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SUMARIO

Págs.

<i>Bening choroidal folds: a difficult aethiological problem to asses</i> L. Barca - G. Vaccari - F. Passani - S. Esente	123
<i>Estudio clínico de la eficacia terapéutica del Tobrex</i> Carmen Barraquer	133
<i>Long term results of myopic keratomileusis - 1982</i> José I. Barraquer	137
<i>Tratamiento quirúrgico del glaucoma trabeculectomía con iridencleisis intraescleral</i> Angel Hernández - María E. Bardeci de Zeman	149
<i>Development of the main electroretinographic components in premature infants during the first weeks of life</i> B. Ricci - B. Falsini - P. Valentini - F. Lacerra - F. Molle - L. Rufi	159
<i>Fracturas del piso orbitario</i> Federico Serrano - Alberto Zeman	167

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BENIGN CHOROIDAL FOLDS: A DIFFICULT AETHIOLOGICAL PROBLEM TO ASSESS

BARCA L.* VACCARI G.* PASSANI F.* ESENTE S.*

SUMMARY

Benign choroidal folds present a controversial aethiological problem.

The authors refer about 6 patients with choroidal folds of unknown origin that failed to improve after treatment. Particular emphasis is given to the ultrasonographic findings, especially in relation to the differential diagnosis of secondary or idiopathic choroidal folds.

The authors accept the possibility that idiopathic choroidal folds, not improved with treatment, have to be related to a subclinic posterior scleritis.

INTRODUCTION

Nettleship¹ was the first to recognize, in 1884, the involvement of Bruch's membrane and choriocapillaris in what had previously been considered to represent atrophy secondary to papillitis.

The clinical picture was one of retinal striae, with light and dark lines radially disposed from the optic disc towards the macula. Various authors have subsequently reported choroidal folds.

Walsh (1947)², Reese (1953)³ included illustration of choroidal folds in their publications, without comprehensive discussion of their significance. In 1959, Hedge and Leopold⁴, presented 5 cases, derived from a search of the existing

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literature, and a further 2 cases which they had observed among their own patients.

Prior to 1960, choroidal folds were considered characteristic though not invariable indicators of expanding orbital lesions. It was not until 1969 that the matter of choroidal folds received due attention and became subject of full discussion.

Norton (1969)⁵, Kroll and Norton (1970)⁶ described 10 cases of choroidal folds of which only 4 were associated with tumors.

In 1972 Gass⁷ described choroidal folds secondary to hipotony.

In 1973 Newall⁸ reported 16 cases of choroidal folds of various aetiologies: 8 due to intra orbital tumors and 8 due to the following: Graves disease, post extraction hipotony, unilateral papilloedema, senile macular degeneration, hypermetropia and uveitis.

In 1979 Bullock and Egbert⁹ reported 11 cases and described experimental findings, including histopathology in animals. In this series, only 7 were associated with intra orbital tumors, the others with hipotony, retinal detachment and papilloedema.

In 1977 Cappaert et al.¹⁰ described 6 cases of which 2 were ascribed to acute posterior scleritis but no apparent cause was found in the remaining 4.

The aim of this paper is to describe 6 cases of choroidal folds, which failed to improve after treatment, out of the 10 we observed in 1982.

Particular emphasis is given to the ultrasonographic findings; the role of ultrasonography in the differential diagnosis with respect to folds secondary to intra orbital tumors and those thought to be idiopathic will be discussed.

AETHOIOLOGICAL THEORIES

Many authors have discussed the aetiology of choroidal folds, but no single theory has emerged.

Some causes have been eliminated, including abnormal scleral rigidity, previous choriocapillaris disease, exudative detachment of choroid, pigment epithelium or sensory retina, inner limiting membrane or vitreous.

BENIGN CHOROIDAL FOLDS: A DIFFICULT AETHIOLOGICAL PROBLEM TO ASSESS

The theory currently favoured now suggest there is an abnormal adhesion of Bruch's membrane to the underlying choriocapillaris with a relative reduction of its normal elastic properties. This explains why choroidal folds are associated with such a diversity of ocular pathologies.

Any condition which can cause thickening of the choroid or the underlying sclera can cause choroidal folding in the presence of abnormal adhesion between Bruch's membrane and choriocapillaris.

The pigment epithelium passively follows the new contours of Bruch's membrane while the choroid becomes more or less thickened corresponding with peaks and valleys to the folds.

In the absence of these abnormal adhesions Bruch's membrane and the pigment epithelium slide easily over the choriocapillaris as determined by their elasticity, thus, choroidal folds are not seen in association with choroidal congestion or scleral thickening of whatever cause.

Choroidal folds have been described in the following conditions: primary or secondary orbital tumors, orbital pseudo tumors, orbital cellulitis, dysthyroid exophthalmos, posterior scleritis, choroidal malignant melanoma, metastatic choroidal tumors, endophthalmitis, uveitis, phthisis bulbi following scleral laceration or longstanding detachment, severe post operative hipotony, papilloedema, extreme hypermetropia and disciform degeneration of the macula.

CASE REPORT

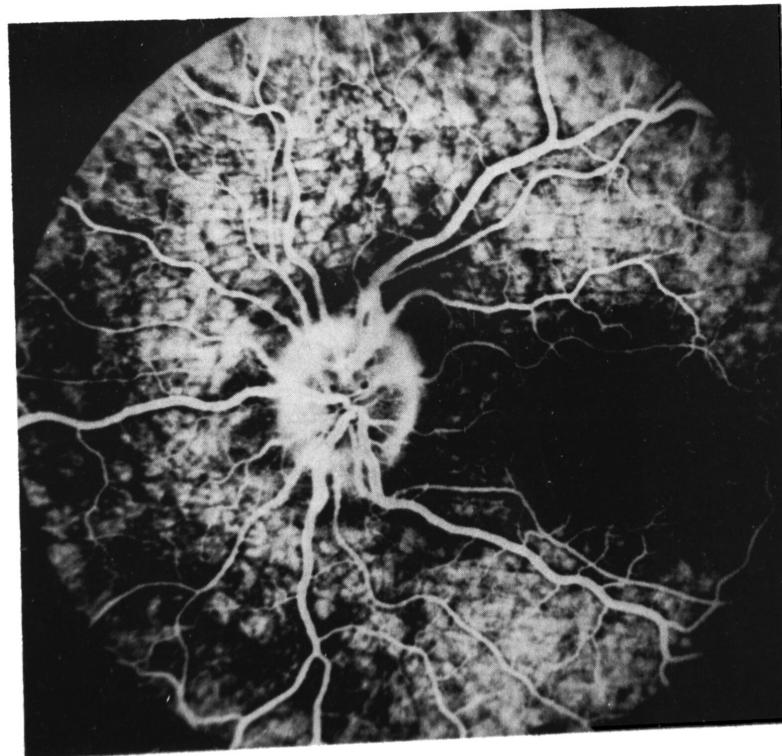
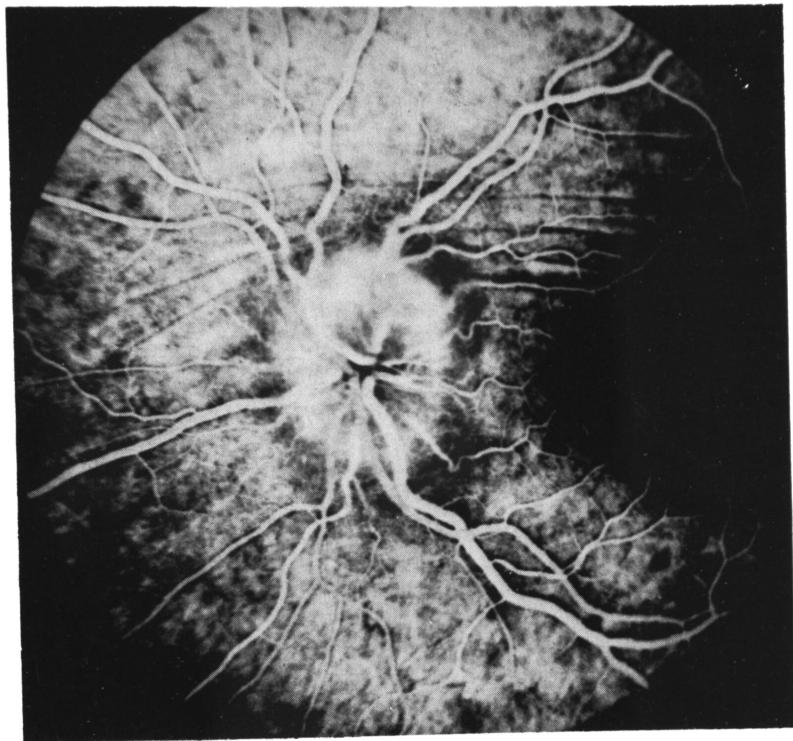
In 1982, 10 patients with choroidal folds were reported to our Department, after a routine eye examination for further testing.

Using the fluorescein angiography the choroidal folds appeared like light and dark lines localized principally to the posterior pole, the lines were virtually parallel but various in lenght and width. (Figs. 1, 2).

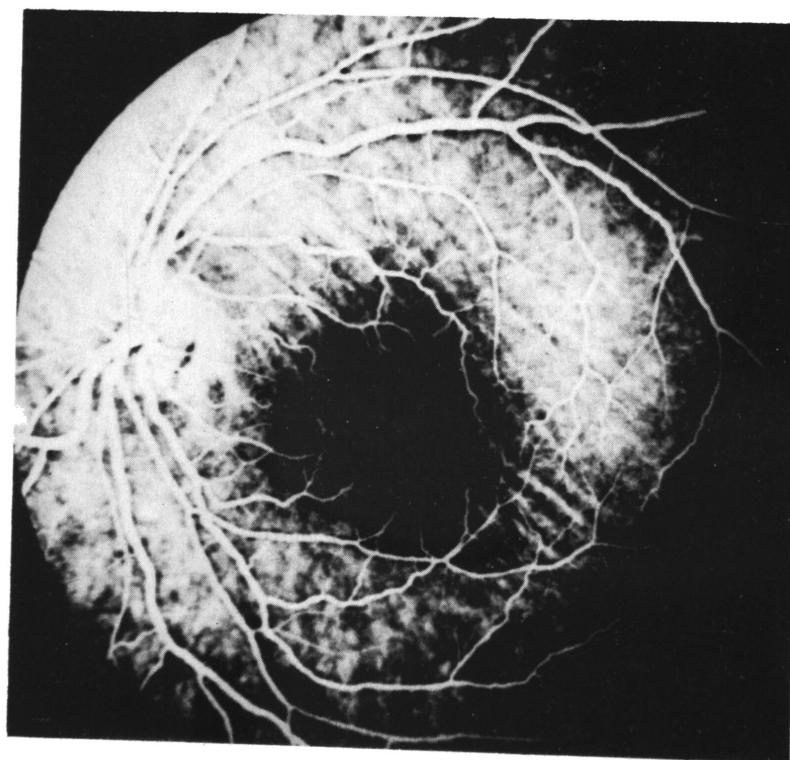
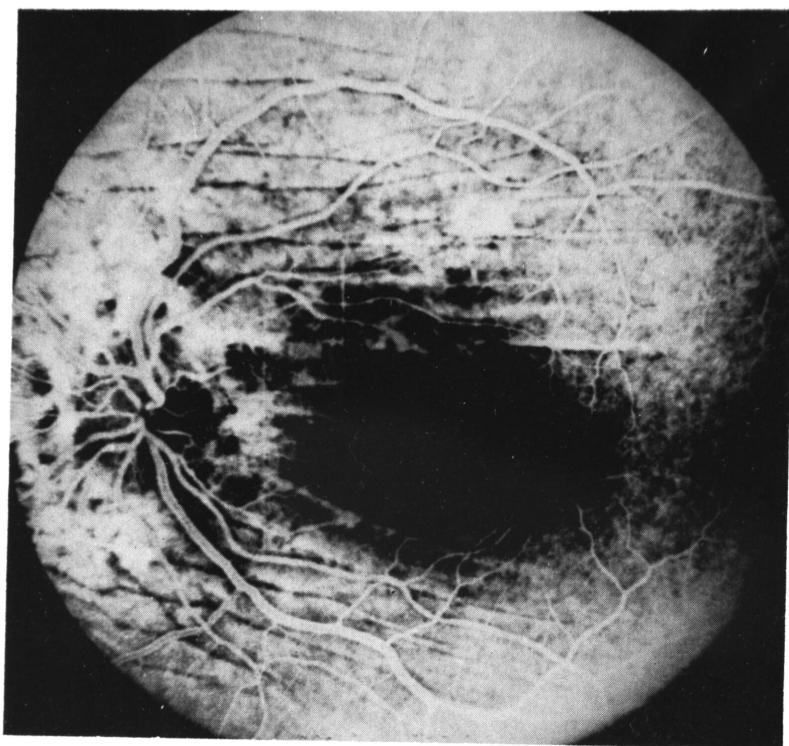
Horizontal, vertical and oblique choroidal folds were present. (Figs. 3, 4, 5). No leakage of contrast medium was present. A rapid B-contact scan was performed allowing resolution of the retino choroido scleral envelope.

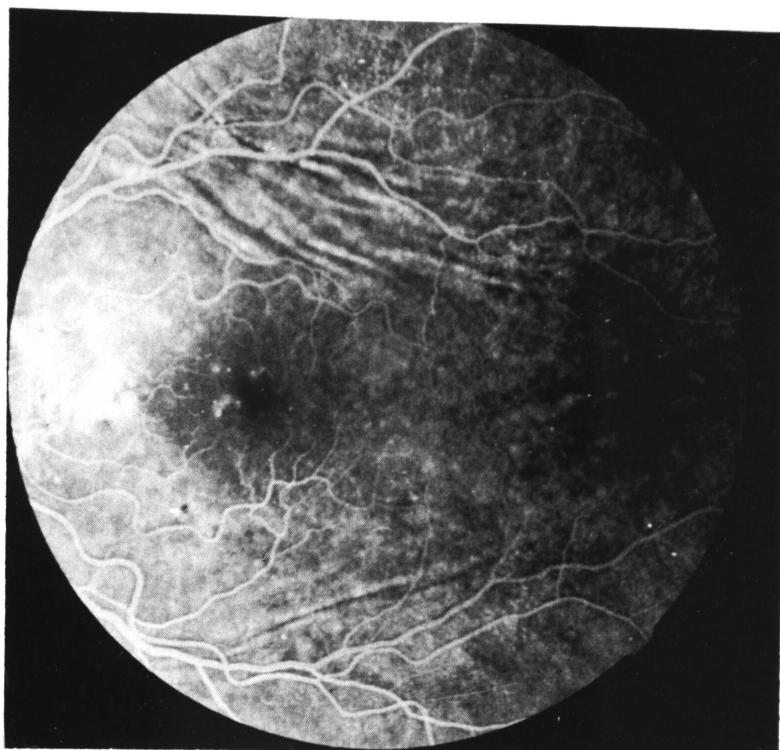
This was distinct from the retrobulbar fat and was of measurable thickness. The posterior pole, in the presence of choroidal folds appeared ultrasonographi-

BARCA L. - VACCARI G. - PASSANI F. - ESENTE S.



BENIGN CHOROIDAL FOLDS: A DIFFICULT AETHIOLOGICAL PROBLEM TO ASSESS





cally to have flattening of the normally curvilinear aspect of the retroequatorial retina. (Figs. 6, 7).

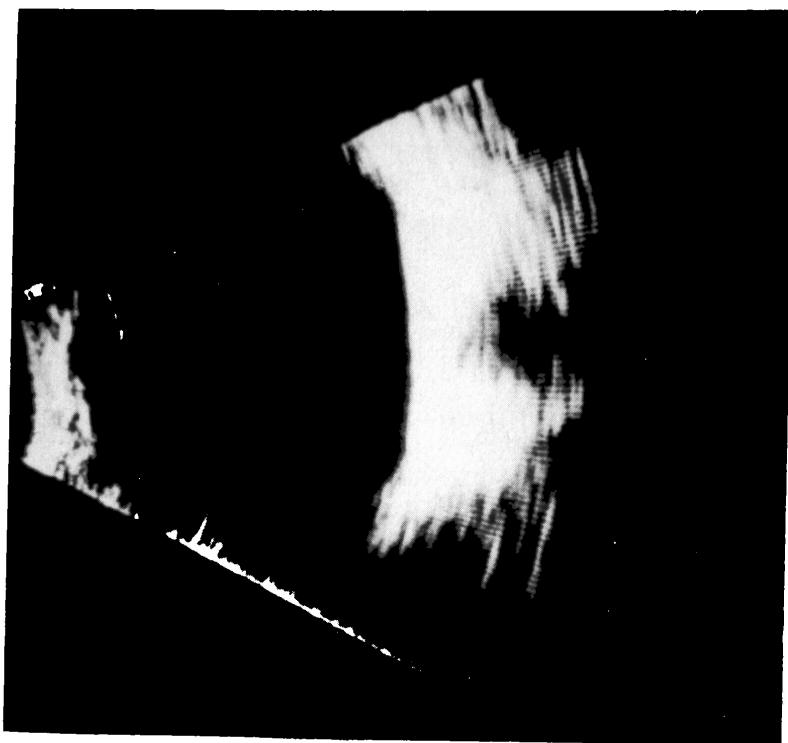
In addition, there was a concomitant thickening of the choroido sclera. It was generally impossible to specify whether the observable thickening seen echographically was due to the choroid, sclera or to both structures together.

It was, however possible to differentiate through the B-scan examination the appearance of localised orbital inflammation, as with acute posterior scleritis, or more diffuse inflammation, as with pseudo-tumor, or the presence of a concomittant orbital tumor.

In 6 of the 10 patients it was impossible to evidence any of these alterations; they were treated with systemic steroids according to the usual schemes, but no improvement were noticed.

Of the 4 other cases of choroidal folds we treated, one was associated with an orbital tumor that was removed, and the remaining 3 involved a papilloedema and improved after treatment.

BENIGN CHOROIDAL FOLDS: A DIFFICULT AETHIOLOGICAL PROBLEM TO ASSESS



CONCLUSION

Choroidal folds are often missed at routine ophthalmological examination as signs of ocular pathology.

Accurate fundus examination using the binocular indirect ophthalmoscope or the Goldmann lens with Slit-lamp, should reveal choroidal folds if present. Where there is doubt, fluorescein angiography can demonstrate the typical light and dark lines which run horizontally or vertically in the retroequatorial retina.

It is thus possible to identify choroidal folds using the fore mentioned techniques.

Having diagnosed choroidal folds, the concomitant pathology should be determined as it is only in rare cases that no ocular or orbital cause is recognised. To this end, the use of the contact B-scan is of great value not only demonstrating and localising choroidal folds, but also to exclude or confirm the presence of a solid mass or inflammatory process of globe or orbit.

Our findings support the concept of scleral shrinkage causing benign choroidal folds, when no causal pathology is recognised, as first proposed by Norton 1969⁵.

Ultrasonic evidence supports the hypothesis that acute phase of scleritis can cause choroidal folds associated with retro scleral inflammation signs.

Chronic, mild, asymptomatic, inflammation of posterior sclera, clinically undetected, can, on the other hand, cause choroidal folds without any other associated finding. In this occasion, any treatment fails in changing ophthalmoscopic, angiographic and echographic appearance as it is happened in our patients.

For this reason we tend to accept the possibility, proposed by Cappaert et al. (1977)¹⁰, that, excluding any causal aethiology and having failed any treatment, the choroidal folds have to be related to a subclinic posterior scleritis.

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ESTUDIO CLINICO DE LA EFICACIA TERAPEUTICA DEL TOBREX*

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La Tobramicina es un antibiótico del grupo de los aminoglucósidos, soluble en agua y activo *in vitro* contra gran número de gérmenes tanto Gram (+) como Gram (-) *Estafilococos*, *Estreptococos*, *Escherichia coli*, *Klebsiella*, *Pneumoniae*, *Enterobácter*, *Aerogenes*, *Proteus Mirabilis* y otras especies de *Proteus*, *Hemofilus Influenzae* y *H. Aegyptius*, *Moraxella*, *Lacunatta*, *Salmonella*, *Shigella* y algunas *Neisserias*. Su capacidad de inhibición del crecimiento bacteriano "in vitro" de algunas especies de *P. Aeruginosa*, es de 2 a 4 veces mayor que la del Sulfato de Gentamicina; sin embargo, en los estudios clínicos realizados en humanos se ha comprobado que la eficacia es muy semejante entre los dos productos.

Por ser un antibiótico nuevo en nuestro medio, llevamos a cabo un estudio sobre la eficacia terapéutica de la Tobramicina en infecciones oculares superficiales poniendo especial énfasis en sus reacciones adversas.

METODO

Se llevó a cabo una evaluación clínico-terapéutica de la eficacia y efectos colaterales de la Tobramicina 0.3% solución oftálmica.

Se incluyeron en el estudio pacientes con diagnóstico de inflamación ocular superficial aguda, de origen presumiblemente infeccioso. A todos se les clasificó de acuerdo a un protocolo y a todos se les inició el tratamiento únicamente con la droga en estudio.

* Este estudio se llevó a cabo por solicitud de Laboratorios ALCON de Colombia, en el Instituto Barraquer de América.

El antibiótico fue administrado por 10 días, indicándole al paciente de aplicarse dos gotas en el ojo afectado cada dos horas durante las primeras 48 horas y luego dos gotas cada cuatro horas durante los ocho días siguientes mientras estuviera despierto.

Los pacientes fueron examinados los días 1, 3, 5, 7 y 11. Si durante el curso del tratamiento la infección empeoraba o se presentaban reacciones colaterales, el paciente podía ser excluido del estudio a juicio del médico tratante para instalar otro tratamiento que fuera más indicado.

La evaluación clínica se llevó a cabo por medio de cambios en los parámetros anotados en el protocolo al comienzo del estudio: SINTOMAS (incomodidad, dolor ocular, lagrimeo, fotofobia y alteración de la agudeza visual). SIGNOS (Eritema y edema parpebral. Hiperhemia conjuntival parpebral y bulbar. Secreción o exudación conjuntival. Afección epitelial de la córnea. Infiltrados estromales en la córnea).

RESULTADOS

Se incluyeron en el estudio 50 ojos con diferentes grados de inflamación, pero ninguno de ellos presentaba compromiso corneal. Solo dos ojos pertenecientes a un mismo paciente tuvieron que ser excluidos del estudio por falta de respuesta al tratamiento al tercer día. Los 48 ojos restantes (96%) curaron satisfactoriamente en el período de tiempo pre-establecido.

Mejoría clínica

En todos los casos para el 5º. día la secreción y exudación conjuntival junto con el edema parpebral habían desaparecido; los pacientes solo acusaban incomodidad y algo de lagrimeo debido a la hiperhemia conjuntival residual.

Esta hiperhemia fue considerada como signo residual del proceso inflamatorio sufrido y habiendo desaparecido la exudación se decidió administrar a los pacientes un anti-inflamatorio local. El (90%) de los ojos, es decir, 44 ojos fueron considerados clínicamente curados al 7º día y los 4 restantes estaban curados para el día 11.

ESTUDIO CLINICO DE LA EFICACIA TERAPEUTICA DEL TOBREX

Reacciones adversas

Exceptuando los dos ojos que no respondieron al tratamiento, no pudimos observar ningún tipo de reacción adversa a la Tobramicina.

CONCLUSIONES

Por los resultados obtenidos en este estudio podemos considerar que la Tobramicina 0.3% es un antibiótico eficaz y seguro en el tratamiento de infecciones oculares externas. En nuestra experiencia se requirió de la ayuda de un anti-inflamatorio local para acelerar el proceso de curación. No pudimos observar ninguna reacción alérgica al producto.

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4. Monografía de la casa ALCON sobre TOBREX (Tobramicina 0.3%).

LONG TERM RESULTS OF MYOPIC KERATOMILEUSIS - 1982

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This paper presents the statistical results of 23 myopic keratomileusis procedures (K.M.M), performed in 19 patients, with an average followup of 9.29 years (range: 2 - 20.75 years). Since the statistics were gathered from patients who came voluntarily for followup controls between July 2 and December 18, 1982, they represent a random study.

In table I, the cases are numbered by the order in which they were examined. The average results of this table show that although a correction of -9.60 D was requested for the correction of an ametropia of -9.74 D (0.14 D of undercorrection), 1 month postoperatively there was an overcorrection of 0.06 D. This means that there was an overcorrection of 0.08 D in relation to the correction requested. Between this time (1 month postop) and the end of the first postoperative year, the correction obtained was reduced 15.42%, due to the reduction of the edema along the edges of the lenticule.

After the first year, the correction was more stable. The study shows that at the end of the average followup period (9.29 years) the progression of the myopia was 1.85 D; in other words, 0.22 D per year (2.02%).

On the other hand, the non operated eye had an average myopia of -0.63 D, which progressed 0.12 D per year (19.56%) (Table V).

Contrary to what happens in the short term results (2), in the long term results the visual acuity improves in cases of relative amblyopia as well as in those with satisfactory preoperative vision. Table I shows the cases of the study, classified by preoperative visual acuities higher than and lower than 0.40. Here, we can see

Table I

RESULTS OF KERATOMILL FUSIS FOR THE CORRECTION OF MYOPIA

FIVE A B

PREOPERATIVE

C. R. #	Case #	Age	Sph / equiv	C / VA	De request.	Sph / equiv	C / VA	Sph / equiv	C / VA	Years	Sph / equiv	C / VA
PREOPERATIVE												
1	2	3	4	5	6	7	8	9	10	11	12	13
272533	1	36.00	-9.75	0.50	-12.00	+5.75	0.40	+2.50	0.45	4.00	+1.75	0.67
272533	2	36.00	-9.25	0.67	-9.72	-1.25	0.50	-2.75	0.67	2.75	-1.25	0.67
251928	3	8.00	-5.75	0.10	-5.75	0.00	0.62	0.00	0.62	13.00	-2.00	0.67
120890	4	6.00	-6.37	0.80	-7.00	+3.25	0.45	+1.13	0.80	5.00	+0.75	1.00
881113	5	12.00	-7.25	0.10	-8.00	+3.67	0.50	-0.25	0.40	6.25	-1.50	0.40
55344	6	42.00	-8.00	0.70	-8.50	0.00	0.67	-0.25	0.67	11.42	-1.50	0.60
129355	7	38.00	-12.67	0.33	-11.00	+0.25	0.20	+0.13	0.50	3.75	+0.25	0.50
129355	8	38.00	-15.50	0.33	-13.00	-0.87	0.30	0.00	0.20	3.75	+1.25	0.25
282061	9	25.00	-8.62	1.00	-8.00	+2.37	0.80	+1.25	1.00	2.25	+2.50	0.80
282061	10	25.00	-13.25	0.80	-11.48	+1.00	0.67	+0.25	1.00	2.83	-1.75	0.80
54522	11	17.00	-5.50	0.67	-5.25	+1.25	0.40	+1.75	0.40	12.42	+0.50	0.50
60036	12	12.00	-8.25	0.15	-8.00	-3.25	0.15	-2.00	0.30	17.75	-7.36	0.33
209551	13	6.00	-12.75	0.05	-14.00	-4.50	0.05	-4.50	0.50	20.75	-5.12	0.40
261081	14	9.00	-6.00	0.25	-7.50	-0.25	0.29	-2.38	0.50	8.25	-5.75	0.50
256464	15	16.00	-13.67	0.40	-11.00	-0.75	0.50	-7.50	0.67	11.00	-12.00	0.45
203327	16	6.00	-7.50	0.20	-11.00	-0.12	0.15	-3.37	0.67	11.00	-3.75	0.80
282844	17	37.00	-18.37	0.10	-15.00	-0.37	0.50	-0.50	0.67	2.58	-0.62	0.67
206538	18	17.00	-15.75	0.10	-17.00	-7.37	0.10	-7.00	0.10	11.50	-14.25	0.10
85247	19	14.00	-4.50	0.33	-4.50	+0.87	0.33	+0.50	0.67	8.00	+0.50	1.00
203330	20	20.00	-6.75	0.29	-6.26	+0.25	0.50	-1.75	0.80	13.00	-7.25	0.80
63119	21	23.00	-13.00	0.40	-11.87	-0.75	0.40	-5.75	0.50	15.25	-11.37	0.70
63119	22	23.00	-10.75	0.25	-10.00	-0.25	0.40	-4.00	0.80	14.67	-8.12	0.80
63532	23	20.00	-4.75	1.00	-5.00	-0.25	1.00	+0.25	1.00	12.50	-0.75	1.00
AVERAGES		21.31	-9.74	0.41	-9.60	-0.06	0.34	-1.49	0.60	9.29	-3.34	0.63
With VA < 0.4		18.46	-10.14	0.20	-10.08	-0.92	0.31	-1.93	0.52	10.33	-4.13	0.56
With VA > 0.4		24.60	-9.22	0.69	-8.98	+1.06	0.58	-0.91	0.72	7.94	-2.31	0.72

that the group with preoperative visual acuities higher than 0.40 had an average preoperative visual acuity of 0.69, which increased to 0.72 at the end of the followup period, and that the group with preoperative visual acuity lower than 0.40 had an average vision of 0.20, which increased to 0.56 at the end of the followup period.

These findings confirm our previous belief in the fact that the improvement of the visual acuity persists during the postoperative period, even in spite of a myopia increase, so long as there are no myopic sclerocoroidosis lesions. These, however, have not been observed in any of the cases included in the present study.

The statistics of this study are really a complement of those gathered in 1980 - 1981 (2). At that time, the visual acuity of cases with a preoperative vision lower than 0.40 improved with the surgery, and the decrease in visual acuity of cases with a preoperative vision higher than 0.40 was 0.15, 8.7 months postoperatively and 0.04, 14.63 months after the surgery. The present study shows the same results in cases with preoperative vision lower than 0.40, but a definitive average improvement in cases with more than 0.40, at the end of the 9.29 years of followup.

In the present series, the evolution of the visual acuity was:

	Uncorrected	Corrected
Preop	0.03	0.41
1 month	0.25	0.43
1 year	0.29	0.60
9.29 years	0.27	0.63

Table II shows the cause for the myopia progression in the eyes operated. Here we see that at the end of the followup period the axial length of the ocular globe increased 1.72 mm. and the corneal curvature became 0.13 mm. steeper in patients under 21 years of age, while in older patients the axial length increased only 1.02 mm. and the corneal curvature remained stable.

The general average shows that during the 9.29 year followup the axial growth was 1.40 mm. and the corneal steepening was 0.44 mm. When converted into diopters, these data show that 2.72 D were due to axial growth, while only 1.85 D were due to corneal steepening (in other words, by loss of surgical effect). The distribution of these figures by the yearly evolution shows that 0.20 D per year were due to a loss of correction and 0.29 D per year to an increase of the axial length. This, again, agrees with previous statistics¹. However, the fact that

Table II

K M M 1982 - GROUPED BY AGE

CR #	Case #	Age	Axial length	RF obtained (1 year)	Followup (years)	RF	Axial length	Change in RF	Axial growth	Ametropia	
										Preop	Postop.
120890	4	6	25.10	9.25	5.00	9.72	26.16	+0.47	1.06	-6.37	+0.75
209551	13	6	24.00	8.17	20.75	8.00	25.75	-0.17	1.75	-12.75	-5.12
203327	16	6	24.00	9.35	11.00	9.25	28.10	-0.10	4.10	-7.50	-3.75
251928	3	8	23.50	8.33	13.00	8.67	24.00	+0.34	0.50	-5.75	-2.00
261081	14	9	23.25	8.18	8.25	7.98	25.74	-0.20	2.49	-6.00	-5.75
600326	12	12	---	8.40	17.75	8.00	---	-0.40	---	-8.25	-7.76
881113	5	12	25.50	9.00	6.25	8.90	25.98	---	0.48	-7.25	-1.50
85247	19	14	22.00	9.00	8.00	8.92	23.00	-0.08	1.00	-4.50	+0.50
256464	15	16	27.50	8.40	11.00	8.07	29.66	-0.33	2.16	-13.67	-12.00
206538	18	17	29.00	9.15	11.50	8.55	30.00	-0.60	1.00	-15.75	-14.25
54522	11	17	25.50	10.25	12.42	10.25	26.00	0.00	0.50	-5.50	+0.50
203330	20	20	24.45	9.31	13.00	8.90	29.00	-0.41	4.55	-6.75	-7.25
63532	23	20	22.00	8.15	12.50	8.02	23.00	-0.13	1.00	-4.75	-0.75
63119	22	23	23.45	-	14.67	7.95	26.00	-	2.55	-10.75	-8.12
63119	21	23	24.95	8.07	15.25	7.95	27.00	-0.12	2.05	-13.00	-11.37
282061	9	25	26.10	9.80	2.25	9.25	25.86	-0.55	-0.24	-8.62	+2.50
282061	10	25	27.13	9.75	2.83	9.45	26.74	-0.30	-0.39	-13.25	-1.75
272533	2	36	27.15	8.00	2.75	8.17	27.50	+0.17	0.35	-9.25	-1.25
272533	1	36	27.30	10.12	4.00	10.38	28.80	+0.26	1.50	-9.75	+1.75
282844	17	37	28.27	9.62	2.58	9.70	31.58	+0.08	3.31	-18.37	-0.62
129355	8	38	27.21	9.00	3.75	9.50	27.43	+0.50	0.22	-15.50	+1.25
129355	7	38	27.39	8.96	3.75	9.20	26.77	+0.24	-0.62	-12.67	+0.25
55344	6	42	26.00	8.75	11.42	8.75	27.47	0.00	1.47	-8.00	-1.50
GENERAL AVERAGE	21.13	25.49	8.96	9.29	8.85	26.89	-0.06	1.40	-9.74	-3.36	TOTAL
Average < 21 years	12.54	24.65	8.84	11.57	8.71	26.37	-0.13	1.72	-8.06	-4.49	< than 21
Average > 21 years	30.50	26.09	9.02	6.89	8.94	27.10	+0.02	1.02	-11.26	-1.89	> than 21

LONG TERM RESULTS OF MYOPIC KERATOMILEUSIS - 1982

the final radius 1 year postoperatively is 8.96 mm, while 9.29 years postoperatively it is 8.85 mm, shows that the results of the surgery are more stable after the first year. Therefore, the loss of curvature is 0.11 mm 8.29 years postoperatively, which is equal to 0.54 D in the cornea, or 0.065 D per year.

Consequently, if the progression is 1.85 D in 8.29 years and, if of these only 0.54 D are due to corneal steepening, the remaining 1.31 D must be due to axial growth and not to a lack of stability of the procedure after the first year.

The classification of patients into groups with more than and less than 10 D of myopia shows that the average age of the former is 22.89 years, while the average age of the latter is only 16.21 years.

Table III shows that the astigmatism, which was 1.95 D (-0.50 to -5.25) before the surgery, improved to 1.72 D (0.00 to -5.00) 1 year postoperatively and went

Table III
ASTIGMATISM

Case #	Preop (cyl)	1 mo (cyl)	12 mos (cyl)	Followup (years)	Late (cyl)	Change in axis	C / VA
15	-5.25	-5.00	-5.00	11.00	-2.00	5.00	0.45
13	-3.50	-2.00	-2.00	20.75	-2.25	25.00	0.40
5	-3.50	-1.25	-2.25	6.25	-3.00	15.00	0.40
20	-3.50	-1.50	-1.50	13.00	-4.00	5.00	0.80
6	-3.00	-2.50	-2.50	11.42	-2.50	10.00	0.60
9	-2.75	-2.25	-2.50	2.25	-2.00	20.00	0.80
18	-2.50	-1.25	-2.00	11.50	-2.50	20.00	0.10
10	-2.50	0.00	-1.50	2.83	-1.50	55.00	0.80
8	-2.00	-0.75	0.00	3.75	-0.50	5.00	0.25
1	-2.00	-1.25	-2.50	4.00	-0.50	70.00	0.67
14	-2.00	-2.00	-1.75	8.25	-2.50	10.00	0.50
12	-1.50	-0.50	-1.00	17.75	-2.75	20.00	0.33
2	-1.50	-1.50	-1.00	2.75	-2.50	10.00	0.67
21	-1.50	-2.50	-3.50	15.25	-4.24	0.00	0.70
22	-1.50	-0.50	-3.00	14.67	-2.25	0.00	0.80
16	-1.00	-0.75	-0.75	11.00	-2.50	10.00	0.80
11	-1.00	0.00	-1.50	12.42	-2.00	30.00	0.50
19	-1.00	-0.75	-1.00	8.00	-1.00	0.00	1.00
17	-0.75	-0.75	-1.25	2.58	-1.25	25.00	0.67
7	-0.75	-0.50	-0.75	3.75	-0.50	20.00	0.50
4	-0.75	-1.50	-0.75	5.00	-1.50	50.00	1.00
3	-0.50	-1.00	-1.00	13.00	-1.00	85.00	0.67
23	-0.50	-0.50	-0.50	12.50	-1.00	0.00	1.00
AVERAGES	-1.95	-1.33	-1.72	9.29	-1.99		0.63
Change in astigmatism		-0.66	-0.23		+0.04		

JOSE I. BARRAQUER, M. D.

back to 1.99 D (-0.50 to -4.00) 9.29 years postoperatively (almost the same preoperative figures).

Table IV registers the cases by preoperative axial lengths of more than and less than 27.00 mm. The first group had an average preoperative axial length of 27.62 mm, which progressed 0.97 mm. during the course of 5.27 years. The second group had an axial length of 24.27 mm, which progressed 1.66 mm. in 10.98 years. This amounts to 0.184 mm. per year in the first group and to 0.151 mm. per year in the second group and shows that the globes that are more elongated have a higher tendency towards myopic growth than less distended eyes. It should be noted that the first group includes cases No. 15 and 21, which had a postoperative myopia that was very similar to the preoperative myopia, but kept the preoperative visual acuity.

Table IV
AXIAL LENGTH OF THE GLOBE

PREOPERATIVE LATE POSTOPERATIVE

Case #	C / VA	Axial length	Sph. equiv.	Followup (years)	C / VA	Sph. equiv.	Axial length
17	0.10	28.27	-18.37	2.58	0.67	-0.62	31.58
15	0.40	27.50	-13.67	11.00	0.45	-12.00	29.66
7	0.33	27.39	-12.67	3.75	0.50	+0.25	26.77
1	0.50	27.30	-9.75	4.00	0.67	+1.75	28.80
8	0.33	27.21	-15.50	3.75	0.25	+1.25	27.43
2	0.67	27.15	-9.25	2.75	0.67	-1.25	27.50
10	0.80	27.13	-13.25	2.83	0.80	-1.75	26.74
9	1.00	26.10	-8.62	2.25	0.80	+2.50	25.86
6	0.70	26.00	-8.00	11.42	0.60	-1.50	27.47
5	0.10	25.50	-7.25	6.25	0.40	-1.50	25.98
11	0.67	25.50	-5.50	12.42	0.50	+0.50	26.00
4	0.80	25.10	-6.37	5.00	1.00	+0.75	26.16
21	0.40	24.95	-13.00	15.25	0.70	-11.37	27.00
20	0.29	24.45	-6.75	13.00	0.80	-7.25	29.00
16	0.20	24.00	-7.50	11.00	0.80	-3.75	28.10
13	0.05	24.00	-12.75	20.75	0.40	-5.12	25.75
3	0.10	23.50	-5.75	13.00	0.67	-2.00	24.00
22	0.25	23.45	-10.75	14.67	0.80	-8.12	26.00
14	0.25	23.25	-6.00	8.25	0.50	-5.75	25.74
19	0.33	22.00	-4.50	8.00	1.00	+0.50	23.00
23	1.00	22.00	-4.75	12.50	1.00	-0.75	23.00
AVERAGES	0.41	25.49	-9.74	9.29	0.63	-3.34	26.89
Ax. length < 27	0.44	24.27	-7.68	10.98	0.71	-3.06	25.93
Ax. length > 27	0.40	27.62	-13.63	5.27	0.51	-3.33	28.56

LONG TERM RESULTS OF MYOPIC KERATOMILEUSIS - 1982

The evolution of the postoperative corneal radius (Rf), myopia and axial length was:

	Radius	Myopia	Axial length (mm)
Initial	7.62	-9.74	25.49
Rf and Dc Requested	9.74	-9.60	
1 month postop.	9.31	-0.06	
1 year postop.	8.96	-1.49	
9.29 years	8.85	-3.36	26.89

Worthy to be noted is the fact that although the initial radius corresponds to 43.56 D and the final radius corresponds to 37.51 D (in other words, to a correction of 6.04 D), the correction obtained was 6.40 D, in spite of the axial growth.

This disagreement between the corneal and refractive changes should be studied more thoroughly, in order to find its cause, because it also occurs in hypermetropic keratomileusis, in which there was no accurate correlation between the keratometric and refractive modifications.

Table V gathers the evolution of the ametropia during 11.56 years, in the non operated eye of the patients of the study. This evolution was:

	Preop.	Late	Change in 11.56 years
Spherical	-0.14	-1.34	1.20 D
Cylindrical	-0.98	-1.43	0.45 D
Corneal radius	7.84	7.80	0.04 mm
Axial length	22.68	24.43	1.75 mm
Spherical equivalent	-0.63	-2.05	1.42 D

The calculation of the previous and late refraction with the corneal radius and axial length shows a difference of 5.31 D. Since the resulting myopia is only 2.05 D, we suspect there is a factor that compensates for the remaining 3.26 D, originating probably in the lens (deepening of the anterior chamber?).

Table V
MYOPIA EVOLUTION IN THE NON OPERATED EYE

Case #	FIRST EXAMINATION					LAST EXAMINATION							
	Sph.	Cyl.	Axis	VA	RI	Axial length	Followup (years)	Sph.	Cyl.	Axis	VA	Axial length	RF
3	0.50	0.00	1.00	7.59		13.00	0.00	-0.50	180.00	1.00		8.09	
4	-1.50	-1.00	180.00	0.80	7.86	5.00	-4.50	-0.75	180.00	0.80	24.72	7.95	
5	-1.00	-1.00	180.00	0.80	7.86	6.25	-2.25	-2.00	5.00	1.00			
6	0.50	-0.75	180.00	1.00	7.70	23.00	11.42	0.00	-2.25	170.00	1.00	23.48	7.61
11	0.00	-3.00	120.00	0.40	8.00	25.00	12.42	-2.00	-4.00	180.00	0.20	26.00	7.70
12	0.00	-0.25	180.00	1.00	7.70		17.75	-2.00	-1.50	10.00	1.00		
13	1.00	-2.75	180.00	0.80	7.22	21.50	20.75	-1.50	-2.00	180.00	1.00	7.22	
14	0.00	0.00	1.00	7.59		21.25	8.25	-0.50	-0.50	165.00	1.00	23.50	7.70
15	0.25	-3.25	165.00	1.00	7.70	22.25	11.00	-1.50	-2.50	165.00	1.00	7.59	
16	-0.75	0.00	1.00	8.24		22.00	11.00	-1.75	0.00		1.00	25.16	8.25
18	0.00	-1.25	5.00	1.00	8.19	24.00	11.50	0.00	-2.25	15.00	0.80	8.00	
19	0.50	0.00	1.00	8.12		8.00	0.00	0.00			1.00		8.04
20	-1.25	0.00	1.00	8.29		23.14	13.00	-2.50	-0.50	10.00	1.00	26.05	
23	-0.25	-0.50	180.00	1.00	7.66	22.00	12.50	-0.25	-1.25	15.00	0.80	22.10	7.64
AVERAGE	-0.14	-0.98		0.91	7.84	22.68	11.56	-1.34	-1.43		0.90	24.43	7.80

LONG TERM RESULTS OF MYOPIC KERATOMILEUSIS - 1982

In 14 cases, the stereopsis was examined for near vision with the fly test. The results were:

Case No.	Seconds of arc	Case No.	Seconds of arc
5	800	15	800
6	100	16	100
9	40	17	800
10	40	19	Absent
12	200	20	400
13	200	21	60
14	100	22	40

Due to the differences in followup, the surgical technique used in this series was not uniform. Therefore, in most cases the comparison of the different parameters does not produce sufficiently homogenous results to be of value. Only the classification of the results by thickness and diameter of corneal disc resected shows a congruence between the thickness and diameter of the disc and the degree of correction obtained (Table VII).

Although the followup of the groups under comparison differs greatly, this study shows that discs 0.30 mm. thick and 7.25 mm. in diameter provide the most stable results.

The surgical parameters used in this series were the following:

	Averages	Extreme values
Radius of base	8.17	7.00 to 9.67
Diameter of the disc	7.56	6.75 to 8.25
Ed	0.28	0.25 to 0.38 (0.38 in Case No.12)
Ep	0.31	0.20 to 0.40
Rt	5.93	4.27 to 7.71
Ec'	0.12	0.09 to 0.21 (0.21 in Case No.9)

In Table VI the results are gathered by Ed. In this table we can see that the best thickness ranges between 0.30 and 0.33 mm. Greater thicknesses lead to low corrections (because the posterior corneal layers adapt to the anterior layers), and smaller thicknesses provide less stability. The following are the averages obtained in Table VI.

Relation between Ed and correction		
Ed (mm)	Correction (diopters)	Correction %
0.38	-0.87	8.00
0.30-0.33	-10.51	96.01
0.29-0.33	-8.40	88.43
0.28-0.29	-4.93	71.53
0.25-0.26	-3.76	48.54

JOSE I. BARRAQUER, M. D.

Table VI
RESULTS GROUPED BY ED

Case No.	Age	Sph. equiv.	ED	EC	Correction	% Corr.
12*	12.00	-8.25	0.38		-0.89	8.00
9	25.00	-8.62	0.33	0.21	-11.12	131.25
7	38.00	-12.67	0.32	0.10	-12.92	102.27
8	38.00	-15.50	0.32	0.10	-16.75	109.62
21	23.00	-10.75	0.30	0.12	-2.63	18.88
4	6.00	-6.37	0.30		-7.12	110.71
10	25.00	-13.25	0.30	0.12	-11.50	84.76
1	36.00	-9.75	0.30	0.10	-11.50	114.56
18	17.00	-15.75	0.29	0.10	-1.50	16.18
19	14.00	-4.50	0.29		-5.00	111.11
23	20.00	-4.75	0.29	0.09	-4.00	85.00
13	6.00	-12.75	0.28	0.09	-7.63	63.00
6	42.00	-8.00	0.28	0.15	-6.50	82.35
14	9.00	-6.00	0.27	0.13	-0.25	23.33
17	37.00	-18.37	0.27	0.10	-17.75	95.87
20	20.00	-6.75	0.26		0.50	-15.81
5	12.00	-7.25	0.26	0.15	-5.75	81.25
22	23.00	-13.00	0.25	0.11	-1.63	4.21
2	36.00	-9.25	0.25	0.11	-8.00	87.14
3	8.00	-5.75	0.25	0.10	-3.75	65.22
16	6.00	-7.50	0.25	0.10	-3.75	65.91
11	17.00	-5.50	0.25	0.15	-6.00	109.52
15	16.00	-13.67	0.25	0.11	-1.67	-9.09
AVERAGE	21.55	-9.80	0.28	0.12	-6.40	67.18
ED > 0.30	27.29	-10.99	0.31	0.12	-10.51	96.01
ED < 0.30	18.87	-9.25	0.27	0.11	-4.85	57.68
*Excluded						

Two operative complications occurred in the series of this study. One was damage to the lenticule during the optical cut. The damaged lenticule was replaced with a homoplastic lenticule, in the same procedure, with satisfactory results (Case No. 11). The other occurred during the section of the disc with the microkeratome, and left the disc slightly pyriform, but had no consequences (Case No. 14).

The postoperative complications were 1 case of peripheral epithelialization of the interface, solved by simple evacuation, 1 case with a visible peripheral scar and 4 cases which showed a slight Hudson - Sthally line under the pupillary center in the late postoperative period. Two cases with postoperative ametropias higher than the preoperative figures maintained their visual acuity.

In all cases, the refraction and visual acuity was determined by some one other than the surgeon.

Case No.	Complication	Evolution
4	Eptihelialization	Solved
11	Homoplastic KM	Satisfactory
12	Peripheral Scar	Persisted
14	Pyriform resection	Satisfactory
5,18,21 and 22	Hudson-Sthally	Persisted
12	Ectasia by Ed= 0.38	Persisted

CONCLUSIONS

This study shows that the results provided by keratomileusis for the correction of myopia are stable enough to make it indicated in cases of intolerance to high conventional correction and patients with relative amblyopia. It also shows that the surgical results should be considered 1 year postoperatively because, after that, the progression of the myopia depends especially on the axial growth of the globe. Moreover, the study shows that there is an average improvement in acuity, both in amblyopes as in cases with good initial vision.

Table VII shows a summary of the preoperative and postoperative parameters of this series, as well as the extreme values of each one and their standard deviation.

JOSE I. BARRAQUER, M. D.

Table VII
GENERAL SUMMARY - 23 CASES

	Average	Extreme values	Standard dev.
PREOPERATIVE			
Ametropia	-9.74	-4.50 to -18.37	3.928
Astigmatism	-1.95	-5.25 to -0.50	1.215
C/VA	0.41	0.10 to 1.00	0.297
Age	21.13	6.00 to 42.00	
Axial length	25.94	22.00 to 27.30	1.994
Initial radius	7.62	7.05 to 8.33	0.303
Correction requested	-9.60	-17.00 to -5.00	3.354
Final radius request.	9.74	11.93 to 8.30	0.945
POSTOPERATIVE (1 MONTH)			
Final radius 1 month	9.31	11.00 to 8.22	0.760
U/VA 1 month	0.25	1.00 to 0.01	0.215
C/VA 1 month	0.43	1.00 to 0.05	0.234
Spherical equivalent	-0.06	-7.37 to +5.75	2.654
POSTOPERATIVE (1 YEAR)			
Final radius 1 year	8.96	10.25 to 8.00	0.621
U/VA 1 year	0.24	0.80 to 0.04	0.198
Spherical equivalent	-1.49	-7.00 to +2.50	2.765
C/VA 1 year	0.60	1.00 to 0.10	0.240
LATE POSTOPERATIVE			
Years of followup	9.29	20.75 to 2.25	5.332
Final radius	8.85	10.38 to 7.95	0.764
U/VA	0.27	0.67 to 0.02	0.256
C/VA	0.63	1.00 to 0.10	0.240
Spherical equivalent	-3.34	-12.00 to +2.50	4.728
Axial length	26.89	31.58 to 23.00	2.129
Postop. astigmatism	-1.99	-4.25 to -0.50	1.017
Correction obtained	-6.40	-17.75 to +0.50	5.125
Improvement in VA	0.22	0.67 to -0.20	0.265
Axial growth	1.40	4.55 to -0.62	1.381
Change in RF	-0.06	-0.60 to +0.47	0.314

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TRATAMIENTO QUIRURGICO DEL GLAUCOMA TRABECULECTOMIA CON IRIDENCLEISIS INTRAESCLERAL

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Analizamos el resultado obtenido en el tratamiento quirúrgico del glaucoma en pacientes afectados, durante el período de tiempo comprendido entre 1971 y 1981 en la clínica Barraquer de Bogotá - Colombia.

DESCRIPCION DEL TRATAMIENTO:

La intervención quirúrgica practicada la denominamos **Trabeculectomía con iridencleisis intraescleral** porque combina los dos procedimientos y consta de los siguientes tiempos quirúrgicos³:

Anestesia general con pentrane o flotane.

Colocación de los campos quirúrgicos y blefarostato colibrí.

Incisión conjuntival con base en el fondo de saco. Hemostasia con varilla de vidrio calentada a la llama a 90 grados centígrados.

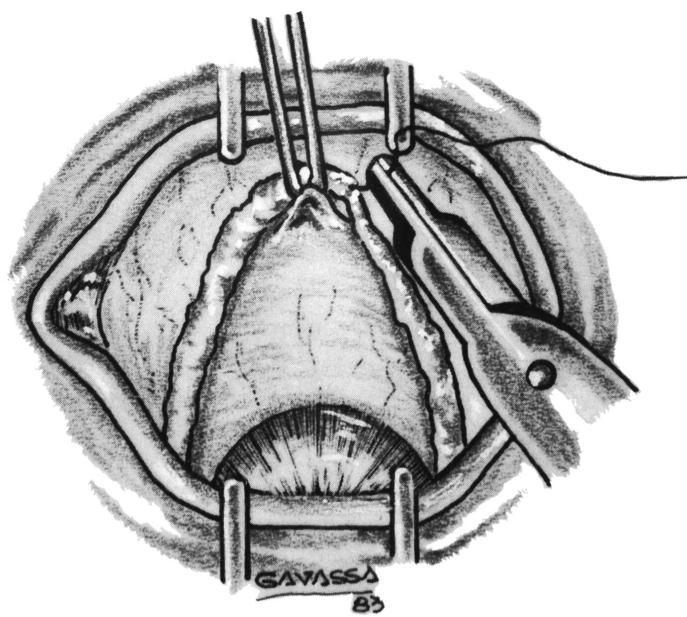
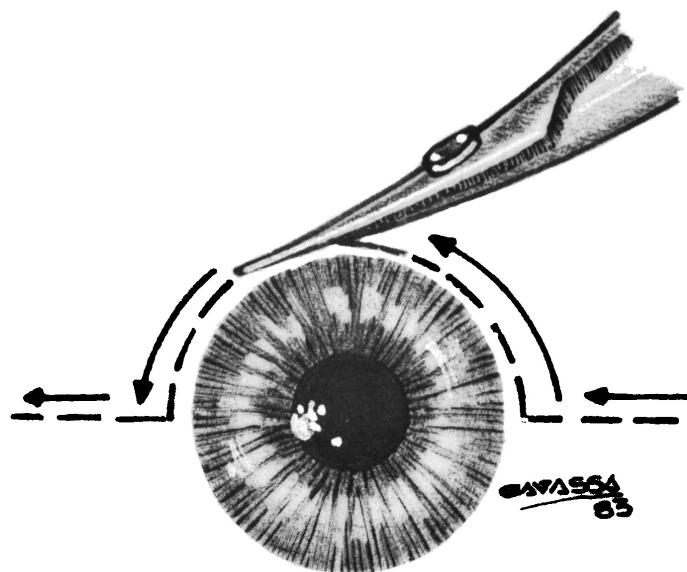
Colocación de un punto de seda 4/0 en el músculo recto superior que sirve a la vez para mantener el ojo en posición hacia abajo y para replegar hacia el fondo de saco superior el colgajo conjuntival.

1. Jefe del Departamento de Glaucoma de la Clínica Barraquer - Bogotá, Colombia.

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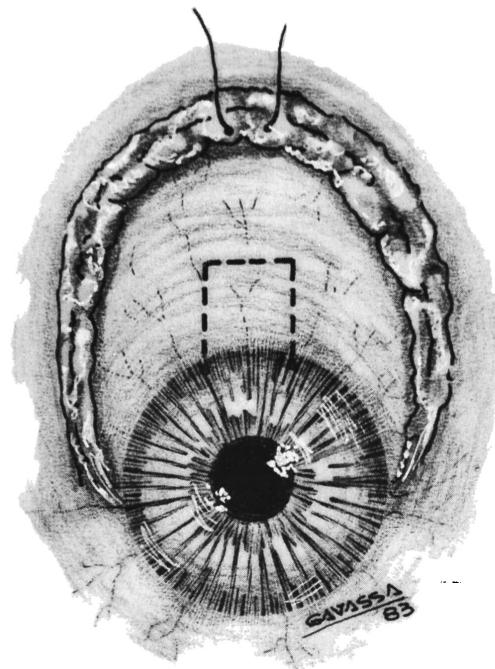
3. Técnica quirúrgica presentada por el Dr. José I. Barraquer M. durante el VI Congreso del Horacio Ferrer Eye Institute de Miami, Fla. Marzo 3 - 5 de 1973.

Symposium on Glaucoma - Edited by Olga Ferrer, M. D. - Charles C. Thomas Springfield - Illinois. pp. 193 - 1973.

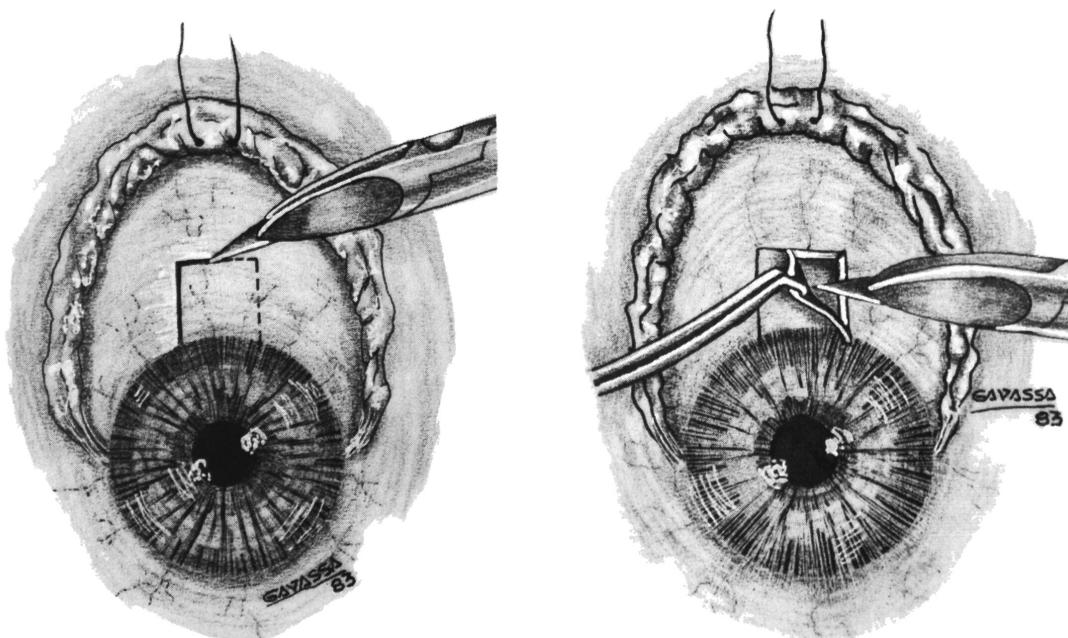


Demarcación de una lengüeta en la esclera de aproximadamente 5 mm x 5 mm
a una profundidad de más o menos la mitad del espesor escleral.

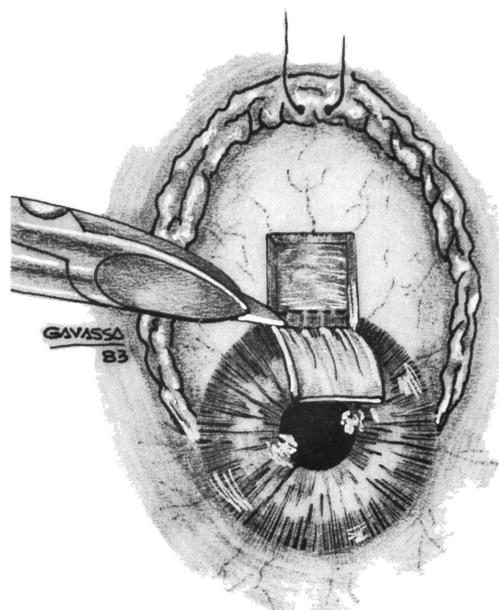
TRATAMIENTO QUIRURGICO DEL GLAUCOMA



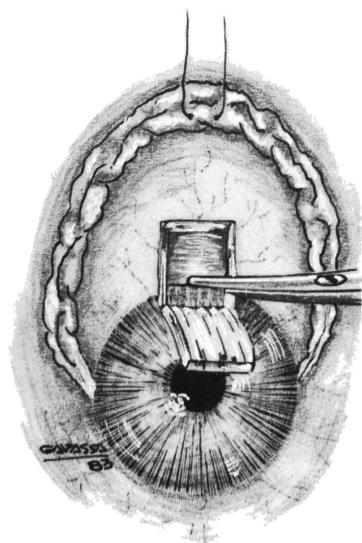
Talla de la lengüeta hasta el limbo quirúrgico y sobrepasando un poco en el tejido corneal (1 mm).



Sobre esta franja de tejido corneal penetraremos en la cámara anterior con un fragmento de cuchilla de afeitar. Cuando se trata de la técnica combinada con la

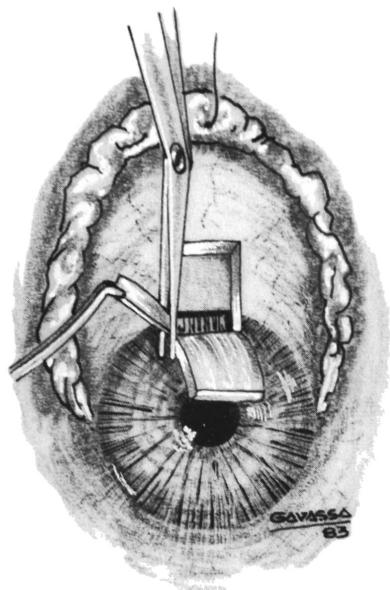


extracción de la catarata, ampliamos el colgajo corneoescleral con una tijera hasta 180 grados y procedemos a la faquectomía. Practicamos también una doble iridectomía periférica, una a cada lado de la esclerectomía para evitar que la hialoides o el vítreo bloquen la filtración en algún momento y la iridencleisis es elaborada partiendo de una de las dos iridectomías.

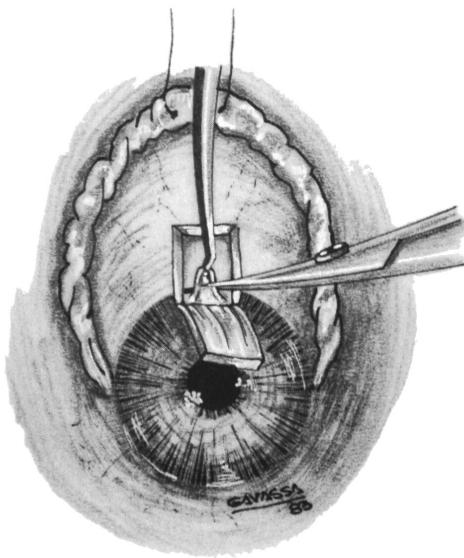


Se recorta un fragmento del trabéculo en forma cuadrangular con unas tijeras de Vannas o con un punch escleral.

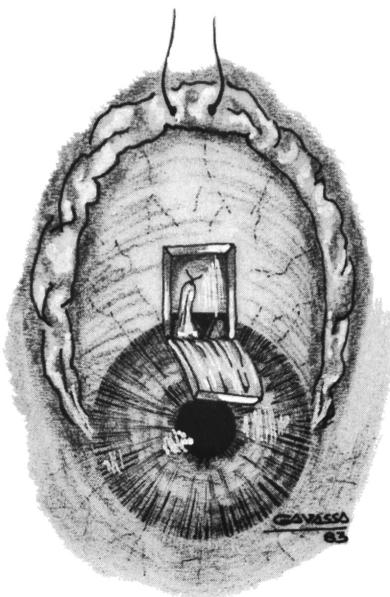
TRATAMIENTO QUIRURGICO DEL GLAUCOMA



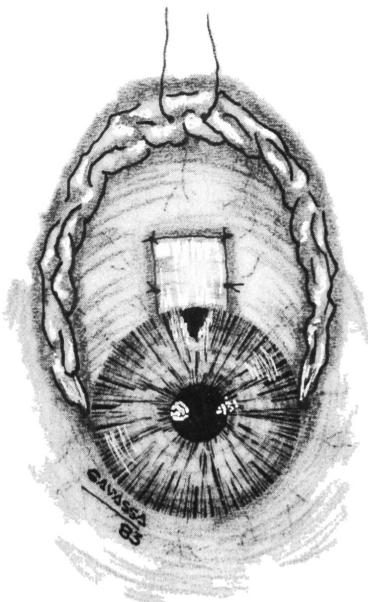
Una vez se hernia el iris por el orificio escleral se practica una iridectomía periférica de tal manera que no se corte todo el fragmento de iris sino se deja prendido por un extremo o se deja adherido a la raíz si se trata de una iridectomía en sector.



Este fragmento de iris así cortado se coloca en la entrecara escleral de la lengüeta a manera de iridencleisis y se fija a la esclera profunda con un punto de nylon 10/0 para evitar que se retraija nuevamente hacia la cámara anterior y garantizar su posición intraescleral



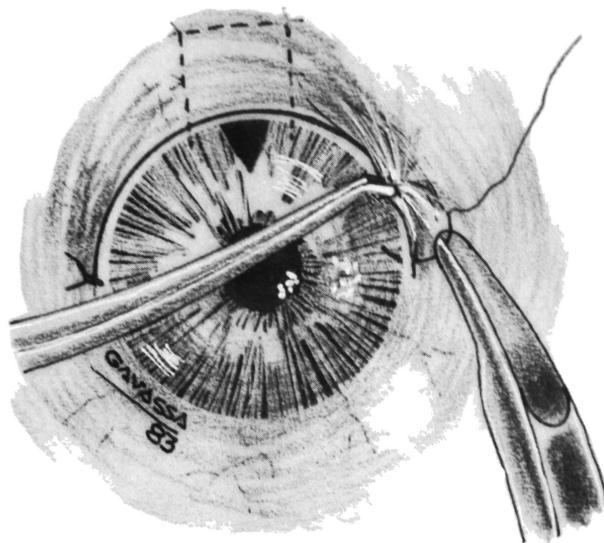
Sutura de la lengüeta escleral con cuatro puntos de nylon 10/0 enterrando los nudos en la esclera para evitar que perforen la conjuntiva posteriormente.



Reformamos la cámara anterior con una solución de acetilcolina para centrar la pupila y una burbuja pequeña de aire. Sutura de la conjuntiva con dos puntos de nylon 10/0 anclados en el limbo corneal.

Instalación de un midriático suave como escopolamina o mydriacyl y aplicación subconjuntival de gentamicina y decadadrón.

TRATAMIENTO QUIRURGICO DEL GLAUCOMA



El estudio analiza 127 ojos operados, de los cuales 100 fueron tratados con la técnica simple y 27 con la técnica combinada con extracción del cristalino cataratoso.

RESULTADOS:

Analizaremos separadamente los 100 casos de intervención simple y los 27 de intervención combinada.

En todos los casos se intentó primero el tratamiento médico y la decisión de intervenir quirúrgicamente fue tomada solamente en aquellos pacientes en quienes el tratamiento médico no obró satisfactoriamente ya sea porque la presión intraocular permaneció elevada o porque los daños ocasionados en el campo visual continuaron en aumento.

INTERVENCION SIMPLE (100 casos)

To. Pre-op.	To. Post.=op Inmed. 1 mes	To. Post-op. tardío + 6 meses	Tiempo de seguimiento
29.02 mm	13.89 mm.	14.87 mm.	3.45 años

TENSION OCULAR EN POST-OPERATORIO TARDIO

Sin Medicac.	Con Medicac.	No curaron
73 = 73%	16 = 16%	11 = 11%

TRABECULECTOMIA + CATARATA (27 casos)

To. Pre-op.	To. Post-op. Inmed. 1 mes	To. Post-op. Tardío + 6 meses	Tiempo Seguimiento
30.63 mm.	16.33 mm.	16.26 mm.	3.12 años

TENSION OCULAR EN POST-OPERATORIO TARDIO

Sin Medicac.	Con Medicac.	No curaron
17 = 63%	8 = 29.5%	2 = 7.5%

COMENTARIOS

En nuestra observación personal hemos concluido que es realmente necesario cierto grado de filtración alrededor de la lengüeta escleral ya sea en forma subescleral, intraescleral o subconjuntival para que el resultado de la trabeculectomía sea eficiente. En un buen número de casos, cuando la ampolla subconjuntival no es evidente, la tensión intraocular tiende a elevarse en el post-operatorio mediano o tardío. Pero cuando hay un buen grado de filtración a manera de una tenue ampolla subtenoniana la presión se conserva dentro de límites satisfactorios.

Nosotros creemos como lo creen también muchos otros cirujanos de glaucoma, que este cierre de la filtración es debido a la adherencia producida entre las dos caras de la esclera que forman la escotilla ya sea porque la cicatrización es muy activa o porque la baja de la presión intraocular generada por el trauma quirúrgico no permite un flujo suficiente de humor acuoso a través de la fistula filtrante para crear una filtración permanente.

Es por esto por lo que hemos considerado que la colocación de un fragmento de iris dentro de las caras de la lengüeta escleral a manera de iridencleisis puede colaborar a mantener la permeabilidad de la fistula pretendida con la intervención quirúrgica.

Los resultados obtenidos nos llevaron a pensar que la variación efectuada a la técnica corriente de la trabeculectomía nos da mayores garantías en el tratamiento de aquellos glaucomas que precisan una filtración considerable para el buen control de la presión intraocular.

TRATAMIENTO QUIRURGICO DEL GLAUCOMA

Concluimos también que el procedimiento es más eficaz en el caso de la técnica simple que cuando se practica combinada con extracción del cristalino quizá tal vez por el mecanismo de obstrucción que la hialoides y el vítreo ejercen sobre la esclerectomía en este segundo grupo.

RESUMEN:

Se analizaron dos grupos de pacientes: 100 casos afectados de glaucoma y 27 casos de pacientes afectados de catarata y glaucoma.

Fueron tratados quirúrgicamente con trabeculectomía e iridencleisis intraescleral el primer grupo y con trabeculectomía e iridencleisis intraescleral más extracción intracapsular del cristalino cataratoso el segundo grupo.

En ambos grupos el porcentaje de curación oscila alrededor del 90% en un período de tiempo de seguimiento superior a los 3 años.

DEVELOPMENT OF THE MAIN ELECTRORETINOGRAPHIC COMPONENTS IN PREMATURE INFANTS DURING THE FIRST WEEKS OF LIFE

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Roma, Italia.

Horsten and Winkelman (1960) first showed that the electrical activity of the retina is present in premature babies at birth. In a further study Rouher and co-workers (1967) pointed out the existence of the photopic retinal function in newborns born prematurely. The Samson-Dollfus (1967) and Nagata (1977) experiences showed, in order to demonstrate the electroretinographic maturation process in prematures, an increasing a-and b-wave amplitude in relation to gestational age.

These previous results indicate that the ERG registration in premature infants may be considered an useful index of maturity at birth, especially if this study is performed with an averaging computerized technique and supported by other available investigations such as VEP, Forced choice preferential looking, EEG, etc.

However at present time it isn't still clearly recognized the evolution of the main ERG's components in prematures of different gestational ages during the first weeks of life.

The aim of our study is to investigate the development of the photopic and scotopic electroretinographic components in the first weeks of extrauterine life in infants born prematurely.

SUBJECT AND METHODS

We have examined 8 premature babies (gestational age: 29-36 weeks; birth weight: 1020-2400 g). The arterial PO₂ ranged from 60 to 100 mmHg during the

period of our examination. The other metabolic parameters were within the normal limits.

The ERG's registration were performed at birth and repeated weekly from 4 times at least to 6 times after birth at different post-natal ages. After the ERG registration we performed an indirect ophthalmoscopy to exclude any vitreo-retinal abnormality clinically evident.

In no case was general anesthesia used nor pharmacological sedation, but the ERG was recorded after the feeding in condition of wake and after instillation of 0.4% Novesine. The pupils were dilated with repeated instillations of 1% cyclopentylate and 5% phenilephrine. The eyelids were maintained opened by a sharp speculum that we have shaped.

The newborns were always maintained into their incubators during the examination.

The active electrode was a small spiral of AgCl₂ which contained inside a sharp cotton flake imbued with physiological salt solution and maintained on the corneal surface. The reference electrode was placed in the auricular lobe and a mass electrode was applied in the centre of the forehead.

The ERG was recorded with the electroretinograph PANTOPS M₂ (Biophysic Medical Co.). The electroretinographic responses were averaged (N = 256) by a computer of average transients with a memory of 12 bits (4096 levels). The flash stimuli were provided by a continuous luminous source consisting of an halogen lamp and transmitted by optic fibers. We have used flashes of white light of 2.000 lux intensity and orange monochromatic light of 517 Å wavelength, 5 msec. duration and 4 Hz frequency. The extremity of the optic fibers was placed 5 cm above the infant's eye. The pre-amplifiers used have a differential entrance, a gain of 2.000 x 20, a band-pass from 0.2 to 1.000 Hz and an impedance of 2 x 1 MOhm.

The premature infant was dark adapted for a period of 30 minutes, both eyes being covered with opaque eye pads. After the registration of a static ERG with white light, the infants were submitted to a continuous white stimulus (2.000 lux intensity) for a period of 3 minutes. Immediately after we registered an ERG response every 2 minutes for a period of 16 minutes utilizing the orange stimulus.

Age (weeks)		Cases						
		1	2	3	4	5	6	7
29	28msec/20 uV	-	-	-	-	-	-	-
30	20 "/20"	28msec/30uV	-	-	-	-	-	-
31	n. r./n. r.	28 "/20 "	-	-	-	-	-	-
32	n. r./n. r.	24 "/30 "	26msec / 30uV	28msec / 40uV	-	-	-	-
33	26 "/50"	26 "/30 "	n. r./ n. r.	30 "/ 40 "	-	-	-	-
34	n. r./n. r.	26 "/30 "	n. r./ n. r.	26 "/50 "	26msec / 30uV	30msec / uV	30msec / 20uV	n. r./n.r.
35	-	-	n. r./ n. r.	24 "/50"	24 "/40 "	28 "/40 "	28 "/30 "	26 "/30 "
36	-	-	-	24 "/30 "	24 "/40 "	26 "/40 "	n. r./ n. r.	26 "/40 "
37	-	-	-	-	-	28 "/40 "	26 "/40 "	26msec / 30uV
38	-	-	-	-	-	28 "/30 "	-	24 "/40 "
39	-	-	-	-	-	28 "/40 "	-	26 "/50 "
40	-	-	-	-	-	-	-	24 "/40 "
41	-	-	-	-	-	-	-	24 "/50 "

Table No. 1
A-WAVE AMPLITUDE (MICROVOLTS) AND LATENCY (MSEC.) CHANGES IN SUBSEQUENT POST-NATAL WEEKS IN PREMATURE BABIES. (n. r. = not registered)

Age (Weeks)	Cases							
	1	2	3	4	5	6	7	8
29	140msec / 50uV	-	-	-	-	-	-	-
30	78 " / 40 "	120msec / 50uV	-	-	-	-	-	-
31	86 " / 50 "	82 " / 50 "	112msec / 50uV	115msec / 60uV	-	-	-	-
32	62 " / 70 "	68 " / 60 "	82 " / 40 "	86 " / 50 "	-	-	-	-
33	70 " / 80 "	72 " / 80 "	82 " / 80 "	60 " / 70 "	80msec / 60uV	70msec / 70uV	90msec / 40uV	-
34	70 " / 90 "	66 " / 90 "	60 " / 50 "	60 " / 80 "	68 " / 80 "	68 " / 70 "	68 " / 60 "	-
35	-	-	58 " / 65 "	56 " / 75 "	75 " / 70 "	70 " / 80 "	70 " / 70 "	-
36	-	-	52 " / 65 "	-	62 " / 70 "	60 " / 80 "	65 " / 70 "	70msec / 60uV.
37	-	-	-	-	65 " / 65 "	-	-	-
38	-	-	-	-	60 " / 75 "	-	-	-
39	-	-	-	-	-	-	-	-
40	-	-	-	-	-	-	-	-
41	-	-	-	-	-	-	-	-

Table No. 2
B₂-WAVE AMPLITUDE (MICROVOLTS) AND LATENCY (MSEC) CHANGES IN SUBSEQUENT POST-NATAL WEEKS IN PREMATURE BABIES

RESULTS

We have analysed the photopic and scotopic components of the ERG represented by a- and b₂-waves which resulted quite always well elicited. Their changes in the early weeks after birth are reported in tables n. 1 and n. 2.

1. The a-wave amplitude and latency seem to present almost constant values in subsequent post-natal ages after birth.
2. The b₂-wave presents an increase in the amplitude and a reduction of its latency in subsequent post-natal ages after birth.
3. These changes of the b₂-wave seem to be more remarkable and precocious in prematures of lower gestational age than in prematures of higher gestational age.

DISCUSSION

In present study we recorded an adapto-electroretinogram with a standard averaging technique in premature infants starting from 29th gestational age week. In these newborns we also registered the ERG weekly from 4 times at least to 6 times after birth. We obtained a remarkable increase of the b₂-wave amplitude and a precocious reduction of its latency in prematures of the lower gestational ages. On the other hand in prematures of higher gestational age these changes seem to be less evident.

Amplitude and latency of the a-wave present moderate changes in subsequent registrations during the early weeks after birth.

Results obtained suggest some speculations. The peculiar evolution of the two main electroretinographic components should be explained considering their origin in different retinal layers. According with Auerbach and Burian (1955) the a-wave represents the activity of the receptors elements, the rods and cones, while Miller and Dowling (1970) and Babel and co-workers (1977) found the b-wave origin in the inner retinal layers where it is probably produced by Müller cells.

As all the layers of the choroid, which provides to the nutritional apport to retinal receptors, are well differentiated by 7th month of foetal life, we think that may explain the almost constant electrical response of the photoreceptors in subsequent post-natal ages. Instead the evolution of retinal circulation, which supplies the inner retinal layers and is developing during the last weeks of intrauterine life, could explain the increase of b-wave amplitude and the

reduction of its latency registered in premature babies after birth. The most remarkable increase of b₂-wave recorded in prematures of lower gestational age during the early weeks of extrauterine life may be also explained by their precocious visual experience. This hypothesis is in part supported by results obtained by Sokol and Jones (1979). These A.A. in fact obtained a shorter latency of P100 VEPs components in 7 premature babies in comparison with full-term newborns, and suggested this phenomenon could be produced by a precocious visual stimulation.

Our results seem to indicate that the particular changes of b-wave in post-natal periods may be utilized to recognize the moments of greatest evolutive risk in premature infants. For that reason we think that regular study of this component of the ERG may be considered an additional and useful method for the surveillance of the anatomic maturation process of retinal tissue in the lower gestational ages.

SUMMARY

We have studied the development of the main electroretinographic components in premature infants during the first weeks of life. The method utilized was the adapto-electroretinogram in condition of wake. The ERG was repeated weekly from 4 times at least to 6 times after birth in 8 prematures.

The amplitude and latency of the a-wave resulted poorly influenced by increased post-natal age. On the contrary we have registered a progressive increase of the amplitude and a similar reduction of the latency of the b-wave in subsequent post-natal weeks in premature babies of lower gestational age. Results obtained indicate that the ERG study may be considered an useful method for the surveillance of the anatomic and functional maturation of the retinal tissue in premature infants.

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FRACTURAS DEL PISO ORBITARIO

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INTRODUCCION

En nuestra sociedad, cada vez más violenta, las fracturas de la órbita han pasado a ocupar un lugar destacado, por sus consecuencias funcionales y estéticas.

En este trabajo, se considerarán solo aquellas fracturas que comprometen el piso de la órbita, ya que ellas conforman una entidad especial por su frecuencia y características clínicas, además de ser esencialmente de competencia del oftalmólogo.

Se analizaron 34 pacientes con fractura de piso orbitario que fueron intervenidos quirúrgicamente. Por estar más expuestos, los hombres abarcaron el 88% de los casos. En general, este tipo de fractura se presenta en sujetos jóvenes, con una edad promedio de 25 años (rango: 15 - 52 años).

ETIOPATOGENIA

La etiopatogenia de las fracturas del piso orbitario es disentible. Los autores americanos definen con el término de "Blow-out fracture" a aquellas fracturas que se producen como consecuencia de un trauma directo sobre el contenido de la órbita, globo ocular y tejidos blandos, causado por un objeto de mayor diámetro que el de las dimensiones óseas orbitarias. Se produce entonces un aumento de la presión intraorbitaria, que causa explosión (Blow-out) del piso

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orbitario y de la pared interna, por ser estas más delgadas y, en consecuencia, más débiles. Las fracturas "blow-out" fueron subdivididas en "puras" e "impuras", en base a si comprometen o no el reborde orbitario.

Los autores franceses consideran que la fuerza responsable de la fractura del piso se ejerce siempre sobre el reborde mismo de la órbita, el cual se dobla sin fracturarse, y transmite la fuerza al piso, fracturándolo aisladamente. De esta manera no niegan el hecho mismo del "blow-out" o explosión; lo que niegan es el origen único de estas fracturas, causado por el sólo aumento de la presión intraorbitaria.

El hecho de que la mayoría de los pacientes afectados son sujetos jóvenes, hace pensar que la elasticidad ósea permite una deformación del reborde orbitario, seguida de una fractura aislada del piso. Por lo anterior, nos inclinamos a pensar que la causa de estas fracturas es un traumatismo sobre el reborde orbitario y no sobre globo ocular.

Aunque los traumatismos que causan estas fracturas son de diversos tipos (Tabla 1), los más frecuentes ocurren por accidentes de circulación (38%) y golpes de puño (21%).

Tabla 1

TIPO DE TRAUMA CAUSANTE DE LA FRACTURA

Accidente de circulación	13
Golpe de puño	7
Golpe con palo	4
Golpe con piedra	1
Golpe con botella	1
Accidente de aviación	2
Golpe de rodilla	1
Cornada	2
Coz de mula	1
Caida de caballo	1

CARACTERISTICAS CLINICAS

Con frecuencia, inicialmente estas fracturas pasan inadvertidas, viniéndose a descubrir sólo semanas o meses después de haber sucedido el trauma desencadenante. En un principio su síntoma clave es la diplopia post-traumática, enmascarada por edema, hematoma y emborronamiento de la visión.

En promedio los pacientes estudiados consultaron al quinto mes del traumatismo, oscilando su primera visita entre el primer día y los 18 meses desde la injuria, en relación a la severidad de las lesiones.

En 21% de los casos se presentó trauma ocular asociado (Tabla 2).

Tabla 2

DAÑO OCULAR ASOCIADO 7 (21%)

1. Hemorragia vítreo , desprendimiento de retina
2. Hemorragia macular
3. Midriasis paralítica , subluxación del cristalino
4. Hemorragia macular , luxación de cristalino , midriasis paralítica
5. Hemorragia vítreo , recesión angular , desprendimiento de retina
6. Leucoma adherente , catarata
7. Sección canalicular completa

Los síntomas y signos varían de acuerdo con la violencia del agente traumatizante y el tiempo transcurrido desde la injuria inicial. La diplopia, síntoma capital en el reconocimiento de estas fracturas, se debe a un encarcelamiento del músculo recto inferior y/o oblicuo inferior o de sus vainas aponeuróticas, al quedar atrapados en los fragmentos óseos. Ante la persistencia de una diplopia post-traumática se debe sospechar una fractura aislada del piso de órbita. Aunque casi siempre hay hipotropia del ojo del lado afectado, si la fractura es muy posterior se puede observar una hipertropia. Estos pacientes tienen una marcada limitación de la elevación y una dificultad de la depresión, cuya severidad es variable. La diplopia, sobre todo en la mirada arriba, es un síntoma constante. Este hallazgo debe complementarse con un test de ducción forzada, cuya positividad es un elemento de primer orden en el diagnóstico.

El enoftalmos es otro signo de gran importancia pues es debido a un descenso del globo ocular, tejidos blandos, músculos y grasa orbitaria que se hernian dentro del seno maxilar al fracturarse el piso.

Cuando el párpado superior pierde su apoyo normal en el globo, se produce una pseudo-ptosis. Las ptosis palpebrales pueden presentarse como consecuencia del traumatismo del músculo elevador de los párpados. Se debe distinguir de la pseudo-ptosis, la cual es un signo de enoftalmos que se recupera al elevar el globo ocular con tratamiento. Puede encontrarse exoftalmos debido a desplazamientos óseos o por hemorragia orbitaria.

Las lesiones del nervio infraorbitario son consecuencia del traumatismo directo de los fragmentos óseos e implican anestesia o hipoestesia de su territorio de inervación.

Los hallazgos clínicos más importantes en estos pacientes se enumeran en la tabla 3.

Tabla 3

Diplopia	28	82%
Enoftalmos	26	76%
Anestesia del territorio del infraorbitario	8	24%

RADIOLOGIA

Las proyecciones básicas para el estudio de la órbita permiten la visión del reborde y el piso orbital. Puede observarse un velamiento del seno maxilar por hemorragia y edema. En el caso de fractura del piso con hernia del contenido orbital al cabo de 10 días se puede comprobar el signo clásico de la "gota pendiente" (semejante a una gota de agua que cuelga del techo del seno maxilar).

Cuando el estudio básico de la órbita nos brinda escasa información, se debe recurrir a la tomografía orbitaria.

En nuestra casuística el estudio radiológico fue positivo en todos los casos. En ella se presentó velamiento del seno maxilar, en 5 casos que tenían una evolución menor de 1 mes desde el traumatismo.

FRACTURAS DEL PISO ORBITARIO

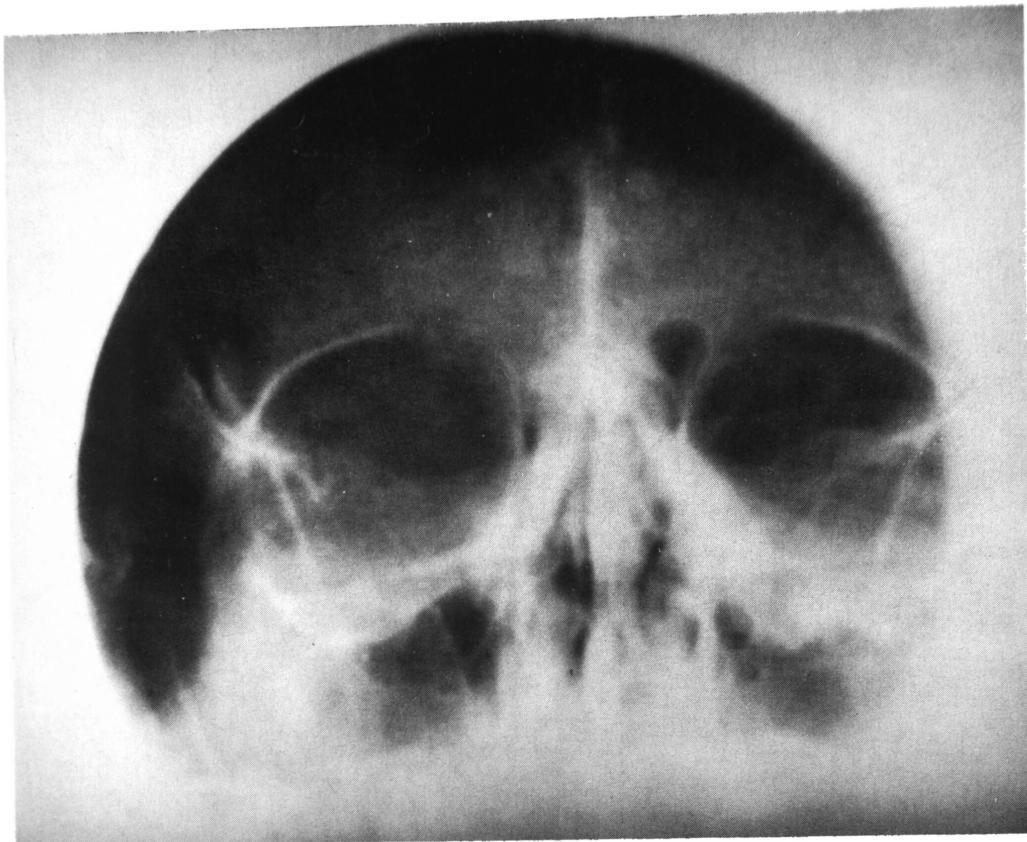


FIGURA 1.

Radiografía en proyección anteroposterior, fractura del piso orbitario izquierdo, signo de la "gota pendiente".

Las fracturas asociadas a las del piso orbitario no son frecuentes. En nuestro estudio fueron encontradas en el 12% de los casos. (Tabla 4).

Tabla 4

Hueso malar	1
Reborde orbitario	1
Techo de la órbita	1
Huesos propios de la nariz	3

TRATAMIENTO

Cuando una fractura del piso orbitario se confirma radiológicamente, y ella ocurre sin alteraciones funcionales, se debe observar. Por el contrario, la intervención está indicada en los casos de diplopia persistente con test de

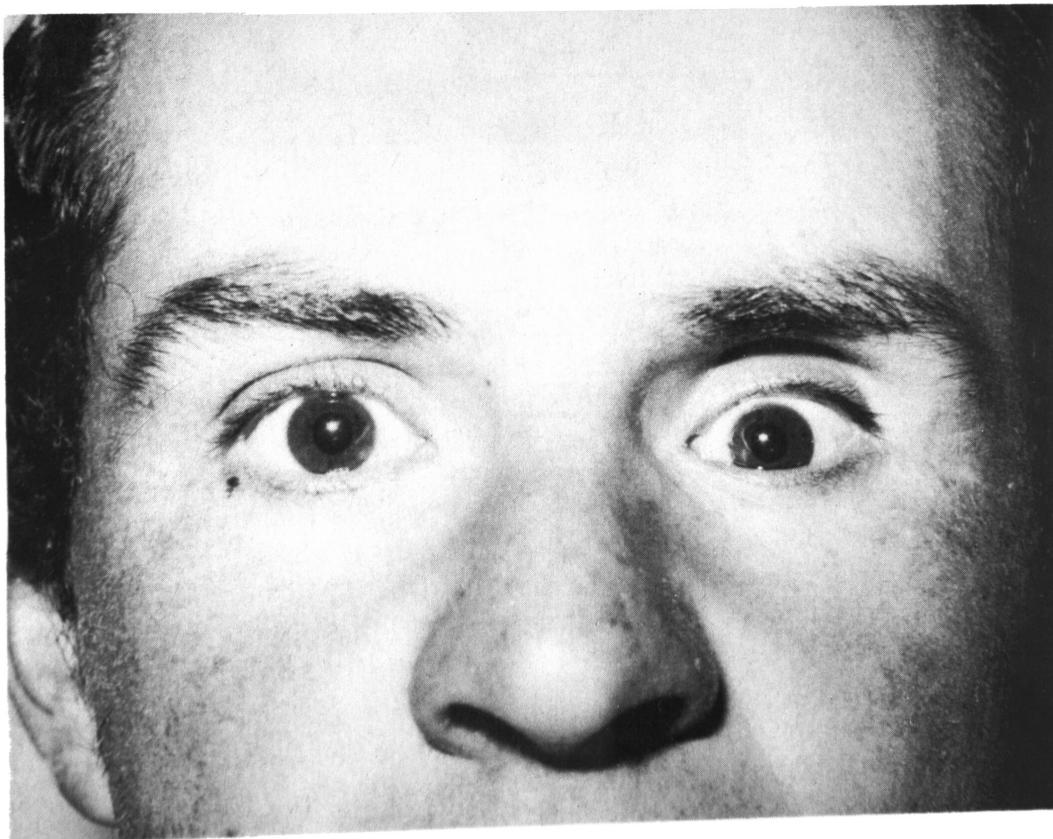
ducción forzada positivo y estudio radiológico negativo. La segunda indicación es la enoftalmía acompañada de diplopia, más aún si se presenta anestesia del infraorbitario y un estudio radiológico positivo.

Los objetivos del tratamiento quirúrgico son:

1. Elevar el globo para corregir el enoftalmos.
2. Liberar los tejidos orbitarios, grasa, músculos o sus vainas aponeuróticas y destruir las adherencias para suprimir la diplopia.
3. Despejar el nervio infraorbitario para resolver la anestesia de la mejilla.

Decidida la cirugía, se practica el test de ducción forzada, mediante un hilo de seda que se pasa en el recto inferior, antes, durante y al finalizar la cirugía, para comprobar la liberación de los tejidos comprometidos.

Nosotros utilizamos una incisión subciliar, dejando un colgajo de piel amplio. Las fibras del orbicular se separan longitudinalmente hasta el septum.



*FIGURA 2.
Fractura del piso de la órbita izquierda con enoftalmos.*

FRACTURAS DEL PISO ORBITARIO

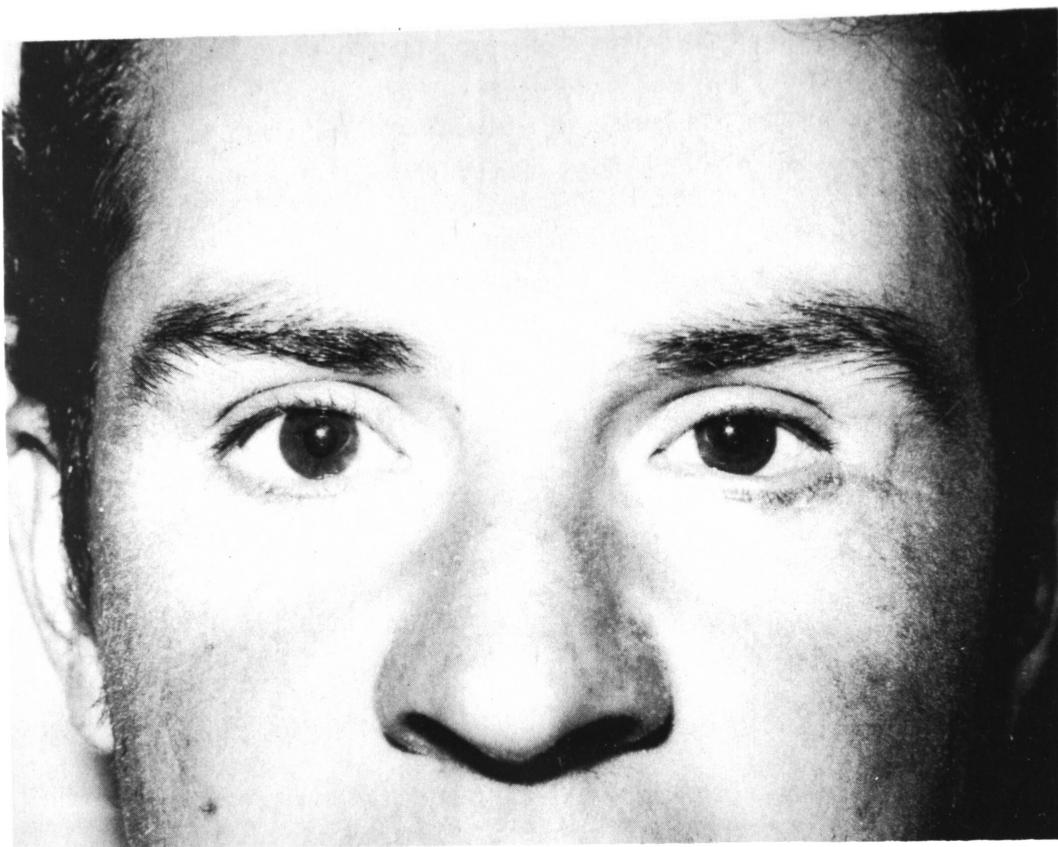


FIGURA 3.
Resultado postoperatorio.

Una vez que se llega al periostio, éste se corta y levanta a nivel del reborde orbitario inferior. Se explora el piso y se libera el contenido orbitario. La pérdida de sustancia ósea puede ser reparada con un implante o injerto óseo.

Por ser muy bien tolerada, en todos los casos utilizamos implantes de silicona, los cuales se tallan de acuerdo a cada caso particular.

Se sutura en tres planos y se deja un vendaje compresivo durante las primeras 24 horas. Esta técnica se utilizó en todos los pacientes que presentamos, asociándola, en dos casos, a liberación de simblefaron inferior y en 1 caso a colocación de un tubo de Jones.

COMPLICACIONES

En general, las complicaciones no son frecuentes (Tabla 5). Puede presentarse una hiper corrección del enoftalmos, la cual inicialmente se debe a edema de los

tejidos, pero desaparece en pocos días. De persistir después de un mes, es necesario reintervenir y retallar el implante.

Tabla 5
COMPLICACIONES

Hipocorrección del enoftalmos	2
Hiper corrección del enoftalmos	1
Anestesia del infraorbitario	3
Infección	1
Expulsión del implante	0
Daño de la bomba lagrimal	0
Ptosis secundaria	3

Las infecciones son raras y habitualmente ceden con el tratamiento médico.

IMBALANCES MUSCULARES

En el post-operatorio inmediato es frecuente que el paciente refiera diplopia. El compromiso de un recto inferior en una fractura, puede causar alteraciones de su función por varias semanas, e inclusive meses, después de ser liberado. Esto resulta en una hipertropia y diplopia post-operatoria.

En la casuística que presentamos, 12 pacientes presentaron diplopia después del primer mes y 5 después del tercer mes. Aunque la diplopia post-operatoria fue tratada con oclusión, fue necesario recurrir a cirugía estrabológica en 7 pacientes.

El objetivo de esta cirugía es obtener fusión en posición primaria y en infraversión, aún a costa de perder la fusión en supraversión.

CONCLUSIONES

La fractura del piso de órbita constituye una entidad cuya frecuencia se ha incrementado con el creciente número de accidentes, especialmente automovilísticos, y en relación al aumento del índice de criminalidad. La sospecha de esta fractura aumenta cada día, por el mejor conocimiento que de ella tenemos. El retraso en el tratamiento disminuye su éxito, a pesar de lo cual se obtuvieron resultados satisfactorios.

FRACTURAS DEL PISO ORBITARIO

RESUMEN

Se presentan 34 pacientes con fractura de piso de órbita y se analizan su etiopatogenia, rasgos clínicos y radiológicos. Se describe la técnica quirúrgica empleada en todos los casos y se enumeran las complicaciones y su tratamiento.

Se subraya la necesidad de considerar tanto el aspecto funcional como el estético en los pacientes con fractura de piso de órbita.

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