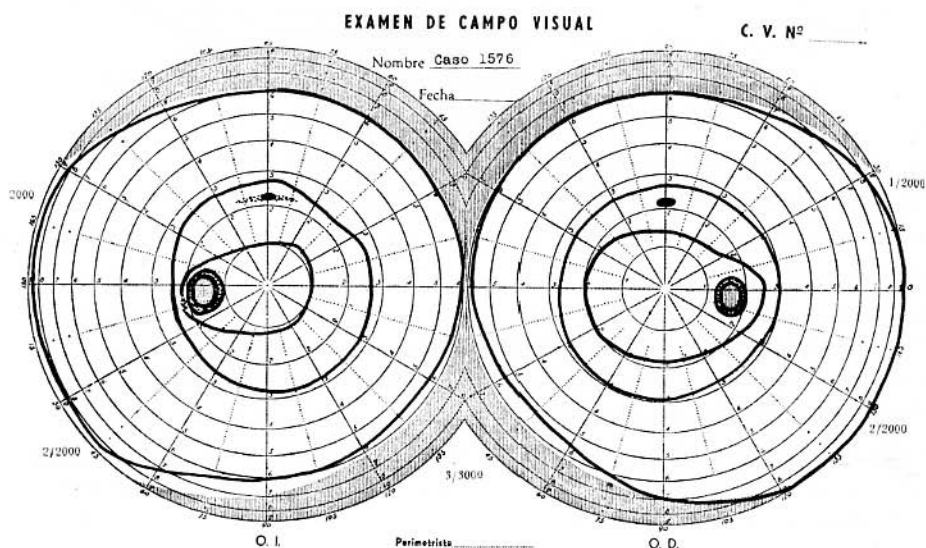


Fig. 3.

El test de Amsler presenta en casi todos los casos sus alteraciones características, metamorfomas, escotomas translúcidos, ampliopia de fijación, etc., etc., sin alteración patognomónica pero con frecuencia notable.

El examen de la visión cromática investigado con las tables de Harding, Rand y Ritter (H.R.R.) no ha demostrado nada particular aunque en muchos casos no es normal.

La interpretación del escotoma del vertical superior abre un campo de investigación y experimentación oftalmológica que es digno de profundizar.

El elemento objetivo de observación casi constante que acompaña los signos campimétricos descritos es la papila pálida.

La papila pálida ha reconocido infinidad de etiologías pues baste recordar para ello que es siempre el estado previo obligado de atrofia o hipotrofia de papila cuya gama etiológica es igualmente vasta.

Con respecto a la vinculación probable en el orden clínico oftalmológico podemos mencionar (en estudio) el estado glaucomatoso y preglaucomatoso. Recordemos que la contracción de las isópteras campimétricas en el sector externo es signo precoz de glaucoma crónico simple.

En el orden clínico general en la gran mayoría de los casos, un porcentaje que supera holgadamente toda coincidencia se encuentran alteraciones típicas del neurovegetativo.

CONCLUSIONES

Hemos comprobado la existencia de un nuevo signo campimétrico: el escotoma del vertical superior y la contracción supero-externa de la isóptera 1/2000.

Se ha encontrado este signo vinculado en forma principal al estado de papila pálida.

En el orden general se ha constatado como la vinculación más constante, alteraciones del sistema neurovegetativo correlacionadas con facilidad en el criterio de Bergman.

(Hemos comprobado 100 campos visuales en que no existía el escotoma del vertical superior y hemos encontrado trastornos del N.V. en solo 2% de los casos).

Se han constituido por lo tanto, elementos de juicio suficientes para un nuevo síndrome.

Rivadavia 7047

NOTA: Los gráficos de campo visual han sido obtenidos con optotipos 3|300, 2|2000 y 1|2000 sin excepción.

SOBRE UM CASO DE FIBROMA PURO, DA ORBITA

POR

CLOVIS PAIVA, M. D., ALVARO GOMES, M. D.

Recife, Brasil

"The diagnosis by clinical methods of the presence of a tumor of the orbit is always difficult, and of the type of tumor frequently impossible". Duke-Elder, 1952.

O laudo histopatológico de um tumor de orbita, por nós removido e encaminhado para o exame de rotina, veio pôr-nos em contacto com uma raridades clínica, até então por nós insuspeitada um caso de Fibroma puro, da orbita.

Ao compulsar a literatura a respeito dos tumores da orbita tomamos conhecimento da baixa incidencia dos fibromas, nas estatísticas dos varios autores que têm se ocupado do assunto.

Isto animou-nos a trazer á luz o seu relatório.

O Fibroma é um tumor mesenquimal, de caracter benigno, de evolução lenta e de extrema raridade. Lipomas, Mixomas, Condromas e Osteomas são os outros tipos de tumores mesenquimais, benignos, que com mais frequencia do que o Fibroma podem ocorrer na orbita. Dos referidos tumores mesenquimais existe, apenas, um do tipo maligno que é o Sarcoma.

Birch-Hirschfeld (1930) fazendo uma revisão de literatura, até 1930, sobre tumores da orbita, catalogou apenas 30 casos de Fibroma, incluindo neste numero as formas mixtas (fibro-adenoma, angio-fibroma, fibro-mixona e neuro-fibroma).

Fowler e Terplan (1934) em trabalho publicado nos Archives of Ophthalmology, relataram um caso de Fibroma da orbita e 16 outros, também de fibroma, que encontraram na literatura mundial, descritos por outros autores.

Stokes e Bowers (1934) em 35.869 doentes matriculados até 1930 na New York Eye and Ear Infirmary, encontraram 9 casos de tumor da orbita, sendo 1 de Fibroma.

Schrech (1934) não encontrou casos de Fibroma entre os 259 tipos de tumores da orbita por ele examinados.

Dandy (1941) em 24 casos de tumores da orbita operados pela via transcraniana identificou um deles como sendo de Fibroma.

Reese (1941) descrevendo 174 casos de exoftalmia unilateral apresenta uma delas como motivada por Fibroma da orbita.

Forrest (1949) em 222 tumores orbitarios (184 primarios e 38 secundarios) encontrou 2 casos de Fibroma.

Guy Offret (1951) no seu importante livro sobre "Les Tumeurs Primitives de L'Orbite" relata o caso da uma sua paciente que era portadora de Fibroma puro, bilateral, da orbita. Ela não tinha exoftalmia, porem apresentava ptose bilateral das palpebras.

Almeida (1954) não refere no seu trabalho "Tumores da Orbita", casos de Fibroma.

Machado (1956) em 74.700 pacientes matriculados nos seus arquivos, encontrou 17 casos de tumores da orbita, não existindo, entre eles, o Fibroma.

Ozorio (1956) em 12 pacientes com tumores primitivos da orbita, registrados na Clinica Oftalmologica da Faculdade de Medicina de Porto Alegre (periodo de 1934 a 1956) não refere a ocorrencia de Fibroma.

Nos arquivos do nosso Serviço Oftalmologico (privado e hospitalar) não tivemos tido, até então, casos de Fibroma da orbita.

O Fibroma, segundo Duke-Elder, (1952) usualmente aparece entre os 16 e os 30 anos de idade; raramente depois. O seu desenvolvimento é muito lento e so ao cabo de muitos anos de evolução é ele capaz de determinar sintomas clinicos, do geralmente por compressão de estruturas visinhas e pela exoftalmia. Quando localizado na orbita o seu crescimento pode ser para diante, produzindo exoftalmia progressiva, irreductivel e de eixo que varia de acordo com a sua implantação. Quando o crescimento do tumor é para traz, geralmente o nervo optico sofre um processo de atrofia por compressão.

O Fibroma pode ser congenito como nos casos de Feyer, Cosmettatos, Perls e Steiner, citados por Offret (1951) e talvez o nosso proprio caso.

O tumor atinge, muitas vezes, tamanho consideravel. São encapsulados e apresentam estrutura fibrilar caracteristica.

Os sinais radiologicos geralmente são negativos em virtude de ser o tumor permeavel aos Raios X. Salvo quando ha calcificação no seu parenquima ou quando ele é congenito, como no caso de Cosmettatos e no nosso proprio, pois determina um aumento da cavidade orbitaria. No nosso paciente havia um afastamento da sutura do malar acarretando o aumento da orbita e uma discreta sombra tumoral no fundo da orbita.

O local de implantação varia muito. Pode ser no periosteo da orbita, nos musculos extrinsecos, na capsula de Tenon ou nas membranas envoltorias do nervo optico. Alguns sao encontrados dentro do cone muscular.



Fig. 1. Fibroma da Orbita.

A nossa observação pertence a um paciente do sexo masculino (P.R. de Lima), com 31 anos de idade, matriculado na Clínica Oftalmologica de Faculdade de Medicina da Universidade do Recife em 2. 9. 1956.

O referido paciente sentiu-se compelido a procurar o nosso Serviço em virtude da exoftalmia (O.D.) de que era portador e que teve inicio ha cerca de 3 anos passados. Referiu, tambem, que de alguns meses para cá vem sentindo dores periorbitarias que se irradiam para a metade correspondente da cabeça e da face.

O seu olho esquerdo apresentou-se ao exame, somatica e funcionalmente normal.

No olho direito, alem da exoftalmia irreductivel e direta, havia estase venosa na conjuntiva bulbar e fundo de saco conjuntival inferior.

No fundo do olho assinalamos discreto engorgitamento venoso. A acuidade visual era de $\frac{1}{2}$ e o campo visual apresentava a isoptera dentro dos limites normais. Os movimentos oculares estavam presentes, embora de amplitude reduzida.

Exames sorologicos para lues, negativos. Hemograma, normal, o mesmo succedendo com as taxas de ureia, creatinina e glicose.

Radiodiagnostico (Dr. Ypiranga de Sousa Dantas): Afastamento da sutura do malar direito determinando aumento da orbita correspondente, fundo orbitario opacificado (Filme Ap). Sombra tumoral perfeitamente nitida não atingindo ao osso. (Filme perfil).

Resolvemos intervir cirurgicamente em 22. 11. 1956. Dentro do cone muscular abordamos um tumor encapsulado, de cor rosada, ovoide, de superficie lisa, medindo aproximadamente o tamanho de um ovo de galinha (5x3 - 5x3) e preso por

frageis bridas aos musculos que com ele mantinham contacto. O tumor foi digitalmente mobilizado e removido com grande facilidade do seu leito hospedeiro.

O resultado do exame histopatologico realizado pelo Dr. Clovis Marques diz o seguinte: "Exame macroscopico: A peça cirurgica consta de uma formação tumoral, medindo 5x3, 5x3 cm., limitada por uma capsula fibrosa, consistência firme. Ao corte era de cor branca e aspecto fasciculado. Exame microscopico: A estrutura tumoral acha-se integrada por elementos alongados de natureza conjuntiva, formando feixes que se orientam em sentidos diversos. Não se observam sinais evidentes de anaplasia. Conclusão: Fibroma. (Figuras 1 e 2).

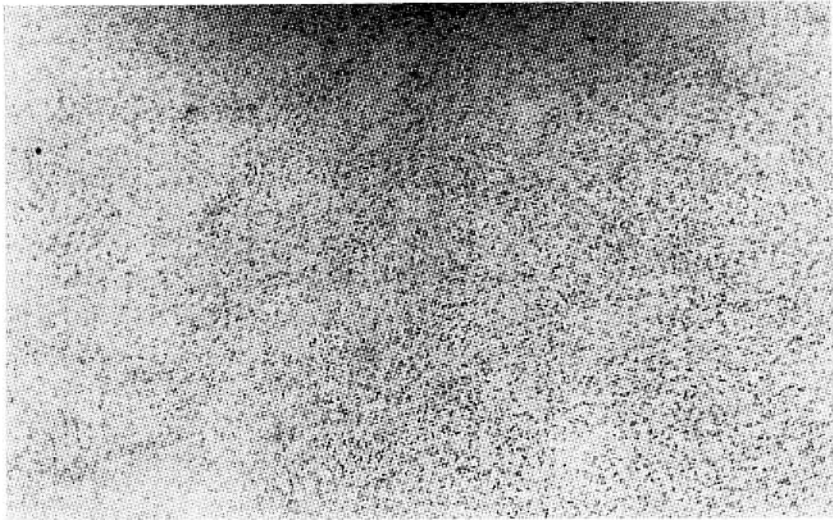


Fig. 2. Fibroma Puro da Orbita.

Universidade Do Recife

BIBLIOGRAFIA

1930. BIRCH-HIRSCHFELD, Fibroma der Orbita. Kurzes Handbuch Ophth. Schieck e Bruckner, vol. 3, 78. Springer Ed., Berlin, 1930.
1934. FOWLER, S. G., TERPLAN, K. L., Fibroma of the Orbit. Arch. of Ophth. 28: 263-271.
1934. SCHRECK, In Fowler e Terplan (Trabalho citado).
1934. STOKES, W. H., e BOWERS W. F., Pure Fibroma of the Orbit. Arch. of Ophth. 21: 278.
1941. REESE, A. B., Orbital Tumors. Am. J. Ophth. 25: 386.
1941. DANDY, W. E., Results following the transcranial operative attack on orbital tumors. Arch. of Ophth. 25: 191.
1949. FORREST, A. W., Intraorbital Tumors. Arch. of Ophth. 41: 198.
1951. OFFRET, G., Les Tumeurs Primitives de l'Orbite. Masson e Cia. 1951.
1952. DUKE-ELDER, S., Text-Book of Ophthalmology. Vol. 5: 5532-5534. Henry Kimpton. London.
1954. ALMEIDA, A., Tumores da Orbita. Rev. Bras. Oft. 13: 147-178.
1956. MACHADO, N. R., Tumores Primitivos da Orbita. Rev. Bras. Oft. 15: 221-340.
1956. OZORIO, L. A., Tumores Primitivos da Orbita. Boletim Informativo da Soc. Oft. e Oto-rino-laring. do Rio Grande do Sul. Ano 1, Nº 4.

PHOTOCOAGULATION OF EYE MEMBRANES

BY

EMILIO RAVERDINO, M. D.

Milan, Italy

If one looks directly at the sun an inflammatory hearth will appear on the macula, which may completely disappear if fixation has been fugacious and there was only little luminosity but otherwise will produce a retinochoroidal scar with a permanente pigmentation, causing the retina to adhere more intimately to the choroid. Te so-called photocoagulating apparatuses (so far it seems there are only two of the kind, one in Bonn and the other in Milan) by producing a light beam similar to that of the sun may provoke, at libitum and in any point of the retina, some scars which are like those produced by the sun; they are therefore used for treating the detachment of the retina or for destroying the centre of retinal diseases.

Raverdino's photocoagulating apparatus has been constructed by Messrs. Prevost in Milan and is providing a lighth beam strong enough to coagulate more or less intenseley the retina according to the time of exposure, producing round white spots with a diameter smaller than the papilla. A light flash of $\frac{1}{4}$ of a second already produces a slight discolouration of the retina; in the rabbit a 10 seconds exposure provokes an intense coagulation which may result in the perforation of the retina during the following days. In view of the quasi identity between the solar spectrum and that of the Prevost apparatus the pathogenic problem of the sun retinitis may be considered to be similar to the ons of artificial coagulations. The problem not yet resolved is whether the coagulation is due to the heat produced by the light beam (Duke Elder) or to a thermic storage at the level of the retinal pigment (Leplat) or if it is not a matter of heat action but of a puraly photodynamic action (Cirincione, Cioceri, Seidenari).

Raverdino is inclined to support this latter opinion as the measurements of these latter Authors, carried out under different circumstances, never have shown on the retina a temperature over 40° C. The use of a photocoagulator may supply some probative data for this problema, while it may also raise many problems for the biologist as well as for the practical oculist. Among these problems the main ones are the following.

1. Which surgical problems of the ophthalmic speciality may be resolved by means of the photocoagulator?
2. The so-called photocoagulation phenomena in the eye membranes and specially in the retina, are they only due to the heat or to other agents?
3. What kind of modifications may take place, especially in the dioptric means of the eye or in other membranes too, which are crossed by a light beam having a spectrum similar to that of the sun?

As for the first point, Meyer Schwickerath (Bonn), taking advantage of his vast clinical experience, has by now resolved nearly all problems regarding the retina and iris membrane. There is no doubt that only after publication of the complete documentation of the many Authors already employing photocoagulation, ophthalmologists will have an idea of the great advantages offered by photocoagulating apparatuses.

Indication of photocoagulation for retinal diseases may thus be summarized: Photocoagulation can be employed for coagulating the rims of any retinal rupture (using a particular care one may even attain very peripheral ruptures), provided the retina is adhering to the choroid and the dioptric means are sufficiently transparent. Photocoagulating treatment is therefore particularly indicated:

1. For retinal ruptures not yet followed by detachment.
2. Whenever there are some areas of the retina subject to rupture or areas of atrophic chorioretinitis liable to form ruptures or detachments. This is more indicated as a prophylactic measure in order to avoid the detachment in the second eye, when there has already been a detachment in the first one.
3. For macular or paramacular ruptures or any other rupture at the back pole nearly unapproachable by the usual surgical means.
4. In the case of multiple ruptures having different seats, and here photocoagulation will follow immediately a first operation for reapplying the retina.
5. For retinal ruptures not yet followed by detachment, in the presence of an actual exudative, inflammatory chorioretinitis, provided the dioptric means are transparent enough in the rupture area.
6. In reoperations which are necessary after an operation which achieved only a partial reapplication of the rupture rim, or whenever there is a new retinal rupture immediately after a first intervention, situated near the coagulated area or far from it, provided the retina is attached.

Finally it can be said, that this new method is liable to bring about a revolution in the classical principles for the treatment of retinal detachment. In the end it would no longer be necessary to reach by surgical means and with some difficulties the ruptures distributed here and there, and specially in the back pole, in

order to coagulate their rims, as photocoagulation will simplify the surgical problem due to the fact that it will be sufficient to reattach the retina and then photocoagulate the rupture. Consequently those methods which were considered exceptional or too simple, like the excision of sclera flaps, the evacuations of subretinal liquids, air injections, will acquire a greater value and more frequent application, a fact which also represents a technical revolution.

Some other indications for photocoagulation in retinal diseases, besides the detachment, are: retinal agiomathosis, which cannot be treated medically but may be cured by massive photocoagulations. Not pre-eminent, superficial retinal tumors, specially if they are not very large and if they are pigmented. Haemorrhagic hearths of the retina with a character of recidivism (haemorrhages of young people, periphlebitis, etc.) and whenever medical treatments have shown to be not sufficient. Meyer Schwickerath, by focussing the light of the apparatus on the iris has been able to pierce this membrane without damaging the cornea. In order not to alter the crystalline these artificial pupils can only be executed in aphacic subjects.

Raverdino has used photocoagulation for the treatment of skin diseases, of diseases of the conjunctiva and the cornea, by focussing the beams for destroying the small xanthelasma or cutaneous neoformations, insisting for some seconds. These coagulations take place due to the heat and are crater-shaped. With the same system also the cornea can be cauterized spot by spot, but coagulation is too fast that one could control its effect. That is why this method is not useful in practice and it is more advisable to concentrate the beams on the conjunctiva or on the cornea through a filter glass, as it will be seen later on.

The essence of the problems inherent to the second point consists in establishing whether all coagulations are due to the heat or whether some of them represent the result of a photodynamic phenomenon, considering the fact that usually coagulation by heat only takes place between 60 and 70° C. Now, with the photocoagulator we obtain, on the conjunctiva as well as on the cornea, some whitish coagulation at temperatures which are much lower than those above mentioned. This phenomenon cannot be observed very well when we concentrate by a lens the light on the conjunctiva or on the cornea, as after one second temperature on the point of concentration already reaches 55° and will attain 60 after 2 seconds and 71° after three sec., but if temperature is reduced by means of a small filter glass having a laminated surface in touch with the tissue, it will be possible to observe some clear and evident superficial white spots even at temperatures which are much lower than those mentioned above. Between 37 and 50°C, according to length of exposure, some white spots being more or less and deep will appear, and their histologic examination will show that they are regressive

cellular alterations. This is more easy to demonstrate on the corneal epithelium and the phenomenon is so distinct that, by varying exposure times, it is possible to obtain a really separated coagulation of the various layers of cell in the corneal epithelium. A veritable coagulation (with the forming of the usual coagulated gelatinous layer) will be attained only when —insisting in coagulating— temperature is over 60°C. But even in this case the Bowmann membrane and the last layer of the corneal epithelium can be maintained unimpaired. As the first regressive alterations of the cells start at sub-fever temperatures it is clear that they cannot be ascribed to heat but to photodynamisms only. From the therapeutic point of view these superficial or adjustable in depth photocoagulations have shown to be useful in the case of keratitis bullosa, of denticritic and viral keratitis, as they are remarkably shortening the course of illness and destroying the ill epithelium without producing deep scars, which may in future be responsible for visual damages. Of course the photodynamic phenomenon is more visible in the case of photocoagulation of the retina, due to the great quantity of photodynamic substances of the retina itself (Santamaria, De Vincentiis). But also with these applications it can be shown that, by introducing a thermoelectric bit in a sac between sclera and choroid in a rabbit, during a coagulation protracted even for 4 seconds, temperature never surpasses 40°C. This is confirming the experiences made by Ciaceri, Cirincione, Seidenari's observations and Duke Elder's studies.

All this is therefore indicating that on the retina as well as on the cornea and on the conjunctiva a very intense light beam may produce well visible cellular alterations.

The third point is comprising already ascertainable phenomena and problems arising when consider the many reactions intense light is able to produce on the different chemical componets of substances and liquids contained in the eye liquids in a normal pathologic stage.

It is difficult to foresee what difficult experiments biologist may organize in order to study the behaviour of the different organic liquids, of the blood and the manifold tissues under the influence of a long and protracted illumination. Thanks to the photocoagulator Viale's wellknown investigations on the biologic actions of radiations are subject to be widened and to become more exact, considering that whole body of doctrine which has now been elaborated by various Institutes and particularly by Ciaranfi's School about the action of photodynamic substances.

Nor is it possible to tell at present what advantages will be offred to the studies and applications of the oculistic speciality. But it may already be stated that in

PHOTOCOAGULATION

some cases irradiation of the vitreum being turbid for one reason or the other and stuffed with pigmented corpuscles has brought about a modification and even a clearing of the vitreum itself. Nor can we doubt of this action if we know the great activity of light on colloids (and cornea as well as vitreum are colloids) and if we know that many of the substances contained in the turbid vitreum are extremely sensitive to light, like, for instance, cholesterol.

That is why apparatuses which are simple and handy like the photocoagulatos already used are representing not only a definite progress for the oculistic speciality, but also a useful instrument for investigating fundamental biologic problems.

Main Hospital

Confesiones de un Operado

MODALIDADES SENSO-PERCEPTIVAS EN UN AFAQUICO

POR

EDMUNDO RICO, M. D.

(Profesor de Clínica Psiquiátrica de la Universidad Nacional)

Bogotá, Colombia

*"Al Profesor don Ignacio Barraquer, de
Barcelona, en el septuagésimo quinto ani-
versario de su natalicio".*

En la vida de algunos hombres quedan flotando recuerdos, reminiscencias verdaderamente imborrables. Por sobre modo, de aquellas épocas enmarcadas en incertidumbre. De mí sé decir que el once de julio de 1957, señala hito gratísimo, porque en la mañana de ese día —merced a brillante intervención oftalmológica— mis ojos, al tornarse, en un solo tiempo operatorio, bilateralmente afáquicos, recuperaban la visión perdida.

Es fenómeno harto conocido que quienes de tiempo atrás van quedándose ciegos —sabiendo, desde luego, que el único tratamiento radica en la cirugía— nó por ello pierden la ilusión, hasta cuasi los postreros instantes, de licuar las opacidades o esclerosis de sus cristalinos, apelando a fórmulas galénicas o magistrales, colirios sui-generis, grageas homeopáticas e inclusive, a toda una gama torturante de métodos bioeléctricos.

¡Qué vamos a hacer! Así es, así será siempre la condición humana. Porque nada ni nadie desmoraliza en tamaño grado la personalidad como el asistir, impotente, a la fuga inmisericorde de la luz y del color, de la forma y del espacio para sumirse, de cuajo, en sórdida obscuridad.

Ciertamente que lo más doloroso, lo más corrosivo de este desmoronamiento paulatino del Yo, son los oleajes físicos de la angustia, compañeros inseparables de aquella otra resonancia psíquica que es la ansiedad.

Para fortuna mía, topé con un cirujano oculista, engastado en no menos diestro psicólogo. A la par que, previamente, estudiara mis reacciones y defensas orgánicas, calaba —con discreta elegancia de hombre de mundo— dentro de las entretelas nerviosas de mi psiquismo, advirtiéndome, previniéndome y enseñándome luego a corregir los trastornos senso-perceptivos inherentes a la extracción de cristalino.

* * *

Al día siguiente del acto operatorio —verificado bajo la doble tranquilidad del “coctel lífico” y de una aquinesia perfecta —maniobra quirúrgica donde en una sola sesión, quedéme afáquico de ambos ojos— se me autorizaba moverme en el lecho, sentarme, dejarme afeitar y hasta tomar un baño del cuerpo si lo apetecía.

Provista, aún, mi memoria con una serie de leyendas y consejas antañeras, referentes a que en las operaciones oculares (notoriamente las habidas en los cristalinos) era menester guardar, durante muchos días, inmovilidad absoluta, vacilé algún tiempo, en acatar las insinuaciones del oftalmólogo.

Mas, bien pronto, sacudiendo, avergonzado, semejantes telarañas arcaicas, reaccioné y entreguéme, entonces, a ejecutar, en veces, con ayuda de la enfermera, cuanto se me ordenaba.

Para mí, para mi idiosincracia, lo suplicante fue permanecer vendado por espacio de ocho días. Detesto la obscuridad. Quizás, ello se deba, entre otros motivos, al asco o desprecio que inspiran ciertas conciencias entenebrecidas por estenosis del sentido moral.

Confieso, sin sombra de hipérbole, que el día más feliz de mi existencia, ha sido aquél cuando ya portador de mis primeros lentes, torné a ver como en mejores tiempos. Al sentirme inundado de claridad circundante; al avizorar, al través del ventanal abierto, las casas, calles y carruajes aledaños, no pude menos de evocar —por contraste paradójico— las palabras pre-agónicas de Goethe: “Luz, más luz...” Solamente que con ellas, el pensador de Weimar, despedíase de la vida, mientras ahora, yo, exigua molécula vegetando en un repliegue andino de Monserrate, renacía a la luz, a esta luz de los ojos, que fisio-psicológicamente, es emanación crucial del espíritu.

Durante el tiempo transcurrido en la clínica, —visitado, de continuo, por cordiales amistades cuyos lazos afectivos tonificábanse en la confianza de algunos whiskys— no escatimaba yo, horas meditativas para educar mi voluntad (rehecha por la facoerisis) hacia las dificultades que en adelante como afáquico, habría de superar, costárame lo que me costara.

El oftalmólogo solía repetirme, paciente y atinadamente: “Usted debe acostumbrarse poco a poco, con calma, a dominar la falsa orientación, la aberración esférica y la carencia de orientación, que vienen a ser como el trípode que en las dos primeras semanas importunan al afáquico. Ud. franqueará pronto esas etapas desagradables. Ya lo verá.”

Mi fuero interno sabía que esta amable sugestión hallaba terreno propicio. Por lo demás, la experiencia y sinsabores humanos de antemano enseñan que, en ocasiones, “querer es poder”.

* * *

Y, así fue en verdad. Media hora después de usar los lentes correctores, aprendía la marcha en terreno plano. Salí de mi habitación encaminándome hasta el extremo del corredor sin ayuda de nadie; varias veces hice el mismo recorrido, saludé a varias personas, sintiéndome y pisando tierra firme, ni más ni menos que si fuese un conquistador español. El primer paso estaba dado y “lo difícil en la vida es el primer paso”, reza un proverbio francés.

Las sinergias funcionales para movimientos automáticos y pertinentes como tomar una cuchara, escribir, anudar la corbata, afeitarme, etc., tampoco demandaron mayor esfuerzo.

En cambio, la tal “aberración esférica” resultó terrible para mis senso-percepciones visuales: apenas salido del sanatorio, encontré en sus umbrales, al profesor Jorge E. Cavellier, cuya estatura airosa e inconfundible, se me transformó en esos momentos en la de “algún descomunal gigante” que dijera Don Quijote. Así que, al estrechar en mis brazos agradecidos, aquella talla inmensa, cóncava y retorcida como árbol centenario, sentí cuando depositaba sus brazos afectuosos sobre mis hombros, que un boxeador hercúleo se me venía encima, no teniendo otra válvula de escape sino dejar brotar mi agradecimiento ecuménico, en esas burbujas de vapor de agua, que los poetas llaman lágrimas.

Otra sorpresa de la maldita “aberración esférica”, me esperaba: mi amigo don Josué Murillo, propietario tradicional de “La Gran Vía”, y descendiente santafereño de los fundadores de “La Gruta Simbólica”, me había enviado, espontáneamente, su lujoso automóvil. Y, ¿qué percibieron mis ojos al través de los lentes? Nada menos que un colosal barco negro cóncavo y quebradizamente deforme, cuyo conductor, ataviado por las mismas características, me pareció ser el Aquerón que conducía su barca fantástica, hacia las procelosas zonas del Infierno dantesco. En tal percance recordé —el hombre es surtidor inconciente de vivencias— al reverendo Hermano Cristiano Didier, quien ahorcara sus hábitos, cuando en el Instituto de la Salle, al iniciarnos en los estudios elementales

nos decía: “no olviden ustedes, los ateos, que Dios se les presenta en efemérides especiales de la “justicia celeste”.

Y, ciertamente que al contemplar aquel hermoso vehículo, transformado, ahora (como en algún delirio mescalínico) por el aberrante complejo esférico, en monstruosa cuanto flexible artesa, veía, así mismo e imaginariamente, al voluble ex-Hermano Didier convertido en desmesurado pingüino merced a “circunstancias especiales de la justicia celeste”.

* * *

Cuando llegué a mi residencia, intentando apearme del coche brindado por “el chato Murillo”, otra de las dificultades afáquicas púsose aquí de manifiesto: la falta de orientación. Tuve la certeza de que entre el estribo del vehículo y la calzada, había un abismo. Manos amables me ayudaron a descender penosamente.

Pero el vértigo subió de punto al querer franquear las tres amplias y muy suaves gradas que conducen al zaguán del edificio. Carente de todo control, mis piernas incoordinadas parecían atacadas de ataxia locomotriz progresiva.

Ya dentro de la mansión, los objetos otrora tan conocidos y familiares, sufrían deformaciones, cambios sustanciales que no por ser pintorescos en su enmarañado pergeño, dejaron de impresionarse muy de veras. Nuevamente en muebles, libros y cuadros surgieron curvaturas, sinuosidades, escorzos e inflexiones de todas categorías. El escritorio, aparecía ondulante, ahuecado y retorcido mientras las sillas volviéronse combas, esféricas, increíblemente redondas. Todo, pues, desajustado y discordante como en alguna estructura esquizofrénica.

Así que, ya en mi casa y por espacio de una semana, la aberración esférica, unida a la carencia y falsa orientación me hicieron vivir, si bien es cierto en molestas zozobras, no por ello, igualmente, en divertidas ficciones ópticas: los pasillos v. g. alargadamente curvilíneos conducían a puertas siempre cóncavas, pandas, abarquilladas que —vistas de lejos— unas veces mostrábanse inmensamente estrechas y otras, enanas, pequeñísimas, a tal punto que para franquearlas, hube menester de agacharme casi hasta el suelo aturbonado sartal de reflejos defensivos.

En ocasiones, los pies, manos y nariz apercibíalos enormes. Y, mi espíritu nunca en verdad se regodeó tanto como en el trance de ciertas circunstancias fisiológicas, cuando veíame en la necesidad de verter al exterior, el ritmo del filtro renal.

En estas coyunturas —extrañamente deformadas por la ilusión óptica— y en donde todo lo corpóreo alcanza guarismos de magnitud y arqueo, acudía a mi

caletre el episodio aquel protagonizado por Gargantúa desde las torres de Nuestra Señora de París, y cuyo desenlace, nos lo narra don Francisco de Rabelais en este saleroso comentario: “Y sonriendo destacó, Gargantúa, su bella braguita, sacó al aire su méntula y los meó tan copiosamente que ahogó a doscientos sesenta mil cuatrocientos diez y ocho, sin contar en esta cifra las mujeres ni los niños.”

* * *

Fatigado, a la postre, con tamañas extravagancias dependientes de la faco-erisis, díme, entonces, a zanjar bajo riguroso entrenamiento y no menos tozudos ejercicios disciplinarios, la adaptación paulatina a las lentes correctoras.

El edificio que habito consta de tres pisos —unidos no por ascensor— sino por escaleras en espiral. En altas horas del silencio nocturno cuando ya los inquilinos de los demás apartamentos dormían o estaban ausentes, iniciaba yo, fuertemente aferrado a la baranda, con mi aberración esférica y mi ataxia locomotriz a cuestras, el ascenso de la contorsionada, de la torcida escalera.

Al principio, aquella prueba cónica fue tremenda. Las dos primeras noches, únicamente subí y bajé hasta el primer piso; las otras dos al segundo, y en seguida, escalaba la cúspide del tercero. Ulteriormente, logré descender como ascender, sin apoyarme en el barandaje, y sin aberración esférica ni tabes dorsal, los tres pisos del edificio.

A los diez y ocho días de operado la inanición así como el confinamiento en la residencia, me sofocaban. Cierta que ahora, volvía a leer con maravillosa diafanidad. Empero, resolví reabrir mi consultorio, siendo intensa la sorpresa al comprobar, objetivamente, en el examen de los enfermos que todos mis movimientos y maniobras clínicas, se habían readaptado.

Fui luego saliendo a la calle y, aunque lo hacía con cautela, no exenta de timidez, en breves días lograba encarrilarme dentro de la multitud, en esa multitud que en tratándose de corridas de toros —y al decir de Blasco Ibáñez— “es la verdadera, la única fiera”.

Quedábame otro paso por dar y... lo dí: manejar el automóvil. Primeramente me hice conducir, en dos ocasiones, hasta la “Clínica de Nuestra Señora de la Paz” con el auxilio de mi discípulo Pepe Conde. Y, una espléndida mañana de las postrimerías del mes de agosto de 1957, tomé entre mis manos el timón —no sin alguna emotividad— pero venciendo, finalmente, la penúltima via-crucis del afáquico.

Porque existía un postrer escalón para liquidar: decirle adiós a los espejuelos correctores. Es hecho sabido que la pérdida de los cristalinos acarrea la estrechez

del campo visual, proveniente del escotoma en anillo como de la exactitud precisa de las lentes afáquicas. De otro lado, la facoerisis, reduce la visión periférica, y los lentes bifocales que intensifican la central, amargan, desconciertan, sobremanera, la personalidad de no pocos operados.

Y, éste fue precisamente el caso mío. No me advine con los quevedos bifocales. Por ello, cuatro meses después de intervenido, tuve la suerte de amoldarme, en corto lapso, a los LENTES DE CONTACTO. A fe que el hallazgo de estas lentes representa para quienes las usamos, fecha indeleble en los anales de optometría.

En resolución: con voluntad tenaz y continua; con la añoranza constante de que sin la facoerisis, se hubiese perdido irremediamente la vista; con un algo de amor e interés por la vida física y espiritual; con fe creciente en el oftalmólogo-cirujano que, a más de destreza operatoria, está adobado por un clínico y un hombre, la reeducación social, profesional y moral del afáquico, se alcanza en casos como el mío— fácilmente. En mi concepto, ello es cuestión del temperamento de cada quién. En la recuperación del afáquico, todo depende del operador y el resto del carácter e idiosincracia del operado.

Apartado Aéreo N° 72-02

OCULAR MIGRAINE AND LATENT HETEROPHORIA

BY

RAYMOND R. ROY, O. D.

Portland, U. S. A.

Migraine headache is no respecter of persons. It is found in every civilized country today, and can be traced through literature until man's earliest beginnings. The condition has been more or less a futile challenge to physicians for centuries.

The cuneiform tablets of the Early Sumerian Period of 6000 years ago contain a good medical description of this "disease of the temples". They referred to it as a pressure on one side of the head and associated with "eye troubles".

The Ebers' Papyrus which was an account of medicine from old Egypt, tells the story of Ra, the elder Great Father who began to suffer from "enu" pain in the head. "Enu" means "traveling" and aptly fits a description given by modern twentieth century patients. The papyrus had several prescriptions which were supposed to give relief of this "pain in the side of the head".

In A. D. 30-90, a renowned Greek physician, Arataeus of Cappadocia isolated from a general group of headaches, a type distinguished by its paroxysmal nature, its severity, its unilateral character, and its association with nausea and vomiting. Fifty years later Galen, the grandfather of medicine, introduced the word *hemicrania* meaning "one-sided". The Romans borrowed the word and translated it into the Latin *hemicranium*. This was later corrupted to the low latin *hemigranea*, then to *emigranea*, *migranae*, *migrana*, the French *migraine* and the English *mygraine*, *megrin*, *migrim* and *megrin*. The French word *migraine* is today universally accepted.

As is so often true of any descriptive term, common usage and acceptance over a period of years many times changes the original meaning. Word meaning goes through what is known as *semantic change*, which means that while retaining earlier meanings, new ones have been added due to specialized uses and differences of various other kinds.

This is by no means a disturbing factor because a new concept may be more descriptive and more inclusive than originally was observed. This is true of the word *migraine* as much more has been observed and is known about the subject today. The original meaning was a unilateral cephalalgia of severe intensity with an accompanying visual aura. Popular usage and concepts, however, have come to include in migraine classification, any severe incapacitating headache of previous undetermined origin, whether bilateral or unilateral.

Proof of this concept is found in the writings of such men as Walter C. Alvarez, professor emeritus at Mayo Clinic, who has said of migraine that it can either be a mild trouble, not worth talking about, or it can be a terrible affliction which prostrates the victim several times a week. He also states that many migraine headaches are not unilateral but are felt all over the head.

Further proof of the modern concept of migraine is found in the case histories of these migraine sufferers. In taking histories of thousands of these medical "orphans", it is common to review numerous past medical diagnoses of migraine, with no due regard to location, time of onset, or associated symptoms. In fact patient reports indicate a very languid attitude on the part of internists and other specialists in reference to the complaint of headache. The current opinion that headaches are largely psychogenic precludes exhaustive and thorough examinations and the patient comes away with the usual prescription for analgesics, tranquilizers, or vasoconstrictors.

MIGRAINE EQUIVALENTS

What was originally known merely as a *migraine headache* has now been classified as *migraine simplex*. Because of the variability of migraine attacks a long list of names have been attached for descriptive purposes. Some of these are, abdominal migraine, ophthalmoplegic migraine, precordial migraine, facio-plegic migraine, and ophthalmic migraine. Some of the common names are: menstrual headache; relaxation headache; Sunday headache; spring headache; summer headache; constipation headache; sick headache, and so on.

All of these descriptive names point out the vast complexity of headache. This is verified in that over two hundred etiological factors have been isolated which produce headache pain. Thus we see that when a patient presents a history of chronic recurrent headache, it is not known if the variations indicate multiple manifestations of a single etiology or if the one person actually has multiple etiology and that "a migraine" might actually be the accumulation of two or more factors.

Having worked with thousands of headache patients in the past twenty-one years I have seen many cases in which the migraine was of multiple etiology

necessitating two or more specialists working together to furnish relief. It is no wonder then, that these people had suffered so long with no relief, as all previous investigations were seeking a single etiological factor. Nevertheless in these cases of multiple causation, it was found that latent heterophoria was the salient factor. Once found, the remaining anomalies were easier to isolate.

OCULAR MIGRAINE

That there may be a connection between migraine and visual anomalies is not a new thought, however the literature must be diligently searched to reveal any mention of such possibility.

Savage (1902) said, "However, genuine sick headache - pure migraine - is sometimes caused by both refractive and muscle errors".

Allen (1900) writing of the symptoms of hyperphoria stated, "Reflex headache is exceedingly common in hyperphoria, and is associated with dizziness, staggering gait, feeling as if one would faint, sharp pains through the head, drawing in the temple, pain in the forehead and also behind the ears".

Duke-Elder (1949) states that, "Not uncommonly gastric disturbances may dominate the clinical picture of eye-strain - chronic indigestion, dyspepsia, nausea and even vomiting. It is this symptom-complex, when it occurs periodically associated with an acute and incapacitating headache, which resembles a true migraine".

The above writers have seen that stress on the ocular muscles can produce severe headaches which in every way fit the description and modern concept of migraine. It is to be noted however that the stress which was found to be the etiology of the migraine was a *manifest* dysfunction — found by a careful analysis of the visual mechanism.

Now, however, *latent* binocular stress or heterophoria may be found by means of a prolonged monocular occlusion test. By utilizing a standardized technique and investigating the possibility of latent heterophoria on migraine patients previously showing no appreciable heterophoria by any testing modality, it can now be shown that a tremendous percentage of migraine patients can be classed as having ocular migraine.

In this work, the history of chronic, severe incapacitating headache along with allied symptoms is used as the basis for utilizing a standardized prolonged monocular occlusion test. In an extremely high percentage, these patients reveal latent heterophoria, which when corrected by means of proper prism lenses, obtain relief heretofore unobtainable.

PROLONGED MONOCULAR OCCLUSION

In 1920, Marlow published a paper entitled, "Prolonged Monocular Occlusion as a test for Muscle Imbalance". This was a monumental work which opened the door into one of the vast unexplored areas of our knowledge of the function of binocular vision. It evoked a storm of protest, however, from so-called authorities who immediately claimed the test unreliable with very little support and a vast amount of protest, the validity of the prolonged occlusion test was questioned by ophthalmologists and optometrists alike. A typical sentence that carries the finality of the last rites is quoted from Krinsky as recently as 1948; "Biesbarth and Abraham and others exposed the unsoundness of the occlusion test and it required a wealth of literature to finally show that prolonged occlusion is of no value in diagnosis". He then refers to Marlow's 1938 writings as being a final admission that his occlusion was in reality producing artefacts that had no relation to the normal physiology of the eye.

Scobee (1952) devotes one paragraph to Marlow's prolonged occlusion and quotes a few of Marlow's critics, such as Cridland, who said, "it has evoked a voluminous outpouring of unscientific hyperbole and almost hysterical vituperation".

Maddox, objecting to it said, "a delict machine is not so informative as a functioning one, although we can learn something from it".

Abraham (1931) studied six cases in which he occluded each eye in turn and each occluded eye developed hyperphoria. He said bluntly, it is a subjective test for demonstrating Bell's phenomenon and is not a test for latent heterophoria."

Scobee's comment in conclusion of the above quotation, is: "It is clear that the position of rest revealed by prolonged occlusion is close to the physiologic position of rest, but can never attain it because the fixation reflex (monocular) comes into play. The hyperphoria thus revealed in nearly every case strongly suggests a persistence of the protected position of the eyes in sleep, the persistence of any abnormal position being well established. It is a method which demands the greatest caution in its interpretation".

In the face of such overwhelming authority as to the impractical value of monocular occlusion as a diagnostic test it would only be the stouthearted or the curious who would attempt any practical therapy based on occlusion. This was indeed a tragic mistake—a costly mistake which has set back headache therapy by at least three decades. A standardized technique of prolonged monocular occlusion, rightly interpreted, is the key to relief for multitudes of chronic headache sufferers.

Coachman (1948) in a brief paper on Prolonged Occlusion was the first to refute these negative arguments. In refuting the fact that prolonged occlusion was a manifestation of Bell's phenomenon he said, "The occlusion of each eye separately does not demonstrate a Bell's phenomenon as some have claimed, for we do not sleep with one eye and the other closed".

In 1955, I published a paper to show statistical evidence that prolonged occlusion has no parallel with Bell's phenomenon. By classifying over 200 clinical records of occlusion cases it was shown that only 49% reacted in the same manner as Abraham's six cases. Thus we see that inconclusive evidence and prejudice were allowed to creep into scientific journals and inhibit further research in the area of latent binocular imbalance.

LATENT HETEROPHORIA

Phoria measurements are designed to indicate the position which one eye will take in relation to the other eye when the controlling influence of binocular fusion is abolished and all residual binocular stress has been eliminated. In the event that structural symmetry is good and the reciprocal and synergistic relationship of the binocular reflexes is such that there is no vertical, horizontal, or rotational differences, the condition is referred to as orthophoria.

There are two factors in the above definition of phoria measurements which are equally important. First, binocular fusion must be abolished; but secondly, *all residual binocular stress must be eliminated*. Here is the great stumbling-block to the accurate determination of heterophorias.

As true orthophoria is an almost incomprehensible state of perfection, we conclude that heterophoria in some degree must be found in every patient examined.

The great problem is to find the direction of deviation and the degree. If heterophoria exists in any degree, then we know there must be a stress set up in the neuromuscular pattern to counteract this deviation if efficient binocularity is to be maintained.

Here is where *time* becomes an integral factor in phoria measurements. If a hyperphoria has existed for many years, perhaps even a congenital hyperphoria such as found in hypertropia, it may take hours, or days, or even months for this stress to be inhibited to a degree to facilitate a true phoria measurement and correction. The type of simple phoria measurement that most of us were taught to make is generally worthless if we are to adequately diagnose a migraine patient.

DIFFERENTIAL DIAGNOSIS

When bodily and ocular stresses have become deeply imbedded through years of compensatory muscle tension, and when electromyographic research has proven

that in the face of increasing fatigue a muscle requires a more prolonged time for relaxation, it any wonder that our feeble superficial efforts to measure phorias meet with such dismal failure?

Once we realize that *time* is a most important catalyst in the creation of binocular stress, it is easy to see that time may become equally important in relieving that stress. If a patient has utilized a compensatory disjunctive stress for twenty years to maintain single binocular vision, it is only reasonable to assume that this must be relieved for a prolonged period if a true finding is desired. The only way in which binocularity can obtain relief is by occluding one eye. After a designated period of monocular occlusion then phoria measurements are again taken and the difference noted.

Here are a few typical examples from the files of successfully completed migraine patients as an illustration of changes in binocular relationship following a standardized prolonged occlusion test:

<i>Patient</i>	<i>Before Occlusion</i>	<i>After Occlusion</i>
A	1½* Left Hyperphoria 3* Exophoria	8* Left Hyperphoria 8* Exophoria
B	¼* Left Hyperphoria 3* Exophoria	10* Left Hyperphoria 10* Exophoria
C	½* Left Hyperphoria 12* Exophoria	5* Hight Hyperphoria 22* Exophoria
D	1* Left Hyperphoria 6* Esophoria	Negative 27* Esophoria

* Prisms Dioptrics.

In each of the above cases there had been a long history of severe headache which was eliminated after the proper amount of prism was prescribed to relieve the binocular stress. Hence it is easy to see why the etiological factor of these migraine headaches was not found in all of the previous investigations and why the label "psychosomatic" was placed there instead.

CONCLUSION

It has been the purpose of this article to try to show how latent heterophoria can produce neuromuscular stress in the binocular act wich may result in headaches of the severest intensity. It is impossible in this short space to cover the standardized prolonged occlusion technique and the associated test used in differential diagnosis, but it is desired to point out that no patient suffering from chronic headache has had an adequate investigation for etiological factors until a thorough investigation of possible latent heterophoria has been made.

318 S. W. Alder St.

BIBLIOGRAPHY

- ABRAHAM, S. V., Bell's Phenomenon and the Fallacy of the Occlusion Test, *Am. Jnl. of Ophth.* 14:65, 1931.
- ALVEREZ, WALTER C., "How to Live with a Migraine Headache", Newspaper Article.
- COACHMAN, E. H., A Common Extraocular Muscle Imbalance, *The Eye, Ear, Nose and Throat Monthly*, 27: 175-179, 1948.
- DUKE-ELDER, *Textbook of Ophthalmology*, C. V. Mosby Co., 1949.
- MARLOW, F. W., Prolonged Monocular Occlusion as a Test for Muscle Imbalance, *Trans. Am. Oph. Soc.*, 1920.
- ROY, RAYMOND R., Ocular Migraine and Prolonged Occlusion, *Opt. Weekly* Sept. 3-10-17, 1953.
- ROY, RAYMOND R., Prolonged Monocular Occlusion and Bell's Phenomenon, *Jnl. of A. O. A.* June 1955, 26: 626-630.
- ROY, RAYMOND R., Headaches and Binocular Stress, *Opt. Weekly* Oct. 11-18, 1956.
- ROY, RAYMOND R., Symptomatology of Binocular Stress, *Opt. Weekly* May 15, 1958.
- SAVAGE, G. C., *Ophthalmic Myology*, McQuiddy Printing Co., 1902.
- SCOBEE, RICHARD G., *The Oculorotary Muscles*, C. V. Mosby Co., 1952.
- WALKER, KENNETH, *The Story of Medicine*, Oxford University Press, 1955.

TELESCOPIC AND MICROSCOPIC SPECTACLES

BY

J. I. RODGIN, O. D.

Bluefield, U. S. A.

The correction or improvement of subnormal vision by means of special optical devices is now receiving more attention than ever before. Persons from seven to ninety years of age who are almost blind, are being rehabilitated with these special visual aids, enabling them to live normal and useful lives. Many devices such as contact lenses, telescopic spectacles, microscopic spectacles, pinhole spectacles, hand magnifiers, and projection devices are being used in cases of subnormal vision assistance. This paper, however, will be confined to the consideration of two of these optical aids — telescopic and microscopic spectacles.

Small Galilean telescopes for the correction of high myopia were first used in the seventeenth century. However, no practical and satisfactory results were realized until about 1910, when Von Rohr designed for Zeiss in Jena the modern compact form of telescopic spectacles. The use of these telescopic spectacles became wide spread after World War I, for soldiers whose eye injuries during the war had resulted in subnormal vision. This was especially true in Germany where many men were able to continue their work by use of these telescopic spectacles.

The most practical telescopic system used is a miniature Galilean telescope mounted in a sturdy frame and worn like ordinary spectacles. It consists of a positive objective and negative eyepiece separated by the difference in their focal lengths, which produces a relatively flat field without much astigmatism. The erect magnified image is equal to $-f_1/f_2$ when f_1 is the focal length of the objective and f_2 is the focal length of the eyepiece. The angular size of the image is increased without affecting vergence of the incident light so that the system has no refracting power. The purpose of the telescopic system is to enlarge the retinal image, thereby increasing the number of nerve receptors used. The optical aberrations can be reduced so that they do not produce too much interference. The spherical and chromatic aberrations are inconsequential. Coma, oblique astigmatism, curvature, and distortion can be minimized by the proper curvatures and dispersion of the lenses used.

A case of subnormal vision is indicated when the visual efficiency of the patient with ordinary lenses is insufficient for his needs. However, it must be remembered that intelligence and environment also play an important part in determining visual efficiency. Subnormal vision is a relative term and must be applied to each individual case for proper analysis with regard to prognosis before any attempt is made to prescribe telescopic spectacles.

Anomalies and diseases responsible for a condition of subnormal vision are numerous, including irregular astigmatism, keratitis, corneal scars, glaucoma, retinitis pigmentosa, detachment of the retina, central chorio-retinitis, primary and secondary optic nerve atrophy, posterior uveitis. Various physical anomalies, such as coloboma of the iris, lens and choroid, congenital cataracts, and dislocation of the lense are also factors in reducing vision. It is essential that the basic cause of the subnormal vision be known — that is, whether the reduction is due to an irregularity of the refracting surface or to the media not being homogenous (isotropic and transparent) or to a reduction of the nerve receptors (rods and cones). The use of the peripheral retina is essential with telescopic spectacles so that magnification can be meaningful. The differentiation of those pathologies causing a loss of central vision from those resulting in a loss of peripheral vision, is therefore an important factor.

The psychological factor involved in subnormal vision is one which deserves special consideration. Pathological changes causing optical abnormalities usually come to a standstill after a certain amount of damage has been done. However, a further loss of visual perception is imposed upon this immediate loss of visual acuity. A psychological factor is then superimposed, causing the patient to see more poorly as time goes on.

The question of what optical device should be used for the correction of subnormal vision is one which should be carefully analyzed by the examiner. Telescopic spectacles are used primarily in cases of retinal involvements, although they are sometimes used in cases of media involvement, and in those cases where there is both a media and retinal involvement. Before prescribing telescopic spectacles for near, it is advisable to first try aspheric microscopic lenses to see whether this will take care of the patient's needs. Often a high plus "add" may be more advantageous. It is very difficult to walk with telescopic spectacles because of the apparent displacement and the reduced field of vision. Telescopic spectacles are ideal for viewing TV or any form of entertainment where the patient is seated. They can also be used to great advantage for near visual tasks where magnification is so important.

The magnification of telescopic spectacles for distance usually varies from 1.8X to 2.2X. For example, the telescope of 1.8X magnifies the retinal image 1.8.

This means that a patient with 20/200 vision or 10% visual acuity with regular spectacles, would have 18% visual acuity for distance with 1.8X telescopic spectacles. The field of vision obtained with telescopic spectacles for far and near decreases with the magnification. This is an important fact to remember in the selection of the proper telescopic spectacle.

Microscopic spectacles used for near are theoretically simple microscopes, designed in such a way that markedly diverging light from the printed page is made to leave the microscope as parallel light. Since the patients who require this type of lense for reading usually have eccentric fixation or nystagmoid movements, it was imperative to design the system with a large aperture. These microscopic spectacles magnify the retinal image an amount equal to the magnifying power of the microscope. The microscopes are designed with magnification from four to sixteen times. The triple lense microscopic spectacle both optically and cosmetically has proven the most satisfactory type.

The actual working distance with microscopic spectacles is usually only from five to seven centimeters from the eye. The greater the magnification, the shorter this working distance becomes. The useful field, however, remains relatively large even with the highest magnification. The microscopic spectacle was originally designed to care for those patients who had chiefly rod vision.

The fact must be recognized that in using a telescopic spectacle, the external illumination must be varied according to the amount of light that passes through the device and the type of nerve receptors left in the retina. If the patient has the maximum of cone vision present, then the external illumination must be high. When, however, the remaining is rod vision, the intensity of the external illumination must be low.

Since the microscopic spectacle is generally used in cases where rod vision predominates, and since rod vision is most sensitive under low illumination, it is important to keep the illumination on the printed page as low as possible. Usually, the intensity should be reduced to one to three foot candles.

The fitting of telescopic spectacles is more than a mere optical problem, because interwoven with this problem are outstanding psychological factors. This situation must be understood even before one undertakes to examine such a patient. There is usually a definite relation between the duration of the sub-normal vision and the extent of the improvement desired. For example, persons who have had poor vision for a short time are usually not interested in having their visual acuity just improved, but are interested in having their vision improved to the normal visual acuity they had before the pathological condition set in. The examiner must do his best to overcome this attitude, since the patient

with such an attitude will never consent to undergo the necessary training that is absolutely required before he can properly use telescopic spectacles. Persons who have "lost" their vision for a period of one, two, or five years, have the same attitude, psychologically at least, towards an improvement in vision as those whose loss of vision was only recent.

In contrast to those patients who have had their condition for only a short time, are those who have had subnormal vision for five, ten and twenty years. As the years have passed, they have become resigned to the condition of their eyes and have stopped trying to see, or to develop a new interest in vision. The "desire to see" is of paramount importance, and telescopic spectacles are psychological as well as optical aids for subnormal vision. The doctor should adopt the attitude that the patient may be governed in his reactions by conditions that have nothing to do with the optical problem of visual acuity. He should always realize that patients with subnormal vision are subject to changes in attitudes towards themselves and the rest of society. The extent of these changes insofar as the visually handicapped are concerned, depend for the most part on the nature of the affliction, the time of onset, and the age of the patient.

The examination of the subnormal vision cases consist of the following salient parts: (1) case history; (2) objective examination (retinoscope, ophthalmoscope, ophthalmometer, slit lamp); (3) field test; (4) subjective examination at distance with simple lenses; (5) subjective examination at distance with telescopic spectacles; (6) subjective reexamination at near with telescopic or microscopic spectacles.

The psychological factors influencing a patient's attitude should be always kept in mind during the examination. The case history should include age, duration of subnormal vision, facts as to whether the condition has changed in the last five years, two years, or six months, diagnosis and prognosis of other practitioners, information as to whether patient sees better in the daytime or at night, and which eye he sees best with. It is also important to find out how the patient sees in the theater, how he reads the newspaper, whether he can recognize colors, walk around unassisted, what he can do (play cards, see food, etc.), and what he desires most to be able to do. The case history, however, should take as little time as possible.

The preliminary subjective examination should be started with the static findings (if at all satisfactory). The chart is located at ten feet instead of the usual twenty foot distance and the test are made monocularly. Vision and not comfort is the important thought in the subjective test. The minus should be crowded if better vision is obtained, and high cylinders used even if not indicated by the retinoscope and ophthalmometer. A simple trial frame with trial lenses is

used. The subjective routine should then be continued without indicating to the patient in any way that anything different is being done. The telescopic unit, usually 2X, is then inserted in the front cell of the trial frame and the improvement in visual acuity is noted in each eye (the test being made monocularly). Special attention should be given to the illumination of the test card. A lamp with a one hundred Watt bulb and photo-flood bulb is used, and the distance of the lamp from the chart is varied to determine whether the patient requires more or less light for best visual acuity.

While the 2X telescope is still in the trial frame and the distance visual acuity has been noted, the patient is tested at near. The problem of training the patient to read should be carefully studied by the examiner so as to fully appreciate the care that must be exercised during the first examination at near. The examiner then arbitrarily adds a plus six or eight reading addition that fits on the objective of the telescope as a sort of cap. The reading attachment depends on the distance at which the object viewed is held, this distance being equal to the focal length of the reading attachment. Thus, with a plus eight reading attachment (object at 12.5 cms.) the total magnification would be 4X. A special subnormal vision reading card is then placed in the patient's hand. He is then instructed to read aloud very, very slowly, the examiner guiding the card throughout the entire part of the examination. The reading attachment is then changed until least magnification is obtained that enables the patient to do what he most desires (reading, writing, etc.) with due consideration being given to age, the resulting maximum visual acuity at distance and near, and the patient's field of vision. It may be necessary in certain cases to change the telescopic rather than the reading attachment. If the reading lenses are plus fourteen or more, it is advisable to test with microscopic spectacles, when the reading lense is more or less incorporated in the objective of the telescope. The microscope, however, requires considerably less coordination to maintain the field of view than the telescope with high reading add.

The patient is then instructed to return for a second eye examination. This is necessary since there is always a possibility of having overlooked an important point in the previous eye examination. The nervous state of the patient frequently interferes with proper field determinations during the first examination. The patient's attitude perhaps may have given the examiner misleading ideas regarding the use of telescopic spectacles in that particular case. The second examination visit should start with a repetition of the distance and near subjective tests with telescopic spectacles. An effort should be made to refine the distant subjective findings. The rest of the visit should be spent in trying the telescope at practical tasks. In reading books and newspaper print, the lowest possible reading addition should be selected to allow as big a depth of focus as possible,

and as large a field as possible. The patient must be taught to read by moving the printed matter rather than his eyes. If the patient desires to write, he must be taught to do so on paper ruled one inch apart and held at a good working distance. Subnormal vision patients are frequently interested in seeing motion pictures. The examiner should accompany the patient to the theater to observe his responses with different telescopic spectacles and at various distances from the screen.

The only way to be certain that the patient can actually use the telescopic spectacles for walking around is to try it out of doors. The patient should not be instructed in any way as to the particular characteristics of the telescopic spectacles. That is, nothing should be said to the patient as to the field, magnification, and the judgment of distance. The patient should merely be made to understand that this is a lense that is being tried to observe its effect on him. The patient's ability to judge distances, his ability in crossing streets, etc., should be noted. At the end of the second visit, the doctor should acquaint the patient with the practical possibilities of these lenses. The patient should also be advised that he will be required to undergo active adaptation with telescopic spectacles for a period ranging from two weeks to a month. This training period is necessary in order that the patient learn to properly coordinate head, eye, and arm movements. Lack of such coordination will result in the frequent loss of the field and in the inability to maintain sharp focus. The essential element in learning this new coordination is practice.

The psychological complex most usually observed is fear of blindness, because of the horrors associated with being blind, and the fact that most people believe that partial loss of vision will inevitably lead to total loss. This fear of blindness will reveal itself particularly during the first and second examinations, and will persist during the training period, unless the examiner is prepared to eradicate it by assuring the patient, that, all things being equal, his condition will not lead to blindness.

A second marked change in subnormal vision patients that is often noted, is an attitude of dependency that they develop. To help such a patient gain confidence in himself, the doctor must assume the role of an analyst. He must study problems of personality, particularly as they relate to the institutionalized and handicapped.

In almost all these cases, the reading is limited to one eye. The binocular field for near is extremely small and the patient would have to approximately double his convergence. Thus, if the patient normally reads at 25 centimeters then he converges 24 prism diopters (60 mm. P. D.), but with telescopic spectacles, the convergence is 48 prism diopters. This is equivalent to reading at twenty-

five-centimeters with 12 prism diopters over each eye. Reading binocularly with telescopic spectacles can not be done. Usually more than one focus reading addition is required. For example, a plus 6.00 might be used for writing purposes and a plus 10.00 for reading. Telescopic spectacles should be worn as close to the eyes as possible, so that the exit pupil and eyeflense are approximated in order to provide the largest field. The telescopic spectacles should never be tilted because of the increase in aberrations.

Perhaps it would be well to emphasize again the two adverse effects produced by the increased magnification of the telescopes. A restricted field with the distance glasses may affect the safety of the patient while he is out of doors. This important fact should be carefully considered by the examiner before prescribing telescopic spectacles for constant distance wear. Another bothersome effect is the apparent motion noticed by the patient. When a patient turns to look at an object with 2X telescopic spectacles, the object seems to move as he "arrives" at the object in one half the time ordinarily expected. The patient will thus overshoot his mark and must "return" in order to see the object desired. When it was necessary to turn the eyes through 60 degrees without telescopes, he must now turn through 30 degrees with telescopes. This muscle sense conflict must be overcome during the training period with these telescopic spectacles.

Investigations with regard to subnormal vision correction show clearly that such problems lie in the field of rehabilitation. The problems presented with the fitting of telescopic spectacles are twofold: the development of the apparatus to compensate for the loss of function and the emotional readjustment. A consideration of these two problems is essential in the fitting of telescopic spectacles.

401 Bland St.

A NEW OPERATION FOR THE SURGICAL TREATMENT OF SEVERE CONTRACTION OF THE SOCKET

BY

B. W. RYCROFT, M. D.

Sussex, England

In adults the surgical treatment of a contracted socket is necessary either to improve the appearance of the patient, to make it possible for him to wear an artificial eye in comfort, or to relieve his discomfort by the control of constant and chronic discharge. In children surgery is undertaken to enlarge the immature socket so that it will eventually retain a suitable artificial eye in adult life.

In an industrial country such as the United Kingdom the treatment of contracted sockets is a frequent necessity and there are also many examples which are the result of enemy action in two World Wars both amongst civilians and ex Servicemen.

The most successful results are not entirely due to the work of the surgeon, since to the surgery must be added the skill of a first class technician who is able to make suitable modification of standard artificial eyes and contact prostheses.

Principles of Treatment.

In order that a standard artificial eye can easily be worn without extrusion and irritation several conditions of the socket must be satisfied. The lower fornix must be of adequate depth and must not be too shallow; the depth must be equal along the whole length of the fornix and the fornix should not be crossed by conjunctival adhesions. It is more important that the depth of the lower fornix shall be firmly established rather than that of the upper fornix. Nor is it necessary to have a deep socket in order to retain an artificial eye; indeed sometimes this is a disadvantage and in the past too much attention has been given to the depth of the socket.

One feature, however, is essential and that is to remember that the best cosmetic results with movement are obtained when the artificial eye is held firmly between the pressure of the lids in front against the base on which it rests, whether the base is a movable stump, orbital tissue, or a buried implant. Too much stress cannot be placed on this factor and the result is well seen where a thin contact



Fig. 1-2-3-4. F. act 52. Old leucoma adherens with secondary glaucoma and anterior staphyloma. The cornea, anterior uveal tissue and lens were removed; the sclera and conjunctiva were sutured in separate layers and a good stump was obtained (Fig. 1). A contact artificial eye was worn with complete comfort (Fig. 2) and good movement was possible with a satisfactory cosmetic result (Figs. 3 and 4).

artificial eye is simply placed over a movable globe and held firmly against it by lid pressure. In such a case the movements of the artificial eye are free in all directions and far surpass anything which may be obtained with orbital implants.

It is often a wise procedure, in suitable cases, not to enucleate the whole globe but just to excise the cornea, anterior uveal tissue, and lens. The sclera is then firmly closed by sutures and covered by conjunctiva thus constructing a mobile stump. Against this stump a contact shell can be placed with a satisfactory cosmetic result.

We have reached the conclusion that it is helpful to excise the lacrimal gland completely in all cases of enucleation. This prevents the excessive moisture which plagues most sockets of any duration and thereby reduces the possibility of chronic infection. The mucous secretion of the conjunctiva alone is quite sufficient for adequate lubrication of the lining of a socket and artificial eye without the excessive inflow from the lacrimal gland.

Principles of Surgery

There are three phases in reconstruction of a socket, namely, 1, lower fornix reconstruction, 2, upper fornix reconstruction, 3, general reconstruction.

In the reconstruction of a socket which has moderate contraction of one or both fornices it is essential to remove all subconjunctival fibrous tissue which is responsible for the contraction, and this may often involve a deep dissection of the orbit: unless this fibrous tissue is removed completely there will inevitably result contraction of subsequent grafts later. When the fibrous tissue has been entirely removed a raw area is left which it is necessary to graft. An accurate assessment of the amount of tissue required may be obtained by the use of oiled silk which is pressed against the raw area: the blood stain on the oiled silk accurately delineates the raw area and enables accurate grafts to be fashioned from mucous membrane with due allowance for subsequent shrinkage.

The free grafts suitable for socket reconstruction are generally taken from the inside of one or both cheeks and should be as thin as possible: if these mucous membrane grafts are thick they will inevitably ooze later and lead to excess of moisture. Sometimes it is possible to obtain a small piece of conjunctiva from the upper fornix of one or other eye.

Skin is not a suitable tissue for lining a socket and it should never be used in conjunction with mucous membrane as the moisture from the mucous membrane invariably causes excessive desquamation of the skin with the production of an offensive odour. Skin is used when there has been an exenteration of the orbit and where it is not possible to obtain sufficient mucous membrane to line it: in such a case the skin must be kept completely dry or again an offensive odour may result.

The surgical principles for the reconstruction of conjunctival fornices are well known: it is essential that the free graft of mucosa be held firmly against the raw area in the fornix and maintained in position either by a head cap splint or by a gutta percha mould. Our preference is for a gutta percha mould combined with a complete tarsorrhaphy; the mould is retained for at least four months. The tarsorrhaphy is constructed by splitting the whole length of both lids along the grey line: this produces an anterior and posterior flap in the lid tissue. By an intracuticular suture the two anterior flaps are joined together and slightly everted: no tissue is excised and there is no distortion of the lash line. When the tarsorrhaphy has served its purpose it can easily be divided and the edge of the lid quickly resumes a normal appearance. It must be emphasized that the tarsorrhaphy should run from the outer canthus right up to the lacrimal canaliculi if the mould is to be retained in position: if the tarsorrhaphy is merely in the middle third of the lids the mould will surely extrude itself from one or other side of the adhesion.

Sometimes it is possible to obtain a mobile stump in a deep socket where the eye has been removed many years before: this is by the use of a delayed implant. The shrunken Tenon's space is opened up and the muscles are defined: into the area thus exposed an acrylic ball of 14-18 mms. is placed and subsequently buried by bringing together the muscle tissue over it. The conjunctiva covers this stump as a separate layer. There is generally considerable improvement to the base of the orbit and a useful stump is obtained. Occasionally a dermolipomatous graft from the abdomen can be used to fill up the depths of a socket, but more commonly this is used to deal with an exaggerated supra-tarsal sulcus in an upper lid after enucleation.

Total Excision of the Socket

To reconstruct a socket entirely by the use of free grafts at several operations is a time consuming procedure which necessitates much hospitalisation of the patient, and often the eventual cosmetic result is a disappointment. In an industrial country time spent in hospital is important to the economy of the patient and to industry: our experience of total reconstruction by free grafts has persuaded us to think of another method which would be shorter in length of hospital stay, and give a better cosmetic result.

Such a method has been devised by the total obliteration of the socket and the use of a contact prosthetic appliance which is stuck on the skin or carried on a spectacle frame.

Technique of the Operation

This operation is done where there is complete contracture of the socket in all directions and where there is practically no fornix, either above or below, with lateral contraction of the canthi.

CONTRACTION OF THE SOCKET



Fig. 5-6-7. F. aet 48. This patient had marked contractum of the left socket owing to pemphigus: She was quite unable to wear an artificial eye (Fig. 5). The socket was completely excised (Fig. 6). A contact prosthesis was applied (Fig. 7).

Anaesthesia is by the hypotensive method and at present the drug "Arfonad" is the method of choice: by hypotensive anaesthesia bleeding is reduced to an absolute minimum and the differentiation of tissue planes is made easy. Where this type of anaesthesia is contra-indicated the operation table is given a tilt of 45° to the horizontal which also diminishes haemorrhage.

The first step in the operation is to extend the outer canthus by an incision as far as the fronto-zygomatic suture. The lids are then held widely apart on traction hooks and the whole mucous membrane of the socket is exposed. The mucous membrane is then carefully dissected in one piece from the posterior surfaces of both lids and from the depths of the socket. No deep tissue is removed as this will be useful to constitute a soft pad after operation. The lacrimal gland is completely removed from the lacrimal fossa and all bleeding points are carefully stopped, though with hypotensive anaesthesia there is usually very little haemorrhage. The lash lines are then excised together with the lacrimal puncta and



Fig. 8-9. M. aet 14 had bilateral retinoblastoma. After radiation there was marked contracture of both sockets (Fig. 8). Both sockets were excised (Fig. 9) with much increases in comfort, and contact prostheses were mounted on spectacle frames.

CONTRACTION OF THE SOCKET

canaliculi. Deep structures are drawn together with catgut and the raw edges of both lids are carefully sutured by interrupted sutures: all blood is expressed before the final suture is tied. A firm pressure bandage is then applied for four days.

At the end of fourteen days the incision has firmly healed and a smooth skin surface is the result.

It is essential to excise the lacrimal gland in every case or a fistula will probably result.

After the skin has firmly healed the patient is transferred to the care of Mr. Warren of Messrs. Clement Clarke Ltd., Wigmore Street, London, who then provides the prosthesis.

If the patient is in the habit of wearing spectacles the prosthesis is clipped on to the affected side. If, however, the patient does not wear spectacles then the prosthesis is made to adhere to the skin by the use of Mastisol tissue glue.

The advantages of this operation are, 1, that the patient stays in hospital for about ten days and that he is generally up and about on the third day: 2, that the cosmetic result is excellent and far better than could be obtained by several operations on a socket which shows gross contraction: 3, the prosthesis is clean and requires no after care: 4, the risk of repeated anaesthetics is avoided.

In our experience patients who have undergone free grafts for the reconstruction of sockets and then subsequently had excision of the socket much prefer the latter procedure and are well satisfied with it. The illustrations show the cosmetic effect.

I am indebted to Mr. Gordon Clementson, Director of Photography, Queen Victoria Hospital, East Grinstead, for the illustrations.

Queen Victoria Hospital

LENS EXTRACTION IN MARFAN'S SYNDROME

BY

A. BENEDICT RIZZUTI, M. D.

Brooklyn, U. S. A.

In 1896 when Marfan (1) first described the syndrome that bears his name, no mention was made of the presence of any ocular manifestations. The syndrome is now considered to be a hereditary, bizarre clinical entity which can involve the skeletal, ocular, and cardiovascular systems in varying degrees.

Its true etiology is still speculative. Characteristically the affected individual is unusually tall, slender, with long arms and tapering elongated fingers and toes. Achard (2) in 1902 chose the term arachnodactyly to describe the spider-like aspects of the digits. Kyphosis, scoliosis and "pigeon breast" are frequent findings. The cardiac defects may be represented by valvular lesions, congenital heart disease and aortic aneurysms.

In 1924, Ormond and Williams (3) were the first to stress the importance of the ocular lesions, the most common of which are ectopia lentis, iridodonesis and miosis. These findings were present in approximately 50 per cent of reported cases Marfan's syndrome. Ectopia lentis is found in about 75 per cent of cases presenting this disease entity. The subluxated lenses may appear normal but usually show spherophakia, microphakia and varying degrees of lenticular opacities. The lenses are usually displaced superiorly in a symmetrical fashion. The syndrome as rule is not usually detected until about the third or fourth year of life, when the demand for greater visual acuity increases. The syndrome occurs with equal frequency in both sexes.

In 1942, Rados (4) in a comprehensive report, compiled statistics of this syndrome in 204 cases published by a variety of authors. To date, reports of more than 300 cases of Marfan's syndrome have been recorded.

Many experienced surgeons are hesitant in attempting lens removal to improve visual acuity in this syndrome because of the high percentage of surgical difficulties and late complications that can occur. The elusiveness of grasping the abnormal pellet like lens, the great frequency of vitreous loss, and the ever

present danger of retinal detachment are now well recognized. Zeeman (5) pointed out that approximately 10 per cent of cases ended in spontaneous detachment of the retina. Surgical intervention was not a prerequisite in producing this complication.

In spite of these hazards, however, an occasional gratifying result may be obtained. For this reason the following case history is reported. Lens extraction was performed in the two eyes of the same individual who presented a typical clinical picture of Marfan's syndrome. The post-operative course was satisfactory and a visual acuity of better than 20/20 was achieved in each eye.

CASE REPORT

H. M., a white 30 year old male, was first seen on December 31st, 1954. The family history revealed that his father and one brother were also afflicted with Marfan's syndrome. Physical examination of the patient demonstrated the classical triad. Scoliosis and "pigeon breast" were very evident; the heart findings presented extensive valvular lesions. The ocular findings consisted of bilateral ectopia lentis, associated with a slight degree of iridodonesis.

After dilatation with 10 per cent neo-syneprine, both lenses appeared grossly clear and displaced inferiotemporally. The presence of free vitreous lying anterior to each lens was revealed by slit lamp examination. The corrected vision in the right eye was 20/80 with the use of -12.50 diopter sphere -1.75 diopter cylinder, axis 180. The corrected vision in the left eye was 20/100 with the use of -13.25 diopter sphere -2.00 diopter cylinder, axis 30. The patient was promoted to master electrician and demanded greater visual acuity hence operative interference was seriously weighed.

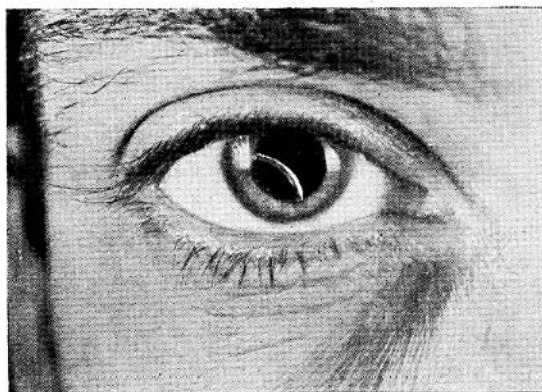
Lens extraction of the left eye was performed on January 3rd, 1955. The corneal section was made with a keratome and prior to performing an iridectomy



Fig. 1. Marfan's syndrome; note elongated, tapering fingers and chest deformity.

MARFAN'S SYNDROME

Fig. 2. Marfan's syndrome; clear lens subluxated inferior temporally, right eye.



superiorly a small amount of fluid vitreous escaped. The superior pole of the subluxated lens was grasped with a Verhoeff forceps and extracted intracapsularly with comparative ease and no further loss of vitreous. Air was introduced in the anterior chamber with guided pressure after firm closure of the corneo-scleral wound was obtained with the use of 5 corneo-scleral sutures.

The patient made an uneventful recovery and was given his final aphakic refraction on March 26th, 1955. Visual acuity of 20/16-3 was obtained with the use of +11.00 diopter sphere +1.00 diopter cylinder, axis 60.

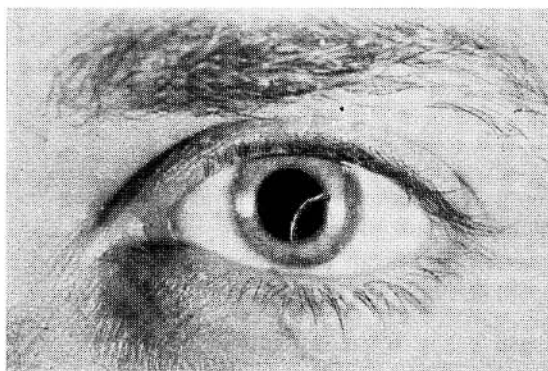


Fig. 3. Marfan's syndrome; clear lens subluxated inferior temporally, left eye.

Lens removal of the right eye was subsequently conducted on January 6th, 1956. After performing a wide iridectomy above, a loss of a small bead of semi-fluid vitreous resulted. The rather clear lens floated somewhat into the wound area and was easily extracted with a lens loop. The few adherent remaining zonular fibers did not offer any unusual resiliency. A slight degree of hyphemia was the only other surgical complication. The post-operative course, however,

was satisfactory. After the final refraction was performed on March 22nd, 1956 the visual acuity had improved to 20/16-1 with the use of a + 10.00 diopter sphere +1.75 diopter cylinder, axis 65°.

The patient was subsequently examined at regular intervals and at no time did he present any late surgical complications or diminution of vision. On October 5th, 1958 he expired while in the line of employment as a result of a heart attack.

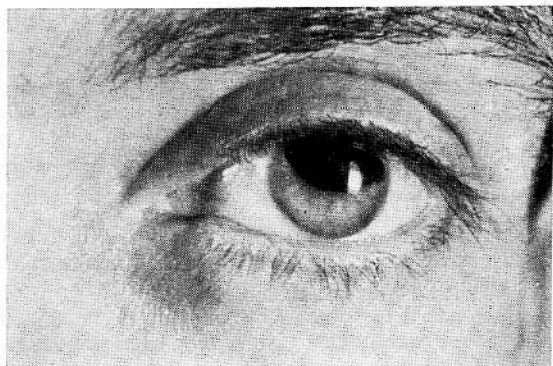


Fig. 4. Marfan's syndrome successful intracapsular lens extraction.

NON-SURGICAL CONSIDERATIONS

Surgical intervention as a rule should be postponed in arachnodactyly with ectopia lentis, if by means of refraction one can improve the visual acuity, either through the aphakic or phakic region of the pupillary zone to 20/70 or better. The refractive error through the phakic area at times may require a minus lens of 15 to 60 diopters. One should be aware of the possible presence of a congenital type of amblyopia with or without manifestations of strabismus. Myopia with astigmatism may be unusually pronounced and of lenticular origin. The subluxated lens across the pupillary zone can produce an annoying type of monocular diplopia and of necessity must be eliminated.

In cases where surgery is not feasible and when refraction does not improve the vision sufficiently, a weak mydriatic instilled daily may offer further improvement. Frequent tonometric and tonographic studies help rule out the existence of an underlying glaucoma.

SURGICAL INDICATIONS

1. Marked impairment of visual acuity.
2. Progressive lens changes.

3. Disturbing monocular diplopia.
4. Dislocation of the lens in the anterior chamber which may eventually lead to severe acute congestive glaucoma.
5. Dislocation of the lens in the vitreous resulting in frequent attacks of cyclitis and secondary glaucoma.

Biomicroscopic study should be carefully conducted of the iris, lens, vitreous and zonular area prior to contemplating any surgical procedure. This is best accomplished after dilatation with a mydriatic. The lens structure, extent and type of subluxation should be given careful consideration. The quality and position of any free lying vitreous, anteriorly situated to the displaced lens may lead one to alter the surgical procedure.

At times one is able to inspect the abnormal zonular area. It is a well recognized fact that although the zonule may be degenerated, the remaining adherent fibers can at times portray an unusual degree of resiliency elasticity. It is possible for a lens that eludes a spoon or where traction is lost with either the forceps or suction cup, to snap back with startling force and lose itself in the vitreous chamber. These factors and many others should be carefully weighed prior to considering any surgical intervention.

SURGICAL PROCEDURES

- I. Iris Surgery (optical improvement)
 - A. Sphincterotomy
 - B. Iridotomy
 - C. Iridectomy
- II. Lens Surgery
 - A. Discission (young individuals).
 - (a) use of one needle knife.
 - (b) use of two needle knives.
 - B. Intracapsular Extraction
 - (a) use of forceps.
 - (b) use of loop or spoon.
 - (c) use of suction.
 - (d) use of cautery.
 - C. Extracapsular Extraction.
 - D. Especial Cases.
 - (a) dislocation of lens in anterior chamber.
 - (b) dislocation of lens in vitreous.

IRIS SURGERY

No one particular surgical procedure should be universally adopted in surgery of a subluxated lens. Sphincterotomy or iridotomy are best performed in the lower nasal quadrant of the eye for optical improvement. A 3 or 4 mm. radial incision is usually found to be adequate in cases where the lens is displaced upward.

In cases of inferior subluxations a wide iridectomy superiorly at times produces a rather astonishing satisfactory optical result. These procedures should be attempted after proper visual evaluation following dilatation of the iris. It is often found, however, that these surgical procedures may prove to be disappointing for they are not easy to perform technically due to improper lens support and furthermore that they do not attack the main source of the difficulty.

LENS SURGERY

Young individuals respond better than adults to surgical intervention. Before the age of 25 years discission of the lens with the use of 1 or 2 needle knives can offer satisfactory visual results. The one knife technique is not recommended because of difficulty encountered in penetrating the usually small globular dislocated lens. This surgical procedure is difficult to perform in that the unsupported lens eludes the point of the knife.

Unless a wide rent is made in the lens cortex, the absorption of lens fibers are unduly delayed. Reese was of the belief that this phenomenon was an inherent characteristic peculiar to congenital subluxated lenses. Discission of the lens with the use of two needle knives as first described by Bowman in 1852 is still the operation of choice. It was popularized by Knapp⁶ and recently modified by Chandler⁷, Kravitz⁸, et al.

One needle knife is introduced through the sclera just posterior to the limbus at a point opposite the free border of the lens. The lens is pierced and held firmly in place. Another similar knife is introduced in the same manner a sector's distance away to puncture the lens capsule and cortex. The handles of the two knives are slowly approximated, the opening in the lens is enlarged and each knife is then cautiously removed in turn. Proper opacification of the lens fibers result but occasionally this procedure has to be repeated one or more times.

In the older age group intracapsular extraction with a wide iridectomy is the operation of choice. Free vitreous is frequently found to be present anterior to the lens and there may be some spill after making the corneal section. The proper iridectomy avoids the drawn up or hammock shaped pupil that usually follows.

It is possible to grasp the free border of the lens with a forceps to execute the proper extraction.

Gentleness and minimal amount of surgical manipulation helps prevent excessive loss of vitreous and usually leads to fewer post-operative complications. The lens spoon or loop when carefully used has its greatest value when the lens is seen to float rather freely towards the wound area. One must be careful of the zonular fibers which may show a live, elastic band response in cases where the lens slips from the spoon or the forceps. The lens may snap back with startling force into the vitreous chamber.

The hand or motor driven erisophake are often found to be inadequate in extracting the lens because of poor adaptation of the suction cup to the anterior lens surface in the presence of free vitreous. Barraquer⁹ suggested that in cases where the vitreous can be swept away with a Marten's hair brush that proper contact can then be made and the lens can thus be extracted by suction. The use of electrodiathermy for extracting the lens as advocated by Lacarrere¹⁰ has not been very popular. The Lacarrere electrodiaphake is used to pinion the lens. The instrument or electrome is released by means of a spring and enters the lens capsule and cortex to a depth of 3 mm. When the current is turned on, there results a coagulation of the lens protein which fixes the lens firmly and permits it to be extracted intact.

Dislocation of a subluxated lens in the anterior chamber may lead to acute secondary glaucoma and requires immediate extraction. In cases where recurrent dislocation of the lens in the anterior chamber occurs and then the lens slips back in position due to a relative intact zonule, Chandler⁷ performs a prophylactic peripheral iridotomy or iridectomy. By this means, pupillary block and secondary rise in intra-ocular pressure are avoided, which he believed was the cause for the lens to be dislocated in the anterior chamber.

When secondary glaucoma and recurrent attacks of cyclitis result, lens extraction is mandatory. In cases where the eye is quiet, it is wiser to avoid surgical intervention and keep the patient under careful observation. An aphakic correction at times can afford the patient useful vision for a considerable time. The use of a Hague Lamp which employs ultraviolet light is of much benefit in locating the position of the lens. The Verhoeff "floating technique" in which a stream of normal saline is directed into the vitreous chamber could be of much help in floating the lens upward so that it can be easily grasped and spooned.

CONCLUSIONS

1. Dilatation of the pupils in children under 3 years of age with defective vision should be a routine procedure to eliminate the possible presence of ectopia

lentic. After the proper preliminary study of the position of the lens and vitreous, the contemplated type of surgical interference should be carefully weighed.

2. The surgical results of lens extraction often prove disappointing because of associated ocular abnormalities.

3. In young individuals the operation of choice is dissection of the lens with the use of two needle knives, as originally advocated by Bowman. In older persons, intracapsular extraction with a wide iridectomy is the preferred operation. This may be accomplished with the use of a lens forceps, lens spoon or loop and rarely with the use of a suction cup.

4. Extracapsular extraction should not be attempted, for its results are exceedingly disappointing in Marfan's syndrome.

SUMMARY

The various surgical techniques in Marfan's syndrome have been reviewed and evaluated. A case report is illustrated in which two eyes were operated on in the same individual, one with the use of lens forceps and in the other using a lens loop. The results were unusually satisfactory. Vision of better than 20/20 was obtained in both eyes. The patient maintained useful vision and presented no post-operative complications after a follow-up period of 3 years. Eventually he expired of an acute cardiac distress while at work.

160 Henry Street

REFERENCES

1. MARFAN, M. A., Un cas de déformation congénitale des quatre membrés, plus prononcée aux extrémités, caractérisée par l'allongement des os, avec un certain degré d'amincissement, Bull. et mém. Soc. méd. d. hôp. de Paris 13: 220, 1896.
2. ACHARD, M. D., Arachnodyly, Bull. et mm. Soc. méd. d. hôp. de Paris 19: 834, 1902.
3. ORMOND, A. W., and WILLIAMS, R. G., A Case of Arachnodyly with Special Reference to Ocular Symptoms, Guy's Hosp. Rep. 74: 385, 1924.
4. RADOS, A., Marfan's syndrome. (Arachnodyly coupled with dislocation of the lens), Arch. Ophth. 27: 477 (March) 1942.
5. ZEEMAN, W. P. C., Ueber Ectopia pupillae et lentis congenita, Klin. Monatsbl. f. Augenh. 74: 325, 1925.
6. KNAPP, A., Operative treatment of congenital subluxation of the lens, Trans. Amer. Ophth. Soc. 39: 83-85, 1941.
7. CHANDLER, P. A., Surgery of the lens in infancy and childhood, Arch. Ophth. 45: 125-138, 1951.
9. BARRAQUER, JOAQUIN, Personal communication.
10. KIRBY, D., Surgery of Cataract, Philadelphia. Lippincott, 1950, p. 484.

RESULTADOS DE MAS DE UN AÑO DE EXPERIENCIA CON EL EMPLEO DE LA ZONULOLISIS ENZIMATICA DE BARRAQUER

(Comentario a propósito de 111 casos)

POR

ALEJANDRO SALLERAS, M. D.

Buenos Aires, Argentina

Don Ignacio Barraquer cumple 75 años. Como contribución a su homenaje, hemos elegido este trabajo en el que resumimos nuestros resultados con tres técnicas creadas por su familia: la incisión y sutura de José, la zonulolisis enzimática de Joaquín y el erisifaco de Ignacio (como se dice en España). Las obras de don Ignacio son numerosas; su clínica no parece de este mundo, pero para nosotros su mayor mérito consiste en haber sabido dejar que sus dos hijos llegaran a ser lo que son.

Introducción:

Desde el 7 de mayo de 1958 hasta la fecha de enviar para su publicación este estudio, hemos efectuado 142 operaciones de catarata en pacientes de más de 20 años, 31 de ellas sin emplear quimotripsina y 111 usando quimotripsina.

En este trabajo solo nos ocuparemos de discutir las ventajas e inconvenientes del empleo de la zonulolisis enzimática en pacientes de más de 20 años ya que consideramos que por debajo de esta edad las numerosas extracciones intracapsulares que se obtienen, van seguidas de un porcentaje demasiado elevado de pérdidas de vítreo.

Material:

Del páncreas de las terneras se extraen diversos tipos de quimotripsina: ALFA, BETA, GAMMA, DELTA y PI. La alfa-quimotripsina tiene un mayor poder de difusión y una mayor estabilidad que los otros tipos.

Tres acciones caracterizan a esta última enzima:

1) *Actividad Endopeptidasa*: Rompe las uniones peptídicas dentro de la molécula proteica atacándolas a nivel del último carboxilo de los aminoácidos aromáticos.

2) *Actividad Exopeptidasa*: Libera aminoácidos que tienen grupos amino libres al final de las moléculas proteicas.

3) *Actividad Esterasa*: Hidroliza ciertos ésteres no específicamente.

Empleamos en todos nuestros casos alfa-quimotripsina (Quimotrase) de LABORATORIOS P. E. V. Y. A. (Barcelona-España), en diluciones al 1/5000 ó 1/10000, con preferencia por la primera concentración.

Es sabido que la solución de esta enzima pierde su efecto a las pocas horas estando a la temperatura ambiente (20% en 4 horas), mientras que conservada a 5° C., retiene un 60% de su actividad durante un mes. En algún caso, por carecer del producto hemos usado soluciones conservadas en esta forma, en concentraciones mayores con buen resultado. Preferimos sin embargo, soluciones recién preparadas. Consideramos importante destacar que la solución debe emplearse en lo posible a una temperatura próxima a los 37° C. para que su actividad sea óptima. Decimos esto porque hemos observado en varios casos que la enzima era inefectiva cuando se la empleaba sin haberla entibiado o se la había calentado demasiado. Usamos en cada operación según la edad del paciente y el estado biomicroscópico del cristalino, de 1/4 a 1 cc. de la solución.

Técnica quirúrgica:

Incisión: Hemos efectuado varios tipos de incisiones en esta serie.

TECNICA I: (En los primeros 31 casos). Incisión corneal inmediatamente por delante del limbo con cuchillo o queratomo y tijeras, herida que cerrábamos con 5 a 8 puntos corneales borde a borde con seda virgen.

TECNICA II: (En 12 casos). Incisión a cuchillo dejando una pequeña lengüeta escleral de 1 a 2 mm. para pasar luego de 7 a 11 puntos esclero-corneales que quedan sub-conjuntivales.

TECNICA III: (En 68 casos). Disección de un colgajo conjuntival amplio, cauterización suave de la esclera en la zona donde irá la incisión, paracentesis con aguja de Wessely a las 9 horas tanto en el ojo derecho como en el izquierdo y a 1,5 mm. del limbo; se hace la sección con la tijera con tope de José Barraquer que permite efectuarla biselada sin sacarla introduciéndola a las 9 horas y sacándola a las 3 horas. Esta sección, que deberá mantenerse siempre a 1,5 mm. del limbo, se sutura luego con 9 a 11 puntos esclero-corneales de seda virgen que quedan sub-conjuntivales.

Iridectomía: Periférica.

Irrigación de la cámara posterior: Con una cánula de punta roma y una jeringa conteniendo un par de cc. de la solución, procedemos de la siguiente manera: a) En pacientes con edades comprendidas entre los 20 y los 50 años, irrigamos la zónula en los 360° de su circunferencia; b) Después de los 50 años irrigamos solamente la zónula inferior de 4 a 8 horas con el objeto de facilitar el comienzo de la versión.

Observación de la Acción Zonulolítica: Después de colocar un punto previo, observamos con detención la profundidad de la cámara anterior y la superficie anterior del cristalino y del iris. Al cabo de unos pocos minutos generalmente, la cámara anterior se hace menos profunda, la superficie anterior del cristalino más convexa y la periferia del iris se hunde si la acción zonulolítica es muy efectiva. En ocasiones, cuando la cápsula o corteza anterior del cristalino presentan algún detalle que se destaca, hemos observado al efectuar las diversas maniobras que siguen a la irrigación de la enzima, éste cambia de posición, indicando una movilidad anormal del cristalino. Si solo se observan algunos o ninguno de los signos antes mencionados, habitualmente repetimos la irrigación, sobretodo en los pacientes jóvenes.

Extracción del Cristalino: Desde hace muchos años usamos sistemáticamente la ventosa, (erisifaco de Ignacio Barraquer) instrumento que consideramos ideal cuando efectuamos zonulolisis previa, ya que en la mayoría de los casos no es necesario hacer ninguna presión como cuando se usa la pinza. En la gran mayoría de los casos efectuamos una versión a cielo abierto sin mayor presión ni contrapresión. Cuando la zonulolisis no ha sido muy efectiva, también hacemos la extracción a cielo abierto pero con mayor tracción. Solo en aquellos casos en los cuales, al intentar voltear el cristalino notamos que existe un cierto grado de resistencia zonular que impide la versión, empleamos la técnica clásica de tracción y contrapresión como lo hiciéramos durante tantos años antes del descubrimiento de la enzima. En los enfermos operados de queratoplastia o que padecen de distrofia endotelial incipiente, usamos alfa-quimotripsina para limitar al máximo el trauma mecánico de la córnea y efectuamos la extracción sin versión.

Instilación de Acetilcolina: Terminada la extracción y anudado el punto previo, instilamos Acetilcolina al 1% con una cánula de punta roma, tratando al mismo tiempo de reponer el iris cuando es necesario.

Puntos Complementarios: Tratando de no vaciar la cámara anterior, colocamos los puntos complementarios antes mencionados cuidando de no hacerlos penetrantes. La cantidad de los mismos varía con la edad y docilidad del paciente.

Reformación de la Cámara Anterior: Preferimos hacerlo con solución fisiológica balanceada. Creemos que la solución fisiológica nos permite saber con mayor seguridad que la inyección de aire, si la incisión está bien cerrada, pues ésta se elimina con mayor dificultad cuando la filtración es mínima.

Sutura del Colgajo Conjuntival: Continua con seda virgen.

Post-Operatorio: Efectuamos vendaje monocular, levantamos al paciente a las 24 horas y lo damos de alta de la clínica al tercer día.

Análisis de los pacientes de esta serie:

En tres cuadros con sus comentarios correspondientes, haremos una breve reseña de algunos datos importantes de nuestros operados.

CUADRO 1

<i>Edades</i>		
20	a 30 años	5 casos
30	a 40 años	2 casos
40	a 50 años	7 casos
50	a 60 años	26 casos
60	a 70 años	34 casos
70	a 80 años	31 casos
80	a 90 años	6 casos

Como puede observarse en el CUADRO 1, la mayoría de nuestros enfermos está por encima de los 60 años. Podría argumentarse que ésta no es una serie ideal para demostrar la efectividad de la enzima, cosa que no pretendemos con este trabajo. Solo queremos comunicar aquí los resultados altamente satisfactorios obtenidos con el empleo sistemático de la enzima a todas las edades después de los 20 años y desvirtuar la presencia de graves complicaciones inmediatas o alejadas como consecuencia del uso de este nuevo proceder.

CUADRO 2

<i>Biomicroscopia de las cataratas</i>	
Inmaduras	67
Intumescentes	23
Maduras	15
Hipermaduras	5
Luxadas	1

El análisis del CUADRO 2 muestra el gran predominio de las cataratas inmaduras (más del 50%) cuya zónula es más resistente.

CUADRO 3

<i>Afecciones acompañantes a la catarata</i>	
Uveitis inactiva	4
Lesión macular	3
Glaucoma	3
Sinquisis centelleante	1
Retinopatía pigmentaria	1
Retinopatía diabética	5
Glaucoma operado	3
Exfoliación capsular	3
Uveitis heterocrómica	2
Miopía elevada	10
Enclavamiento de iris	1
Atrofia de papila	3
Coloboma congénito	1

Las afecciones mencionadas en el CUADRO 3, demuestran el criterio no selectivo del autor. En 22 de estos casos las lesiones preexistentes justificaban que la agudeza visual no fuera mayor de 0,6.

Estudio de la acción inmediata de la Zonulolisis

En los tres cuadros siguientes y a continuación de cada uno de ellos pasaremos revista a los resultados de la extracción del cristalino, analizaremos estadísticamente la efectividad de la zonulolisis y explicaremos las razones en las cuales nos basamos para hacer esta diferenciación; finalmente, enumeraremos los casos en donde hubo pérdida de vítreo y analizaremos las causas que las motivaron.

CUADRO 4

<i>Extracción del cristalino</i>	
Intracapsulares "in toto"	108 (97,3%)
Con rotura capsular	3 (2,7%)

Atribuimos al empleo de la enzima el alto porcentaje de extracciones intracapsulares "in toto", ya que en una serie similar de 117 casos operados también por nosotros, motivo de una conferencia sobre complicaciones en el II Curso Internacional de Oftalmología Especial del Instituto Barraquer, las extracciones "in toto" alcanzaban al 85,5% y las roturas capsulares al 15,5, mientras que en los casos motivo de este trabajo, los porcentajes son 97,3 y 2,7 respectivamente. En las tres roturas capsulares (Casos 26, 96 y 109) está consignado en el protocolo operatorio que la zonulolisis fue inefectiva por las causas que explicaremos en seguida:

CUADRO 5

Grado de efectividad de la zonulolisis		
Muy efectiva	en	60 casos
Efectiva	en	13 casos
Poco efectiva	en	6 casos
Inefectiva	en	9 casos
No consignada	en	23 casos

La efectividad de la zonulolisis depende de una serie de factores difíciles de precisar todavía, como son: concentración de la solución, temperatura de la misma, tiempo transcurrido entre su preparación y empleo, forma de irrigación de la cámara posterior, tiempo de permanencia de la solución dentro del ojo y finalmente, grado de resistencia zonular.

Decimos que la zonulolisis es *muy efectiva* cuando a los pocos minutos de la irrigación, el cristalino está completamente luxado nadando sobre la hialoides y la extracción se efectúa a cielo abierto sin tracción ni contrapresión.

Llamamos a la zonulolisis *efectiva* cuando se observan solo algunos de los signos de la acción enzimática, como disminución de la profundidad de la cámara anterior y aumento de la convexidad de la cara anterior del cristalino. La facoerisis puede hacerse a cielo abierto, sin contrapresión y con ligera tracción.

Consideramos que la zonulolisis es *poco efectiva* cuando no se observa ninguno de los signos de zonulolisis antes descritos y para la extracción del cristalino es necesario efectuar tracción y contrapresión, pero este sale con mayor facilidad que la que hubiéramos sospechado si no hubiéramos empleado la enzima.

Cuando la extracción se efectúa con maniobras de gran tracción y fuerte contrapresión, decimos que la acción zonulolítica fue *inefectiva*.

Del examen del CUADRO 5 se desprende que en el protocolo de la operación no se consignó en 23 casos el grado de efectividad de la zonulolisis.

En los 88 casos restantes, la zonulolisis fue más o menos efectiva en el 82,9% en tanto que su efectividad fue escasa o nula en el 17,1%. Atribuimos la poca efectividad de la zonulolisis en estos casos a defectos en la técnica de su preparación y empleo (excesivo calentamiento o temperatura insuficiente, poco tiempo de permanencia "in situ").

A pesar de ser partidarios del empleo de la enzima sistemáticamente, aun en los pacientes de mayores edades, no la hemos empleado en 31 de nuestros 142 operados, debido a que por razones de importación en ciertos momentos nos ha faltado.

CUADRO 6

Causas de pérdida de vítreo

CASO Nº 1: Por adherencias hialoideo-cristalinianas.

CASO Nº 65: Previa a la extracción por rotura de la hialoides con la cánula al irrigar la cámara posterior con alfa-quimotripsina.

CASO Nº 72: Por adherencias hialoideo-cristalinianas.

CASO Nº 91: Por coloboma congénito del cristalino.

Atribuimos la pérdida de vítreo del caso 1 como la de algún otro enfermo que no figura en esta serie por tener menos de 20 años, a las adherencias hialoideo-cristalinianas y a la falta de la rigidez escleral característica en los jóvenes, que hace que el tono normal de los músculos extraoculares exprima el contenido ocular al faltar la contención que significa el cristalino.

En el caso 65, un movimiento brusco de la cabeza de la paciente en el momento en que para irrigar la cámara posterior con quimotripsina, la cánula estaba en el meridiano de las 8 horas, hizo que ésta se introdujera a través de la zónula y rompiera la hialoides. El vítreo salió por delante del cristalino a pesar de lo cual pudo efectuarse una extracción intracapsular con ventosa.

La pérdida de vítreo en el caso 72 es en cierto modo inexplicable, porque a los 44 años, generalmente el ligamento hialoideo-capsular está roto.

Tenemos una película filmada de esta extracción en la que se ve perfectamente la adherencia del vítreo a la cara posterior del cristalino y como lo acompaña en su salida.

En el caso 91 la pérdida de vítreo, lógica, se produjo inmediatamente después de la paracentesis.

En resumen, en un solo caso la pérdida de vítreo está vinculada al empleo de la alfa-quimotripsina, pero no a su acción química sino al mecanismo de su introducción. En realidad, más bien a una falla en la preparación de la enferma cuya premedicación no era la deseable.

Las pérdidas de vítreo, que representan un 3,66% no están vinculadas por lo tanto en ningún caso, a la acción química de la enzima. En los 117 casos de la serie anterior (1957) el porcentaje de pérdidas de vítreo era de 3,4%.

En los dos cuadros siguientes nos ocuparemos de las complicaciones post-operatorias y de los resultados.

CUADRO 7

<i>Complicaciones post-operatorias</i>				
	<i>Técnica I</i>	<i>Técnica II</i>	<i>Técnica III</i>	<i>Total</i>
Estriada	22	8	36	66
Iritis	10	—	8	18
Discoria	—	—	1	1
Desprofundización de la C. A.	1	—	3	4
Fístula	—	1	—	1
Vaciamiento de la C. A.	4	5	9	18
Cámara anterior poco profunda	—	3	12	15
Desprendimiento coroideo	—	—	3	3
Glaucoma secundario	—	—	2	2
Rotura tardía de la hialoides	—	—	1	1
Oclusión pupilar	—	1	—	1
Sinequias anteriores	2	2	3	7
Sinequias posteriores	—	1	1	2
Distrofia endotelial	1	—	—	1
Sinequias corneo-vítreas	—	1	—	1
Neuritis óptica	—	1	1	2
Bloqueo pupilar	2	—	1	3
Hípema	1	1	13	15

El elevado porcentaje de queratitis estriada se explica porque involucramos en este rubro a todo pliegue corneal profundo por discreto que él sea. Por otra parte, todos los que empleamos el erisifaco de Barraquer, tenemos que soportar un porcentaje más elevado de queratitis estriada para compensar los enormes beneficios que este instrumento trae aparejados. Todas las queratitis estriadas han regresado rápidamente, de manera que ninguna de ellas representó una verdadera complicación para el ojo. Comparado el porcentaje de esta serie (59%) con el del año anterior mencionado (69%), vemos que no podemos atribuir a la alfa-quimotripsina la responsabilidad de esta complicación transitoria.

El 16,2% de iritis de mayor o menor intensidad (rotulamos como iritis a todo enturbiamiento de la cámara anterior ostensible macroscópicamente, aunque con los medicamentos habituales no deje rastros) de la presente serie, en contraposición con el 22% de la antes mencionada, descarta la posibilidad de que estas sean provocadas por la enzima.

Toda intervención quirúrgica produce un aumento de la densidad óptica de la cámara anterior. Muchos colegas por razones obvias restringen el examen de estos enfermos a la lámpara de hendidura en los primeros días del post-operatorio y quizás por esa razón sus porcentajes de iritis son inferiores a los nuestros.

Las discorias y las sinequias posteriores consignadas, deben atribuirse a las iritis mencionadas.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
1	16707	A. M. 27 años	I	Inmadura Traumática	Técnica I Intracapsular Zonulolisis**** Vítreo*	Iritis grave	+5+1,5/100:	1.
2	16766	R. L. 60 años	I	Intumescete Sineq. Ant. Atrofia iris inferior.	Técnica I Intracapsular Zonulolisis***	Iritis grave Bullas cornea- les - Distrofia Endotelial	+7+1,5/175º:	0,4.
3	15196	J. L. 49 años	I	Inmadura Glaucoma Lesión macular	Técnica I Intracapsular Zonulolisis**** Iredec. total	Estriada*	?	?
4	16944	T. M. 55 años	D	Intumescete Tracoma Nubécula central.	Técnica I Intracapsular Zonulolisis****	Iritis**	+10+5/120º:	0,9.
5	16789	J. C. 59 años	D	Inmadura	Técnica I Intracapsular Zonulolisis****	Estriada*	+10,5+2,5/120º:	1.
6	16854	T. S. 78 años	D	Morgagniana Sinquisis Centelleante	Técnica I Intracapsular Zonulolisis***	Estriada*	+11+2,5/30º:	0,5.
7	16815	D. F. 80 años	I	Inmadura Retinopatía Pigmentaria	Técnica I Intracapsular Zonulolisis*** Iridodiálisis	Estriada** Iritis*	+12:	0,4.
8	10005	M. A. 76 años	D	Inmadura Glaucoma operado Atrofia de papila.	Técnica I Intracapsular Zonulolisis* Iredec. total	Estriada Superior***	+10+2,5/0º:	0,2.
9	16803	B. G. 61 años	I	Inmadura Uveítis grave Sineq. Post.	Técnica I Intracapsular Zonulolisis*	Estriada* Iritis* Hipertensión Transitoria	+10:	0,6.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
10	10955	C. P. 65 años	I	Inmadura	Técnica I Intracapsular Zonulolisis*	Estriada**	+11:	1.
11	17411	P. L. 75 años	D	Inmadura	Técnica I Intracapsular Zonulolisis***	Estriada	+10+1/150°:	1.
12	17235	M. S. 72 años	D	Inmadura Dispersión pigmentaria	Técnica I Intracapsular Zonulolisis***	Iritis* Bloqueo pupilar Hipertensión Transitoria	+12+1/170°:	1.
13	14842	V. C. 82 años	D	Inmadura	Técnica I Intracapsular Zonulolisis	Vació C. A. a los 12 días Hernia II-III	+11+1/120°:	1.
14	17143	M.G. 50 años	I	Inmadura	Técnica I Intracapsular Zonulolisis	Vació C. A. a los 9 días Sineq. Ant. 10 y 12hs.	+16:	1.
15	17493	M. M. 78 años	I	Intumescete	Técnica I Intracapsular Zonulolisis	Estriada Bullas corneales	+13:	1.
16	11135	A. M. 80 años	D	Inmadura Glaucoma Lesión macular	Técnica I Intracapsular Zonulolisis	Iritis* Estriada* Bullas corneales	+12:	0,1.
17	17638	J. O. 74 años	I	Inmadura Exfoliación Capsular	Técnica I Zonulolisis Irodotomía	Bloqueo pupilar Iritis	+13+1/0°:	0,9.
18	17481	P. P. 71 años	I	Madura	Técnica I Intracapsular Zonulolisis	Estriada	+12+1,5/150°:	1.
19	13926	L. T. 68 años	D	Madura Retinopatía Diabética	Técnica I Intracapsular Zonulolisis - - -	Estriada*	+11+1/0°:	0,8.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
20	17623	A. G. 20 años	I	Intumesciente Heterocrómica	Técnica I Intracapsular Zonulolisis***	Estriada	+12+4/80°:	0,7.
21	17739	E. P. 54 años	I	Inmadura Miopía elevada	Técnica I Intracapsular Zonulolisis	Estriada***	-1/100°:	0,5.
22	17597	C. L. 63 años	D	Intumesciente	Técnica I Intracapsular Zonulolisis	Estriada** Hernia vítrea IV.	+11:	0,9.
23	17861	P. R. 62 años	D	Inmadura	Técnica I Intracapsular Zonulolisis	Estriada° Iritis	+12+2/150°:	1.
24	17698	A. T. 75 años	D	Inmadura Miopía elevada Lesión macular	Técnica I Intracapsular Zonulolisis	Estriada	s. c. 1/20.	
25	16795	M. C. 67 años	D	Madura	Técnica I Intracapsular Zonulolisis	Estriada Iritis Hipema	+10+2/30°:	0,8.
26	15288	R. H. 64 años	D	Inmadura	Técnica I Extracapsular Zonulolisis - - -	Estriada	+10:	1.
27	17916	G. F. 53 años	I	Inmadura	Técnica I Intracapsular Zonulolisis*** Iridodiálisis Superior	Estriada Vacío C. A. a los 12 días Sineq. Ant.	+13:	1.
28	15387	C. C. 55 años	I	Inmadura	Técnica I Intracapsular Zonulolisis****	Estriada	+13+1/135°:	1.
29	17671	V. A. 69 años	I	Inmadura	Técnica III Intracapsular Zonulolisis**	Iritis	+11+2/0°:	0,5.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
30	12919	C. R. 59 años	I	Inmadura	Técnica III Intracapsular Zonulolisis***	Poca C. A. a los 20 días Bloqueo pupilar	+11+1,5/20º:	1.
31	10612	F. S. 45 años	I	Inmadura	Técnica III Intracapsular Zonulolisis***	Sin C. A. a los 17 días - Queda con C. A. poco profunda	+11:	1.
32	18619	L. M. 63 años	D	Intumescete	Técnica III Intracapsular Zonulolisis***	Estriada	+8:	1.
33	18636	C. L. 28 años	D	Intumescete	Técnica III Intracapsular Zonulolisis***	Estriada Sin C. A. a los 3 días	+10+0,75/55º:	1.
34	18701	J. O. 78 años	D	Intumescete Retinopatía diabética	Técnica III Intracapsular Zonulolisis* -	Estriada Desprendimiento coroideo	+10+2/80º:	1.
35	15025	M. V. 73 años	I	Intumescete Retinopatía diabética	Técnica III Intracapsular Zonulolisis* -	Estriada - Vacío C. A. a los 17 días. Desp. coroideo, Hipema; Sineq. Anteriores	+11+2/170º:	0,1.
36	18262	J. Q. 76 años	D	Inmadura	Técnica II Intracapsular Zonulolisis*	Sin C. A. (1 mes) Oclusión pupilar (Pupilotomía)	+12:	1.
37	18644	P. F. 62 años	D	Intumescete Subluxada Traumática Enclavamiento inferointerno	Técnica II Intracapsular Zonulolisis***	Estriada* Poca C. A.	?	?
38	18363	J. D. 72 años	D	Intumescete	Técnica II Intracapsular Zonulolisis***	Estriada* Sin C. A. al 19º día. Sineq. Post.	+9+3/50º:	0,8.
39	11670	E. B. 80 años	I	Inmadura Lesiones miópicas en fondo	Técnica II Intracapsular Zonulolisis***	Fístula	+3,5+2/45º:	0,6.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
40	16571	A. S. 68 años	I	Inmadura	Técnica III Intracapsular Zonulolisis***	Tardó en formar C.A. Estriada* Discoria	+11+2/20°:	1.
41	18398	J. S. 63 años	I	Inmadura	Técnica II Intracapsular Zonulolisis***	Sin C. A. desde el 1º día (15 días). Sineq. Anteriores	+9+3/20°:	1.
42	18764	B. P. 65 años	D	Inmadura Exfoliación capsular	Técnica II Intracapsular Zonulolisis****	Poca C. A.	+11:	1.
43	10968	M. R. 70 años	D	Intumescete	Técnica III Intracapsular Zonulolisis****	Estriada* Poca C. A. a los 12 días. Sineq. Anteriores	+9+2,5/95°:	0,7.
44	18419	E. D. 72 años	D	Intumescete	Técnica III Intracapsular Zonulolisis***	Estriada*	+12:	1.
45	17764	J. G. 78 años	D	Inmadura Ret. Diabética Glaucoma	Técnica II Intracapsular Zonulolisis***		+10+2/165°:	0,4.
46	18992	O. G. 58 años	D	Inmadura Miopía elevada	Técnica III Intracapsular Zonulolisis****	Estriada* Desprofundiza C. A.	+3+3/150°:	0,8.
47	18888	R. C. 63 años	I	Intumescete	Técnica III Intracapsular Zonulolisis**		+11+2/0°:	0,9.
48	19131	R. G. 68 años	D	Madura	Técnica II Intracapsular Zonulolisis***	Estriada*** Hipema. C. A. no muy profunda	+12:	1.
49	18941	I. S. 72 años	D	Inmadura Miopía elevada	Técnica III Intracapsular Zonulolisis***		+4+3/0°:	1.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
50	18694	L. P. 55 años	I	Inmadura Operada de Glaucoma agudo. Atrofia de papila	Técnica III Intracapsular Zonulolisis***	Hipema	+11+3/170°:	0,5.
51	19160	T. T. 72 años	I	Intumescete	Técnica III Intracapsular Zonulolisis**	Hipema Desprofundiza C. A.	+14,5+2/65°:	0,7.
52	19204	A. C. 59 años	D	Inmadura	Técnica III Intracapsular Zonulolisis***	Estriada* Poca C. A.	+12+1/65°:	1.
53	19556	E. G. 77 años	I	Hipermadura Exotropia	Técnica III Intracapsular Zonulolisis**	Estriada*	+10+2/130°:	0,6.
54	17860	J. M. 56 años	D	Madura	Técnica III Intracapsular Zonulolisis***	Estriada Poca C. A. Neuritis	+10:	0,3.
55	19516	G. S. 66 años	D	Inmadura Uveítis antigua Atrofia papila	Técnica III Intracapsular Zonulolisis...	Estriada*	+10:	1/20.
56	19402	J. R. 69 años	D	Intumescete	Técnica III Intracapsular Zonulolisis**	Estriada*	+11+1/120°:	0,5.
57	19328	D. G. 68 años	I	Inmadura	Técnica III Intracapsular Zonulolisis****	Estriada* Pliegues Infero- externos	+8+2/100°:	0,3.
58	10348	A. L. 69 años	I	Madura	Técnica III Intracapsular Zonulolisis****		+10+4/30°:	0,9.
59	14842	V. C. 83 años	I	Inmadura	Técnica III Intracapsular Zonulolisis--	Estriada Hipema Trau- mático	+11:	1.

Nº	R. G.	Nombre	Ojo	Biomicroscopía	Operación	Complicaciones	Refracción	Visión
60	19488	A. T. 59 años	I	Inmadura	Técnica III Intracapsular Zonulolisis***	Estriada** Poca C. A.	+12+1,5/0º:	1.
61	19823	F. D. 64 años	I	Intumescente	Técnica III Intracapsular Zonulolisis****	Estriada Hipema Poca C. A.	+15:	1.
62	19766	C. P. 62 años	I	Inmadura	Técnica III Intracapsular Zonulolisis*	Estriada*** Iritis	+13+1,5/60º:	1.
63	19781	A. P. 68 años	I	Inmadura Miopía elevada	Técnica III Intracapsular Zonulolisis**	Estriada Desprofundiza C. A. Hipema	+3:	0,5.
64	18676	C. D. 71 años	D	Madura Injerto corneal Glaucoma operado	Técnica III Intracapsular Zonulolisis			s. e. C. D.
65	20076	N. F. 62 años	I	Intumescente	Técnica III Intracapsular Zonulolisis*** Pérdida de vítreo	Estriada	+11+1/40º:	1.
66	19810	R. R. 62 años	I	Inmadura Nubéculas	Técnica III Intracapsular Zonulolisis**		+6+0,75/65º:	0,5.
67	19503	S. D. 60 años	I	Inmadura Ret. Diabética	Técnica III Intracapsular Zonulolisis***	Vació C. A. a los 17 días Desp. Coroideo	+11:	0,4.
68	20108	V. C. 68 años	I	Intumescente	Técnica III Intracapsular Zonulolisis**	Estriada Poca C. A. Sineq. Post.	+13:	1.
69	20134	M. F. 64 años	D	Inmadura Exfoliación Capsular	Técnica II Intracapsular Zonulolisis***	Estriada* Hipema Glaucoma	+10+2/170º:	0,7.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
70	15192	A. L. 63 años	I	Intumescete	Técnica III Intracapsular Zonulolisis***	Vació C. A. a los 23 días	+14+1,5/175º:	1.
71	20070	J. C. 53 años	I	Inmadura Miopía - 20 D.	Técnica III Intracapsular Zonulolisis***	Estriada Hipema Trau- mático. Vació C. A.		s. c. 0,2.
72	20377	I. P. 44 años	D	Inmadura	Técnica III Intracapsular Zonulolisis* Pérdida de vítreo	Iritis. Sineq. Ant. Glaucoma secundario Estriada*** Ciclodialísis	+9+1/175º:	0,8.
73	10384	C. M. 78 años	D	Madura	Técnica II Intracapsular Zonulolisis	Estriada***	+11+3/165º:	1.
74	20116	R. G. 57 años	D	Morgagniana	Técnica II Intracapsular Zonulolisis***	Estriada* Sin C. A. del comienzo. Sineq. Ant. 3 hs.	+12+0,5/20º:	0,5.
75	18667	P. R. 71 años	I	Inmadura Diabética	Técnica II Intracapsular Zonulolisis***	Estriada**	?	?
76	20149	J. O. 78 años	D	Hipermadura	Técnica II Intracapsular Zonulolisis**	Estriada*** Vació C. A. Sineq. córneo-vítrea (sinequiotomía)	+11+2/150º:	0,3.
77	20479	S. M. 74 años	D	Madura Exotropía	Técnica III Intracapsular Zonulolisis****	Estriada** Vació C. A. al 6º día. Pliegues internos	+10:	0,5.
78	15610	G. M. 61 años	I	Inmadura	Técnica III Intracapsular Zonulolisis****	Estriada Poca C. A.	+11:	0,7.
79	20648	C. P. 31 años	D	Inmadura Heterocromía	Técnica III Intracapsular Zonulolisis***	Poca C. A.	+10+1/160º	1.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
80	15412	P. B. 50 años	D	Inmadura Uveítis grave	Técnica III Intracapsular Liberación sinq. Zonulolisis**	Hipema	+12+2/60°:	1/30.
81	19979	T. C. 59 años	D	Inmadura	Técnica III Intracapsular Zonulolisis inf.***	Iritis Hipema Poca C. A.	+12+3/0°:	1.
82	20626	R. M. 65 años	I	Madura	Técnica III Intracapsular Zonulolisis inf.**	Estriada*	+10+0,75/0°:	1.
83	20623	C. G. 72 años	I	Madura	Técnica III Intracapsular Zonulolisis inf.*	Estriada** Hipema Estriada Temp.	+12+2,5/70°:	1.
84	11670	E. B. 80 años	D	Inmadura Lesiones miópicas	Técnica II Intracapsular Zonulolisis*** (Estaba luxada)	Estriada	+3+2°160/:	0,6.
85	17739	E. P. 54 años	D	Inmadura Miopía elevada Lesión macular	Técnica III Intracapsular Zonulolisis -	Estriada		s. c. 0,1.
86	17910	G. F. 53 años	D	Inmadura	Técnica III Intracapsular Zonulolisis****		+12+1/150°:	1.
87	17411	P. L. 75 años	I	Inmadura	Técnica III Intracapsular Zonulolisis***	Estriada Hipema	+10+1/150°:	1.
88	19516	G. S. 66 años	I	Inmadura Uveítis grave	Técnica III Intracapsular Zonulolisis**	Estriada Hipema	+10:	1/20.
89	19204	A. C. 59 años	I	Inmadura	Técnica III Intracapsular Zonulolisis	Poca C. A. Rotura tardía hialoides	+12:	1.

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
90	S. A.	A. I. 52 años	D	Madura Exotropia Lesión macular	Técnica III Intracapsular Zonulolisis*		+10+1,5/90º:	0,2.
91	S. A.	E. B. 53 años	I	Inmadura Colomboma congénito de iris Cristalino coroides	Técnica III Intracapsular Zonulolisis* - Pérdida de vítreo	Iritis**	+7:	0,1.
92	S. A.	N. T. 28 años	D	Inmadura Patológica	Técnica III Intracapsular Zonulolisis***	Sinequias Anteriores?	+9+1,5/0º:	1.
93	S. A.	C. P. 65 años	I	Madura	Técnica I Intracapsular Zonulolisis****	Desprofundiza C. A.	+6+3/0º:	1.
94	S. A.	C. C. 77 años	D	Madura	Técnica III Intracapsular Zonulolisis		+10+3,5/170º:	0,7.
95	S. A.	M. N. 49 años	I	Intumescete	Técnica I Intracapsular Zonulolisis**	Vació C. A.	+16+1,5/30º:	0,8.
96	S. A.	J. L. 55 años	I	Intumescete	Técnica I Extracapsular Zonulolisis - - -	Estriada* Iritis	+12,5:	1.
97	20867	A. R. 58 años	I	Inmadura	Técnica III Intracapsular Zonulolisis****	Poca C. A. Larga duración	+12+1,5/0º:	0,9.
98	20318	G. C. 60 años	D	Inmadura	Técnica III Intracapsular Zonulolisis***	Estriada Sin C. A. (20 días)	?	?
99	20999	V. V. 70 años	I	Inmadura Diabético	Técnica III Intracapsular Zonulolisis***	Hipema Iritis*	?	?

ZONULOLISIS ENZIMATICA

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
100	19479	M. A. 59 años	D	Inmadura	Técnica III Intracapsular Zonulolisis****	Vació C. A. a los 28 días	?	?
101	21101	C. M. 54 años	D	Intumescente Sub-luxada traumática Corectopia	Técnica III Intracapsular Zonulolisis inferior	Estriada*	+8+1,5/140°:	I.
102	21246	M. C. 77 años	I	Inmadura	Técnica III Intracapsular Zonulolisis inferior***	Estriada* Poca C. A.	?	?
103	21307	D. L. 45 años	I	Inmadura Miopia elevada	Técnica III Intracapsular Zonulolisis****	Estriada*	?	?
104	Mendoza	A. D. 40 años	D	Inmadura Endocrina	Técnica II Intracapsular Zonulolisis***	Estriada*	?	?
105	Mendoza	A. D. 40 años	I	Inmadura Endocrina	Técnica II Intracapsular Zonulolisis inefectiva	?	?	?
106	Mendoza	E. D. 39 años	D	Inmadura	Técnica II Intracapsular Zonulolisis****	?	?	?
107	Mendoza	S. C. 78 años	I	Madura	Técnica II Intracapsular Zonulolisis****	?	?	?
108	Mendoza		D	Hipermadura	Técnica II Intracapsular Zonulolisis----	?	?	?
109	S. A.	N. T. 24 años	I	Madura	Técnica II Rotura capsular (Quedan restos****) Zonulolisis...	Iritis*	?	?

Nº	R. G.	Nombre	Ojo	Biomicroscopia	Operación	Complicaciones	Refracción	Visión
110	21305	J. M. 53 años	D	Inmadura Miopía elevada	Técnica II Intracapsular Zonulolisis...	Estriada*	+11+2/175°	0.5.
111	20718	M. L. 73 años	I	Inmadura	Técnica II Intracapsular Zonulolisis		+11+1/0°	1.

En dos casos (12 y 17) ambos con incisión corneal (Técnica I), se produjo un bloqueo pupilar debido a la incompetencia de la iridotomía para mantener el pasaje normal de los líquidos de la cámara posterior a la anterior. Ambos cedieron inmediatamente con la atropinización.

En un solo caso tuvimos una distrofia endotelial (vinculada a la práctica de la incisión corneal?) en un paciente predispuesto. (Caso 2).

No hemos observado diferencias en el número e intensidad de los hipemas, que en esta serie llegan al 13,5%, con el empleo de la alfa-quimotripsina. Ellos se acrecientan con el empleo de las incisiones esclero-corneales (Técnicas II y III) desapareciendo casi completamente con las incisiones corneales. (Técnica I).

Solo hemos notado una complicación relativamente frecuente desde que usamos la zonulolisis enzimática; (pero también usamos mucho más frecuentemente la incisión escleral con o sin cauterización previa /Técnicas III y II/) la presencia de cámaras anteriores transitoria o permanentemente bajas.

Decimos que la cámara anterior *se desprofundiza*, cuando habiendo sido de profundidad afáquica durante varios días, aparece con menor profundidad en una curación y llega a ser muy baja en la siguiente, para luego recuperar lentamente la profundidad normal de los operados de catarata o quedar ligeramente disminuída. Atribuimos este hecho a una disminución tardía de la producción de humor acuoso, del "flow" (F) quizás provocada por la alfa-quimotripsina. Descartamos la presencia de una filtración porque en estos casos, el colgajo conjuntival permanece plano sin edematizarse.

Hablamos de *vaciamiento de la cámara anterior* cuando esta desaparece bruscamente, ya sea en forma espontánea o como consecuencia de un trauma y existen signos evidentes de filtración (edema del colgajo conjuntival o eliminación de humor acuoso por la herida). Esta complicación se debe a una mala coaptación, a la presencia de una incisión poco biselada, a la existencia de algún punto penetrante, a una cauterización previa excesiva o a un trastorno en la cicatrización de la herida. Solo en el último de los casos puede ser culpada la quimotripsina de esta complicación.

Entendemos por *cámara anterior poco profunda*, aquella que desde el primer día del post-operatorio no tiene la profundidad afáquica y no habiendo signos de filtración la atribuimos a una disminución en la producción de humor acuoso, debida al trauma sobre el cuerpo ciliar de la cánula profundamente introducida o a una acción inhibitoria de la enzima?

Sin embargo, si analizamos nuestra estadística, veremos que la frecuencia de estas complicaciones está directamente vinculada al tipo de incisión empleada.

Las sinequias anteriores de nuestra estadística, están vinculadas en su totalidad a aquellos casos en los cuales hubo vaciamiento de la cámara anterior o ésta permaneció poco profunda durante un período de tiempo más o menos prolongado.

En sólo dos casos hemos observado la existencia de un glaucoma secundario (casos 69 y 72). El primer caso lo atribuimos a la exfoliación capsular que padecía el enfermo y el segundo, a la presencia de sinequias anteriores en un paciente en el cual se perdió vítreo y del que nos hemos ocupado más arriba.

Como consecuencia de iritis tórpidas, hemos llegado en un caso a una oclusión pupilar, que mejoró llegando a visión normal, mediante una simple pupilotomía (sección de la membrana pupilar). (Caso 36).

En dos casos (9 y 12) observamos hipertensión transitoria inexplicable que desapareció sin el empleo de medicación. En otros dos casos (48 y 54) anotamos una neuritis óptica que ya ha sido descrita por otros autores (Reese) sin el empleo de la quimotripsina.

En 1 caso (89) se produjo una rotura tardía de la hialoides pupilar, que la vinculamos a una dilatación pupilar enérgica que le provocamos a una paciente que había quedado con sinequias posteriores.

Finalmente en un solo caso (39) observamos una fistulización con drenaje al espacio sub-conjuntival producida posiblemente por un defectuoso cierre de la herida.

En resumen, ninguna de las complicaciones post-operatorias documentadas por nosotros puede ser, directa o indirectamente, atribuída a este nuevo procedimiento ya que todas existían en mayor o menor grado en otras series publicadas por nosotros y en las cuales no usábamos la enzima.

CUADRO 8

		<i>Resultados</i>	
<i>Astigmatismo</i>		<i>Agudeza visual</i>	
Sin astigmatismo:	31 casos		
De 0 a 1D:	17 casos	De 0,8 a 1:	56 casos
De 1 a 2D:	29 casos	De 0,2 a 0,8:	30 casos
De 2 a 3D:	13 casos	De menos de 0,2:	12 casos
De más de 3D:	4 casos	Se ignora en:	13 casos
Se ignora en:	17 casos		

Atribuimos el alto porcentaje (51%) de astigmatismos inferiores a 1D al empleo de la incisión esclero-corneal con sutura múltiple sub-conjuntival efectuada en la mayoría de nuestros casos (Técnicas II y III).

Del examen del cuadro 8 fluye que la frecuencia y el grado de astigmatismo post-operatorio no son influenciados por el empleo de alfaquimotripsina, ya que en un 82% de los casos en los que el astigmatismo resultante está consignado en la historia clínica, éste es inferior a 2 dioptrías.

Las agudezas visuales finales de esta serie, se comparan favorablemente con otras hechas sin el empleo de la enzima, ya que en un 90% de los casos los pacientes han resultado con visión útil (más de 0,2) y en un 58,9% la visión es normal (0,8 a 1). No se han considerado en este cómputo 13 casos en los cuales por diversas razones: recientemente operados, enfermos que no siguieron concurriendo a la consulta, etc., no se pudo efectuar una refracción definitiva.

En 31 casos la agudeza visual al cabo de un cierto tiempo no llega a 0,6. Debemos aclarar, que de acuerdo con el estudio pre-operatorio y en razón de la presencia de otras afecciones oculares acompañantes a la catarata, en 22 casos se sabía que la agudeza visual sería inferior a 0,6. En 6 casos, la agudeza visual no llegó a 0,6 y a pesar del estudio minucioso de los mismos no se pudo determinar la causa (¿ambliopía?). En solo dos casos la agudeza visual inferior a 0,6 se debió a complicaciones operatorias y post-operatorias, ninguna de ellas atribuibles al uso de la enzima (2 y 76). En el caso 2 la disminución visual se debe a una distrofia endotelial en una paciente de 60 años operada con incisión corneal y quien tenía alteraciones pre-operatorias que hacían sospechar un endotelio frágil. En la enferma 76 la operación fue sin inconvenientes, pero en el post-operatorio vació la cámara anterior, se produjeron adherencias córneo-vítreas y pliegues en la Descemet que cedieron bien a una sinequiectomía con inyección de aire. En la última refracción practicada, su agudeza visual era de 0,3, pero tenemos la impresión de que todavía tiene que mejorar mucho.

CONCLUSIONES

- 1) El empleo sistemático de la zonulolisis con Alfa-quimotripsina, incrementa extraordinariamente el porcentaje de extracciones "in toto" (97,3% en esta serie contra 84,5% en una anterior).
- 2) El uso de quimotripsina no modifica los porcentajes de pérdidas de vítreo (3,6% y 3,4%).
- 3) La efectividad de la enzima varía considerablemente por muchos factores. Sería aconsejable buscar su standarización.
- 4) Como fruto del menor trauma mecánico en la extracción, las complicaciones graves son menos frecuentes.

- 5) Solo hemos hallado una complicación nueva para nosotros; la desprofundización de la cámara anterior, que atribuimos a una disminución en la producción de humor acuoso (F) posiblemente por una acción de la enzima sobre el cuerpo ciliar. (19 casos: 17,1%).
- 6) La Alfa-quimotripsina no altera macroscópicamente, ni inmediata ni tardíamente, ninguna de las demás estructuras oculares (iris, vítreo, retina, etc.)
- 7) Los resultados inmediatos y alejados del empleo de la zonulolisis enzimática se comparan muy favorablemente con los de otras series en las cuales no se usó la enzima.

Melo 1788

DES AVANTAGES DE L'INTRODUCTION INTRA-TENONNIENNE D'UN IMPLANT SCLÉRAL DANS LA PROTHÈSE OCULAIRE

PAR

RAYMOND DE ST. MARTIN, M. D.

Toulouse, France

De tous temps, les opérateurs se sont efforcés de remédier à la difformité que provoque l'ablation d'un oeil et qui résulte de l'enfoncement dans l'orbite de la pièce de prothèse et de son immobilité, ainsi que, par voie de conséquence, de la formation, au dessous du sourcil, d'un sillon plus ou moins profond qui accentue encore la défiguration.

Il serait sans intérêt d'énumérer ici les moyens imaginés pour atteindre ce but et qui tendent tous, par l'introduction dans la cavité de *TENON* d'un corps étranger bien toléré, d'un volume aussi important que possible, à constituer au fond de l'orbite un moignon susceptible de repousser en avant la pièce de prothèse et de la faire participer à sa propre motilité.

Qu'il nous suffise de rappeler que l'on peut classer les implants orbitaires qui ont été successivement préconisés en deux catégories, selon qu'ils sont constitués par des substances *inertes* (billes de verre ou d'or, de tantale, de nylon, d'acrilic) ou *organiques*, celles-ci pouvant être à l'état frais (graisse, oeil de lapin, cartilage costal, tendon d'Achille, sclérotique) ou *stérilisées et conservées* (cartilage de veau formolé, têtes de fémur de nouveau-nés, sclérotique).

Quel que soit le type adopté, l'implant doit répondre aux desiderata suivants:

Volume aussi approchant que possible de celui de l'oeil enlevé;

Tolérance par les tissus orbitaires;

Mobilité maxima.

Ces conditions obtenues après l'exentération du globe, par la simple inclusion intra-sclérale d'une bille de verre ou d'acrilic, sont beaucoup plus difficiles à

réaliser après l'énucléation, et l'on doit reconnaître qu'aucun des procédés successivement proposés ne donne une entière satisfaction. En effet, les implants inertes ne sont tolérés que sous un volume réduit, sinon leur élimination est fatale à plus ou moins brève échéance.

Les implants vivants (cartilage costal, graisse, tendon) véritables auto-greffes, se résorbent malheureusement la plupart du temps en totalité ou en grande partie et aboutissent à un moignon minuscule, rétracté au fond de l'orbite; ils sont donc pour ces raisons, pratiquement, abandonnés.

Fort heureusement, d'importants progrès ont été réalisés depuis quelques années avec les implants en acrylique. Cette substance est bien tolérée par les tissus, ce qui réduit les risques d'élimination, et peut dès lors être utilisée en pièces plus volumineuses que par le passé, ce qui améliore leur mobilité. On accentue encore celle-ci par leur tunellisation qui permet de suturer les muscles droits au devant d'eux.

Certains modèles, munis de broches maintenues hors de la conjonctive et s'articulant avec la prothèse oculaire procurent des résultats plus parfaits encore.

Ces avantages légitiment donc la préférence de la majorité des opérateurs pour cette catégorie d'implants. Assez nombreux cependant sont ceux qui marquent encore de la défiance à l'égard des broches, car celles-ci peuvent favoriser, quelques précautions que l'on prenne, l'élimination plus ou moins tardive de la pièce, par nécrose de la conjonctive à leur contact, ou par infection.

Ce risque nous paraît assez sérieux pour nous faire renoncer aux prothèses à broches, malgré leurs avantages esthétiques.

Les implants acryliques d'ailleurs, et quelle que soit la tolérance tissulaire dont ils sont l'objet, ne doivent pas moins être considérés comme des corps étrangers toujours sujets à l'élimination. Nous leur préférons donc les *implants organiques* enfouis, et, parmi ceux-ci, l'implant constitué par la sclérotique entière.

Ceux-ci présentent à notre avis, de nombreux avantages.

La sclérotique n'est pas résorbable, elle est parfaitement tolérée, et rapidement réhabilitée par du tissu conjonctif. Elle fait ainsi corps avec le contenu de l'orbite, ce qui, joint à son important volume, contribue le mieux possible, au résultat fonctionnel et esthétique: esthétique, par la disparition ou tout au moins l'effacement parfois très important du sillon sus-palpébral; fonctionnel parce que, suturée aux muscles droits, elle acquiert la meilleure mobilité qu'il soit possible d'obtenir.

Elle peut être utilisée à l'état frais, lorsque'elle provient d'un oeil énucléé que l'on réimplante dans la cavité de Tenon après l'avoir vidé de son contenu et " pré-

paré"; elle peut l'être également à l'état de pièce extraite depuis longtemps (des mois et des années) et conservée en flacon stérile.

Ces deux utilisations donnent des résultats fonctionnels identiques. Nous préférons la seconde pour les raisons suivantes:

1—La sclérotique conservée, aussi bien tolérée qu'à l'état frais, est toujours disponible et se prête aux interventions d'urgence: il suffit d'en posséder une réserve.

2—La sclérotique fraîche est parfois inutilisable, par exemple dans les cas de tumeur intra-oculaire ou lorsqu'elle a subi un traumatisme trop important.

Les raisons qui plaident en faveur de l'implant scléral ont été exposées, il y a déjà de longues années (LEMOINE et VALOIS) (1). Nous rappellerons cependant car elles expliquent et justifient l'excellence de cet implant qui résulte, répétons-le de son volume et aussi de la mobilité qu'il acquiert plus que tout autre.

En effet, la capsule de Tenon contracte des adhérences avec la partie antérieure de la sclérotique. Par ces adhérence pré-musculaires et latérales, les muscles et les tendons font corps avec la capsule qui, d'autre part, s'insère à la sclérotique au devant des tendons et dans toute la largeur des intervalles tendineux. Il en résulte que le muscle ne s'implante pas seulement sur le globe par son atache, mais aussi, par la large insertion supplémentaire de la capsule. Nous pouvons dès lors entrevoir que la contraction d'un muscle agit, non seulement sur son extrémité tendineuse, mais encore sur une partie de l'entonnoir ténonien.

Pour avoir un moignon régulier et mobile, il suffira donc d'obtenir des adhérences entre la capsule et lui-même.

Cette condition ne peut être réalisée qu'avec un greffon vivant non résorbable (cartilage ou sclérotique).

L'expérience nous a montré que le cartilage à l'état frais est fréquemment résorbé et, à l'état conservé, souvent éliminé.

La sclérotique au contraire, aussi bien conservé que fraîche, a toujours été bien tolérée. Elle se montre donc très supérieure au cartilage et c'est à elle que vont nos préférences.

Nous décrivons deux techniques d'implantation sclérale: l'une avec sclérotique fraîche; l'autre avec sclérotique conservés.

La première a fait l'objet d'un travail récent du Pr. HERVOUET (2), la seconde a été exposée par nous-même (3) plus récemment encore.

(1) LEMOINE et VALOIS, Enucléation avec greffe sclérale. Ann. d'Ocul. 1922, p. 175.

(2) F. HERVOUET, L'enucléation - Eviscération. Bull. S. F. O. 1958, page 508.

(3) R. de StMARTIN, La prothèse sclérale intra ténonienne. Bull. S. F. O. Juin 1959.

1^o—*Implantation de sclérotique à l'état frais.*

“L'énucléation, écrit HERVOUET, est opérée suivant la technique habituelle, les 4 chefs musculaires étant bien disséqués sur au moins 1,5 ctms. La prothèse est alors mise en place, le pôle postérieur en avant”.

Cette prothèse a été préparée de la façon suivante.

“On prélève un globe de cadavre et on enlève avec soin tout ce qui n'est pas la sclère elle même. On introduit à son intérieur une grosse bille de verre ovale puis on la referme par une suture bord à bord au catgut chromé. L'implant est alors retourné, la section du nerf optique se trouvant maintenant en avant. Au couteau de Parker on pratique ensuite aux 4 pôles de l'implant, 2 incisions parallèles de 6 mm, éloignées chacune de 2 mm. Les incisions supérieures et inférieures sont distantes de 1,5 ctms. environ et les incisions latérales, suivant les cas, de 1,5 à 1,8 ctms.

Ce temps opératoire préliminaire peut être réalisé soit, aussitôt avant l'intervention, soit la veille, soit même plusieurs jours auparavant, la greffe étant alors conservée dans l'alcool et rehydratée une heure avant l'intervention”.

La pièce étant introduite dans la capsule de TENON comme il a été dit, les 4 chefs musculaires sont passés, les uns après les autres, dans le pont scléral découpé à chacun des 4 points cardinaux du greffon. Chaque muscle engagé doit dépasser le pont scléral assez largement, de façon à pouvoir être ramené en arrière, les sutures devant être, par sécurité, rigoureusement musculo-musculaires.

Le tissu sous-conjonctival, puis la conjonctive, sont ensuite refermés en deux plans.

2^o—*Implantation de sclérotique conservée.*

Notre technique diffère assez notablement de la précédente ce qui nous oblige à la décrire avec quelque détail.

a—*Préparation de l'implant.*

On utilise un oeil énucléé ou prélevé pour kératoplastie.

On résèque la cornée ainsi qu'une collerette de la sclérotique limbique en donnant à l'ouverture une forme ovalaire horizontale.

L'oeil est alors vidé de son contenu puis, retourné en doigt de gant, frotté avec une gaze pour supprimer tout vestige d'uvée.

Cette toilette effectuée avec beaucoup de soin, la sclère est de nouveau retournée. On introduit dans sa cavité une olive de plastique et on la ferme par quatre points en U à la soie forte. On noue ensuite une boucle de soie noire très fine (fil de Kalt) sur l'insertion des 4 muscles droits pour pouvoir la repérer ultérieurement.

L'implant ainsi préparé est mis, 48 heures, dans une solution de formol à 2% puis, 8 jours, dans l'alcool à 90°.

Il est alors conservé, jusqu'à son utilisation, dans l'alcool à 60°. La conservation en flacon bien bouché est, pratiquement indéfinie. Il suffira de le rehydrater quelques heures avant l'emploi, en le plongeant dans du sérum que l'on renouvelle 2 à 3 fois.

b—*Inclusion de l'implant.*

1—*Enucléation.*

Il est essentiel d'éviter toute hémorragie. La section du nerf optique sera donc faite à l'anse métallique. La section des muscles demande une attention particulière. On s'efforcera de conserver, autant que possible, leurs attaches à la capsule et aux ailerons. On les dénudera donc tout juste assez, pour passer un fil dans chaque tendon et le sectionner aux ciseaux ainsi que les insertions pré-équatoriales de la capsule.

Un tampon adrénaliné au une éponge de thrombase est introduit dans la cavité tandis qu'on marque au bleu de méthylène, sur l'implant le siège de l'insertion des muscles.

2—*Mise en place de l'implant.*

La sclère conservée est alors introduite dans l'entonnoir de Tenon, nerf optique en arrière, c'est à dire au fond de l'entonnoir.

La ligne de sutures sclérales est orientée horizontalement et les marques des anciennes insertions se placent en face des 4 muscles droits. On suture alors chacun d'eux à l'ancienne insertion, comme lors d'une intervention pour strabisme.

L'opération s'achève par une suture en deux plans: capsule, d'abord, par points en U, au catgut chromé; conjonctive ensuite, par un solide surjet.

Les deux interventions diffèrent par la qualité et la préparation de l'implant, par son mode d'introduction dans la cavité de Tenon, enfin par celui de la suture des muscles.

L'une et l'autre présentent l'avantage de la bonne tolérance des tissus. Quant au volume et à la mobilité de la prothèse, et bien que nous n'ayons pu comparer les résultats obtenus avec les deux techniques, il semble que la seconde doit être plus satisfaisante. Au point de vue mobilité d'abord, les muscles n'étant pas raccourcis, leur implantation s'effectuant sur les anciennes insertions correspondantes, et les expansions latérales de la capsule faisant rapidement corps avec l'implant; au point de vue volume, ensuite, qui est certainement ainsi plus important et, par conséquent, au point de vue esthétique.

Si cependant le sillon palpébral persiste et nuit à la perfection de la prothèse, il est possible de la réduire notablement par un greffon dermique.

Nous préférons ne pas procéder à cette greffe au moment même de l'énucléation, comme certains la conseillent, mais seulement plusieurs mois plus tard, lorsque la prothèse a pris sa place définitive.

Toulouse, France

VISION AND ASTRONAUTICS

BY

INGEBORG SCHMIDT, M. D.

Bloomington, U. S. A.

Since the earliest times the astronomers obtained their information about celestial bodies through direct visual observation. In the last decades photography started to play a role as an objective method and at the present time, in some instances, radio astronomy is applied. But it is still recognized that vision cannot be fully replaced by objective methods. For instance it is nearly impossible to register details of the surface of the planet Mars during a "Clearing" of its cloudy atmosphere on a photographic film, when it lasts for a fraction of a second only, whereas the human eye is able to catch some impressions. Astronomy takes advantage of the scientific results of visual physiology and the latter gains from astronomy, e. g. our visual acuity standard is based on the statement about the resolvability of two stars which have a separation of one minute.

With the development of astronautics the visual problems are to some extent the same as those of astronomy yet in certain respects the *conditions of seeing* are different: the astronomer observes the celestial bodies through our atmosphere. In the daytime the sky appears bright due to light scattering particles in the air. This diffused light is of a blue color because the short wavelengths are scattered more than the long wavelengths. The stars are not visible because of lack contrast. At night, even in a moonless night, the sky is never entirely black. That is due mainly to airglow in the upper atmosphere. This phenomenon is light emitted by atomic oxygen, nitrogen and sodium brought into an excited state by ultraviolet radiation during the day. Also zodiacal light, which is light reflected from micrometeorites, starlight and galactic light, both direct and scattered, contribute to the luminance of the night sky. The average luminance of a moonless sky is of the order of 10^{-4} cd/m² (nit). Due to attenuation by the atmosphere through absorption and scattering all celestial bodies appear by about 20% darker than outside the atmosphere.

In space, which for practical purpose begins at an altitude of about 150 km, the atmosphere is too rarefied to scatter light appreciably. The sky, therefore,

appears permanently dark except of a faint brightness provided by direct starlight, galactic light and zodiacal light. The luminance of the sky in space is of the order of 10^{-5} nit. Stars, sun, and moon are permanently visible and appear brighter than on Earth and also whiter because short wavelengths are not scattered out. More stars are visible than through the atmosphere. The illuminance by the sun at the outer limit of our atmosphere is equal to about 140000 lux, whereas at the earth surface it is only slightly above 100000 lux even under the through the cone of the Earth shadow, there is always extreme brightness on that most favorable conditions.

On Earth we have a day-night cycle. In space, except when the astronaut passes side is deep shadow except when, depending on the position and distance, it is partly brightened up by moonlight and by earthlight. This photo-scotic condition (Strughold), extremely dark shadows outside and inside the cabin poses special visual problems for the astronaut.

The *visual tasks* to be performed are mainly detection and observation of oncoming space crafts, satellites, meteorites and of planets as prospective landing targets, including the planet Earth, since to the orbiting astronaut the Earth itself attains the properties of a luminous celestial body. It causes the strange situation that the surround is bright below and dark above, the reverse of the situation at the Earth surface, where in the primary position of the eyes, the lower part of the retina is usually adapted to a greater luminance than the upper part.

The visibility of an object, the clearness with which an object stands out from its surroundings, is a function of *luminance contrast*, which is defined by the rela-

$$C = \frac{L_o - L_b}{L_b}$$

tionship $C = \frac{L_o - L_b}{L_b}$ where L_o is the luminance of the object, L_b the lumi-

nance of the background. The liminal contrast value, the threshold contrast, depends on a number of variables, e. g. the background luminance, the size of the object, and psychological factors. Data for contrast thresholds are available in the literature (Blackwell). Small light sources, subtending about 1 minute of arc or less for the light adapted eye, or up to 10 minutes of arc for the dark adapted eye, are a special case of contrast thresholds insofar as the visibility is then affected proportionally to the illuminance E . at the plane of the eye. The relationship $E = I/d^2$ is known as the law of inverse squares, I representing the intensity of the observed point light source, d its distance. When the eyes of the astronaut are adapted to the sky luminance of 10^{-5} nit, the threshold illuminance E_t equals 2×10^{-9} lux. This value corresponds to a star of 8th magnitude. Knowing also the intensity I , for instance in the case of an artificial satellite, its visual range can

be computed, that is, the farthest distance at which it should be just visible. The intensity of a spherical satellite with specular reflection can be computed from a

formula $I = \frac{E_s R r^2}{4}$ where E_s is the illuminance by the sun, R the average reflectance of the satellite, r its radius. For a white diffusing spherical satellite and also

for planets the intensity is given by the formula $I = E_s R r^2 \frac{2}{3} \cdot \frac{\sin \alpha + (\pi - \alpha) \cos \alpha}{\pi}$

where α is the phase angle, subtended at the center of the satellite or planet by the direction to the sun and to the observer. When seen through the atmosphere the intensity values must be multiplied by its total transmittance.

The next problem is the recognition of details in order to identify the luminous object. Here another function of the eye will be utilized, namely *visual acuity* which comprises different functions, e. g., the detection of single spots, of single lines, the separation of two or more lines or spots, seeing of contours, and breaks in contours and distinction of more or less familiar forms. All these functions depend on a multitude of variables. One of the most important factors affecting visual acuity is illumination. Visual acuity does not only depend upon the luminance of a small central area containing some details, but also upon the luminance of the surround, which is the chief determinant of the adaptation level of the eye. For low and medium luminances of the central area, maximal visual acuity is obtainable when the luminance of the surrounding area is the same as that of the central. In case of high luminances, e. g., when the astronaut observes the surface of Mars with an average luminance of 2350 nit or of Venus with 50000 nit, the adaptation level must be somewhat below that of the observed surface to achieve maximal visual acuity, as follows from Fig. 1 after Foxell and Stevens. The astronaut should have a possibility to obtain the desired level of adaptation in order to recognize as much details as possible.

Another function of our eye supporting recognition of details is *color vision*. We observe yellow-red and green areas on Mars, a great red spot and other colored features on Jupiter, white and yellow belts on the golden ball of Saturn, etc. We will consider only a few of the factors affecting color vision, namely the luminance of the observed area, the adaptation state of the eye, the size of the object, and assume normal color vision of the observer. The threshold illuminance for correct recognition of colors is about 10 lux. The distance from the sun where correct color discrimination is not possible any more because the illuminance by the sun is not sufficient would be about 18 billion km from the sun or about three times the distance of the planet Pluto from the sun. The colorless world

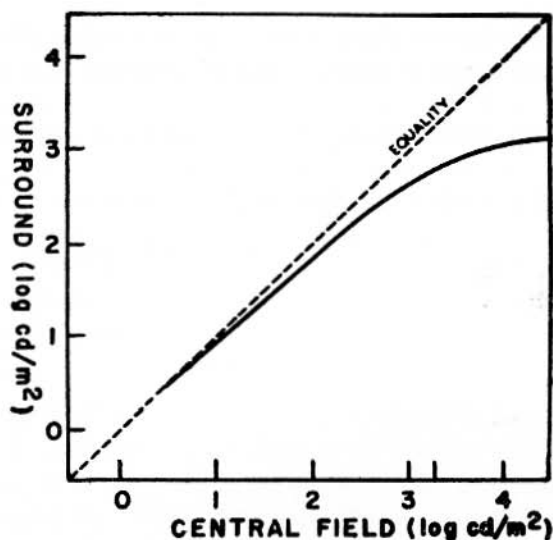


Fig. 1. Luminance of surround for maximum visual acuity (central field 0.5° , surround 120°) (redrawn after Foxell and Stevens).

of interstellar space with a black, star studded, sky can be called a *hypophotic zone* (Strughold) in comparison to the euphotic zone which in respect to solar illumination is favorable to space operations and to life on planets.

Not only the luminance, but also the size of the colored area is of importance for a correct recognition of colors. By compiling the data of the literature Fransworth found a product of area times intensity below which color vision become tritanomalous and a somewhat lower product below which it is tritanopic. In the tritanomalous stage orange-red and bluegreen and violet are about colorless and hardly distinguishable from each other. Because of the tritanomaly of the fovea, Syrtis Major, a large dark area on the northern hemisphere of the planet Mars will be seen with unaided eyes in its real colors with certainty only when the astronaut approaches the planet to about 652000 km that is about $1\frac{1}{2}$ times the distance of our moon from Earth.

In order to observe colors the eyes must be bright adapted. Dark adaptation, which the astronaut obtains when scanning the sky, is unfavorable insofar as it makes dim colors appear unsaturated and causes a shift in their luminosity, known as Purkinje phenomenon.

Even when luminance and size of the observed area are sufficient and the eyes are adapted to brightness, we cannot be certain that the colors perceived are real. For decades astronomers have argued the question of whether or not the dark green and bluishgreen areas of Mars are really green and bluegreen or merely a phenomenon of contrasts. By simulating the conditions of observation

of Mars in the laboratory it has been shown that production of the Martian coloration by means of contrast phenomena is definitely a possibility (I. Schmidt). These findings cannot be used as evidence against the existence of vegetation of Mars, however, since, first the vegetation may have some dark color, different from green, and secondly, as we know from observations on earth, distant green areas do not appear green, but unsaturated bluegreen or even blue, due to the absorption by the atmosphere and to an additional coloration by scattered indirect sunlight, the bluish air light. Since the atmosphere on Mars is not very transparent for short wavelengths, such phenomena can take place also on that planet. Moreover, dark areas in the vicinity of large sunlit areas, for instance snowfields, assume a bluish contrast color to the yellowish bright areas (for more details see Middleton). This may occur also on the surface of Mars, the atmosphere of which by attenuation may render the sunlight yellowish.

From a certain high luminance value on colors lose in greenness and redness and become increasingly yellowish and bluish. Color vision of a normal person becomes similar to that of a red-green blind. On further increasing luminance everything becomes achromatic, colorless. The lower limit, above which color vision is appreciably disturbed, is, according to Segal, beyond 6000 trolands. A troland is measure of retina illuminance, obtained by multiplying the luminance of the observed area in nits with the pupillary area in mm^2 , e. g., a retinal illuminance of 6000 trolands is produced when an area having a luminance of 1,910 nits is observed through an artificial pupil of 2 mm. diameter. Thus, when observing Mars through the atmosphere (1880 nit) color vision would not be effected whereas when observing in outside the atmosphere (2350 nit) it probably would.

When the astronaut with unaided eyes takes a look into the bright sun, or at near distance from the planet Earth glances toward its surface (having an average reflectance of about .35, Earth reflects a great portion of the incident sunlight) a *dazzling glare* would result causing discomfort to the observer because of a blinding after-image and disabling him for any visual tasks. The astronaut will be particularly prone to such glare since his eyes, when scanning the sky will be dark adapted and the pupils will be wide. When there is a glare source in the field of view, e. g. the sun from a great distance, which would permit vision while it is present, it may produce a veiling effect through scattered light by the eye media and thus reduce contrasts. The effect of the veiling glare depends on the angle between the visual line and the direction of the glare source. In the presence of high intensity light sources subtending a small angle, the rest of the visual field is affected as if a veiling luminance L_1 were present over the visual field. If φ is the angle between the visual line and the direction of the glare source, E the illuminance at the eye from the glare source, then $L_1 = \frac{E}{\varphi^2 \kappa_1}$. For L_1 in

nits, E in lux equals 13.7 ∓ 1.6 (cited after a survey by Rose). Rose computed that the eyes of the astronaut would still suffer from glare up to a distance of 1.8×10^9 km from the sun that is beyond the orbit of Saturn. At that distance the illumination from the sun is about 1000 lux.

Special attention must be given to possible hazards to the eyes when observing the sun without appropriate protection. With sufficient energy a retinitis solaris or even a burn of the retina can occur, resulting in a *helioscotoma* in the visual field (Strughold). Such burns are known from observation of solar eclipses with an insufficiently smoked glass. Outside the atmosphere the danger is greater and increases of course in heliopetal direction, for instance on an expedition into the region of Venus. From data available about similar effects from atomic flashes (Byrnes, Brown et al.) it can be estimated that an exposure time of the order of 15 seconds or less of the eye to the solar radiation in space at earth's distance might be sufficient to cause retinal burns. On a flight in the direction of Venus the critical time of exposure would become shorter. (It may be of interest that in the optical blinking reflex, a time of 0.15" elapses from the onset of the stimulus until the lids are closed (Lawson) and that time required to contract the pupil equals 0.45 to 0.7 seconds (Petersen).

Depth perception is not of great value in space except during approach and hookup between two spacecrafts, because the distances are too great. The binocular cue of stereopsis is useful only up to about 200 meters. The so-called empirical or secondary factors of depth perception will be helpful to some extent: for instance geometrical perspective: an apparent slight curvature of the obviously parallel belts of Jupiter support the impression of a globe, a partial overlay permits us to conclude that the blue clouds on Mars are higher than the yellow clouds, the size of known objects, e. g. satellites or spacecraft may be helpful to judge their distance. Motion parallax which is enhanced by high speed (Rose) may play a role. When observing the surfaces of celestial bodies, the transition between light and shade gives the surfaces a quality of shape in the third dimension. A gradual transition of shading yields an impression of a curved surface, an abrupt shading that of an angled surface. Fig. 2 shows two photographs of the same area on the moon. One must know the direction of the incident light, otherwise an optical illusion may take place. In the right picture the sunlight is incident from the left. We perceive craters with elevated edges. In the left picture the sunlight is incident from the right, but we have the impression that it is also incident from the left and the craters appear as mounds. By rotating the figure the illusion disappears.

The use of the human eye in the control of the spacecraft is limited by the distance the vehicle travels during the time necessary for *perception and reaction*.

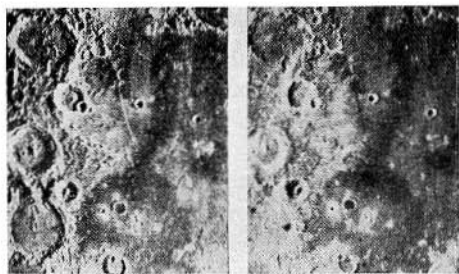


Fig. 2. (a) Moon last quarter. (b) Moon first quarter (after F. L. Whipple).

(a)

(b)

According to a survey by Strughold the latent period of a simple optical perception is 0.1 seconds at low luminances and in the periphery of the retina, in the fovea and in daylight 0.05 sec. The reaction time for a fixation movement is about 0.18 sec. Recognition takes 0.2 to several seconds. To permit evasive control action an object must have a visual range exceeding the distance travelled during the chain of latencies. The spacecraft would have also mechanical latencies before a change in the flight path occurs. Since within a time of 1 seconds a spaceship travels 7 km, but a meteorite 40 km (its visual range may be shorter than 40 km), there is permanent danger of collision with meteorites.

Still the visual sense is the sole sense which serves the astronaut for orientation concerning his position and movement in space in this world of silence and weightlessness, where the mechanoreceptors, the pressoreceptors and the otolithic organ cannot provide any information.

SUMMARY

The visual observation of the sky by an astronaut differs from that by an astronomer insofar as it is not done through the light absorbing and light scattering medium which our atmosphere represents. The sky appears dark with the celestial bodies permanently visible, whiter and brighter than when observed through the atmosphere. A day-night cycle is lacking. Brightly illuminated area bordering deep shadows. The functions of contrast sensitivity, visual acuity and color vision are discussed in view of recognition and observation of oncoming spacecraft, satellites, meteorites and of planets as prospective landing targets, including the planet Earth. Since the eyes of the observer are dark adapted when scanning the sky and the pupil is wide, a glance toward the sun and even toward Earth may cause a dazzling glare or at least a veiling glare disturbing vision. Special attention must be given to avoid retinal burns. Depth perception is not of great value because the distances are too great. Due to the high speeds on one side and sensorial and mechanical latencies on the other side there will be permanent danger of collision of a spacecraft with meteorites.

Bloomington

REFERENCES

- BLACKWELL, H. R., Contrast Thresholds of the Human Eye, *Journal of the Optical Society of America* 36: 624-634, 1946.
- BYRNES, V. A., BROWN D. V. L., ROSE, H. W., and CIBIS, P. A., Choroiretinal Burns Produced by Atomic Flash. *Archives of Ophthalmology* 53: 351, 1955.
- FARNSWORTH, D., Tritanomalous Vision as a Threshold Function. *Farbe* 4: 185-197, 1955.
- FOXELL, C. A. P., and STEVENS, W. R., Measurements of Visual Acuity. *British Journal of Ophthalmology* 39: 513-537, 1955.
- MIDDLETON, W. E. K., *Vision through the Atmosphere*. University of Toronto press, 1952.
- ROSE, H. W., Monocular Depth Perception in Flying. *Journal of Aviation Medicine*, 23: 242-245, 1952.
- ROSE, H. W., Perception and Reaction Time. Chapter 18 in *Physics and Medicine of the Atmosphere and Space*. Ed. by O. O. Benson Jr., and H. Strughold, J. Wiley & Sons. New York, 1959.
- SCHMIDT, I., Spaces of Potential Visibility of Artificial Satellites for the Unaided Eye. *Astronautica Acta* 3: 373-387, 1958.
- SCHMIDT, I., Visual Problems in Observing the Planet Mars. *Proceedings of the Lunar and Planetary Exploration Colloquium*, April 25, 1959, Santa Monica, Calif.
- SCHMIDT, I., The "Green" Areas of Mars and Color Vision. (to be published in *Astronautica Acta*).
- SEGAL, J., *Mechanismus des Farbensehens*. Translated from French, Fischer, Jena, 1958.
- STRUGHOLD, H., *The Human Eye in Space*. (to be published in *Astronautica Acta*).
- STRUGHOLD, H., *Chains of Latencies in Vision*. USAF School of Aviation Medicine. Randolph AFB, Texas, 1950.
- WHIPPLE, F. L., *Earth, Moon and Planets*, Blakiston Co. Phila, Toronto, 1946.

THE VITREOUS IMPLANT

BY

C. DEE SHAPLAND, M. D.

London, England

INTRODUCTORY

That degenerative changes in the vitreous body play an important part in the etiology of simple detachment of the retina has been increasingly recognized during the past decade—thought, in reality, this is no new thought as it was first suggested by Leber as long ago as 1882 and later stressed by Gonin, Lindner and Von Sallman. Indeed, the development of the various forms of globe shortening operation has been due to a tacit understanding of the importance of vitreous detachment and contraction in the causation of retinal detachment, but it is to Dr. Donald Shafer of New York that we are indebted to this latest development in retinal detachment surgery—the planting of human vitreous in eyes affected with complicated retinal detachments.

TECHNIQUE

The doner eye is an enucleated human eye preferably removed within six hours of death. Immediately after removal the eye is placed in a penicillin streptomycin suspension in sterile liquid paraffin (500,000 units penicillin, 500,000 units streptomycin, 5,000 gm. sterile liquid paraffin), where it remains for two hours. Then, with all aseptic precautions, the eye is removed from its container, rinsed in Ringer's solution, and a culture taken from the limbus and bulbar conjunctiva, after which it is placed in a glass bottle containing sterile liquid paraffin and stored in a refrigerator at 4° C.

The recipient eye is prepared for the vitreous injection before any surface diathermy or other operative procedure has been undertaken by making a small vertical opening in the conjunctiva and Tenon's capsule 7 mm. behind the limbus in the infero-temporal quadrant just below the insertion of the external rectus. The exposed sclera is cleaned and a small incision some 3 mm. long, centred at 7 mm. behind the limbus and parallel to the meridian at that point, made with a

Graefe knife just down to the pars plana of the ciliary body. A mattress suture of 00 silk or plastic thread, doubly armed with Grieshaber 81/7 needles, is inserted across the small incision and the two threads in the base of the incision withdrawn with a scleral hook — the two loops so formed enable the assistant to separate the lips of the little wound at the time of the vitreous injection. The detachment operation, whether diathermy or scleral resection or a combination of both, is now proceeded with up to the stage of evacuation of the sub-retinal fluid.

The surgeon now changes over to a second trolley which contains the donor eye and a separate set of instruments. The cadaver eye is well rinsed in sterile normal saline so as to remove all paraffin globules and then held firmly in a large gauze swab in the left hand. The sclera is cleaned at a point some 14 mm. behind the limbus and between any two recti. A cautery at dull red heat now makes an opening at this site, circular in shape, about 3 mm. in diameter and shelving more deeply towards its centre, the cautery being applied intermittently until the choroid and retina are just perforated (Fig. 1). The opening should be just sufficient

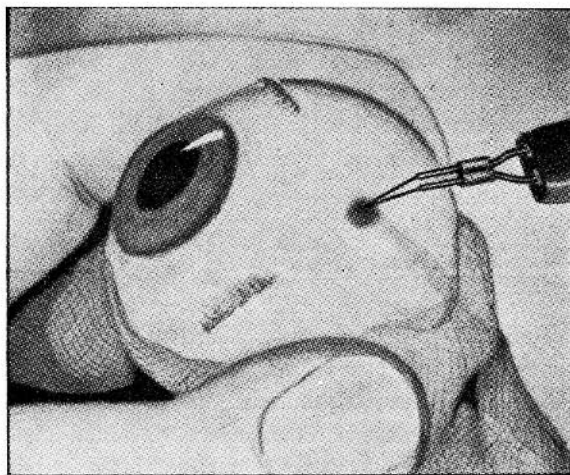


Fig. 1. Cautery opening into donor eye.

size to accommodate the nozzle of a 2 c.c. record syringe which is now applied to the opening and with firm pressure on the globe with the left hand and suction on the plunger of the syringe with the right as much as 2 cc. of vitreous can be aspirated (Fig. 2). A special needle of 18 British Standard Gauge has been made to my design by C. Davis Keeler which has the advantage of not only a sharp point but of a cutting edge on each side of this, so that it slips easily through the small incision in the recipient's eye, and also a stop 12 mm. there from so that the needle cannot be inserted too far (Fig. 3). This is now placed on the nozzle of the syringe and the surgeon returns to the recipient eye to make provision for

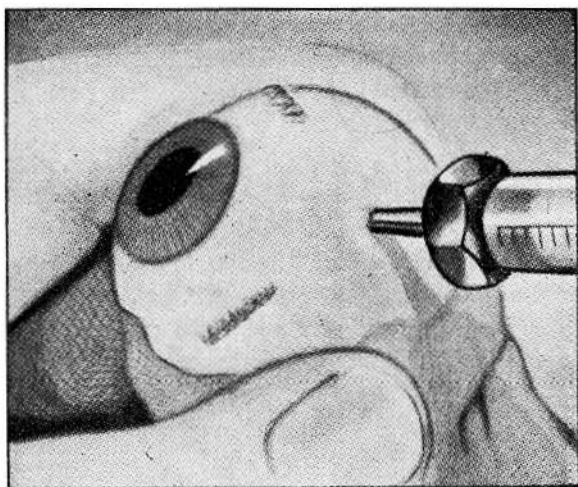


Fig. 2. Aspiration of vitreous from donor eye.

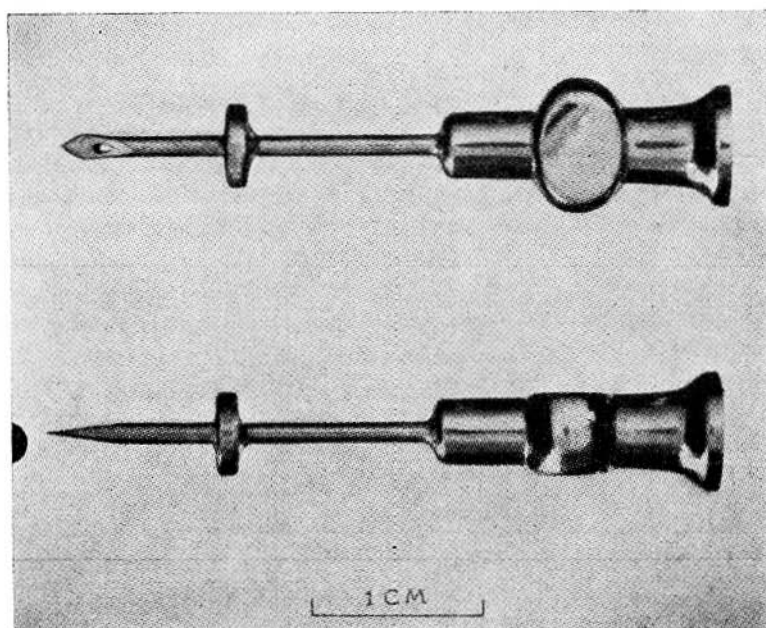


Fig. 3. Vitreous implant needle (Shapland).

the evacuation of the sub-retinal fluid — this I do with a catholysis current of 2 ma., and a 0.5 mm. diameter needle over the site where the main bulk of the sub-retinal fluid is lying. It is very important that there should be an obvious free of sub-retinal fluid before the vitreous is injected.

The vitreous aspirate is now planted into the recipient eye by inserting the 18 gauge needle through the little opening previously made in the sclera and directing it towards the centre of the globe. The eye being soft at this stage the lower lip of the incision is supported by the assistant making traction on the lower loop of the mattress suture whilst the surgeon supports the upper lip with a pair of Jayles forceps (Fig. 4). The vitreous is slowly injected until the eye becomes quite firm and a gush of sub-retinal fluid can usually be seen escaping from the site of the catholysis puncture. The needle is held in situ while the assistant releases the loop of the mattress suture and makes one half of a surgeon's knot — this he then ties off tightly as the surgeon withdraws the needle.

It is of interest to note that if the donor eye be now opened a much more compact vitreous remains in the anterior portion of the globe and if the nozzle of the 2 cc. record syringe is applied to this it will not become aspirated into the syringe on making suction but will adhere to the nozzle and it, together with the retina and sometimes the lens as well, can all be withdrawn from the eye on the nozzle of the syringe.

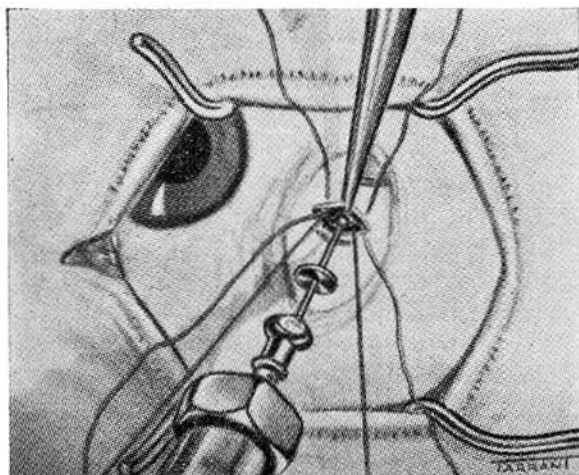


Fig. 4. Injection of vitreous into recipient eye.

RESULTS

Between August 13th, 1956 and June 11th, 1959 I have performed a vitreous implant on 34 cases, 27 males and 7 females. Half of these (17) were myopic

detachments, 9 aphakic detachments whilst the remaining 8 occurred in emmetropic of hypermetropic eyes, these latter mainly traumatic of inflammatory detachments. Twenty of these cases were blind in the fellow eye from previous retinal detachments and were therefore to be regarded as desperate cases, and, indeed, I usually only employ the vitreous implant as a last resort after more orthodox detachment surgery — diathermy or lamellar scleral resection has failed. I have, however, often combined the vitreous implant with other surgical procedures at the same session — 8 times with diathermy, 6 with a lamellar sclerectomy, on 3 occasions with light coagulation, twice with catholysis punctures over the hole and once with an embedded polyethylene tube. I have now employed multiple vitreous implants in 5 cases — 5 in one, 3 in a second and 2 in the remaining three, making a total of 43 implants to date.

It is important that the donor eye should be quite fresh — excised not longer than 24 hours. In two cases in which I used vitreous from eyes which had been kept for four days — both of them were early cases my first and fourth and each after receiving second injections reacted violently — a panophthalmitis occurring in the first and a low grade endophthalmitis in the second. At that time it was not clear whether this was an infective or allergic process but both responded to intensive local and systemic antibiotic therapy, so presumably were infective. In Barcelona last September Shafer assured me that he had seen no allergic reactions with subsequent vitreous implants and I have since employed multiple implants on 3 further cases — one receiving 5, one 3 and the third 2 with no untoward reactions, but in all these the donor material was under 24 hours old.

In taking the vitreous aspirate from the donor eye I now employ the vitreous implant needle instead of applying the nozzle of the record syringe to the globe as originally described — the needle is inserted towards the centre of the globe through an elliptical area at the equator sterilised by a cautery at dull red heat — it is unusual to obtain more than 2 cc. of vitreous aspirate and I use the same needle for the injection into to the recipient eye so as to avoid losing any of the aspirate.

Complications of this procedure have been few — and, apart from the two cases of endophthalmitis, not serious. There were three cases of secondary glaucoma and all occurred in aphakics, one lasted from the time of the implant for 3 weeks but eventually responded to diamox and pilocarpine, in the remaining two the raised tension appeared on the fourth day in one and on the sixth day in the other and both responded to diamox 250 mmg. four hourly within 3 days. Curiously enough two of these three cases were cured so a fleeting secondary glaucoma is certainly not of bad import.

With regard to results 6 of the 34 cases were cured — 3 of the 9 aphakic detachments, 2 of the 13 myopic detachments and one of the inflammatory detachments in a hypermetrope. Although a temporary improvement in a retinal detachment is frequently seen following a vitreous implant it is rarely maintained for long unless the retina is completely replaced and although a further six cases do appear to have derived some permanent benefit from this procedure — one aphakic, 2 myopic and 3 detachments in emmetropic eyes, the rest must be classed as failures.

In my experience the type of case which does best with vitreous implant is a sub-total detachment in which no definite unsealed retinal hole can be found, indeed, one in which a collection of sub-retinal fluid appears to be trapped and has failed to become absorbed. At the time of the vitreous implant it is most important to puncture over the approximate centre of this loculus and to be sure of a free flow of sub-retinal fluid before injecting the vitreous aspirate.

In conclusion the vitreous implant would appear to have a small but definite place in the treatment of the more serious cases of retinal detachment — myopic and aphakic detachments which have not responded to orthodox surgery, and also it is worth trying in those with vitreous traction bands. In my experience to date it is, rather, surprisingly, the aphakic detachments which have responded the best.

London, England

L'EXTRACTION INTRACAPSULAIRE CHEZ LES PETITS ENFANTS ET LES ENFANTS A L'AIDE DE L'ALPHA-CHYMOTRYPSINE

PAR

E. B. STREIFF, M. D.

Lausanne, Suiza

Depuis juin 1958, nous employons à la Clinique ophtalmologique de Lausanne la méthode d'extraction intracapsulaire à l'aide de l'alpha-chymotrypsine (Quimotrase) d'après J. Barraquer. Nos cas sont peu nombreux comparés à ceux d'autres cliniques. Nous nous sommes bornés à employer cette méthode seulement dans des cas précis: cataractes intumescents, cataractes juvéniles et préséniles, cataractes avec myopie forte et en particulier cataractes congénitales.

Nous avons suivi les indications de J. Barraquer: irrigation sous-irienne au moyen d'une canule-aiguille mousse coudée ou courbe (J. Weiss, Londres, ou Fl. Fischer, Freiburg) dirigée dans les différents quadrants et, enfin, à travers le colobome basal. L'extraction est faite soit à la pince, soit à la ventouse de Harrington, 2 à 3 minutes après l'irrigation. En fin d'opération, nous avons pratiqué le lavage de la chambre antérieure au moyen d'Acéchole. Nous avons augmenté le nombre habituel des sutures cornéo-sclérales (6-8).

L'intérêt de la méthode nous paraît surtout résider dans le fait de pouvoir opérer des enfants en bas âge. Car si l'on part du principe que l'extraction de la cataracte congénitale doit être précoce, afin que la vision centrale puisse se développer plus ou moins normalement, il est, nous semble-t-il, important d'éviter d'une part plusieurs narcoses et d'autre part des réactions uvéales parfois prolongée comme c'est le cas lorsqu'on fait l'extraction soit linéaire, soit par dissections multiples.

Nous avons opéré des deux yeux à distance de 2 - 3 semaines, sous narcose générale, 4 garçons âgés de 5 mois, de 1 an, de 10 ans et de 13 ans. Chez les garçons de 10 et 13 ans, nous avons eu à un oeil l'apparition du vitré dans la plaie, mais il a été facilement réduit au moyen d'eau distillée d'après l'indication de E. Moutinho.

Les autres yeux de tous ces enfants n'ont présenté aucune complication et, fait remarquable, nous n'avons constaté la moindre irritation, même le lendemain de l'extraction. La cicatrisation s'est faite de première intention, la cornée est toujours restée claire.

Chez trois enfants, nous avons observé un léger déplacement pupillaire vers le haut, ce qui est probablement dû au fait que l'enfant pleure et serre les paupières lors des changements de pansement. Nous nous sommes demandé s'il ne serait pas préféralle de faire une iridectomie totale chez les enfants en bas âge. Chez le quatrième garçon, nous avons fait une akïnésie du facial par injection de 2 cc. d'alcool à 40° et la pupille est restée ronde et centrée pendant une dizaine de jours; ensuite elle s'est déplacée vers le haut, comme chez les trois autres enfants.

Les contrôles ultérieurs de ces yeux n'ont pas montré jusqu'ici d'altérations particulières ni de la cornée, ni du vitré, ni du fond de l'oeil (recul de 6 mois à 1 an). Il nous paraît peu probable que des altérations dues à l'alpha-chymotrypsine puissent encore se manifester. Nous ne croyons pas non plus que le danger du décollement rétinien soit augmenté, bien au contraire, car cette méthode d'extraction évite précisément toute traction.

Il est de toute importance de ne pas appliquer cette méthode dans n'importe quel cas de cataracte congénitale. Si les formes zonulaires ou les formes molles totales ne posent pas de problèmes, il faut se méfier de certaines cataractes totales blanches ou des polaires postérieures, surtout si elles sont unilatérales, car nous savons qu'elles peuvent présenter une persistance de l'artère hyaloïde à la capsule postérieure. L'extraction totale dans ces cas entraînerait l'arrachement de la rétine.

Le nombre de cataracte congénitale opérée chez l'enfant n'est pas encore très élevé, surtout en dessous de l'âge de 2 et 1 an. Les cas publiés jusqu'à ce jour ne semblent pas avoir présenté de complications particulières, ce qui est confirmé par nos résultats.

Cependant, J. Barraquer qui avait dans une première grande série (14 cas) obtenu des résultats favorables, nous met en garde contre certaines complications qu'il a observées dans une deuxième série de cas (1er. Colloque Maghrébin d'Ophthalmologie, 3-6 mai 1959).

Chez les adultes jeunes entre 31 et 58 ans (12 cas) et au dessus de 60 ans (15 cas), nous avons observé une forte réaction du globe oculaire avec troubles vitréens intenses dans trois cas seulement. Il s'agissait de myopies fortes de 60, 70, 71 ans. Il est possible que le vitré liquéfié et dégénéré du myope sénile soit plus sensible à l'effet de l'alpha-chymotrypsine. Notons cependant que 2 autres cas

de myopie et que dans les cas cités le vitré s'est éclairci en quelques semaines après l'opération, sans compromettre le résultat fonctionnel final.

Il nous faudra attendre des résultats opératoires plus nombreux et un recul plus grand pour établir la valeur définitive de cette méthode. Malgré tout, nous croyons en son avenir, même et surtout chez l'enfant en bas âge.

Clinique Ophthalmologique

BIBLIOGRAPHY

- BARRAQUER, J., *Kl. Mbl. f. A.* **133**, 609-615, 1958.
HRUBY K., *Kl. Mbl. f. A.* **134**, 527-531, 1959.
PAUFIQUE L., *Bull. Soc. opht. de France* **1958**, 570-572.
REMKY H., *Kl. Mbl. f. A.* **133**, 616-619, 1958.
WEEKERS R., LAVERGNE G., et STASSART-HOURLAY Cl., *Bull. Soc. Belge opht.* **1958**, 539-543.
ZORAB E. C., *Brit. J. Ophth.* **43**, 202-203, 1959.

RESTORATION OF SEALED OFF FISTULAE AFTER OPERATIONS FOR GLAUCOMA SIMPLEX

BY

H. WEVE, M. D.

Zeist, Nederland

After operations for glaucoma simplex based on Lagrange's principles like Elliot's trephining, Holth's iridencleisis or Preziosi's cauther-perforation not always a lasting subconjunctival filtration is obtained. This is mostly due to the fact that the conjunctiva adheres strongly to the bulbus. This abnormal adhesion is caused by cicatrization of the subconjunctival connective tissue.

There are two categories of such failures:

A—There exists a fistula in the limbus as is shown by a cystlike bulging of the conjunctiva, imitating a filtration cushion, but this cyst is sealed off in all directions by the surrounding adherent conjunctiva.

B—No such bulging formation is developed and we may assume that the original perforation of the bulbus is also closed.

All of us will have experienced that simple repetition of the operation by trephining or by cauterisation after Preziosi is rarely successful.

I found two methods of dealing with such cases, both based on the same principal that after diathermy coagulation cicatrices usually are weak and supple.

Here follows a description of those technics:

A—Cases with a cystlike prominence of the conjunctiva over the spot where anterior chamber has been opened by perforation of the cornea, but where the tension of the eye is nevertheless very high.

1. After anaesthesia of the conjunctiva by installation of drops of novocaine a subconjunctival injection is given of about $\frac{1}{2}$ cc. of hyason (hyafuronidase of N. V. Organon - Oss). We use a fine needle, entering the conjunctiva at a spot where it does not adhere to the bulbus. During the injection the needle perforates further until the region around the cystlike formation is infiltrated. (Fig. 1).

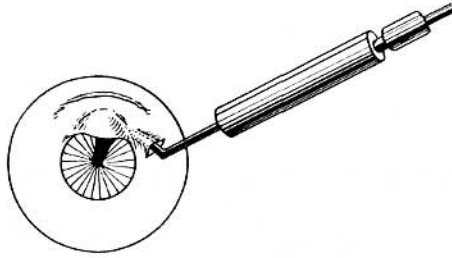


Fig. 1.

2. This follows an injection of 1% novocaine (without adrenaline!). As rule $\frac{1}{2}$ cc. is sufficient.

3. A small incision in the conjunctiva is made at about the same spot, where the needle of the first injection entered in a normal mobile part of the conjunctiva. Through this small opening a special diathermy-electrode is introduced under the conjunctiva and is slowly forced subconjunctivally in the direction of the cyst with wriggling movements, whilst care is taken not to perforate the conjunctiva. The electrode has the shape of a Heine spatula as used in cyclodialysis. (*) Care should be taken that the diathermy current is relatively weak, just sufficient to "assist" the wriggling spatula in undermining the conjunctiva and perforating the cyst, but without causing visible "white" coagulation of the conjunctiva. As soon as the cyst is opened fluid from the anterior chamber is drained off. The opening of the cyst should be made as wide as possible. It is not necessary to close the little incision of the conjunctiva afterwards by a suture, but there is no objection against doing this.

In the next days subconjunctival injection of hyason may be repeated, but I have also seen good results without it.

In my opinion the lasting good results is due to the fact that the surface of the sclera as well as the subconjunctival tissue have been slightly coagulated by the high frequency current and this prevents a quick cicatrization and there by enabling the formation of a lasting subconjunctival fistula.

B—In case there is no cystlike formation another technique is followed. Now a flap of the conjunctiva is prepared from a large incision at about 6 mm distance from the cornea. After this the surface of the bulbus is very weakly coagulated in whole the area by gently rubbing over it a diathermy electrode under

(*) Eventually such a spatula can be used if a thick rubber tube is placed over its steel and an assistant contacts it with a diathermy electrode.

low current. (Fig. 2) Care should be taken to use a weak current as too strong coagulation might damage the eye as a whole by overheating of the vessels. On the other hand one needs not to fear a somewhat stronger coagulation in this area between cornea and ora serrata as it can only assist us in lowering the tension of the eye ("cyclodiathermia"). After this surface coagulation a new opening in the cornea is made either by trephining or by electrocauterisation. The conjunctiva is then closed in the usual manner.

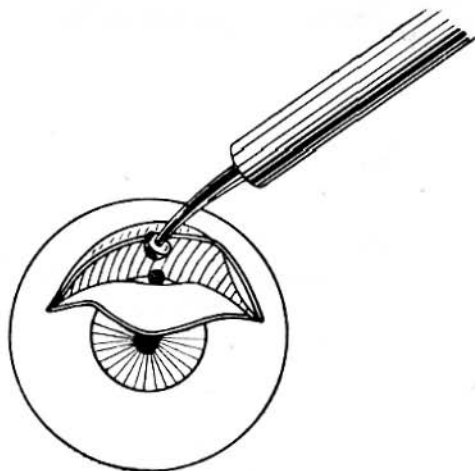


Fig. 2.

In all the cases where I have used this technique the result has been highly satisfactory even to such extent that I intend to use it as standard technique already in the first operation of cases of glaucoma simplex.

Now and then I have observed a zone of "exudative" detachment (or was it a *solutio chorioideae*?) in the coagulated area, but this disappeared spontaneously within one or two weeks.

I will add a remark that may be useful. After perforating operations of the bulbus such as operations for glaucoma, cataract or detachment we usually inject subconjunctivally 25000 units of penicilline + novocaine. These injections cause strong adherence of the conjunctiva to the bulbus and therefore should never be given in the neighbourhood of filtering scars! In case of glaucoma-operations in the upper part of the bulbus we therefore give the injection of penicilline only in the lower part.

For the same reason we also avoid injection of adrenaline in the neighbourhood of filtering scars.

The number of cases treated successfully in the here described way is not large enough to give percentages, but the fact that until now we have mostly had good results seems to justify this preliminary communication of our modification of operations for glaucoma simplex.

I am well aware that this small communication, published to honour a surgeon as Prof. Ignacio Barraquer is not in proportion to his great merits, but as it seems to be of some practical value I dare nevertheless to dedicate it to him as a small token of my great respect for his contributions to ophthalmic surgery.

Utrechtseweg 26

Nuevos Instrumentos
News Instruments

THE PHORO-ACCOMMODOMETER
AND OFFICE INSTRUMENT FOR
ACCOMMODATION AND CONVERGENCE RESEARCH

BY
MERRIL J. ALLEN, O. D.

Bloomington, U. S. A.

At the present time are no available office testing equipment or procedures that can measure the status of accommodation and the simultaneous status of convergence. This paper gives a description of a simple instrument which combines the principles of a Badal and Scheiner optometer with a simple phorometer.

The prototype instrument, figure 1, has been in use for over three years and has proven its practicality for special accommodation and convergence studies on routine clinical patients. It has a 10.00 D. accommodative range and a convergence and divergence range of 30 D. These values have proven satisfactory for accurately obtaining the ACA* ratio by means of phoria measurements without instrument limitations in almost every case.

Figure 2 is a schematic of the optical system utilized in the phoro-accommodometer. A colimator and a Risley prism are mounted before the left eye and are used for measuring convergence. A Badal optometer seen through M by the right eye provides the stimulus to accommodation, and a vernier optometer seen by reflection at M permits the measurement of the accommodative response.

For the left eye, lens L_4 images the vertical filament S_3 at infinity. The Risley prism R, receives parallel light from the lens L_4 and changes its direction in proportion to the power set into the prism. Since the rays are parallel entering and leaving the prism, no interpupillary distance adjustment is necessary as long as the target can be seen within the field of the prism. The light source S_3 is a retinoscope "streak" bulb illuminated only for occasional very brief flashes so that the patient's left eye has no target for the majority of the time and assumes its phoria position.

* The ACA ratio is the amount of convergence change induced by 1 diopter of accommodative change. A value of about 4 prism diopters/1 D. is considered to be the normal ACA ratio.

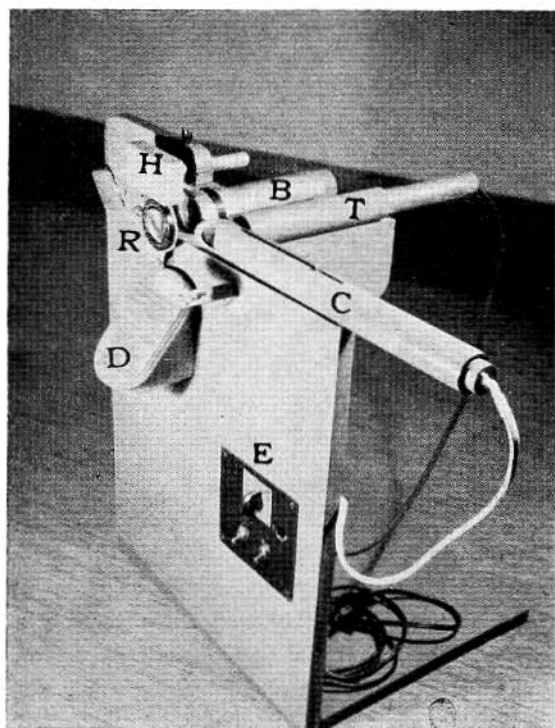


Fig. 1. The Phoro Accommodometer. The parts are as follows: H is an adjustable forehead rest; D is an inclined plane chin rest; R is the Risley prism before the left eye; B is the collimator housing which can be swung upward out of the view of the left eye; T is the target housing which can be swung to the right out of the view of the right eye; C is the housing of the vernier optometer; E is the electrical control panel (placed conveniently for the subject to operate during a special experiment); the base and upright are made of plywood; the metal piece causing a shadow between C and D is a hinged eye positioning device whose position in the photograph has no significance.

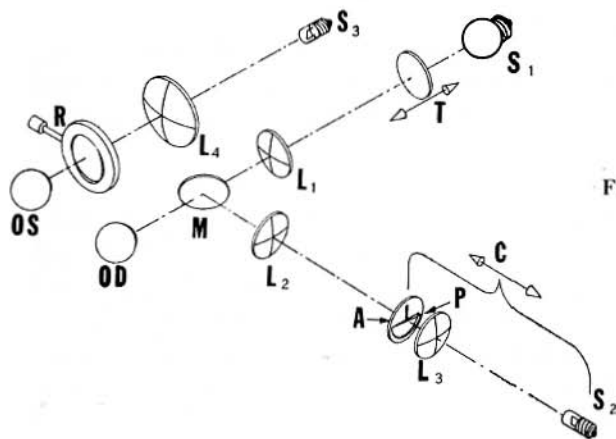


Fig. 2. The Optics of the phoro-accommodometer.

In front of the right eye is a beam splitting mirror, M. Beyond M at 10 cm. from the entrance pupil of the eye is L_1 , a +10.00 diopter lens. Through lens L_1 the patient can see a reduced Snellen letter target, T, illuminated by the light source S_1 .

When T is placed 10 cm. from L_1 , the stimulus to accommodation is zero. T may be moved forward or backward from this position and each centimeter of movement will produce a 1 diopter change in the stimulus to accommodation. Simultaneously the right eye receives light from L_2 reflected from the mirror M. L_2 is + 10.00 D. and is optically 10 cm. from the entrance pupil of the eye.

"A" is a metal disc with a .2 mm vertical slit about 1 cm long cut through it. Cemented to the back side of A is either a by-prism or a single prism as indicated on the figure by the letter P. Immediately behind the slit and prism is lens L_3 which colimates the light from the retinoscope streak bulb S_2 .

The assembly A through S_2 , indicated by C on the diagram, is a vernier optometer which indicates zero diopters accommodative response when A is 10 cm from L_2 . Each centimeter movement away from this point changes the amount of indicated accommodation by 1 diopter. Assembly C is used to measure the accommodation while the patient focuses on the target T seen through the mirror. The patient sees a letter target and also a fine vertical line (dis A) by reflection from M. If assembly C is adjusted to the conjugate focus of the right eye, the vertical line will appear to the patient to be a continuous line. C is not at the conjugate focus of the eye, the patient will see the upper half of the line displaced laterally from the lower half of the line.

Prism P. is a 2 prism diopter base horizontal and works satisfactorily when used as indicated in the diagram. (An optically superior system would be to use an upper 1 prism diopter base left and a lower prism of 1 prism diopter base right. The two prisms must be edged so that the dividing line between them is as thin as possible). The spacing between A and L_3 is not critical. The spacing between L_3 (+ 10.00 D.) and S_2 is 10 cm.

SUMMARY AND CONCLUSIONS

An office type research instrument called a phoroaccommodometer is described. It is a simple, inexpensive instrument with good accuracy for measuring the accommodative response to a stimulus, the accommodative amplitude, the ACA ratio, the effect of convergence on accommodation, instrument myopia, psychic accommodation, psychic convergence and etc.

It is a vernier optometer combined with a Badal optometer and a prism phorometer.

The prototype instrument is in the Division of Optometry and a second instrument is in the office of C. W. Morris, O. D., Fort Wayne, Indiana.

Experience to date has shown the phoro-accommodometer to be a practical means of applying the principles of research haplosopes in an office.

Bloomington, Indiana

TREPANO PARA LA QUERATOPLASTIA EN DOS PLANOS *

POR

JOSE I. BARRAQUER M., M. D.

Bogotá, Colombia

Para la práctica de la queratoplastia penetrante en dos planos tal como fue descrita por el autor en Corneal Grafts de B. W. Rycroft Pag. 98, hemos diseñado un nuevo modelo de trepiano que se adapta a un mango corriente de trefina.

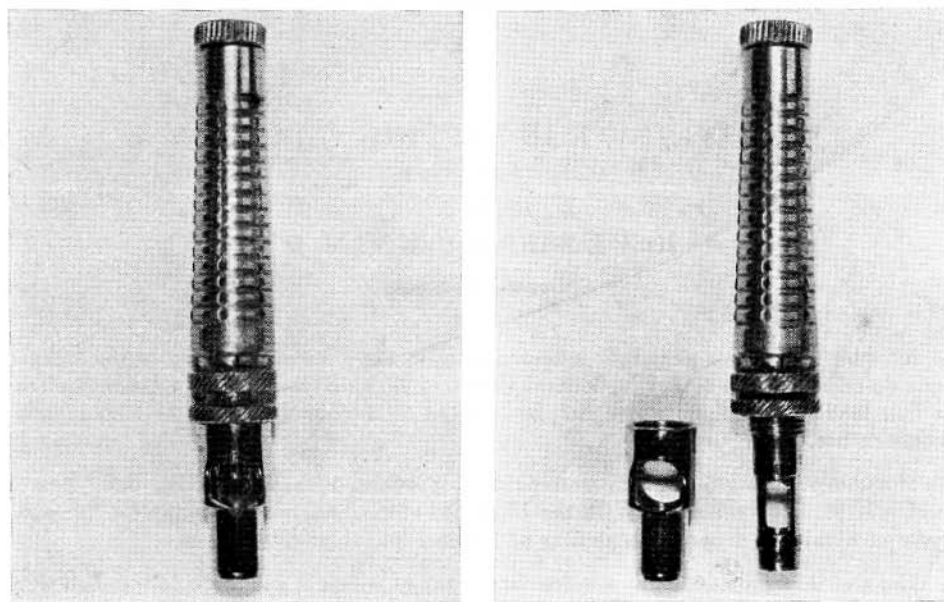


Fig. 1-2. Trépano para queratoplastia en dos planos.
Trepine for Steping Grafts.

* Construído por Moria, 108 Boulevard Saint-Germain, París.

El trepano interior de 5 milímetros cortante va provisto de una guarda exterior perfectamente concéntrica con el de $6\frac{1}{2}$ milímetros no cortante para permitir el perfecto centraje de la incisión de los planos posteriores.

La guarda mediante una rosca puede deslizarse en sentido del eje del instrumento a fin de proteger el filo del trepano cuando éste está en reposo o de permitir que el filo sobresalga 3 o 4 décimas de milímetro en el momento de utilizarlo.

Este instrumento debe utilizarse conjuntamente con un trépano ordinario de $6\frac{1}{2}$ milímetros con pistón interior.

Si bien, las anteriores son las dimensiones más corrientes puede construirse en las dimensiones que se deseen.

La diferencia entre la dimensión del trepano interior y la guía exterior debe ser de $1\frac{1}{2}$ milímetros para los tamaños pequeños y de 2 milímetros para las dimensiones mayores.

Hasta la fecha las dimensiones mayores que hemos empleado con esta técnica han sido de 6,1 milímetros para la sección de las capas posteriores y 8,1 milímetros para la sección de las capas anteriores.

TREPHINE FOR STEPING GRAFTS *

BY

JOSE I. BARRAQUER M., M. D.

Bogotá, Colombia

In order to perform penetrating keratoplasty in two planes according to the technique described by the author in Corneal Grafts of B. W. Rycroft p. 98, we have devised a new model of trephine which can be adapted to the handle of any of trephines in common use.

The sharp cutting 5 mm. inner trephine is provided with a protecting outer guard which is perfectly concentric with the blunt non-cutting $6\frac{1}{2}$ mm. trephine for the purpose of obtaining a perfect centering of the incision of posterior planes.

The guard is provided with a screw by means of which it can slide in the direction of the axis of the instrument for the purpose of protecting the sharp edge of the trephine when this is at rest or else to allow this edge to protrude 3 o 4 tenths of a millimeter at the moment al which it is used.

* Made by Moria, 108 Boulevard Saint-Germain, Paris.

NUEVOS INSTRUMENTOS

This instrument is to be used conjunctly with a $6\frac{1}{2}$ mm. ordinary trephine with an inner piston.

Although the trephine here described is made by the said dimensions, it can be made of any other dimensions, at will.

The difference between the dimension of the inner trephine and that of the outer guard should be $1\frac{1}{2}$ mm. for small sizes and 2 mm. for the larger sizes.

Up to the present time the greatest dimensions we have used with this technique have been 6.1 mm. for sectioning the posterior layers and 8.1 mm. for sectioning the anterior layers.

Apartado Aéreo 11056

NUEVA TIJERA PARA LA TALLA DEL COLGAJO EN LA OPERACION DE CATARATA *

POR

JOSE I. BARRAQUER M., M. D.

Bogotá, Colombia

Para llevar a cabo la incisión en la operación de la catarata en una forma regular y sin necesidad de retirar la tijera de los labios de la herida describí en los "Archivos de la Sociedad Americana de Oftalmología y Optometría" volumen I, pag. 183 una tijera acodada de láminas rectas que tenía la peculiaridad de tener un tope móvil entre sus ramas a fin de que durante la talla del colgajo corneal la

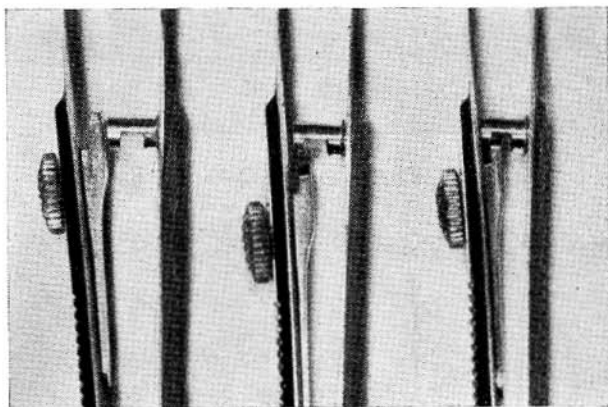


Fig. 1. A: Tope en posición para la talla del colgajo corneal.

Butt in position for cutting the corneal flap.

B: Para la talla del colgajo conjuntival.

Butt in position for cutting the conjunctival flap.

C: Posición del tope para guardar la tijera cerrada.

Butt in position to keep scissors closed.

* Este instrumento es construido por Moria, 108 Boulevard Saint-Germain, París, y E. Franz, Av. José Antonio P. de R., 562, Barcelona.

tijera no cerrase completamente y de esta forma poder tallar el colgajo en una forma continua sin retirar el instrumento.

El modelo que presento hoy tiene las mismas características pero se ha modificado la construcción mecánica del tope (fig. 1).

En la fig. 1-A podemos ver el tope en la posición requerida para la talla del colgajo corneal. En la fig. B la posición para la talla del colgajo conjuntival y en la fig. C para guardar la tijera cerrada.

La figura 2 muestra la tijera de perfil para apreciar la angulación de sus ramas y el dispositivo de tope. La tijera de frente abierta y en la misma posición cerrada.

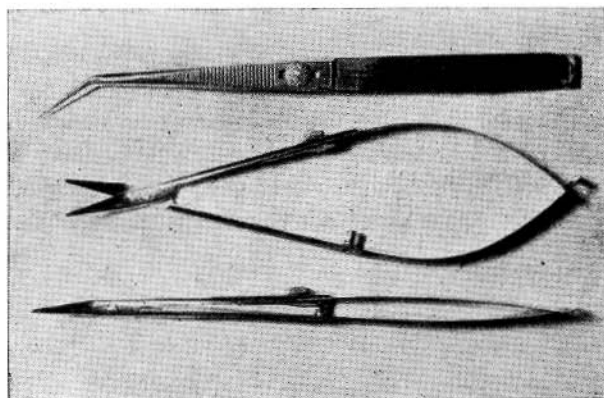


Fig. 2. Tijera para la talla del colgajo en la operación de catarata.

NEW SCISSORS FOR CUTTING THE FLAP IN CATARACT OPERATIONS *

BY

JOSE I. BARRAQUER M., M. D.

Bogotá, Colombia

To accomplish the cutting of a uniform incision in cataract operations, without the need of removing the scissors from the lips of the wound, I described in the "Archivos de la Sociedad Americana de Oftalmología y Optometría" Vol. I, p. 183, an angled scissor which have straight blades and a butt between its handle. This butt, which can be removed at will, prevents the scissors from closing completely at the point during the cutting of corneal flap. In this manner the incision for cutting the flap can be continued throughout its extent without removing the instrument.

The model herein described has exactly the same characteristics as those of the one previously described, but the mechanical construction of the butt has been modified (Fig. 1).

Fig. 1-A shows the butt in the position which is required for the cutting of the corneal flap. Fig. B shows its position during actual cutting of the conjunctival flap and Fig. C shows the position of the scissor when they are closed.

Fig. 2 shows the scissors in a profile position to permit appreciation of the degree of the angle of its blades and the mechanism of the butt. The scissors are shown from the front both open and closed.

Apartado Aéreo 11056

* This instrument is made by Moria, 108 Boulevard Saint-Germain, Paris, and by E. Franz, Av. José Antonio P. de R., 562, Barcelona, Spain.

DIRECCIONES UTILES

- KURT MORCHER**
Reinsgurgstr. 91
Stuttgart-W
Alemania Occidental
Lentes de cámara anterior modelo Danheim.
- W. M. BLOSS**
63, San Eusebio
Barcelona
España
Lente pantoscopio de Cardona para gonioscopia y examen de fondo.
- LEAL**
Arzobispo Apaolazza 25
Zaragoza
España
Instrumental quirúrgico oftalmológico.
- OERTLI**
Zylistrasse 8
St. Gallen
Suiza
Instrumental quirúrgico e implantes de Nylon según Bangerter.
- FISBA**
Rorschacherstrasse 19^a
St. Gallen
Suiza
Pleöptophor.
- MAW'S**
Aldersgate Hause
Bernet-Hertfordshire
Inglaterra
Zonulysin Alfa Quimotripsina para uso oftálmico.
- P. E. E. Y. V. A.**
Molins de Rey
Barcelona
España
"Quimotrase" Alfa Quimotripsina para uso oftálmico.
- PFORTNER**
Juncal, 2345
Buenos Aires
Argentina
Lentes de contacto,
Ojos artificiales.
- CARL ZEISS**
Oberkochen/Wurt
Alemania
Aparatos oftalmológicos.
- RAYNER & KEELER**
100, New Bond Street
London W. I.,
Inglaterra
Lentes para cámara anterior.

DIRECCIONES UTILES

MAISON MORIA
108, Boulevard Saint-Germain
París—Vle.
Francia

Instrumental quirúrgico.
Tijera con tope, hemiqueratomy y pinza colibrí según
J. I. Barraquer M.

OCULUS:
Dutenhofen (Weta-lar)

Aparatos par exploración, tonómetro de Aplanación
de Goldman.

FRANCISCO YBAÑEZ (REMDIX)
Casasnovas, 44
Barcelona

Aparatos electromédicos. Seda virgen. Erisífaco Barraquer. Pinceles de pelo de marta. Implantes para enucleación.

	<i>Página</i>
SCHMIDT INGEBOG, M. D., <i>Vision and Astronautics</i>	291
SHAPLAND C. DEE, M. D., <i>The Vitreous Implant</i>	299
STREIFF E. B., M. D., <i>L'extraction intracapsulaire chez les Petits Enfants et les Enfants a l'aide de L'alpha-chymotrypsine</i>	305
WEVE H., M. D., <i>Restoration of sealed off Fistulae after Operations for Glaucoma Simplex</i>	309
NUEVOS INSTRUMENTOS — NEWS INSTRUMENTS	313
DIRECCIONES UTILES	325
SUMARIO	327



Electro - Keratome for the Dissection of Lamellar Grafts

RAMON CASTROVIEJO, M.D.

Procedure is shortened, and minimally traumatized grafts of even thickness and as large as permitted by the donor's eye are obtained. This result was difficult to achieve by older methods, especially with larger grafts.

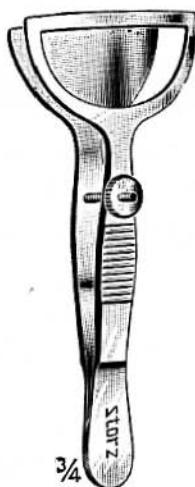
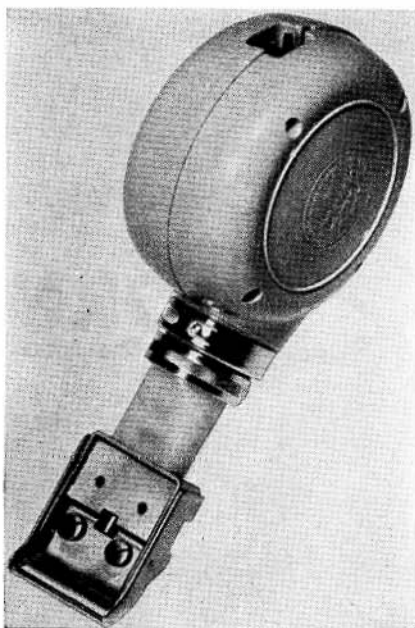
- * Small enough to observe the donor eye for which it is used only.
- * Edge of cutting blade visible through open roller guards, enabling surgeon to start and complete graft at exact margins.
- * Uses small, disposable, double-edged razor blade. Simple mechanism.
- * Shims are provided in thicknesses of 0.1, 0.2, 0.3, 0.4 and 0.5 mm., permitting graft from 0.1 to 1.5 mm. thick. This method so accurately controls the thickness that two grafts of 6 mm. diameter have been removed from a single donor eye.

Instrument also useful for cutting buccal mucous grafts for plastic repair in cases of symblepharon or extensive scarring of the conjunctiva. The mucous membrane can be taken from the buccal surface of lip or cheek. It cuts the grafts so thin that they need no further thinning, which was impossible with previous instruments.

Head of instrument is Stainless Steel. Motor is rotary type, AC or DC, 110.

E-2950—Keratome, CASTROVIEJO: head only with one dozen blades, Stainless Steel \$ 150.00
Foot Switch 14.50
Norelco Motor 11.50

Complete \$ 176.00
E-2952—Extra Blades for E-2950, dozen 1.50

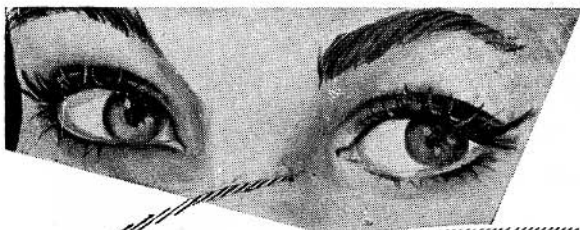


E-2530 — Lip Plate, CASTROVIEJO:

used in connection with the Electro-Keratome for cutting of buccal mucous grafts . . . \$ 17.50

STORZ INSTRUMENT COMPANY - 4570 Audubon Ave., St. Louis 10, Mo.

New York Showroom
809 Lexington Ave.



Una garantía de
perfección en
lentes de contacto
y ojos artificiales.

CORNEALENT

el lente de contacto de mayor tolerancia



OJOS ARTIFICIALES
GONIOLENT - LENTES-GOLDMAN
LENTES ILLIG
IMPLANTES PARA ENUCLEACIONES
CAJAS DE PRISMAS
ANTEOJOS PARA AMBLIOPIA
PRISMA BEHRENS

laboratorios

Pförtner

Juncal 2345 - T. E. 84-2410
Buenos Aires - República Argentina

ra-ra published

8 importadores
colizamos
precios especiales

Lentes de Contacto Semi-terminados

Ofrecemos stock de Lentes de Contacto semi-terminados en todos los radios de curvatura y dioptrías, perfectamente tallados.

Los lentes vienen listos para ser llevados al diámetro por Ud. requerido.

Independiéndose de las esperas del laboratorio brindando un servicio de 24 horas a sus pacientes y colegas.

Usted ganará prestigio, tiempo y dinero.

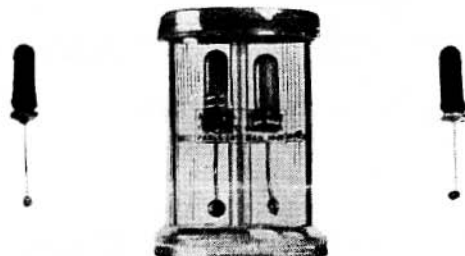
Venta mínima 500 blocks.

También suministramos Lentes de Contacto terminados en cantidad, al diámetro requerido.

Descuentos especiales por mayores cantidades.

LABORATORIOS FOCUS VL
EMILIO MITRE 51
BUENOS AIRES — ARGENTINA

INFORME SOBRE LOS ERISIFACOS DE SANTALICES Y GUILLAUMAT



GUILLAUMAT
Erisifaco

SANTALICES
Erisifaco

Estos dos tipos de erisifacos para extracción total son el producto de las investigaciones de GUILLAUMAT (París) y SANTALICES (Madrid).

En la actualidad estamos en capacidad de suministrar los dos tipos (cada uno tiene sus ventajas).

GUILLAUMAT (Erisifaco)—Copa de presión poco profunda (menor riesgo de romper la cristaloides) SUCCION PODEROSA.

SANTALICES (Erisifaco)—Copa de punta roma que facilita el paso a través de la pupila.—SUCCION MEDIA.

Las peras de succión son iguales en los dos tipos. La superficie externa de las copas ha sido cuidadosamente terminada.

ESTERILIZACION—Se obtiene mediante inmersión de los instrumentos en líquidos o atmósferas bactericidas. Formol, amonio cuaternario, cetavlon, biocidan, etc.

Podemos suministrar un recipiente especial elaborado de plexiglas para la esterilización mediante líquidos, de los modelos anunciados.

MAISON MORIA - 108, BOULEVARD SAINT GERMAIN - PARIS - VIe - FRANCIA

Contact lenses should be part of your professional wardrobe

THE USES of Contact Lenses have become varied and many, and the ever widening use of Contact Lenses is due, without question, to the growing body of research and techniques in which the Plastic Contact Lens Company has been an important force.

Research, Quality, Service in Contact Lenses—Accepted by the
Ocular Professions

USES OF CONTACT LENSES:

- | | |
|--|---|
| As a Diagnostic Aid | Telescopic Contact Lens Fitting |
| Aphakia | To Improve Appearance |
| a. Monocular | Sports |
| b. Binocular | Occupational |
| Keratoconus | Iris Color Change |
| Irregular Cornea | Color Contact Lenses |
| Scarred Corneas | Special Uses of Contact |
| Cosmetic Contact Lens Fitting To Scarred and Unsightly Eyes. | Lens as in Gonioscopy, X-Ray Treatment, Skin Diving, etc. |

Write Department F. for your copy of the W/J Instructional Manual—without obligation.



THE PLASTIC CONTACT LENS COMPANY 59 E. Madison St., Chicago 3, Ill.
RECOGNIZED AUTHORITIES • PRACTICAL • LATEST TECHNIQUES • CLINICS

ARCHIVOS
DE LA
SOCIEDAD AMERICANA
DE
OFTALMOLOGIA Y OPTOMETRIA

— PUBLICACION TRIMESTRAL —

Disponemos de algunos ejemplares del Volumen Primero (1958)

Colombia: \$ 60.00
Extranjero: U.S.A. 10.00

Números atrasados sueltos:
Colombia: \$ 17.00
Extranjero: U.S.A. 2.50

Suscripción Anual:
Colombia: \$ 50.00
Extranjero: U.S.A. 8.00

Para pedidos y suscripciones dirigirse a la Secretaría:
Apartado Nacional 700 - Chapinero, Bogotá, Colombia