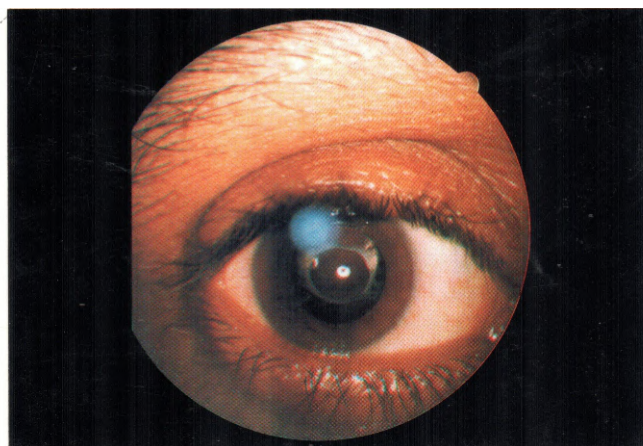


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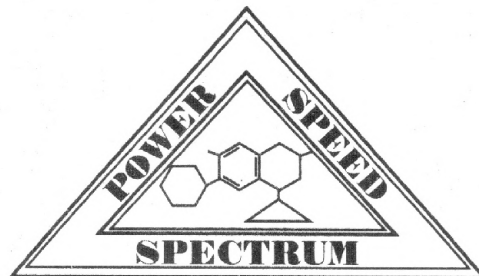
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At Page 77, Figure-1: Capsular phimosis 6 mos after phacoemulsification

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EDITORIAL

Taking Up the Gauntlet Against the Gruesome, Grave, Glaucoma

See also pp.....79-89

Glaucoma has been metaphorically described as a thief in the night that silently robs one, of the eyesight when it strikes. In fact it is a thief stealthily at work day and night, robbing the unsuspecting millions around the world, of the greatest of gifts from God--the gift of eyesight. Glaucoma is a multifaceted group of diseases characterized by progressive optic nerve damage leading to disabling visual field loss from compromised function of retinal nerve fibers manifested by increased cupping of the optic disk.

Various risk factors have been incriminated for development of glaucoma. Raised intraocular pressure is a primary risk factor. Family history of glaucoma increases the risk of developing the disease 15-to 20-folds¹. Primary open-angle glaucoma (POAG) has a prevalence of 0.4-4.1% in whites². A 4-fold higher risk of developing glaucoma is seen in blacks compared to whites. Similarly blacks are 6 times more likely to become blind from POAG than are whites¹. Age is a well-known risk factor in glaucoma. The Rotterdam Prospective Cohort Study showed an overall prevalence of 1.1% for POAG, with a prevalence of 0.2% in 55-59 yr age group, 3.3% in 85-89 yr age group, with males 3 times more likely than females to develop POAG³. The Beaver Dam Study showed an overall incidence of 2.1%, with 0.9% in 43-54 yr age group and 4.7% in those > 75 yrs, and no sex difference⁴. The Barbados Study had a prevalence of POAG of 7% in blacks, 3.3% in mixed-races and 0.8% in whites⁵. A Japanese study had an overall prevalence of 3.6% in > 40 yr age group, a very low (1.4%) prevalence of ocular hypertension (OH) and a very high (2%) prevalence of normal tension glaucoma (NTG) compared to whites⁶. Severe myopia and previous ocular trauma are also additional risk factors, as is arterial hypertension in developing POAG⁷, whereas arterial hypotension is implicated in patients with normal tension glaucoma (NTG)

Glaucoma is a serious blinding disease of global importance. According to WHO (1990) figures, 45 million people are blind globally. Glaucoma is the third leading cause of blindness in the world, after

cataract (16 million) and trachoma (6 million) and is responsible for 5 million blind people the world over. These figures are estimated to double in 20-25 years⁸. World figures for those affected by glaucoma (22.5 million) and those suspected to have glaucoma (105 million) are even more alarming. The prevalence of glaucoma in the U.S. is 1,361,000 (2% of population > 45 years of age), of which 36% are in 45-64 yr age group and 64% in > 65 yrs. The annual incidence of glaucoma is 190,000 (0.0015% in 45-64 yr age group and 0.00047% in > 65 yr age group)⁹. The figures for Pakistan based on the National Health Survey of Pakistan (1990-94) indicate the prevalence of blindness as 1.78% (2.5 million). Glaucoma is the fourth commonest cause of blindness nationally and is responsible for 3.9% of the total blind in Pakistan (82,677 cases)¹⁰.

Socioeconomic burden of glaucoma on society and healthcare resources has been studied in various countries and comprises three elements: direct, indirect and intangible costs. The former are borne by the healthcare system. Indirect costs are mainly production losses and subsidizing expenses to the invalid. Intangible costs related to pain and suffering and reduced quality of life are difficult to measure. In the UK, 1990 measurable costs of glaucoma were £133.5 million, of which direct medical and surgical costs were £62.4 million, residential care cost £25.8 million and productivity losses £45.3 million⁹. In the U.S. these costs are in excess of \$ 1 billion per annum. In Sweden the 1995 direct costs from glaucoma amounted to 375 million Swedish Krona⁹. With increasing longevity the population >65 yrs of age will be putting more strain on society with an overall increase in the incidence of glaucoma.

In most countries, treatment of POAG is primarily medical. The current management strategies, until lately, were aimed at reduction of the primary modifiable risk factor--the intraocular pressure (IOP), by topical eye drops containing beta-adrenergic antagonists, cholinergic agonists, adrenergic agonists or carbonic anhydrase inhibitors (CAIs). If topical medications,

individually or in combination, are not sufficiently effective. systemic CAIs are used but systemic side effects limit their use. The recently developed prostaglandin analogue eye drops have been shown to reduce IOP effectively by a newly discovered (in 1966)¹¹ uveoscleral pathway which is responsible for 10-20% of aqueous outflow¹². These have been shown in studies in the U.K.¹³, U.S.A.¹⁴ and the Scandinavian countries¹⁵ to reduce IOP at 6 months by 28-34% of the baseline IOP. The development of these prostaglandin eye drops seems as revolutionary as the development of beta-blockers in 1978. Once a day application also addresses the major cause of inadequate control of IOP—the lack of compliance with up to 50% of patients failing to take antiglaucoma medications correctly¹⁶.

Failure of medical therapy leaves only the options of laser trabeculoplasty (LTP) or surgical trabeculectomy (TRAB) with or without antimetabolites. The results of LTP are poor in darker races and its effectiveness progressively declines by about 11% annually¹⁷, with only 30% of treated patients maintaining satisfactory long-term control of IOP¹⁸.

Primary surgical treatment (TRAB) has been seen to result in less progression of preexisting visual field loss, compared with initial medical treatment, probably because of lower IOP in the surgical group over the study period^{19,20}. The concept of "Target pressure" customized to each individual patient as the desirable level of IOP was developed after it was seen in several studies on fistulizing surgery that visual fields continued to deteriorate even when IOP had been reduced to within the "normal" range at the rate of 15% at an IOP of 14/15 mmHg and of 60% with IOP of 19mmHg²¹. So there is a catch 22 situation in shooting for the "Target pressure". Since visual field and optic disk changes take a long time to manifest, how do we select a target pressure **now** to come out with flying colors 20,30 or 50 years later? If we consider patients with IOPs of 25mmHg as ocular hypertensives and choose not to treat them, are we missing the "window of opportunity"? And if we choose to treat them for that pressure level, since raised IOP is considered to be the primary risk factor for developing glaucoma, are we overtreating a lot of patients who would turn out to be true ocular hypertensives after all? On the other hand, if we set a target pressure of 15 mmHg for a patient who would turn out (later) to be suffering from normal tension glaucoma, are we being lulled into a false sense of security while the patient continues to lose visual field?

These are difficult questions to answer, especially when economic issues also need to be

addressed. Filtering surgery increases the economic burden but initial surgical treatment has been shown to be as cost-effective as the medical treatment, as well as offering more protection to the patient from progressive glaucoma²². Various surveys from specialized glaucoma units indicate variable success rates: 40% chance of IOP control at 5 yrs; 82% at 1 yr and 90% at 5 yrs; and 68% at 36 mos after surgery, without any additional medication—the prime hope of the patients and their surgeons²³. The more recently introduced nonpenetrating filtering surgery (NPFS) such as viscocanalostomy²⁴ or deep sclerectomy and Descemet's window with or without collagen implant²⁵, is sure to enhance the economic burden further. High pass resolution perimetry and scanning laser tomography that can detect progression of visual field loss earlier than the conventional perimetry, even though they may raise the economic stakes even further in the management of glaucoma, may after all turn out to be really cost-effective in the long run.

In most countries primary therapy for high-tension glaucoma (HTG) and primary open-angle glaucoma, at present, is medical, to be followed by surgical treatment if this fails to maintain an IOP at a "target level" of 17 mmHg or thereabouts. Prostaglandin analogues as primary monotherapy or as combination therapy with existing antiglaucoma medications, e.g. beta-adrenergic antagonists, CAIs, adrenergic agonists or cholinergic agonists are a very effective addition to our antiglaucoma armamentarium indeed.

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Peripapillary Subretinal Neovascular Membranes

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ABSTRACT

Photocoagulation indications and visual outcome in a group of twenty-eight eyes of 26 patients with peripapillary subretinal neovascular membranes (SRNM) were evaluated. Thirteen eyes (46.4%) had age-related macular degeneration, seven (25.0%) had angioid streaks, three (10.71%) were idiopathic, two (7.14%) had degenerative myopia, one (3.57%) had optic disc drusen, one (3.57%) peripapillary choroidal nevus, and one (3.57%) had optic disc coloboma. Two of five patients with angioid streaks had bilateral peripapillary subretinal neovascular membranes. If peripapillary SRNM extended under the centre of foveola, no laser treatment was performed. Overall, nineteen of 28 eyes (67.8%) received argon laser photocoagulation. Seven of these 19 eyes (36.8%) experienced increase in visual acuity. Visual acuity remained unchanged in six eyes (31.6%) and decreased in the remaining six eyes (31.6%). Peripapillary SRNM carried a favorable prognosis with appropriate photocoagulation unless subfoveal extension was present.

INTRODUCTION

Peripapillary subretinal neovascular membrane (SRNM) is associated with various ocular conditions including the idiopathic cases^{1,2}, optic nerve head coloboma³⁻⁶, optic disc drusen⁷, tilted optic nerve head⁸, optic pit^{3,9}, sarcoidosis¹⁰, pars planitis¹¹, angioid streaks¹², Harada's disease¹³, toxocariasis¹³, serpiginous choroidopathy¹⁴, pseudotumor cerebri¹⁵, ocular histoplasmosis^{2,16,17}, and age-related macular degeneration^{2,18}.

MATERIALS AND METHODS

We performed a retrospective analysis among the patients who received a diagnosis of peripapillary subretinal neovascular membrane between January 1994 and January 1998 at Ophthalmology Department of Dokuz Eylul University. Twenty-eight eyes of 26 patients comprised our study group. Characteristics and associated retinal conditions of 26 patients were noted in detail (Table-1).

Peripapillary subretinal neovascular membrane (SRNM) was defined as SRNM contiguous with the optic disc or atrophy adjacent to the optic disc^{16,19}. All symptomatic patients with peripapillary SRNM without subfoveal component were photocoagulated by argon green laser, and all treated membranes were located at least in part, in a triangular area bordered by two lines

drawn from the centre of the fovea to each vertical pole of the disc. Best-corrected visual acuities before and after the treatment were noted for each patient. Change in the best-corrected visual acuity was defined as at least two or more Snellen lines change compared to pretreatment visual acuity. If no change or only one Snellen line change was encountered in the best-corrected visual acuity, it was accepted as unchanged.

Fluorescein angiograms were obtained at least 72 hours prior to treatment, and two weeks, six weeks, three months, and six months after the treatment.

RESULTS

There were thirteen men (50.0%) and thirteen (50.0%) women. Mean age was 54.03 ± 11.4 years (range 15 to 79 years). Follow-up period ranged within eight to 57 months (mean: 19.2 ± 3.4). The underlying diagnosis was age-related macular degeneration (thirteen eyes), angioid streaks (seven eyes), idiopathic (three eyes), degenerative myopia (two eyes), choroidal nevus (one eye), optic disc drusen (one eye), and optic disc coloboma (one eye).

Nineteen of 28 eyes (67.8%) had argon green photocoagulation. Eight eyes (five with age-related macular degeneration (ARMD), two with angioid streaks and one with degenerative

Table 1. Characteristics of the study group.

Patient No	Age (years)	Gender	Laterality	Associated Clinical diagnosis	Initial corrected visual acuity	Final corrected visual acuity	Photocoagulation	Subfoveal extention	Status of the fellow eye	Follow-up period (months)	Visual acuity
1.	79, M	R		ARMMD	CF	CF	No	+	ARMMD	57 m.	Unchanged
2.	72, M	L		ARMMD	20/25	20/30	+	No	Drusen	48 m.	Unchanged
3.	72, M	R		ARMMD	20/60	20/25	+(twice)	No	ARMMD	22 m.	Increased
4.	65, M	R		ARMMD	20/50	20/25	+	No	Drusen	22 m.	Increased
5.	66, F	R		ARMMD	20/100	CF	No	+	ARMMD	11 m.	Decreased
6.	62, F	L		ARMMD	20/200	20/200	+	No	Drusen	17 m.	Unchanged
7.	63, M	L		ARMMD	20/30	20/200	+	No	Drusen	16 m.	Decreased
8.	77, F	L		ARMMD	CF	CF	No	+	ARMMD	10 m.	Unchanged
9.	69, F	R		ARMMD	20/200	20/50	+	No	Drusen	11 m.	Increased
10.	66, F	R		ARMMD	20/100	20/30	+	No	ARMMD	16 m.	Increased
11.	70, F	R		ARMMD	20/25	20/25	No	No	Drusen	8 m.	Unchanged
12.	73, F	R		ARMMD	20/125	CF	+	No	Drusen	9 m.	Decreased
13.	67, M	R		ARMMD	CF	CF	No	+	ARMMD	16 m.	Unchanged
14.	47, M	L		Angioid streaks	20/25	20/200	+	No	Angioid streaks	20 m.	Decreased
15.	65, M*	R		Angioid streaks	20/200	CF	No	+		45 m.	Decreased
		L		Angioid streaks	20/30	CF	+(twice)	No			Decreased
16.	34, F	R		Angioid streaks	20/25	20/30	+	No	Angioid streaks	17 m.	Unchanged
17.	30, M	L		Angioid streaks	20/40	20/200	+	No	Angioid streaks	9 m.	Decreased
18.	42, M*	R		Angioid streaks	20/50	20/60	+	No		11 m	Decreased
		L		Angioid streaks	CF	CF	No	+			Unchanged
19.	42, M	R		Idiopathic	20/50	20/25	+	No	Normal	10 m.	Increased
20.	47, F	R		Idiopathic	20/200	20/100	+	No	Normal	11 m.	Increased
21.	26, M	R		Idiopathic	20/50	20/20	+	No	Normal	16 m	Increased
22.	35, F	R		Degen. myopia	20/100	20/100	+	No	Normal	10 m.	Unchanged
23.	38, F	R		Degen. myopia	20/100	20/100	No	+	Normal	16 m.	Unchanged
24.	15, F	R		O. disc drusen	20/60	20/60	+	No	O. disc drusen	10 m.	Unchanged
25.	27, M	L		O. disc coloboma	20/30	20/30	Refused	No	Normal	14 m	Unchanged
26.	42, F	R		Choroidal nevus	20/25	20/20	+	No	Normal	49 m.	Unchanged

M: Male; F: Female; ARMMD: Age-Related Macular Degeneration; Degen. myopia: Degenerative myopia; O. disc.: Optic disc CF: Counting

Fingers; *: Cases with bilateral Peripapillary SRNM

myopia) were not treated due to extension of SRNM under the foveola. One patient with disc coloboma and peripapillary SRNM declined treatment. One eye with ARMD and one eye with angioid streaks required retreatment as there was angiographically proven persistence of leakage six weeks after the treatment.

The change in best-corrected visual acuities at the last visit was compared to pretreatment visual acuities in the treated group. At the initial visit, the best-corrected visual acuities were 20/40 and better in eight eyes and between 20/200-20/50 in twelve eyes. At the last visit the best-corrected visual acuities were 20/40 and better in nine eyes and between 20/200-20/50 in nine eyes, and <20/200 in two eyes. In the untreated group, at the initial visit, the best-corrected visual acuities were 20/40 and better in two eyes, between 20/200-20/50 in three eyes, and < 20/200 in four eyes. At the last visit the best-corrected visual acuities were 20/40 and better in one eye, between 20/200-20/50 in one eye, and < 20/200 in six eyes.

Seven of twenty treated eyes (35.0%) experienced an increase in best-corrected visual acuity, varying between two and six Snellen lines, whereas visual acuity decreased in six (30.0%) eyes, and no change in visual acuity of the remaining seven (35.0%) eyes was recorded (Figure-1). In eight eyes without any treatment no obvious change was detected in the best-corrected visual acuities during the follow-up period.

DISCUSSION

Though peripapillary SRNM has a relatively favorable course compared to macular SRNM, still, peripapillary SRNM can cause a significant decrease in visual acuity. When peripapillary SRNM is diagnosed, first, underlying clinical cause should be elucidated; second, photocoagulation options should be evaluated. Sabates et al²⁰ found that 8% of the lesions were located in peripapillary region in 375 consecutive cases with senile macular degeneration. In our study group, thirteen of 26 eyes (50.0%) had age-related macular degeneration. Age-related macular degeneration seems to be the most frequent cause in peripapillary SRNM. In the present study in seven eyes with ARMD, four had increase in visual acuity, two decrease in visual acuity and the remaining one eye had no visual change.

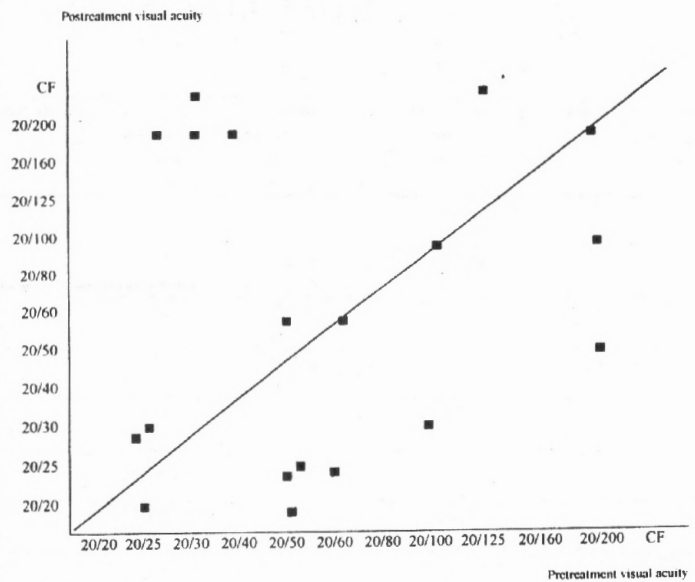


Fig-1: Scattergram of the pretreatment and post-treatment visual acuities

Singerman²¹ studied eight patients with active extrafoveal choroidal neovascular membranes related to angioid streaks. One eye of four patients was treated with argon laser. Both eyes of two patients received treatment. Two patients were not treated. Strikingly, four patients received multiple treatments at different times for recurrence of neovascular membranes. Visual acuity of the treated eyes remained either stable or slightly improved, with one exception. On the other hand the majority of untreated eyes lost vision. During their follow-up recurrences occurred in four of the treated eight eyes demonstrating that these cases should be followed indefinitely. They suggested that carefully selected cases appeared to benefit from complete obliteration of choroidal neovascularization by argon laser photocoagulation. In our study group, seven of five eyes with peripapillary SRNM and angioid streaks received photocoagulation therapy except two eyes with subfoveal extension. In one eye due to persistence in fundus angiography appearance retreatment was considered. In four eyes visual acuity decreased despite anatomically successful laser therapy. Only in one eye visual acuity remained unchanged. Gelisken et al²² had treated 30 eyes of 24 patients with angioid streaks and subretinal neovascular membranes by photocoagulation. Of 30 eyes, 16 showed either ameliorated or unchanged visual acuity. Twelve of

the remaining 14 eyes retained a visual acuity of 20/200 or better. In 11 patients, the fellow untreated eye showed central macular degeneration with loss of central vision. The follow-up period ranged from two months to 16 years (mean, 3.4 years).

Macular Photocoagulation Group had concluded that argon laser photocoagulation is useful in preventing severe visual loss in patients with idiopathic choroidal neovascular membrane (CNVM). In our group, supporting this view all three eyes with idiopathic peripapillary SRNM had an increase in visual acuity after laser treatment.

Jalkh et al²⁴ applied laser photocoagulation for extrafoveal choroidal new vessels in 19 eyes associated with degenerative myopia. Visual acuity improved only in two eyes (11%), stabilized in four eyes (21%), and deteriorated in 13 eyes (68%). In the present study only in one eye with peripapillary SRNM and degenerative myopia laser photocoagulation was performed and in the follow-up no change in the visual acuity was detected.

Harris et al²⁵ found peripapillary choroidal neovascular membranes with optic disk drusen in seven eyes of 57 patients. Six of the seven eyes had retained visual acuities of 6/12 (20/40) or better without any therapy. Thus, despite potential submacular hemorrhage, the authors did not recommend routine photocoagulation for peripapillary choroidal neovascular membranes secondary to optic disk drusen. Laser photocoagulation was applied in our only such patient and no change in the visual acuity was encountered.

Dailey⁴ and Yedavally⁵ proposed photocoagulation in their cases with peripapillary SRNM with optic disc coloboma. Although our only such patient declined treatment, no visual change was noted.

Some laser surgeons are unwilling to treat eyes with peripapillary SRNM as the natural course of these lesions, especially when far from the fovea, is somewhat favourable²⁶⁻²⁹. Complications of photocoagulation include, thermal-induced retinal vasculitis and sudden visual loss due to heat transfer into the optic nerve head at sites of macular nerve bundle entry³⁰,

recurrence³¹⁻³², photocoagulation scar enlargement³¹⁻³⁴, paracentral scotoma, inadvertent foveal photocoagulation, retinal pigment epithelial tear³⁵⁻³⁶, hemorrhage and choroidal ischemia³⁷⁻³⁸.

Due to photocoagulation complications cited above, asymptomatic patients with good visual acuity can be followed-up closely so that treatment may be performed if SRNM extends under the fovea.

Flaxel et al³⁹ reviewed the clinical course of 12 eyes of seven patients with very large SRNMs. Ten eyes received laser treatment, and two received no treatment. Of the ten eyes receiving laser treatment, six showed stabilization of visual acuity, whereas in four the neovascular membrane progressed beneath the fovea with severe visual loss. In the two untreated eyes, the subretinal neovascular membrane progressed beneath the macula with loss of central vision. In another study, 113 eyes of 112 patients having either peripapillary SRNM or SRNM that was located nasal to the fovea and larger than 750µm in the longest diameter, associated with either ocular histoplasmosis or idiopathic causes, were assessed. Forty-nine of 113 eyes had peripapillary SRNM. Although the treatment benefit for eyes with peripapillary lesions was not statistically significant, the authors still recommended considering treatment of eyes with peripapillary lesions²⁶.

In our series, all treated eyes had final best-corrected visual acuities of 20/100 or better and 71.4% had visual acuity of 20/40 or better. Our results are comparable to the study of Turcotte et al⁴⁰. Of the total of 28 eyes, they treated 21 eyes by krypton red laser, five eyes by argon green and two eyes with a combination of argon green and krypton lasers. Twenty-one (75%) either improved or remained within one line of baseline best-corrected visual acuity. They also noted optic disc changes in four eyes (14%) which were either sectorial or diffuse optic nerve pallor. In our group of 14 patients with treatment, no complication was detected. We believe that at least symptomatic peripapillary SRNMs should be treated and that peripapillary SRNMs have a relatively favourable prognosis except angioid streaks and ARMD following argon laser photocoagulation unless SRNM extends under the fovea.

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Ophthalmic "Pastpourri"

SILAS WEIR MITCHELL AND RESEARCH IN OPHTHALMOLOGY EYESTRAIN AND HEADACHES

Silas Weir Mitchell had another profound effect on ophthalmology. With William Thomson, a Philadelphia ophthalmologist, he was the first to recognize the frequent causative relation of eyestrain to headache. His views on this controversial subject became gospel to many ophthalmologists of his time. Foremost in the ranks of the disciples was George Milbury Gould of Biographic Clinics fame.

Jehangir Durrani
MD, FACS, FRCOPHTH

Synder C. Our Ophthalmic Heritage. Little, Brown and Company. Boston. 1967;p 124

Reduction in Anterior Capsular Opening Area After Phacoemulsification (Capsular Phymosis)

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ABSTRACT

Fifty-seven eyes of fifty-seven patients were studied for reduction in the size of the anterior capsular opening after phacoemulsification with PMMA posterior chamber intraocular lens implant. The diameter of Anterior Capsular Opening (ACO) was measured with slit-lamp beam and the area was calculated. After six months of follow-up average reduction in the area of ACO was 13.4% from baseline, minimum reduction was 6% and maximum was 35%. None of the patients developed decreased visual acuity or subjective symptoms due to reduction in ACO area. The risk of developing severe constriction of ACO after phacoemulsification with PMMA lens implant in non-predisposed individuals is minimal.

INTRODUCTION

Phacoemulsification with posterior chamber intraocular lens implant is rapidly becoming the standard treatment for age-related cataract, but the large rim of the anterior capsule left after capsulorhexis has its merits and demerits. Capsulorhexis ensures in-the-bag placement of the intraocular lens, adds to the stability of the implanted intraocular lens and large leaflets of the anterior capsule provide support for implantation of posterior chamber intraocular lens in the event of posterior capsular rupture with insufficient support from the posterior capsule. However, sometimes progressive decrease in the size of already small anterior capsular opening (ACO) can occur and can be severe enough to interfere with patient's vision requiring treatment. There are various risk factors identified with increased incidence and severity of constriction of anterior capsular opening. These risk factors include pseudoexfoliation syndrome, retinitis pigmentosa, diabetes mellitus, myotonic dystrophy and silicone intraocular lens with plate-haptics¹⁻⁵.

This study was carried out to determine the decrease in the size of anterior capsular opening after phacoemulsification with PMMA posterior chamber intraocular lens implant and to see the effect of this constriction on patient's vision.

MATERIALS AND METHODS

The study was carried out at the Institute of Ophthalmology, Mayo Hospital, Lahore, from December 1998 to November 1999. A total of 57 eyes (57 patients) of both sexes and all ages that underwent uncomplicated phacoemulsification and PMMA single-piece posterior chamber lens implant were included in the study. Continuous Curvilinear Capsulorhexis (CCC), circular in shape, was carried out using 26 gauge bent needle under viscoelastic (hydroxymethylcellulose). The patients were followed-up for a minimum period of six months. Evaluation of the area of anterior capsular opening was made at the first postoperative day, two weeks, six weeks and six months after surgery. During the follow-up visits patients had their corrected visual acuity checked, and were also inquired about the quality of vision.

Diameter of the anterior capsular opening was measured with slit-lamp beam at 90° and 180° and surface area of the anterior capsular opening was calculated using the formula:

$$\text{Surface Area (mm}^2\text{)} = \frac{1}{4} \pi b h$$

where b is the diameter in horizontal meridian in mm and h is the diameter in vertical meridian in mm.

RESULTS

Out of a total of 57 patients, 28 (49.1%) were males and 29 (50.9%) were females. The age distribution is shown in Table-1.

Table-1: Age distribution

Age range (Yrs)	No. of Patients	Percentage
30-40	08	14.0
41-50	08	14.0
51-60	19	33.0
> 60	22	39.0

Initial capsulorhexis diameters on the first postoperative day ranged between 4.2 mm and 5.7 mm, while the average CCC diameter was 4.9 mm and the average ACO area was 18.86 mm².

At two weeks after surgery, maximum decrease in ACO was 7.6%, minimum was 2.7%, while average decrease was 5.8% from baseline ACO area measured on the first day after surgery.

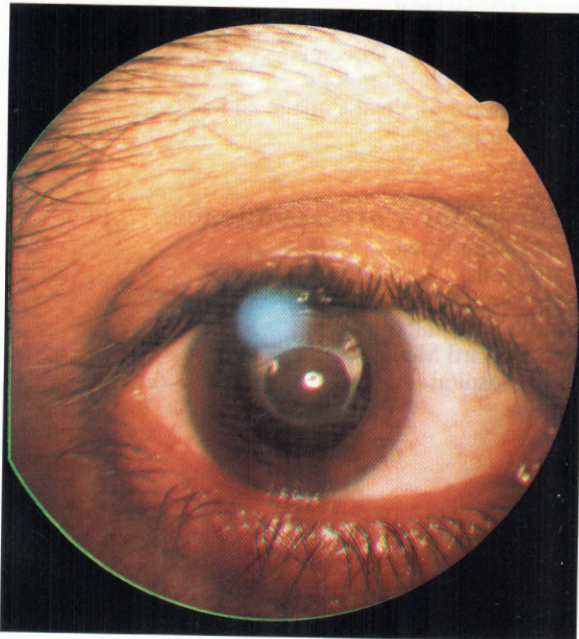


Fig-1: Capsular phymosis 6 months after phacoemulsification through Continuous Curvilinear Capsulorhexis (CCC).

At six weeks after surgery, 45 (78.9%) patients had less than 15% decrease in ACO area, while three patients showed reduction ranging between 20% – 31 % (Table-2 & Fig.1).

Table-2: Reduction in ACO area at six weeks (%)

ACO area reduction from baseline (%)	No. of Patients	Percentage of total Patients
4.7-10	17	29.8
11-15	28	49.1
16-20	09	15.8
21-30	03	05.3

After six months of follow-up, maximum ACO area reduction noted was 35% and minimum 6%, while average reduction was 13.4%. Details are shown in Table-3. None of the patients had visual problems that could be attributed to constriction of anterior capsular opening. The amount of reduction was not related to the age or sex of the patients.

Table-3: Reduction in ACO area at six months (%)

ACO area reduction from baseline (%)	No. of Patients	Percentage of total Patients
06-10	15	26.3
11-15	30	52.3
16-20	07	12.3
21-35	05	08.8

DISCUSSION

Gimbel and Neuhann first described the method of continuous curvilinear capsulorhexis (CCC). Capsulorhexis has played a very significant role to make phacoemulsification a safe procedure with minimal complications. As compared to can-opener capsulotomy a much larger margin of anterior capsule is left behind. Histological studies show that fibrous tissue from leftover anterior capsule contains activated fibroblasts with contractile ability and there is lack of TGF- β which is the most important cytokine in modulating fibroblasts⁶. In addition to shrinkage of the anterior capsule, there is also outgrowth of membrane from margins of the anterior capsule to the center of the anterior capsular opening. There have been reports of severe contraction of capsulorhexis leading to reduction in visual acuity requiring treatment with Nd: Yag capsulotomy or surgical removal of

fibrotic membrane. In our six-month follow up of patients no case of severe constriction of anterior capsular opening (capsular phimosis) was noted requiring treatment. Great majority of patients (78.9%) had reduction in ACO area that was less than 15% of the baseline ACO area measured on the first postoperative day. Most of the reduction in ACO area occurred in the first six weeks after surgery. It appears that severe constriction of the anterior capsular opening is associated with certain risk factors as mentioned earlier. None of our patients belonged to high risk groups. In a majority of the patients undergoing phacoemulsification with posterior chamber intraocular lens implant, especially PMMA lens, the risk of capsular phimosis is small. However, there is definitely some reduction in the area of ACO, and as the rim of the anterior capsule becomes opacified, this may contribute to the difficulty in visualizing the peripheral retina in these patients. A larger capsulorhexis may be performed in high risk patients to reduce the risk of severe ACO constriction.

CONCLUSION

There is some reduction in the anterior capsular opening area after phacoemulsification with PMMA posterior chamber intraocular lens implant. Most of the constriction occurs during the first six weeks postoperatively. However, the risk of severe ACO constriction may be limited to a small group of predisposed patients

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Additive Effect of Latanoprost to Topical Beta-Blockers in Lowering IOP in Primary Open-Angle Glaucoma and its Ocular Tolerance

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ABSTRACT

This ongoing study was carried out at Glaucoma Research Unit, Eye Unit I, Services Hospital, Lahore, from Feb. 1999 to date. In this trial, ocular tolerance and additive effect of 0.005% latanoprost to topical 0.5% timolol maleate were evaluated in patients with primary open-angle glaucoma whose intraocular pressure (IOP) was above the individual target level despite maximal topical beta-blocker therapy. 0.005% latanoprost was added once a day in the evening. Forty eyes of 25 patients were included with a mean IOP, before the addition of latanoprost, of 20.28 mmHg. Six eyes presented with IOP in the range of 11 to 16 mmHg, while 25 eyes presented in the range of 17-22 mmHg, and IOP in the range of 23-30 mmHg was observed in 9 eyes. Patients were followed at the 1st, 2nd and 3rd months. All the patients had minimum follow-up of three months. Reduction in IOP induced by latanoprost was 3.6 mmHg in the first group at the end of the 1st month. In group 3, reduction in IOP was 7.6 mmHg at the end of 1st month. Same trends were observed at the end of 2nd and 3rd months. Overall mean reduction in IOP induced by latanoprost was 27% at the end of 1st month, 30% at the end of 2nd month and 33% at the end of 3rd month. IOP remained stable thereafter in those patients who were followed-up for 6 months using latanoprost along with the beta-blocker. A few patients had conjunctival hyperemia, itching, burning and foreign body sensation. These symptoms were mild and transient.

INTRODUCTION

Glaucoma is one of the major causes of irreversible blindness and primary open-angle glaucoma (POAG) accounts for about 60-70% of all cases of glaucoma. Raised IOP is still being considered the primary risk factor for the development of POAG and the aim of treatment is to reduce IOP to an individual target level which might prevent optic nerve head damage and subsequent loss of visual function. The treatment of POAG is primarily medical. The current mainstay of medical management is usage of topical beta-blockers, cholinergic agonists and adrenergic agonists to decrease IOP.

Antiglaucoma drugs can act on aqueous humour dynamics to lower IOP in three ways:

- Decrease aqueous humour production in the ciliary body.
- Increase aqueous humour outflow through trabecular meshwork.
- Increase aqueous humour outflow via uveoscleral pathway.

Latanoprost is a prostaglandin analogue (PG-F₂α derivative). It is a pro-drug, which is hydrolysed in the cornea to hydrophilic acid of latanoprost. It has different and a newly described mechanism of action as compared to other antiglaucoma drugs. It decreases the IOP by increasing the uveoscleral outflow via relaxation of the smooth ciliary muscle, degradation of extracellular matrix and remodelling of collagen and change of cell shape in the extracellular matrix. Prostaglandins are additives to the ocular hypotensive agents that reduce aqueous production such as beta-adrenergic-blocking agents and carbonic anhydrase inhibitors. Duration of action is 24 hours. So latanoprost 0.005% is administered once a day, usually in the evening.

PATIENTS AND METHODS

In this ongoing study, 40 eyes of 25 patients were included who were established cases of POAG and all patients were using 0.5% timolol

maleate 12 hourly and despite that IOP level was above the individual target level. Latanoprost 0.005% was added once a day in the evening at 6.00 P.M. Follow-up was done at the 1st, 2nd and 3rd month and so on. All the patients had minimum follow-up of 3 months. Patients were asked about any untoward effects induced by latanoprost and follow-up was done noting the visual acuity, IOP, C/D ratio and iris colour.

5 patients had positive family history of glaucoma, while 8 patients had associated diabetes mellitus and hypertension. Baseline evaluation of each patient was done before inclusion in the study and this included the assessment of the visual acuity, slit-lamp biomicroscopy, particularly noting the iris colour, applanation tonometry, ophthalmoscopy, gonioscopy and perimetry.

RESULTS

The patients in this study were men and women between 30 and 76 years of age with the mean age of 53.4 years. Fourteen (56%) were males and 11(44%) were females (Table-1).

Table-1: Sex Distribution

Sex	Number	Percentage
Male	14	56
Female	11	44
Total	25	100

Thirty eyes had visual acuity in the range of 6/6 to 6/12, while 10 eyes had visual acuity in the range of 6/18 to 6/60. C/D ratio in 18 eyes was in the range of 0.4 - 0.6, while 22 eyes presented in the range of 0.7-0.9. It was noted that majority of the eyes presented with higher C/D ratio. Both the visual acuity and C/D ratio remained stable throughout the study. Mean IOP before the addition of latanoprost was 20.28 mmHg and 6 eyes presented with IOP in the range of 11-16 mmHg, while 25 eyes presented in the range of 17-22 mmHg and IOP in the range of 23-30 mmHg was observed in 9 eyes. (Table-2).

Table-2: Pre-Xalatan IOP Range

IOP mmHg	No. of Eyes
11-16	6
17-22	25
23-30	9

Reduction in IOP induced by latanoprost in group I was 3.6 mm Hg at the end of 1st month, while in group II reduction in IOP was 7.6 mm Hg at the end of 1st month, i.e. almost twice that of group I. Same trends were observed at the end of 2nd and 3rd months. Overall mean reduction in IOP induced by latanoprost was 27% at the end of 1st month, 30% at the end of 2nd month and 33% at the end of 3rd month (Table-3). IOP remained stable thereafter in patients whose follow-up was done for 6 months using latanoprost along with beta-blockers.

Table-3: Reduction in IOP Induced by Xalatan

IOP Range (mmHg)	No. of Eyes	1 st month	2 nd month	3 rd month
11-16	6	3.6	4.02	4.0
17-22	25	5.4	6.08	6.96
23-30	9	7.6	8.00	9.5
Mean reduction in IOP		5.53	6.09	6.82
Mean reduction %		27%	30%	33%

Conjunctival hyperaemia was most common followed by itching, burning and foreign body sensation. These symptoms were mild and transient. No increase in pigmentation of iris was seen after 3 months.

DISCUSSION

Glaucoma continues to be a major disease and a leading cause of blindness throughout the world. Many patients with POAG do not obtain satisfactory control of IOP with maximal topical therapy of 0.5% timolol maleate, which decreases IOP by reducing the aqueous humour production. Latanoprost reduces IOP by increasing uveoscleral outflow so it should be additive to ocular hypotensive agents that reduce aqueous production such as beta-adrenergic blocking agents.

Rulo et al¹ studied the effect of latanoprost for the first time in 20 patients with mean IOPs of 24-28 mm Hg after washout of all ocular medications. Patients were treated twice daily for 1 week with either timolol 0.5% or latanoprost 0.006%. IOP was reduced 9 mm Hg (31%) in the latanoprost-treated group and 6 mm Hg in the timolol-treated group. After 1 week of combined treatment, IOP was reduced another 3 mmHg (13 to 14%) in both groups.

The additive effect of latanoprost was studied by Villumsen et al² in 30 open-angle glaucoma patients with an IOP of 22 mmHg or greater despite treatment with twice daily timolol 0.5%. These patients were randomly assigned to receive additional treatment with either 0.5 µg latanoprost or placebo. After 7 days of combined treatment IOP was reduced by 17% compared with treatment with timolol alone.

In a 3-month study by Alm et al³ the additive effect of once or twice daily latanoprost 0.006% in combination with twice daily timolol was evaluated in 50 patients whose IOPs were inadequately controlled by timolol alone. After 3 months of combined treatment, latanoprost lowered IOP by 28% to 37% compared with the baseline of 25 mmHg on timolol alone. Interestingly, latanoprost was more additive to timolol when applied once daily than when given twice daily.

Foreign body sensation, stinging, itching, and burning and mild conjunctival hyperaemia were noted in studies by Alm et al⁴ and Watson et al⁵.

Most side effects of latanoprost observed in our study were mild and transient. All participating patients had homogeneous brown iris colours and there was no sign of increased iris pigmentation in any of the patients included in the study.

CONCLUSION

Latanoprost 0.005% is well tolerated and an effective additive ocular hypotensive agent that may provide significant further reduction of IOP in patients with POAG receiving the maximum tolerated dose of topical beta-blockers.

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Effects of Xalatan (0.005% Latanoprost) in Primary Open-Angle Glaucoma

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ABSTRACT

Purpose: To report the effects of topical latanoprost 0.005%, in three patients of primary open-angle glaucoma.

Method: Detailed medical and ophthalmic history of the patients under observation was obtained. Detailed physical and ophthalmic examination was carried out. Investigations, such as complete urine examination, complete blood examination, and random blood sugar were carried out. In the ophthalmic examination, visual acuity, slit-lamp examination, fundus examination, Goldmann perimetry and applanation tonometry were performed. One drop of Xalatan was instilled in each eye in the evening.

The cases were followed-up according to the following schedule:

One day after starting treatment with Xalatan eye drops and then one week, two weeks, four weeks, eight, twelve, and sixteen weeks after treatment with Xalatan. On each visit detailed ophthalmic examination was carried out giving special attention to IOP, Iris pigmentation, hyperpigmentation of eyelashes, conjunctivitis, anterior uveitis, choroidal effusion, cystoid macular oedema, facial rash or other allergic manifestations. On each visit each patient was questioned about subjective symptoms such as any foreign body or stinging sensation, itching, burning, tearing or pain in the eye.

INTRODUCTION

Primary open-angle glaucoma (POAG) accounts for about 90% of all cases of glaucoma¹. It is thought that optic nerve ischemia or mechanical optic nerve damage may result from elevated IOP. POAG usually has insidious and asymptomatic onset initially causing midperipheral visual field impairment.

Measuring the IOP (tonometry), testing visual fields (perimetry) and examining the optic nerve head for cupping and pallor (funduscopy) make the diagnosis of POAG.

High myopia, previous history of eye trauma and diabetes mellitus are high risks². Arterial hypertension is also a risk factor for POAG³.

Xalatan is a topical solution containing latanoprost (PGF₂ – isopropylester 0.005%) as the

active ingredient. It is a prostaglandin analogue. It binds with prostanoid receptors on the cornea from where it is absorbed into the mucosa and ultimately goes into systemic circulation where it is hydrolyzed in the blood (Pharmacia Upjohn).

In 1994, Rulo described that latanoprost does not cross the blood-retinal barrier⁴.

In 1995 Alm and in 1996 Camras concluded that latanoprost may enhance iris colour by increasing the amount of brown pigment of the iris^{5,6}.

Sudesh et al described corneal toxicity associated with latanoprost in four cases. They explained that the development of dendritiform epitheliopathy as a sign of corneal toxicity has been previously described with the use of topical antivirals, antibiotics, β -blockers and the preservatives in contact lens solutions⁷.

Camras et al came to the conclusion that PhXA34 (prostaglandin analogue) caused a significant dose-dependent reduction of IOP beginning at 4 and 8 hours after the first application of 0.01% and 0.003% doses, respectively and lasting throughout the 6 days of treatment⁸.

Grierson et al described a case that developed eye colour changes with latanoprost. They have done histopathology of the fine structure of an iridectomy specimen from a suspected case, and found that the stroma between the anterior border and the sphincter muscle was hypercellular and the cells contained melanin granules and some of them were pigmented⁹.

Azuara-Blanco et al, noted that the use of latanoprost lowered the IOP in most glaucomatous patients already receiving maximal tolerated medical treatment¹⁰.

Linden et al concluded that long-term treatment with latanoprost has no clinically important effects on the permeability of blood-aqueous barrier and IOP will return to pretreatment level within a few weeks after discontinuation of the treatment¹¹.

Johnstone found hypertrichosis and increased pigmentation of eyelashes and adjacent hair in the region of the ipsilateral eyelids of the patients treated with unilateral topical latanoprost¹².

Rowe et al reported ocular hypotony, choroidal effusions, recurrent cystoid macular oedema and facial rash after 1-4 week's treatment with latanoprost¹³.

Krohn and Hove described iris cysts associated with topical administration of latanoprost¹⁴.

Case-I

A 50-year-old male was seen on 26-11-98 with increased intraocular pressure in both eyes. He was using Blotim 0.5% eye drops B.D. in both eyes for the last six months. On history and examination he was found to be nondiabetic, normotensive, nonasthmatic and with no family history of glaucoma. He gave history of diamox sensitivity. He was a high myope using -15D glasses (both eyes) and his vision with glasses was

R 6/24, L 6/24. Slit-lamp examination revealed no conjunctival congestion, no corneal oedema or keratitis, no anterior chamber activity. On gonioscopy the angles were wide open. Lens and vitreous were clear. Cup/disc ratio was R 0.5, L 0.5 and both discs were oval, with extensive myopic crescents.

The visual fiends were constricted. He was not willing for trabeculectomy or to use diamox (due to allergy) and pilocarpine (due to impairment in vision). His IOP with Goldmann applanation tonometry was R 26, L 26 on the first day of examination, on 26-11-98. He was advised to withhold the previous antiglaucoma medication and revisit after one week for reexamination.

After one week all his examination was the same except IOP which was R 30, L 30 with Goldmann applanation tonometer. He was advised to use Xalatan (0.005% latanoprost) one drop once in the evening in each eye.

After one day of Xalatan eye drops the IOP was R 17, L 17. After one week the IOP was R 16, L 15. After 2 weeks R 18, L 16; after 4 weeks R 16, L 18; after 8 weeks R 18, L 17; after 12 weeks R 16, L 16 and after 16 weeks of Xalatan eye drops the IOP was R 15, L 16.

He complained of slight discomfort in his eyes during the first week of Xalatan eye drops treatment but gradually his complaint subsided in a few days. No other subjective symptoms as F.B. sensation, stinging sensation, itching, burning or tearing were observed.

The side effects like hyperpigmentation of iris, hypertrichosis, anterior chamber activity, macular oedema or choroidal effusion were not seen during the entire follow-up period.

Comments and results

The average decrease in IOP with Xalatan eye drops (once in the evening) was 45%. There were no side effects seen on the lids, conjunctiva, cornea, anterior chamber or fundus. Once daily use of eye drops was convenient to the patient and IOP was nicely controlled.

The IOP control was comparable with the study of Ziai, Dolan and Kacere who worked on

aqueous dynamics of PhXA41 in normal and ocular hypertensive patients¹⁵.

Case II

A 52-year-old male presented on 18-02-99 with history of bilateral cataract extraction with IOL implantation last year. He had no history of diabetes mellitus, hypertension, asthma or any family history of glaucoma. On examination uncorrected vision was R 6/12, L 6/24; with pH R 6/6 P; L 6/12P; with glasses (R 6/6 P, L 6/6 P).

Visual fields were normal in both eyes. Slit-lamp examination revealed that iris colour was dark brown in both eyes and the conjunctiva was normal in both eyes. No anterior chamber activity was seen, both IOLs were properly placed and the vitreous was clear. On gonioscopy angles were wide open in both eyes. C/D ratio was 0.4 in both eyes.

He was using Blotim 0.5% (BD), Diamox tab (1xTDS), KCL tab (1xBD) and Pilocarpine 2% (6 hourly). IOP by applanation tonometry was R 23, L 21 mm Hg.

He was advised to withhold all the above treatment, both oral and topical, and come after one week of discontinuation of medicines. After the desired time duration he was examined again in detail. Now his IOP was R 27, L 26. He was advised to use only Xalatan, one drop in the evening in each eye.

On the next day the IOP was R 20, L 20. One week later the IOP was R 18, L 18. Two weeks later it was R 17, L 16. After four weeks R 18, L 17. Eight weeks after starting Xalatan the IOP was R 15, L 16. Twelve weeks after R 17, L 16. Sixteen weeks after Xalatan use the IOP was R 16, L 15.

On each visit he was questioned about subjective symptoms. He complained of F.B. sensation and stinging sensation during the first week of treatment but the problem gradually subsided.

Comments and conclusions

We noted 9.43 mm Hg (35.58%) average decrease in IOP from the baseline. There were no noticeable symptoms except F.B. sensation and stinging sensation in the early days of treatment,

which subsided without any additional treatment. No side effects like hypertrichosis, hyperpigmentation, choroidal changes or macular changes were seen in the patient during each visit and detailed follow-up examination. But after 16 weeks of treatment the patient decided to give up Xalatan drops because of the cost. He decided to undergo trabeculectomy. So regarding our general socioeconomic conditions the cost of medicine is too high.

Case-III

A 48-year-old male presented on 26.02. 99 with the complaint of pain in both eyes. After complete examination, including visual fields, IOP and fundus examination, he was diagnosed as a case of primary open-angle glaucoma. There was no history of diabetes mellitus or hypertension, or family history of glaucoma.

Examination was as under:-

Vision	R 6/18	L 6/12,
with pinhole	R 6/6 P	L 6/6 P and
with glasses	R 6/6	L 6/6

Slit-lamp examination of each eye revealed normal conjunctiva, clear cornea, quiet anterior chamber, clear lens and vitreous. Both eyes had dark brown irides, C/D ratio was R 0.3, L 0.3. IOP with applanation tonometer was R 26 mm Hg, L 26mm Hg

We started topical Xalatan (0.005% latanoprost) one drop in the evening in each eye and called the patient the next day.

One day after treatment his IOP was R 15, L 15mm Hg; one week after treatment it was R 14, L 14mm Hg; two weeks later R 14, L 14mm Hg; four weeks after R 15, L 15mm Hg; eight weeks after treatment his IOP was R 15, L 15mm Hg; twelve weeks later R 14, L 14mm Hg; sixteen weeks after treatment his IOP was R 14.5, L 14.5mm Hg. On each visit subjective symptoms as F.B. sensation, stinging sensation, tearing, burning and eye pain were asked about and there was no such problem.

On each visit during the follow-up period slit-lamp and fundus examination were carried out in addition to IOP recording. We found no

abnormalities or side effects as hyperpigmentation of iris, hypertrichosis, choroidal changes etc.

Comments and results

Xalatan decreased IOP by 11.5mmHg from the baseline (44.2%).

We came to the conclusion that the IOP lowering effect of Xalatan was very remarkable. The side effects as mentioned in different medical journals were not found. The occurrence of side effects may vary from person to person.

Patients very readily accepted the dose regimen. Xalatan proved to be a very efficient pressure-lowering agent with minimal or no side effects in Pakistani patients during the follow-up period.

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Pressure-Lowering Effect of Latanoprost in POAG and Ocular Hypertension

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ABSTRACT

Twenty eyes of ten patients with primary open-angle glaucoma (POAG) and ocular hypertension were treated with 0.005% latanoprost. Satisfactory intraocular pressure drop was obtained in all but one patient. There were few local side effects and no systemic effects were seen. Latanoprost provides safe and effective drop of intraocular pressure in POAG and ocular hypertension.

INTRODUCTION

Glaucoma is more than a pressure disease but at present pharmacological options are limited to lowering of intraocular pressure only. Intensive research in recent years has widened the choice of pressure-lowering drugs. There are four classes of new drugs that appear promising in long-term pharmacological management of glaucoma. These are: topically active carbonic anhydrase inhibitors (dorzolamide), alpha-2 adrenergic agonists (brimonidine), prostaglandins (latanoprost) and ethacrynic acid. Amongst these newer pressure-lowering agents latanoprost (Ph X A41) has an entirely different mode of action. It increases the uveoscleral outflow by increased percolation of aqueous humor through ciliary muscle, suprachoroidal space and sclera. This different mode of action makes it potentially additive to other currently available drugs.

MATERIALS AND METHODS

The study started on 11-02-1999. The patients were selected from outdoor department of the Institute of Ophthalmology, Mayo Hospital, Lahore.

The study was carried out with the following aims and objectives:

1. To study the pressure-lowering effect of latanoprost in primary open-angle glaucoma and ocular hypertension.

1. To report any side effects observed during the study or follow-up period.

The patients that fulfilled the following criteria were included in the study:

1. Patients aged 18 years or more.
2. Patients with uniocular or binocular primary open-angle glaucoma or ocular hypertension.
3. Patients already on a single pressure-lowering drug were eligible for study after a medication-free period. The drug washout period was five days for cholinergic drugs and three weeks for beta-blockers.

Patients with any of the following were excluded from the study:

1. Known sensitivity to vehicle components (Benzalkonium chloride).
2. History of angle-closure glaucoma.
3. History of argon laser trabeculoplasty (ALT) or surgery within three months prior to the start of study.
4. Closed or barely open angles.
5. Ocular infection/inflammation within three months of the start of study.
6. Any condition preventing reliable applanation tonometry.
7. Pregnancy and nursing mothers.

Latanoprost eye drops (50 microgram/ml) were administered once daily by the patients at about 8:00 p.m. No other drugs, topical or

systemic, known to affect IOP were allowed during the study period

PATIENT EVALUATION

Intraocular pressure measurement was done using Goldmann applanation tonometer. All the measurements were done by the same observer preferably using the same instrument. At each measurement three readings were taken and average of these was used for statistical analysis. At the baseline and concluding visits IOP was measured at 9:00, 13:00 and 17:00 hours, while at two and six weeks visits only 9:00 measurement was made.

Visual fields were documented with automated static perimetry at the start and the conclusion of treatment. Visual fields done within six months before the start of the study were also acceptable.

Iris photographs were taken at the start and the end of treatment period to document any change in iris color after 12 weeks of treatment.

Visual acuity was measured at each visit and refraction was done. Detailed slit-lamp examination and dilated fundus examination were also performed at the first and the concluding visits. Gonioscopy was done at baseline to exclude narrow-angle.

At each visit blood pressure and pulse rate were measured after at least 10 minute's rest and patients were questioned about any side effects, both local and general.

TREATMENT SCHEDULE

Patients were treated with latanoprost for a period of twelve weeks. An additional follow-up visit was scheduled at 14 to 16 weeks to observe any side effects. After the baseline evaluation and the start of treatment the patients were assessed at two, six and twelve weeks. All the topical or systemic drugs known to affect IOP were not used during the study period.

By September 1999 a total of 10 patients were studied. Six patients had primary open-angle glaucoma, while four patients were diagnosed as having ocular hypertension. Seven patients completed the study. Two patients dropped out as they failed to appear for follow-up. In one patient

latanoprost was discontinued after three weeks due to unsatisfactory IOP control.

RESULTS

The baseline IOPs at the start of the study ranged between 23mmHg and 30mmHg. Average IOP was 27.1mmHg (Table-1).

Maximum IOP drops observed were 10.3mmHg and 39% of baseline IOP (Table-2). Minimum IOP drop seen was 2.6mmHg (10.5% of baseline IOP). Average IOP fall seen was 8.2mmHg (29.8%). IOP fall at the conclusion of treatment (12 weeks) is shown except for patient No.6. In this patient treatment was discontinued after three weeks and these three-week readings were taken as final.

SIDE EFFECTS

No significant side effects, local or systemic, were seen during the study and follow-up periods. Only one patient reported mild local irritation with transient hyperemia that disappeared after a few weeks of use. No change in iris color was seen after twelve weeks of latanoprost use. No change in heart rate or blood pressure was noted after latanoprost use.

DISCUSSION

Initial results from this small series show 0.005% latanoprost eye drops to be effective in lowering intraocular pressure in primary open-angle glaucoma and ocular hypertension when used as monotherapy. Average intraocular pressure drop was 29.8% from baseline IOP that is equal to or better than the control achieved with beta-blockers^{1,2}. Only in one study patient the drug was discontinued due to unsatisfactory control of intraocular pressure. Intraocular pressure drop ranged between 10.5% and 39%. Patients may vary in their responses to pressure-lowering effect of latanoprost. Intraocular pressure drop achieved with initial treatment was maintained throughout the twelve weeks of therapy. While latanoprost is effective in lowering intraocular pressure when used as a single drug, its different mechanism of action makes it a very suitable drug as an additive to beta-blockers where beta blockers alone cannot achieve satisfactory intraocular pressure control. Combination of latanoprost with pilocarpine has

Table-1: Baseline IOP

Patient No.	INTRAOCULAR PRESSURE IN mmHg					
	9:00		13:00		17:00	
	Right	Left	Right	Left	Right	Left
1	27	29	28	30	28	30
2	28	28	28	28	27	28
3	24	26	23	26	23	28
4	28	27	28	26	28	26
5	28	26	27	28	29	27
6	25	24	25	26	27	24
7	29	29	28	28	27	28
8	29	28	27	26	29	26

Table-2: IOP drop from baseline

Patient No.	Average baseline IOP		IOP drop mmHg		% Drop	
	Right	Left	Right	Left	Right	Left
1	27.6	29.6	7.6	8.0	27.5	27.0
2	27.6	28.0	9.6	8.4	34.8	30.0
3	23.3	26.6	7.0	7.3	29.6	27.4
4	28.0	26.3	10.0	8.7	35.7	33.0
5	28.0	26.3	10.4	10.3	37.0	39.0
6	25.6	24.6	3.6	2.6	14.0	10.5
7	28.0	28.3	10.0	9.3	35.7	32.8
8	28.3	26.6	8.3	10.0	29.3	38.5

not been found to be additive because these two drugs have opposing effects on uveoscleral outflow of aqueous. An interesting side effect observed after latanoprost use in previous studies was a change in iris color. Increase in iris pigmentation is due to increase in the amount of melanin. No proliferative activity of melanocytes has been observed. This change has been shown to occur only in heterogeneously light-colored irides like green-brown or blue/gray-brown³. This change in iris color is especially a problem when using latanoprost in one eye only. Most of our population has brown irides. None of our patients showed a change in iris color after twelve weeks of latanoprost use. Being a prostaglandin there has been a concern that latanoprost may initiate or reactivate inflammatory response or may lead to cystoid macular edema⁴. None of our patients

showed anterior chamber activity or cystoid macular edema after latanoprost use. In this study patients with history of inflammatory disease or surgery within the previous three months were excluded. No change in heart rate or alteration of blood pressure was seen in any patient. Unlike beta-blockers where systemic side effects are a major problem latanoprost is remarkably free from systemic side effects.

CONCLUSION

Latanoprost when used alone in the treatment of primary open-angle glaucoma or ocular hypertension is very effective in lowering intraocular pressure. It has very good potential for additive pressure-lowering effect when used in combination with beta-blockers. There is minimal

systemic toxicity with latanoprost and few local side effects. Latanoprost is a very useful addition to pressure-lowering drugs used in the treatment of glaucoma and has a potential for becoming a frontline anti-glaucoma drug.

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Ophthalmic "Pastpourri"

SILAS WEIR MITCHELL AND RESEARCH IN OPHTHALMOLOGY ADVANTAGES OF ANTIMETROPIA

Mitchell's eyes merit a special remark. He had very remarkable eyesight and attributed this to the fact that one eye was farsighted and the other eye nearsighted. When he wished to look at something in the distance he would use the farsighted eye and suppress the nearsighted eye. For near work he reversed the process. This meant he was equipped with an extraordinary range of vision. Although he lived until he was eighty-five, he never wore eyeglasses or suffered from any eye problems. He often said the Almighty should equip all human beings as he had been equipped.

Jehangir Durrani
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External Dacryocystorhinostomy with Silicone Tube Intubation

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ABSTRACT

A prospective randomized study on external dacryocystorhinostomy with silicone tube intubation was conducted at the Department of Ophthalmology, Unit-II, Dow Medical College and Civil Hospital, Karachi. A total of sixty-two consecutive procedures were carried out between April 1997 and October 1999. Forty-eight patients were females and 14 were males. The study was conducted to determine the efficacy of this procedure to overcome the nasolacrimal duct blockage. These patients were followed-up for a mean period of 12 months. Of the 62 procedures, 54 (87.09%) were successful, being patent at the end of the follow-up period, while 8 (12.91%) patients continued to have symptoms. The details of the procedures are highlighted and the causes of failure in these 8 cases are analyzed.

INTRODUCTION

Watering eye resulting from obstruction of the nasolacrimal duct is a common ophthalmic problem¹. The main presenting symptom of chronic dacryocystitis is epiphora. Dacryocystitis is a common infection of the lacrimal sac. In adults, dacryocystitis results from nasolacrimal duct obstruction. It is usually unilateral in nature². Bilateral acquired nasolacrimal duct obstruction secondary to Wegener's granulomatosis, sarcoidosis and chronic lymphocytic leukemia have been noted³.

The traditional surgical approach to distal nasolacrimal duct obstruction is external dacryocystorhinostomy (DCR)⁴. It is not a common procedure for most general ophthalmologists to perform⁵. The success rate of external DCR has been reported at between 80% to 99% depending upon the surgeon's experience⁶⁻¹⁴.

Toti¹⁵ first described the external DCR in 1904. This was modified by others. Ohm¹⁶ sutured the anterior and posterior flaps of nasal mucosa with the lacrimal sac. Dupuy-Dutemps and Bourguet¹⁷ described mucosal anastomosis with suturing of the mucosal flaps. Iliff⁹ sutured a rubber catheter into the sac. Older¹⁸ advocated routine use of silicone tube intubation as a useful adjunct to external DCR procedure.

External DCR is designed to create a surgical fistula between the lacrimal sac and middle meatus of

the nose through a short circuit made in the lacrimal bone and the nasal mucosa leading to drainage of ocular secretions in the nasal cavity bypassing the nasolacrimal duct. Lacrimal intubation increases the long-term success rate of external DCR^{5, 19}.

A prospective study on external DCR was conducted at the Department of Ophthalmology, Dow Medical College and Civil Hospital, Karachi, between April 1997 and October 1999. Sixty-two consecutive procedures performed during this period were included in this study. These patients were followed-up for a mean period of 12 months. Successful patency at the end of follow-up period was achieved in 54 cases (87.09%), while 8 cases (12.91%) remained symptomatic. The causes of failure are analyzed.

PATIENTS AND METHODS

Patients with epiphora and chronic dacryocystitis were entered prospectively at random into the study between April 1997 and October 1999. All the patients were recruited to the study from the eye outpatients, Unit-II, Dow Medical College and Civil Hospital, Karachi. The patient's age, sex, lateralization and the indication for surgery were noted. These patients of chronic dacryocystitis were evaluated and selected for surgery on the basis of the following:

1. Regurgitation test
2. Jones dye test
3. Probing
4. Syringing

5. Nasal examination
6. Dacryocystography, where indicated

SURGICAL TECHNIQUE

All procedures were performed under general anesthesia. The surgical techniques used were as follows:

Good hemostasis achieved by:

1. Intermittent positive pressure ventilation
2. Nasal packing soaked with xylocaine 4% and adrenaline (1:1000)
3. Head tilt 15° - 20° upwards
4. Surgical site was infiltrated with 2% xylocaine and adrenaline (1: 100,000)
5. Mopping, minimal
6. Suction, where necessary
7. Bipolar cautery, only when necessary

A straight, vertical incision, 5mm lateral to the inner canthus was made to avoid the angular vessels, approximately 20mm in length, 2-3mm above the medial palpebral ligament. The orbicularis muscle fibers were separated with blunt dissection. Four, 4/0 black silk traction sutures were applied for adequate retraction and good exposure of the surgical field. Incision of the medial palpebral ligament revealed the lacrimal sac which was retracted laterally with a flat retractor. The periosteum overlying and medial to the anterior lacrimal crest was exposed. The periosteal incision was made just medial and inferior to the bony insertion of the medial canthal tendon. The osteotomy, approximately 12x10mm in size, oval in shape, wider below, with smooth edges and round corners, was created with bone trephine and punch, limited posteriorly by the posterior lacrimal crest, superiorly by the medial palpebral ligament and inferiorly by the nasolacrimal duct. The lacrimal sac and the nasal mucosa were opened in a longitudinal fashion to form the anterior and posterior flaps. While making lacrimal sac flaps, the anterior flap was left small and the posterior larger, whereas in the nasal mucosa, the anterior flap was left large and the posterior small. First, the posterior flaps were sutured with two or three 5-0 vicryl sutures and the silicone tube was inserted. The Watzki sleeve was applied to the silicone tube just above the plane of exit from the nostril to secure it. The anterior flaps were sutured. The periosteum and orbicularis muscle were closed in separate layers with 5-0 vicryl sutures. The skin incision was closed with subcuticular, continuous 4-0 black silk suture. Nasal packing was applied. After surgery the patients used

antibiotic eye drops three times daily for two weeks and ointment at night and oral antibiotics for one week. The first follow-up was the day after surgery, then at one week, and one month with subsequent visits at 2-3 months intervals. At 6 months after surgery, silicone tube was removed. A successful outcome was defined as resolution of symptomatic epiphora and negative regurgitation test. Patients having persistent postoperative epiphora were classified as failed DCR and were evaluated with dye test, syringing, diagnostic probing and/or dacryocystography.

RESULTS

Sixty-two operations of external DCR with silicone tube intubation were performed between April 1997 and October 1999 in a prospectively randomized fashion on sixty-one unselected patients (Table-1) with primary acquired nasolacrimal sac or duct obstruction. There were 48 females and 14 males. The female to male ratio was > 3 to 1. Patients ranged in age from 16-71 years (Table-2).

Table-1: Patient Data

Characteristics	External DCR with silicone tube intubation
No. of Patients/Operations	61 / 62
Age (Years)	
Mean	53
Range	16-71
Sex	
Male	14
Female	47
Laterality of surgery	
Right	20
Left	42

Table-2: Age Distribution

Years	No. of Patients
10-19	01
20-29	05
30-39	08
40-49	12
50-59	22
60-69	12
70-79	01
Total	61

We had only one patient who underwent a bilateral procedure. The removal of silicone tube was scheduled at six months after surgery but in two

patients the tube was removed earlier because of accidental injury to the tube and infection. The operation was classified as successful if the patients had no symptoms at the end of follow-up period. Of the sixty-two procedures, 54 (87.09%) were successful (Table-3), while 8 (12.91%) patients continued to have symptoms (Table-4). Patients were followed-up for a mean period of 12 months.

Table-3: Comparison of Success Rate

S.No	Author	Success Rate %
1	Current Study (1999)	87
2	Dareeshani et al ²⁵ (1996)	94-98
3	Ashraf ²⁴ (1996)	100
4	Kundi et al ²³ (1985)	97
5	Halepota ²² (1985)	90
6	Hartikainen et al ⁴ (1998)	91
7	Tarbet et al ²¹ (1995)	83
8	Dupuy-Dutemps and Bourguet ¹⁷ (1921)	95.70

Table-4: Causes of Failure of External DCR

S.No	Literature review	Current study (No. of Patients)
1	Bony ostium closure ²¹	02
2	Fibrous closure of ostium ²⁷	03
3	Common canalicular obstruction ²⁷	03
4	Ostium opening into anterior ethmoidal sinus ³²	Nil
5	Ethmoidal sinus disease ³²	Nil
6	Active systemic disease ¹⁰	Nil
7	Sump syndrome ³³	Nil

DISCUSSION

The aim of DCR is to leave the patient with a patent unscarred rhinostomy in order to create a low pressure lacrimal bypass system and hence relieve his or her epiphora, dacryocystitis or mucocele. DCR has been accepted as a highly successful procedure in dealing with epiphora from nasolacrimal duct obstruction⁶⁻¹⁴. It is prudent to remember the conclusion by Welham and Wulc¹¹ that "it is often not enough, and never adequate, simply to punch a hole in the nose and line it with tubing in the hope that mucosa will grow round it before a fibrous scar can form". We

performed external DCR with the technique of Dupuy-Dutemps and Bourguet¹⁷, in which the anterior and posterior flaps are sutured to achieve a controlled epithelium-lined anastomosis. The external DCR, with various modifications, has consistently yielded success rates of approximately 90%^{6-14,19-21}. In our experience external DCR with silicone tube intubation is a useful modified procedure that possibly increases the success rate of this procedure. This reflects in the 87% success rate in our patients. This rate compares well with other local²²⁻²⁵ as well as international studies^{6-14,19-21}. Halepota²² reported success rate of 90%. Kundi and associates²³ achieved success rates of 97%. Ashraf²⁴ quoted 100% success in his results. Analysis of Dareeshani and associates²⁵ showed 94-98% success rate. Linberg and McCormick²⁶ showed that a mean intranasal ostium size of 1.8mm was enough to be successful in external DCR. There still are 10% of patients in whom the procedure does not succeed, Roper-Hall²⁷ suggested that fibrous tissue growth, scarring and granulation tissue formation during healing process will decrease or compromise the created surface area of the osteotomy site, leading to surgical failure. Causes of failed DCR include inappropriate size or location of bony ostium, common canalicular obstruction, scarring within the rhinostomy, intervening ethmoid sinus air cells, interference of middle turbinate, sump syndrome and active systemic diseases²⁸⁻³³. Eight (12.9%) of our cases continued to have symptoms of epiphora. The causes of failure in our patients were common canalicular obstruction in three, fibrous closure of ostium in three and two cases with bony ostium problem. McLachlan et al²⁸ propose the higher incidence of common canalicular obstruction as a cause of external DCR failure. Endonasal laser DCR has now come of age⁴. The future of lacrimal surgery is changing but external DCR with silicone tube intubation still remains the gold standard by which other modern methods⁴ are measured. We conclude that external DCR with silicone tube intubation is a superior operation, needing only a few postoperative follow-up visits and with a small cutaneous scar.

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Retinal Detachment: A Beginner's Approach

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ABSTRACT

The beginners in the specialty DETACH themselves from RETINAL DETACHMENT surgery from the outset for various reasons. The article emphasizes that retinal surgery is well within their reach, more so when simple detachment is considered. Preexisting knowledge is reorganized to produce precision and simplicity. It elaborates certain mandatory headings to be kept in mind in diagnosis and surgical management. Diagnostic problems, e.g. in relation to media and indirect ophthalmoscopy are classified as the primary deterrents, whereas persistence or recurrence of detachment is the secondary deterrent for a beginner.

The recommended methodology is based on observations, collected over a period in excess of six years, of retinal surgery in THE ARMED FORCES. It is intended that it provide encouragement for beginners, the purpose being: RETINAL SURGERY MADE EASY.

INTRODUCTION

For many-a-newcomer in our specialty, the posterior segment in general and retinal detachment (RD) in particular, is a thing to be dreaded and if possible avoided. Hence, consciously as well as subconsciously, they detach themselves from RD from the very outset of their careers. The question is: *why do they do so?* Various reasons as to the dropout are shown in the flow chart.

We would like to share an experience, spanning over a period in excess of six years, in managing all types of RD, restricting ourselves to simple/uncomplicated RD management.

The most important aspect of RD management is the psyche with which one approaches such a case, believing that each case of RD is well within one's reach. Furthermore, a retinal surgeon requires lots of patience and persistence. The surgical outcome of all types of surgery is based on a balance between expertise and the facilities available.

It is mandatory that a methodical approach, both in diagnosis and treatment, be followed. Indirect ophthalmoscopy (IO) is a cornerstone in the diagnosis as well as treatment of RD. Its techniques are discussed in detail, keeping in view the abilities of a beginner.

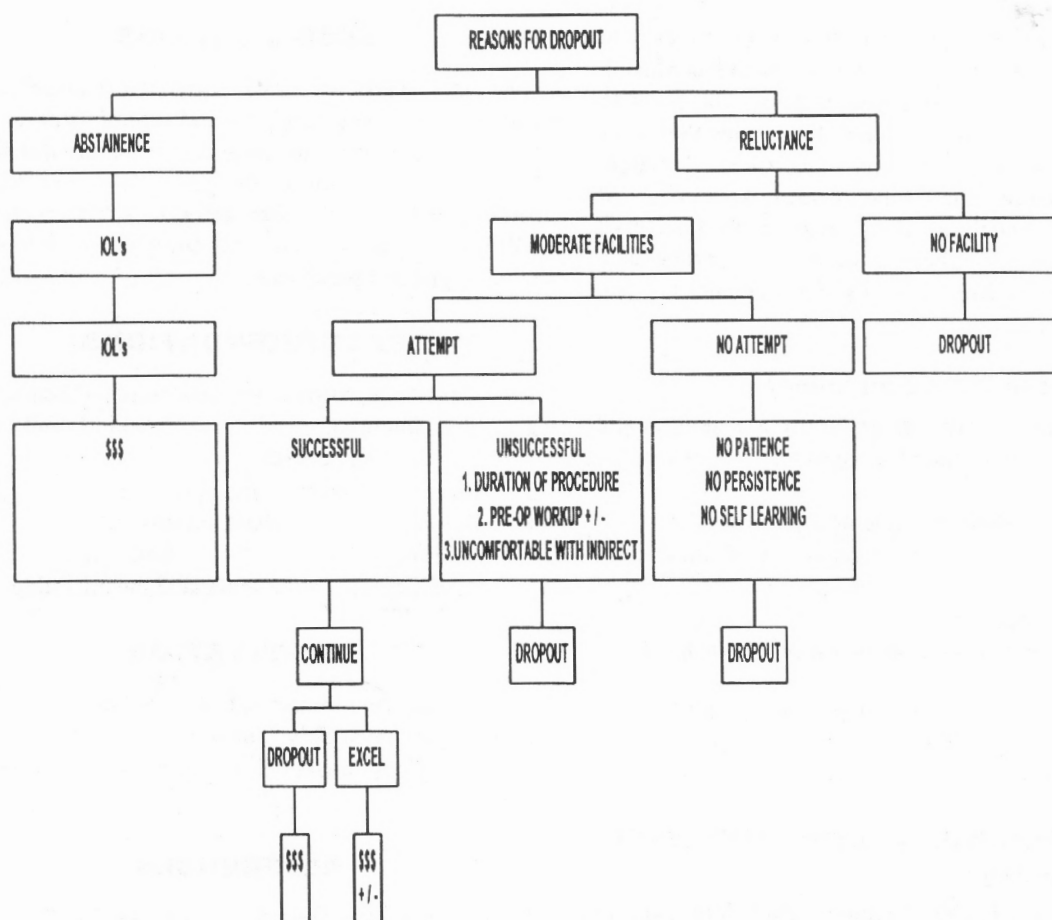
INDIRECT OPHTHALMOSCOPY

IO is the central pivot round which the diagnosis and management of RD revolves. Therefore, in order to

have good results, one should master the technique of IO. How? The answer: ***practice, practice and practice!*** There is no shortcut to hours of working with an indirect ophthalmoscope - as the saying goes "Practice makes a man perfect". So, ***practice, persistence and patience*** are the keys to good RD management.

There are certain determinants, some static and others dynamic, which help one in performing IO. Regarding the static determinants, the foremost is a positive approach/attitude, without which nothing can be achieved. Secondly, the type of condensing lens being used is important. For beginners we would advise the use of a 30 diopter condensing lens. It gives good control, a wide field of vision, makes the configuration of an RD easier to visualize and is friendly with small pupils especially in aphakic eyes.

Regarding the dynamic determinants, the protocol to examine the fundus with an IO includes examination without any instructions to the patient, i.e. in the primary position of gaze. This will help in establishing the configuration of the RD. Then, perform the examination with specific instructions as to where to look. This will help in confirming the extent of the RD and will also provide the subtle details. During IO, initially follow the major blood vessels back to the optic disc; then examine the equatorial region and beyond. As regards the characteristics/dynamic determinants of the RD itself, we have devised a



formula, which we have found to be quite helpful, the need for which is that the "window period" available to the examiner for actually seeing and making a mental impression of the whole pathology is quite short. The formula helps in concentrating thoroughly, with emphasis on headings as follows:

$$RD = \frac{U}{DO} + \frac{E2}{E1} + V$$

Where:

- RD = Retinal detachment
- U = U-shaped tear
- D = Dialysis
- O = Hole
- E2 = Post-equatorial
- E1 = Pre-equatorial
- V = Vitreous state

The numerators have a poorer prognosis as compared to the denominators. We advise our younger colleagues, who are new to this subspecialty, to follow

this formula; they will be much more comfortable. Furthermore, during one's initial days, we advocate that routine scleral indentation be avoided.

The above, in no way, lessens the importance of primary ophthalmic examination, which is mandatory. Furthermore, retinal drawing, again like IO, is yet another cornerstone in diagnosis as well as therapeutics of RD. So, to be good at it *one has to practice!*

TREATMENT

The treatment of RD is surgical - *the goal being to seal off all the communications between the vitreous cavity and the subretinal space (Gonin's principle)*¹ Precision in treatment is mandatory; mental analysis of the problem helps resolve it.

The treatment plan for RD surgery is A to D:

A. Aseptic inflammation induced by cryotherapy

The principle is to produce an artificial buckle by inducing aseptic inflammation by cryotherapy. Here again, orientation while using IO is

important; anteroposterior dimensions are easy to judge, whereas radial dimensions are confusing especially during one's early days - the point to remember is that for radial dimensions the probe movement is in the opposite direction. Furthermore, good topographical knowledge of the eye is helpful along with immaculate cryo probe control. Slipping of the cryo probe is a problem, again especially for beginner's - so, *practice!*

B. Buckle to induce an indent

Two types of explants under use are sponges and bands; we have found sponges to:

1. be safer even for encirclement purposes.
2. be of little worry as regards rise in intraocular pressure (IOP), which was very rare.
3. cover a larger area.
4. give a good quality indent, especially in aphakes.

On the other hand, bands are very useful in cases of multiple tears and for reinforcement of radial explants.

C-D. Clearing off the subretinal fluid (SRF) by Drainage

Drainage of SRF requires strict following of various determinants, both external as well as internal. The external determinants include:

1. selecting relatively avascular sclera for drainage.
2. trying drainage in and around the equator.
3. avoiding of vortex veins and/or large blood vessels.

The internal determinants are:

1. indenting in order to select the deepest area of RD; the poorer the indent the deeper the SRF.
2. Drainage by the flap-valve technique - use of small gauge needles for shallow RDs and large gauge ones for deep SRF, especially in aphakes.

It is advised that beginners drain in all cases, as in 90% of cases drainage leads to a flat macula on the operating table which is very encouraging. Remember always to drain at the site of deepest SRF. So, indentation prior to drainage is mandatory - to see a "Gush" of clear SRF is a lovely sight, whereas seeing blood or a dry tap is really frightening. So, *practice!*

COMPLICATIONS

Over the years we have seen two complications: *choroidal hemorrhage* (38%) and *retinal incarceration* (0.7%). The high hemorrhage rate is mainly due to the higher incidence during the early days. Do not get discouraged; complications do occur - just *practice!* It will help you in bringing your complication rate down both in type and percentage.

RECOMMENDATIONS

1. Develop a positive attitude towards RD cases.
2. The importance of history taking and allied examination prevails.
3. Practice IO as per given guidelines.
4. Beginners are to select inferior RDs with round holes or dialysis.
5. Always aim at a flat macula at the operating table.

OBSERVATION

It has been observed that aphakic eyes with iridotomies invariably show retinal breaks within 1.5- 2.0 clock hours of the iridotomy - Food for thought!

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Abstracts

Edited by Tahir Mahmood

High Concentration of Dexamethasone in Aqueous and Vitreous After Subconjunctival Injection

Weijtens O, Feron EJ, Schoemaker RC, Cohen AF, Lentjes EGWM, Romijn FPHTM, van Meurs JC.

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Local routes of corticosteroid administration, such as eye drops and subconjunctival and peribulbar injections, are generally preferred over systemic administration to achieve optimal drug concentration in the eye, as it is expected that the risk of systemic side effects is reduced. Corticosteroid concentration in the anterior segment of the human eye after administration of various corticosteroid eyedrops have been studied, but information on the intraocular and systemic bioavailability of corticosteroids in patients after subconjunctival and peribulbar injections is limited.

The authors conducted a study to determine the dexamethasone concentration in aqueous, vitreous, and serum of patients after a subconjunctival injection with dexamethasone disodium phosphate and to compare the effectiveness of a subconjunctival injection as a method of delivering dexamethasone into the vitreous with that of two previously tested routes: peribulbar injection and oral administration.

In this prospective study, 50 phakic patients who underwent a pars plana vitrectomy received a single subconjunctival injection with 2.5 mg of dexamethasone disodium phosphate, aqueous solution (after topical anesthesia and a subconjunctival injection with lidocaine) at varied intervals before surgery. An aqueous and a vitreous sample were taken from each patient and serum samples were collected at multiple time points from nine of 50 patients. Dexamethasone concentrations were measured by radioimmunoassay.

The estimated maximum dexamethasone concentration in the aqueous was 856 ng per ml at 2.5 hours after injection, and in the vitreous, 72.5 ng per ml at 3 hours. In serum, a mean maximum concentration of 32.4 ng per ml was measured at approximately 30 minutes after injection.

The authors concluded that subconjunctival injection of 2.5 mg of dexamethasone disodium phosphate resulted in an estimated vitreous dexamethasone peak concentration three and 12 times higher, respectively, than after a peribulbar injection of 5 mg of dexamethasone disodium phosphate and an oral dose of 7.5 mg of dexamethasone. Thus, a subconjunctival injection is the most effective method of delivering dexamethasone into both the anterior and posterior segments of the eye. Systemic drug absorption is considerable and is of the same order of magnitude as after peribulbar injection.

Mooren Ulcer in South India: Serology and Clinical Risk Factors

Zegans ME, Srinivasan M, McHugh T, Whitcher JP, Margolis TP, Lietman T, Jennette JC, Cunningham ET Jr

Am J Ophthalmology 1999;128:205-10

Mooren ulcer is an idiopathic form of peripheral ulcerative keratitis described first by Bowman in 1849 and later by Mooren in 1867. The criteria for the diagnosis of Mooren ulcer include painful ulceration of the peripheral cornea, the frequent occurrence of an overhanging and opacified central corneal edge, and the presence of linear vessels extending from the corneoscleral limbus into the ulcer bed. The ulcer may progress in either a circumferential or a transverse fashion, and in severe cases it may involve the entire corneal stroma. One or both eyes may be affected, either simultaneously or consecutively. Despite extensive destruction of corneal tissue, adjacent scleral loss does not occur.

Several cases of Mooren ulcer have been reported in the setting of systemic hepatitis C infection, and in two cases the ulcer responded to the systemic treatment of hepatitis C infection with interferon. These observations have led to the suggestion that hepatitis C plays a role in the pathogenesis of Mooren ulcer. Moreover, since the original description of Mooren ulcer, it has become clear that rheumatoid arthritis, Wegener granulomatosis, systemic lupus erythematosus, and other autoimmune diseases can produce similar peripheral corneal ulcerations.

The purpose of this study was to investigate the rate of undiagnosed rheumatologic diseases and hepatitis C infection among patients with the clinical diagnosis of Mooren ulcer seen at Aravind Eye Hospital, Madurai, South India.

Twenty-one patients with the clinical diagnosis of Mooren ulcer and 44 control patients underwent a complete ophthalmic history and examination, as well as serologic testing for antinuclear antibodies, rheumatoid factor, antineutrophil cytoplasmic antibodies, herpes simplex virus I antibodies, and hepatitis C virus antibodies.

There were no statistically significant differences in the rates of seropositivity for antinuclear antibodies, rheumatoid factor, antineutrophil cytoplasmic antibodies, herpes simplex virus I antibodies, and hepatitis C virus antibodies between patients with Mooren ulcer and control patients. Two patients with Mooren ulcer and four control patients were found to have a rheumatoid factor titer of greater than 1:20. One of the control patients, but none of the patients with Mooren ulcer, was found to have serologic evidence of hepatitis C infection. A history of corneal trauma, surgery, or infection was reported by 68% of patients with Mooren ulcer, compared with 20% of control patients ($P < .001$). Among patients with Mooren ulcer, bilateral disease occurred in 37% of patients, visual acuity was reduced to light perception in 15% of eyes, and perforation occurred in 19% of eyes.

The authors concluded that nineteen (90%) of 21 patients with the clinical diagnosis of Mooren ulcer were found to have no evidence of an underlying rheumatologic disease by history, examination, or serologic testing, and none was

seropositive for hepatitis C. However, patients with Mooren ulcer were more likely than control patients to report a history of corneal trauma, surgery, or infection.

Efficacy of Unilateral Versus Bilateral Temporal Artery Biopsies for the Diagnosis of Giant Cell Arteritis

Boyev LR, Miller NR, Green WR

Am J Ophthalmology 1999;128:211-5

Giant cell arteritis, also called temporal arteritis, is a multisymptomatic disease that typically affects elderly Caucasians, causing headache, scalp tenderness, jaw claudication, ear pain, fever, malaise, anorexia, and weight loss. Systemic complications include cerebral, ocular, and visceral ischemia. Ophthalmologic complications include transient or permanent loss of vision from such diverse entities as ischemic optic neuropathy, central retinal artery occlusion, choroidal ischemia, and occipital lobe stroke, double vision from extraocular muscle ischemia and ischemic ocular motor nerve palsy, pupillary dysfunction from Horner syndrome or ocular ischemia and hypotony.

In 1990, the American College of Rheumatology established the following diagnostic criteria for giant cell arteritis: age greater than 50 years, new-onset localized headache, temporal artery tenderness, an increased erythrocyte sedimentation rate, and a positive temporal artery biopsy. With this classification, fulfillment of three or more criteria is 93% sensitive and 91% specific for the diagnosis.

Indeed the temporal artery biopsy is the gold standard for the diagnosis of giant cell arteritis but there are reports of false-negative unilateral temporal artery biopsy results in patients subsequently shown to have giant cell arteritis by a biopsy of the contralateral artery. Performing bilateral temporal artery biopsies increases the sensitivity of the diagnosis of giant cell arteritis upto 60%. It is suggested that unilateral biopsy is sufficient for diagnosis, with subsequent contralateral biopsy being useful only in equivocal pathologic results, clinical findings strongly suggesting giant cell arteritis, or both.

The purpose of this study was to determine the utility of unilateral versus bilateral temporal

artery biopsies in detecting the pathologic changes of giant cell arteritis.

The authors performed a retrospective study to determine the utility of unilateral versus bilateral temporal artery biopsies in detecting the pathologic changes of temporal artery biopsy specimens received at the Wilmer Ocular Pathology Laboratory over a 28-year period from 1968 to 1996.

Of 908 specimens examined from 758 patients, 300 specimens were simultaneous bilateral biopsies from 150 patients, 72 specimens were bilateral sequential biopsies from 36 patients, and the remaining 536 specimens were unilateral biopsies from 536 patients. Of the 186 patients who had bilateral simultaneous or nonsimultaneous biopsies, 176 had identical diagnoses on both sides. In four patients, no artery was obtained on one side. In each of the remaining six patients, five of whom had bilateral simultaneous biopsies and one of whom had bilateral sequential biopsies performed 8 days apart, the biopsy specimen from one side was interpreted as showing only arteriosclerotic changes with no evidence of active or healed arteritis, whereas the other specimen was interpreted as showing either probable healed arteritis (three specimens) or possible early arteritis (three cases). In none of the six patients with differing diagnoses between the two sides was one side interpreted as showing definite, active giant cell arteritis. Five of the six patients were subsequently determined to have giant cell arteritis, based on a combination of clinical findings, erythrocyte sedimentation rate, and response to treatment with systemic corticosteroids.

The authors concluded that performing a bilateral simultaneous or sequential temporal artery biopsy improves the diagnostic yield in at least 3% of cases of giant cell arteritis, whereas in 97% of cases, the two specimens show the same findings. Thus, in patients in whom only one artery can be biopsied, there is a high probability of obtaining the correct diagnosis. Nevertheless, although the improvement in diagnostic yield of bilateral temporal artery biopsies is low, the consequences of both delayed diagnosis and treatment of giant cell arteritis as well as the use of systemic corticosteroids in patients who do not have giant cell arteritis are of such potential

severity that consideration should always be given to performing bilateral temporal artery biopsies in patients suspected of having the disease.

Sun Exposure and Pterygium of the Eye: A Dose-Response Curve

Threlfall TJ, English DR

Am J Ophthalmology 1999;128:280-7

Pterygium of the eye is a fibrovascular growth of the conjunctiva, commonly encroaching onto the cornea, usually triangular or wing shaped. Although usually small and relatively benign, pterygia are common, cause considerable irritation, and often recur after surgery. A proportion of cases appear to be inherited, but other factors--including dust, wind, heat, infection, inflammation, and sunlight--have been proposed as causes. The prevalence has been reported to be inversely related to latitude and to be greater among outdoor workers than among indoor workers.

The purpose of this study was to present a quantitative analysis of pterygium and ocular sun exposure, a dose-response curve and to discuss the health-promotion implications of the findings.

A hospital-based, case-control study was conducted in Perth, Western Australia. Case subjects had surgical removal of a pterygium; control subjects had an ear, nose, or throat procedure. A lifetime history of residence, sun exposure patterns, and use of hats, spectacles, and sunglasses was obtained at interview. Measures of potential sun exposure included latitude, daily sunshine hours, and daily global solar radiant energy. The most complex estimate of actual sun exposure was the daily ocular solar radiation dose, calculated from climatic data, time spent outdoors not under shade, and the use of hats and spectacles.

There were strong positive associations between pterygium and measures of potential and actual sun exposure. Associations were as strong for whole-life measures as for those in any specific age range. Pterygium odds ratios increased with exposure level; the odds ratio was 4.0 (95% confidence interval, 1.6 to 10.9) for the highest quarter of the daily sun exposure. The strongest associations were seen for the estimated daily ocular solar radiation dose, with an odds ratio of 6.8 (95% confidence interval, 2.6 to 19.7) for the highest quarter of exposure.

The authors concluded that pterygium is strongly related to ocular sun exposure, with little evidence that exposure during any particular period of life is more important than in other periods; the implication for prevention of pterygium is that ocular protection is beneficial at all ages.

Plateau Iris Syndrome: Changes in Angle Opening associated With Dark, Light, and Pilocarpine Administration

Pavlin CJ, Foster FS

Am J Ophthalmology 1999;128:288-91

Plateau iris syndrome is characterized clinically by persistent angle narrowing in an eye with a patent iridotomy. Angle closure and increased pressure can occur spontaneously or with pupillary dilation. This condition is caused by anteriorly positioned ciliary processes that provide structural support to the peripheral iris. This structural support prevents the peripheral iris from moving posteriorly after iridotomy, thus leaving the angle relatively narrow. It is well known that pupillary dilation can produce an acute increase in intraocular pressure in eyes with this syndrome. Therapy of plateau iris syndrome includes treatment with pilocarpine, which produces pupillary constriction and opens the angle.

The purpose of this study was to report changes in angle configuration associated with dark, light, and pilocarpine administration in plateau iris syndrome.

In 10 eyes of 10 patients with plateau iris syndrome and persistent narrow angles after patent peripheral Nd:YAG laser iridotomy, ultrasound biomicroscopy was used to image variations in angle opening, iris thickness, and trabecular-ciliary process distance. Measurements were taken in the dark, in full room light, and after administration of pilocarpine 2%.

Average angle opening distance increased in the light compared with the dark ($113 \pm 34 \mu\text{m}$ vs $22 \pm 34 \mu\text{m}$, $P = .0001$) and increased further after pilocarpine administration ($171 \pm 52 \mu\text{m}$ vs $113 \pm 34 \mu\text{m}$, $P = .0034$). Average iris thickness decreased in the light compared with the dark ($338 \pm 34 \mu\text{m}$ vs $436 \pm 58 \mu\text{m}$, $P = .0009$) and decreased further after pilocarpine administration ($253 \pm 48 \mu\text{m}$ vs $338 \pm 34 \mu\text{m}$, $P = .0002$). Average

trabecular meshwork-ciliary process distance measurements were smaller than normal and did not change significantly in the light compared with the dark ($481 \pm 42 \mu\text{m}$ vs $464 \pm 44 \mu\text{m}$, $P = .4001$) or after pilocarpine administration compared with light ($451 \pm 67 \mu\text{m}$ vs $481 \pm 42 \mu\text{m}$, $P = .1304$).

The authors concluded that in plateau iris syndrome, anteriorly located ciliary processes support the peripheral iris. Changes in angle opening in dark and light are solely related to changes in iris thickness. Pilocarpine produces iris thinning and is an effective method of opening the angle. Ultrasound biomicroscopy can be used to perform a darkroom provocative test, which provides information on whether the angle anatomically closes in the dark.

Vitrectomy for Chronic Pseudophakic Cystoid Macular Edema

Pendergast SD, Margherio RR, Williams GA, Cox MS Jr.

Am J Ophthalmology 1999;128:317-23

Cystoid macular edema is characterized by hyperpermeability of the macular capillaries, resulting in excessive fluid in the retina and polycystic expansion of the extracellular spaces. Although the mechanism of cystoid macular edema after ocular surgery is not clearly understood, previous studies have implicated vitreomacular traction, excessive ultraviolet light exposure, and anterior segment inflammation with liberation of prostaglandins.

Postoperative cystoid macular edema is a common complication after extracapsular cataract surgery, with angiographic cystoid macular edema occurring in 10% to 30% of patients. Fortunately, the incidence of postoperative cystoid macular edema after cataract surgery has been dramatically reduced by the advent of extracapsular cataract surgery by means of phacoemulsification techniques. However, cystoid macular edema remains the leading cause of unfavorable visual outcomes after cataract extraction. In another prospective study, the incidence of clinical cystoid macular edema (angiographic cystoid macular edema and visual acuity 20/40 or worse) after extracapsular cataract extraction and posterior chamber lens implantation after six months was 1.7%.

Because the natural course of the postoperative cystoid macular edema is to spontaneously resolve, mild cases require no treatment. In eyes with chronic aphakic and pseudophakic cystoid macular edema, various treatment modalities have been advocated, including topical corticosteroids, topical nonsteroidal anti-inflammatory agents, acetazolamide, hyperbaric oxygen exposure, and Nd:YAG vitreolysis of vitreous strands associated with the corneoscleral wound. To date, only topical ketorolac tromethamine 0.5% has been shown to significantly improve visual acuity in prospective randomized studies. In a minority of eyes, clinical cystoid macular edema persists despite aggressive medical treatment.

Cystoid macular edema is more commonly observed in eyes with interruption of the anterior hyaloid membrane and incarceration of the vitreous in the corneoscleral wound. However, in a subset of eyes, chronic pseudophakic cystoid macular edema occurs in eyes with no evidence of vitreous incarceration and is occasionally observed in eyes with an intact posterior capsule.

The purpose of this study was to report the results of pars plana vitrectomy for chronic pseudophakic cystoid macular edema unresponsive to medical treatment.

Retrospective analysis of 23 consecutive eyes of 23 patients with chronic pseudophakic cystoid macular edema was performed. Eyes with vitreous incarceration into the cataract wound or vitreous-cornea contract were excluded from the study.

Preoperatively, all eyes had cystoid macular edema confirmed on fluorescein angiography and were unresponsive to medical treatment. Pars plana vitrectomy was performed using standard techniques and vitreous adhesions to the iris, intraocular lens, or both were lysed if present.

The mean interval between cataract surgery and vitrectomy was 32.3 ± 30.9 months (median, 20 months; range, 3 to 110 months). The vitreous was adherent to the iris or intraocular lens in 12 eyes (52.2%) and was present in the anterior chamber with no evidence of adhesions in seven eyes (30.4%). In four eyes (17.4%) the vitreous was posterior to the iris plane with no adhesions to anterior segment structures. The median preoperative best-corrected visual acuity was 20/200, and the median final postoperative best-corrected visual acuity was 20/60 ($P < .0001$) after a mean follow-up of 30.2 ± 31.2 months (median, 14 months; range, 2 to 109 months). Final best-corrected visual acuity improved by a mean of 3.3 ± 2.6 Snellen lines, with a median percent change of 70% (mean, 57.3%; range 0% to 99%). In all 23 eyes the cystoid macular edema resolved postoperatively by biomicroscopic examination in a mean period of 3.3 months (median, 2 months; range, 1 to 12 months).

The authors concluded that in pseudophakic eyes with chronic cystoid macular edema unresponsive to medical treatment, vitrectomy resulted in resolution of the cystoid macular edema with improved visual acuity in some cases. Clinical improvement may occur in eyes with no apparent vitreous disturbance.

News and Events

THE ROYAL COLLEGE OF OPHTHALMOLOGISTS

**Annual Congress, Harrogate
Tuesday, 23rd to Thursday, 25th May 2000**

The Annual Congress will be held from Tuesday 23rd to Thursday 25th May 2000 at Harrogate International Centre, Kings Road, Harrogate, North Yorkshire, England
For further information and registration please contact:

Miss Heidi Packer, Congress Officer
The Royal College of Ophthalmologists
17 Cornwall Terrace, London. NW1 4QW
U.K.
Fax: +44(0)20 7935 9838

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7TH OPHTHALMOLOGICAL CONGRESS OF SAARC COUNTRIES

**14-16th September 2000
Colombo, Sri Lanka**

For further information contact:
SAARC Congress Secretariat

The Eye Hospital
Deans Road
Colombo-10
Sri Lanka.

Tel: (941) 693911
Fax: (941) 699728 / 693541
e-mail: Acetrvl@eureka.lk

3RD INTERNATIONAL CONGRESS ON ADVANCES IN OPHTHALMOLOGY

(Eye Advances 2000)

Organized by:

**The International Academy for Clinical
Ophthalmology**

Under the aegis of

**Intraocular implant and refractive society: India
and Bomay Ophthalmologists Association
on 20th-23rd September, 2000
at the Taj Mahal intercontinental, Mumbai, India**

For further information contact:

Dr. Keiki R. Mehta / Dr. Kirit K. Modi, Organizing
Chairmen
Sea Side, 147, Shahid Bhagat Singh Road (Near
Colaba Bus Station), Mumbai-400 005, India.

Tel: 215 1303 / 215 0082
Fax: (91) 22-215 0433
e-mail: keiki_mehta@yahoo.com

8TH ANNUAL HYDERABAD OPHTHALMIC CONFERENCE, 2000 ON RECENT TRENDS IN OPHTHALMOLOGY

Saturday, 7th to Monday, 9th October 2000.

Contact Address:

Secretary
Ophthalmological Society of Pakistan
(Hyderabad Branch)
Liaquat Medical College Eye Hospital
Journalist's Colony, Hyderabad-71000

Telephone: 92-221-612090, 611676
 Fax: 92-221-780748, 640598
 e-mail: nlaghari@hyd.netasia.com.pk.

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21ST LAHORE OPHTHALMO

Lahore, Pakistan
 Friday, 24th to Sunday, 26th November 2000

Contact Address:
 Prof. Hamid Mahmood Butt
 Allama Iqbal Medical College &
 Jinnah Hospital
 Lahore, Pakistan
Phone: 92 (042) 5162588
E-mail: hamidbut@wol.net.pk

23RD ANNUAL CONGRESS OF
OPHTHALMOLOGY
PESHAWAR - 23rd - 25th FEB 2001

23rd Annual Congress of Ophthalmology will be hosted by the Ophthalmological Society of Pakistan (N.W.F.P. Branch) at Peshawar on 23rd-25th February, 2001.

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THE ROYAL COLLEGE OF OPHTHALMOLOGISTS DIPLOMA IN OPHTHALMOLOGY EXAMINATION

The Royal College of Ophthalmologist has introduced an examination leading to the award of the Diploma in Ophthalmology (DRCOphth). The examination will be held twice a year, in June and November.

This Diploma is aimed at those not wishing to pursue a career as a consultant ophthalmologist in the United Kingdom. It should, therefore, be of interest to all doctors with an interest in ophthalmology working outside the European Union.

Details are available from the Examination Office,
The Royal College of Ophthalmologists, 17
Cornwall Terrace, London NW1 4QW, UK.
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OSP News

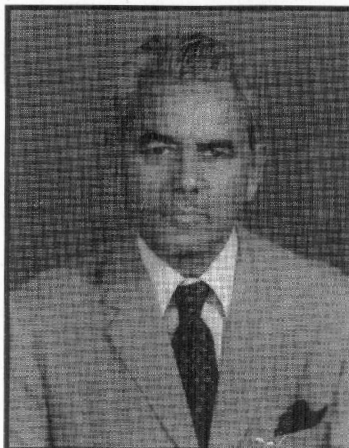
Prof. Raja Mumtaz wrote to Prof. M Hayat Zafar, the recently installed President of the Pakistan Medical and Dental Council that attempts were being made at the PMDC to relegate the teaching of Ophthalmology to earlier years in Medical Colleges in Pakistan and its examination to be held in the 4th year, instead of the final year of MBBS curriculum. Prof. Raja was of the opinion that this would increase the incidence of blindness in the country, since at present the qualifying doctors can at least diagnose and differentiate between conjunctivitis, cataract and glaucoma, but under the new proposal, they would have forgotten to do even that by the time they qualify. This would negate the claims of improving medical education and curricula and would rather expose the shortsightedness and selfishness of our medical and surgical colleagues in promoting their specialties at the expense of Ophthalmology.

The readers are encouraged to let their views be known by writing to the Journal as well as to the PMDC.

Editor-in-Chief

OBITUARY

Dr. Rehmat Ullah Chaudhry Passes Away



Dr. Rehmat Ullah Chaudhry, ex-Secretary Health, Government of the Punjab, passed away on 13th January 2000 at Bahawalpur at 82 years of age. Innalillahe wa innailehe rajeoon.

Dr Rehmat Ullah Chaudhry had a brilliant career:

He started as captain in the Armed forces in the Second World War and served in the British army in Italy for over two years.

He obtained DOMS form the Royal College of Surgeons, London in 1952 and the Fellowship after a few years. He was appointed as Incharge of Ophthalmology Department in Bahawalpur Medical School, Bahawalpur, with special duties as consultant ophthalmologist for His Highness, the Ruler of the State of Bhawalpur. Later on he served as M.S./BV Hospital, Bahawalpur many times and as Director Health Services, Bahawalpur and Khairpur region. During his stay in the South Punjab he served with great dedication and organized Eye Camps all over the deprived and far-flung areas, including adjoining parts of Sindh Province. Many people were blessed with restoration of their eyesight with his efforts.

He was appointed as Project Director of QMC/BVH, Bahawalpur, and also took over as Secretary Health, Government of the Punjab, where he served in that capacity till late 1974. He retired as Administrator and Principal, QMC/BVH late in 1977.

He leaves behind one son, Dr. Tariq Mahmood Chaudhry, Associate Prof. Ophthalmology, BVH/QMC, Bahawalpur and five daughters and sons-in-law that include Dr. Tajammul Hussain Chaudhry, Ophthalmologist, Faisalabad, Prof. G. Sarwar Riaz, Prof of Obs/Gynae, RMC, Rawalpindi, Dr Shabir Ahmad Chaudhry, Assistant Professor Anatomy, RMC, Rawalpindi and Dr Ahsan Mushtaq, Diplomate, American Board of Nephrology, USA.

May God bless the departed soul and give strength and patience to the family and friends to bear the loss. Amen!

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Known hypersensitivity to latanoprost, benzalkonium chloride or any other ingredients in this product. **WARNINGS** XALATAN may gradually change eye color, increasing the amount of brown pigment in the iris by increasing the number of melanosomes (pigment granules) in melanocytes. The change in iris color occurs slowly and may not be noticeable for several months to years. Patients should be informed of the possibility of iris color change. Patients who are expected to receive treatment in only one eye should be informed about the potential for increased brown pigmentation in the treated eye and thus, heterochromia between the eyes. The increased pigmentation may be permanent. **PRECAUTIONS:** Patients may slowly develop increased brown pigmentation of the iris. This change may not be noticeable for several months to years (see WARNINGS). Typically the brown pigmentation around the pupil spreads concentrically towards the periphery in affected eyes, but the entire iris or parts of it may also become more brownish. Until more information about increased brown pigmentation is available, patients should be examined regularly and, depending on the clinical situation, treatment may be stopped if increased pigmentation ensues. XALATAN should not be administered while wearing contact lenses. Patients should be instructed to avoid allowing the tip of the dispensing container to contact the eye or surrounding structure because this could cause the tip to become contaminated by common bacteria known to cause ocular infections.

Patients also should be advised that if they develop an intercurrent ocular condition (e.g., trauma, or infection) or have ocular surgery, they should immediately seek their physician's advice concerning the continued use of the multidose container they had been using. Patients should be advised that if they develop any ocular reactions, particularly conjunctivitis and lid reactions, they should immediately seek their physician's advice. If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart. **ADVERSE REACTIONS** The ocular adverse events and ocular signs and symptoms reported in 5 to 15% of the patients on XALATAN in the 6 month, multi-center, double-masked, active-controlled trials were blurred vision, burning and stinging, conjunctival hyperemia, foreign body sensation, itching, increased pigmentation of the iris and punctate epithelial keratopathy. Local conjunctival hyperemia was observed; however, less than 1% of the XALATAN treated patients required discontinuation of therapy because of intolerance to conjunctival hyperemia. **DOSE AND ADMINISTRATION** The recommended dosage is one drop (1.5 µg) in affected eye(s) once daily in the evening. The dosage of XALATAN should not exceed once daily since it has been shown that more frequent administration may decrease the intraocular pressure lowering effect. Reduction of the intraocular pressure starts approximately 3 to 4 hours after administration and the maximum effect is reached after 8 to 12 hours. XALATAN may be used concomitantly with other topical ophthalmic drug products to lower intraocular pressure. If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart. **HOW SUPPLIED** XALATAN™ (latanoprost solution) Sterile Ophthalmic Solution is a clear, isotonic, buffered, preserved colorless solution supplied in plastic ophthalmic dispenser bottles with a dropper tip and tamper evident overcap. Each bottle contains 2.5ml latanoprost (0.005%) (50 µg/mL). **Storage:** Protect from light. Store unopened bottle under refrigeration at 2° to 8°C (36° to 46°F). Once opened the container may be stored at room temperature up to 25°C (77° F) for 6 weeks.

Further information is available upon request.

How latanoprost pulls the plug on pressure.

New mechanism of action:
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Lowers IOP up to 35%
from baseline.

No long-term drift.

No cardiopulmonary
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One drop a day.

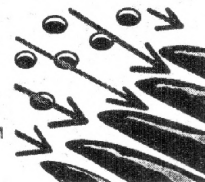
After reviewing the best input on IOP, we decided the solution was better output. So we created Xalatan, a unique prosta-glandin-based drug

that lowers IOP by increasing aqueous humour outflow via the ciliary muscle.

Increased uveoscleral outflow reduces IOP BY UP TO 35%¹

Latanoprost is able to increase uveoscleral outflow by over 100%² for IOP reductions of up to 35% in monotherapy¹. At the same time, Xalatan is easy on your patients, with no cardiopulmonary contraindications and one-drop-a-day dosage. So go with a better solution for long-term glaucoma patient management³. Choose Xalatan.

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Tough on pressure. Easy on people.

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2. Teas CB, Casanova CB, Yokoyama ME. Effects of PRA-AM, a new prostaglandin F_{2α} analog on aqueous humor dynamics in human eyes. Ophthalmology 1993; 100: 1297-1304.

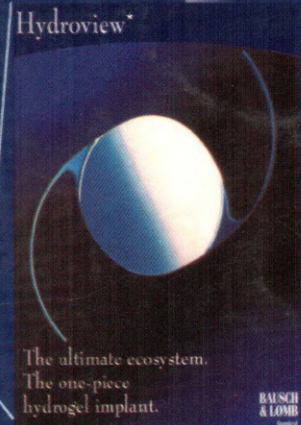
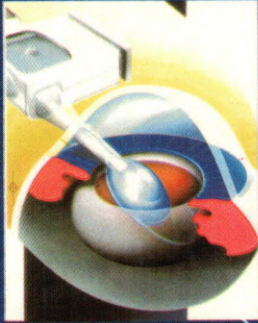
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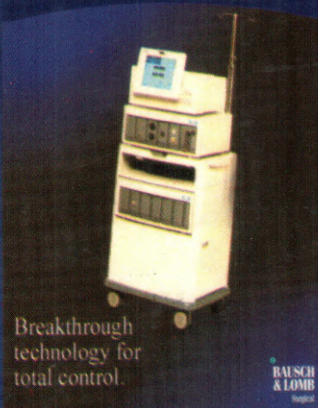
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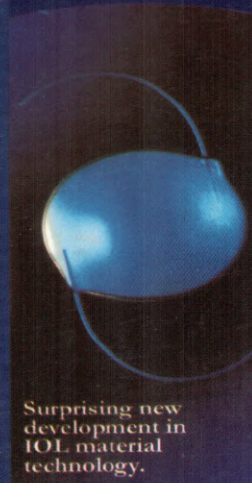
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