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BISMILLAHIR-RAHMANIR-RAHEEM

Editor:
Khalid J. Awan, FPAMS

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Publication and Editorial Staff

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Say: Are the blind and the seeing man equal, or are the darkness and the light equal?
-Holy Qur'an 13:16

Blindness Surveys in Pakistan

Khalid J. Awan, F.P.A.M.S.

Bismillahir-Ruhmanir-Raheem.

فَإِنَّهَا لَا تَعْمَى الْأَبْصَارُ
وَلَٰكِن تَعْمَى الْقُلُوبُ الَّتِي فِي الصُّدُورِ

Truly, it is not the eyes that are blind;
Rather blind are the hearts in the bosoms.
-Holy Qur'an 22:46

Although there are some figures floating around in the lay press, no concrete numbers on the prevalence, types, or causes of blindness exist in Pakistan. During my recent assigment as a visiting professor to the Postgraduate Medical Institute at Peshawar, I learned that under the World Health Organization (WHO) auspices Dr. Mohammad Aman Khan, the very first community ophthalmologist of Pakistan, and his colleague, Dr. Arifa Gulab have just completed a carefully planned blindness survey in the Northwest Frontier Province (N.W.F.P.), the results of which will be published very shortly. In this issue of THE JOURNAL, Drs. Khattak, Khan, Mohammad, and Mulk[1] present their analysis of monocular blindness in the eye patients admitted to the Lady Reading Hospital at Peshawar. (*See page 3.*) Exactly a hundred years ago a similar statistical review on blindness was published in the United States.[2] It is discouraging to note that Khattack and colleagues have come up with a monocular blindness incidence of 13.7%, which is over five times the century old figure (2.6%) of the American publication.

There have been several published reports by Khan and his colleagues[3-6] on traumatic visual loss in the N.W.F.P., and a recent report from Karachi presented a retrospective analysis of a small series of ocular trauma cases.[7] This survey conducting trend, encouraging though it is, is limited to only two locations in the country, and, hence, cannot help delineate any meaningful national variables on blindness. Leaders of ophthalmology in other parts of the country should also conduct blindness surveys in their regions, including the rural population, and pool their results to create a national overall picture of blindness. This is of foremost importance for formulating plans to fight blindness.

The Ophthalmological Society of Pakistan (OSP) can play a very siginificant role in eradicating curable blindness from our nation. I must most emphatically suggest that the Society create a special blindness prevention committee made up of its interested and capable members from all provinces at the coming annual OSP meeting at Peshawar, and hand them a clearly defined agenda of war on blindness.

Statistical information is much important in the planning and financial aspects of attack on blindness. For instance, the Macular Photocoagulation Study in the United States has demonstrated that argon blue-green laser photocoagulation significantly improves the visual prognosis as compared with the natural course of this disease.[8] In another recent American study on racial differences in causes of blindness, age-related macular degeneration affected only whites, though nearly 45% of the patients in this study were American blacks.[9] Obviously, in a non-western country a decision based on western research and reports to invest in highly expensive laser equipment to fight blindness due to age-related macular degeneration in a non-white population would not be cost-effective and proportionately productive. Therefore, before subsidizing any preventive or therapeutic measures, it must be established that the target population does have a significant incidence of the disease against which these measure are intended. To establish this would require properly conducted and analyzed surveys.

The Society should develop, on the pattern of the American Academy of Ophthalmology, a National Eyecare Program, intensive national campaigns for public education and year-round visual screening programs to be held by the ophthalmologists in training. The Society should also work on convincing the government and regional leaders for provision of funds and promotion facilities for these programs.

The importance of surveys on health problems in our country cannot be overemphasized. In addition to providing meaningful figures on prevalence and etiologies of a disease in a community, surveys raise the awareness and sentivity levels of the governments, physicians, media, and the community itself, stimulating a willing participation of all sections of the community in eradication of the disease.

It is also important to learn about the attitudes, socioeconomic circumstances, health beliefs, popular home remedies, and the availability of medical services in a community. Sometimes, a patient may not use the available optimum medical service, because he harbors doubts about modern medicine, cannot financially afford it, other obligations of life keep him from it, or has greater confidence, though misplaced,

in worthless home remedies. I have seen more than once in the United States--yes, even the nations as modern as the US are not immune to superstitious beliefs--a patient walked into my clinic with a horribly inflamed eye with advanced corneal ulcer, after having tried for many days the flabbergasting home remedy of a flax seed, or a small button, or chewing gum placed in the conjunctival sac to achieve cure, without relief, of course. Conversely, some traditional habits may keep a disease at bay in some communities. Hence, it was discovered that daily face-washing may have a dramatic impact on the prevelance or severity of trachoma in Mexico and Malawi.[10] As *Woozoo* (ablution) is a must prerequisite of saying *Salat* (prescribed five-time daily formal prayer) for Muslims, regular washing of face is a social habit of our population.

Our blind themselves cannot be expected to collect data necessary for devising plans to cure or prevent blindness. To do so is our moral, professional, and religious obligation. To ignore just obligations is to join the ranks of those in whom, says Allah, "Truly, it is not the eyes that are blind; rather blind are the hearts in the bosoms."

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End of an Era in Ophthalmology

Khalid J. Awan, F.P.A.M.S.

"See here, there is no period after 'C' in Charles C Thomas," said Dr. Newell softly, pointing to the name of well-known printing company Charles C Thomas, Publisher in one of the references in a manuscript that had just arrived in the Editor's office of the *American Journal of Ophthalmology*, the "AJO". He was going through that day's mail and making necessary comments where appropriate for my benefit, conducting a sort of hands-on course in editing for me.

It was over eight years ago that the Ophthalmlogical Society of Pakistan picked me to found its official journal, the *Pakistan Journal of Ophthalmology*. Although I had by then published nearly six dozen papers, the only contact with editing I had was the rejection slips or letters demanding revisions of my papers from the editors, quite a few of them from Dr. Newell himself. Hence, before embarking on my assignment, I requested Dr. Newell to spend some period of time with him in the offices of AJO to familiarize myself with the editorial operations. During this mini-apprenticeship I would spend all my time with Dr. Newell on his office days, and on other days Mary Borysewicz, Executive Managing Editor of AJO, would assign me to various other departments.

The above apparently minor remark is remarkable in that it immediately instilled in me the importance of meticulousness and accuracy in scientific writing, an impact a lesser teacher could not have achieved even after many lengthy lectures. I observed firsthand what Dr. Derrick Vail, the previous editor of AJO, had said about Dr. Newell, "a scientist, teacher, clinician," and " an expert in new knowledge."

Dr. Frank W. Newell, the Editor-in-Chief of AJO for 27 years, longer than any other editor of this 108 years old venerable publication, has decided to step down this month. He had joined the editorial board of AJO in 1954, and edited its "Research Section" until 1961, when he became the Associate Editor. In June 1965, the Board of Directors of the Ophthalmic Publishing Company, the parent organization of AJO, unanimously elected him as the Editor-in-Chief. In this capacity Dr. Newell not only established himself as an editor *extraordinaire*, but also took AJO to new heights of popularity and prestige, doubling its circulation and making it the most respected ophthalmic journal in the world. He employed all his means and maneuvers to protect the image of AJO.

Dr. Newell also founded the now popular *Survey of Ophthalmology*. He also guided me as the Consultant Editors of THE JOURNAL in its early years. His textbook, *Ophthalmology. Principles and Concepts*, is one of a few top texts in the world. Dr. Newell's remarkable achievements make him a true American hero. He is ophthalmology's hero too, and with his retirement, a glorious and productive era in world ophthalmology has ended. We owe him much gratitude, and wish him a very happy, healthy, and unhurried retirement.

Untreatable Monocular Blindness in Pakistani Eye Patients

M. Naeem. K. Khattak, F.R.C.S., M. Daud Khan, F.P.A.M.S.,
Shad Mohammad, F.R.C.S., and R.A. Mulk, M.B.,B.S.

ABSTRACT: To determine the incidence of untreatable (at least by means currently available in Pakistan) monocular blindness in our hospital-based population, we examined 1,710 consecutive patients admitted to the Department of Ophthalmology, Lady Reading Hospital, Postgraduate Medical Institute, Peshawar from September 17, 1990 to January 1991. Seven percent (235) of these patients had monocular blindness with visual acuity of 3/60 (10/200) or less in the affected eye. Out of these 235 patients, visual loss in 94 (40%) was due to trauma, in 51 (21.7%) due to corneal opacification from various corneal diseases, in 41 (17.4%) due to end-stage glaucoma (33 with primary glaucoma and eight with secondary glaucoma), in 15 (6.3%) from postoperative infection following cataract surgery, in 14 (5.9%) due to optic atrophy, and in the remainder 20 (8.5%) due to miscellaneous causes, including retinoblastoma (four cases), retinal detachment (three cases), panophthalmitis (two cases), spontaneous rupture of an anterior staphyloma (two cases), basal cell carcinoma of eyelid invading the orbit (two cases), squamous cell carcinoma of conjunctiva involving globe (two cases), microphthalmos, proptosis complications, expulsive choroidal hemorrhage during cataract extraction, myopic degeneration, and diabetic retinopathy (one case each). That only one patient had visual loss due to diabetic retinopathy is intertesting.

The overall incidence of monocular blindness in Pakistan can be significantly reduced by adequate and timely preventive and therapeutic measures against ocular trauma and corneal disease, the two leading causes of blindness. The recent heavy influx of the Afghan refugees of Russian agression of their country might having increased the incidence of trauma in our area. (Pakistan Journal of Ophthalmology 8:3-5, January, 1992.)

Significant data on the causes of blindness in general are available from around the world.[1,2,3] Unfortunately, no reliable data on the causes of monocular visual loss in the population of our province are available. However, a few studies on blindness related to ocular trauma in the Northwest Frontier Province (NWFP) were recently published.[4,5]

We set up a prospective study to learn the incidence and etiology of monocular visual loss in hospital-based population in our area. Our aim also was to identify factors leading to such a tragic outcome, and to suggest measures, where possible, for its prevention.

Materials and Methods

All patients admitted to the in-patient unit of the Department of Ophthalmology, Lady Reading Hospital, Postgraduate Medical Institute, Peshawar, between September 17, 1990 and January 31, 1991 were examined. Those patients in whom the visual acuity was less than 3/60 in the worse eye were placed on the study list. Only those patients qualified for the study in whom the blindness was not treateable by any of the currently available means in Pakistan.

The patients with phthisical, enucleated and eviscerated eyes were also included. The details of each patient who met the criteria were entered on a standard proforma.

We divided the patients into three age groups. Group A included patients with age of upto 20 years, Group B had patients between 21 to 50 years in age, and the patients above 51 years of age comprised Group C. We did so to analyze the prevalence of various etiologies in each age group.

From the Department of Ophthalmology, Lady Reading Hospital Postgraduate Medical Institute, Peshawar, Pakistan.
Reprint requests to M.N.K. Khattak, FRCS, at the above address.

Table 1
Causes of monocular blindness as percentage of total patients (1,710)

Cause	Percentage	No. of patients	Male	Female	M/F Ratio
Trauma	40.0%	94	69	25	2.7:1
Corneal pathology	21.7%	51	36	15	2.3:1
Glaucoma	17.4%	41	16	25	1:1.5
Postop infection	6.3%	15	9	6	1.5:1
Optic atrophy	5.9%	14	8	6	1.3:1
Miscellaneous group	8.5%	20	10	10	1:1

Table 2
Causes of monocular blindness in specific age groups (235 patients)

Cause	Upto 20 years			21 - 50 years			51+ years		
		M	F		M	F		M	F
Postop infection	0	0	0	2	2	0	13	7	6
Trauma	37	25	12	32	27	5	25	17	8
Corneal pathology	1	0	1	8	7	2	41	29	12
Glaucoma	3	2	1	8	2	6	30	13	17
Optic atrophy	1	1	0	3	2	1	10	6	4
Miscellaneous group	5	3	2	4	3	1	11	4	7
Subtotal	47 (20%)	31	16	58 (24.6%)	43	15	130 (35.4%)	76	54

Results

Out of a total of 1,710 patients admitted during the study period, 235 patients met the criteria for monocular blindness, which means 7% patients had useful vision in only one eye, the other eye being blind with visual acuity of 3/60 or less.

Out of a total of 235 monocularly blind patients, 148 were men and 87 were women. Group A included 47 patients (20%), 29 men and 18 women; Group B had 58 patients (24.6%), 43 men and 15 women; and Group C 130 patients (55.4%), 76 men and 54 women.

Table 1 contains data on the causes of monocular loss of sight. Table 2 lists causes of blindness according to age groups and sex. The break down of miscellaneous etiologic group is given in Table 3.

Discussion

This study was initiated after we observed an unusually large number of patients customarily labelled as "one eyed" by our outpatient clinic staff. We also intended to determine the probable etiologies that lead to irreversible monocular visual loss in so many among our population. With operating theater standards what they are in our country, our dominant feeling was that the postoperative infection might be the major culprit, but our final figures did not place it at the top of the list of causes. Ocular trauma occupied this place.

It is clear from Table 1 that trauma is responsible for visual loss in 40% of the patients in this study, affecting the male population almost three times more than the female population. All three age groups are affected almost equally by trauma with a slight preponderance in the upto 20-year age group (Table 2). In our culture, the females live in a relatively more sheltered environment, and are, thus, protected from many hazards to which their male counterparts are exposed. The circumstances of trauma ranged from sports, playing, job-related situations, domestic violence to war injuries. Firearms and domestic circumstances were the most common causes of ocular injury. In children, toy pistol was one of the common offenders. In the same age group, penetrating eye

Table 3
Causes of blindness in miscellaneous group (Total patients, 20)

Cause	No. of patients
Retinoblastoma	4
Retinal detachment	3
Squamous cell carcinoma of conjunctiva infiltrating the globe	2
Basal cell carcinoma of eyelid invading the globe	2
Panophthalmitis	2
Spontaneous rupture of anterior staphyloma	2
Microphthalmos	1
Proptosis complications	1
Expulsive choroidal hemorrhage during cataract extraction	1
Myopic degeneration	1
Diabetic retinopathy	1
Total	20

injuries caused by the carelessly discarded disposable syringes with unremoved needles are worth mentioning.[6]

In most patients surgical repair was carried out within 48 hours of injury. However, without the availability of the technical expertise of a vitreoretinal surgeon, eyes with vitreous hemorrhage and retinal detachment fared unversally badly. Interestingly, during our study we did not come across a single patient who had lost sight due to sympathetic ophthalmitis, inspite of our no primary enucleation policy in the majority of our patients. Other factors that account for high visual morbidity due to trauma are delay in presentation, presence of intraocular infection at the time of presentation, and a lack of donor tissue for tectonic corneal grafting.

A lack of donor corneal material is also a very important cause of blindness in the second group of patients, those suffering from corneal ulcers with subsequent extensive scarring. Thus, 21.7% of the patients had visual loss due to dense corneal opacification. Since anterior chamber details were not visible in the vast majority of these patients, secondary involvement of other anterior segment structures could not be ruled out. Bacterial, viral and fungal keratitides all abound in our area. The unavailability of any effective topical antifungal in our country takes an unusually high toll in visual function. Conversely, the injudicious prescribing by the general physicians and also without prescription availability of topical steroids over the counter make the situation worse for ophthalmic patients in more than one ways, particularly in patients with viral dendritic keratitis, glaucoma, or vernal keratoconjunctivitis. Such practices, therefore, need to be strongly discouraged.

A total of 15 patients (6.3%) had lost all vision due to infective endophthalmitis following cataract surgery. Considering our operating theater sterility standards, one would expect to see it happen even in a higher number of patients who undergo eye surgery. cases. We routinely inject subconjunctival tobramycin at the end of every intraocular procedure as a prophylactic measure, and this might account for not making frequency of postoperative eye infections even more disastrous.

Forty-one patients (17.4%) suffered visual loss due to primary or secondary glaucomas. Lens induced glaucoma subsequent to hypermaturity of a cataract, neovascular glaucoma both due to diabetic complications and idiopathic central retinal vein occlusion, and steroid induced glaucoma due to injudicious prescribing and use in vernal keratoconjunctivitis were the most common types of secondary glaucoma.

Optic atrophy accounted for visual loss in 14 patients (5.9%). Two patients gave a clear history of optic neuritis, but with no identifiable systemic disease. It is significant that among the 50+ years age group majority of the patients with optic atrophy were aphakes. In these patients damage to the optic nerve by the retrobulbar needle or from other operative complications cannot be ruled out.

The miscellaneous group (Table 3) included such diverse conditions as retinoblastoma (four patients), old retinal detachment (three cases), microphthalmia (one case), diabetic retinopathy (one case), squamous cell carcinoma of conjunctiva infiltrating the eyeball (one case), basal cell carcinoma of the eyelid invading the orbit (two cases), complications of proptosis (one case), myopic degeneration (one case), expulsive suprachoroidal hemorrhage during cataract surgery (one case), and diabetic retinopathy (one case). It is of interest that only one patient had loss of sight from diabetic retinopathy.

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Camera Clinicals

In this section of THE JOURNAL, *photographic documentation of interesting and challenging observations are persented to the readers. They should make their diagnosis from the given information, and compare their conclusions with the exposition given on page 17.* -Editor.

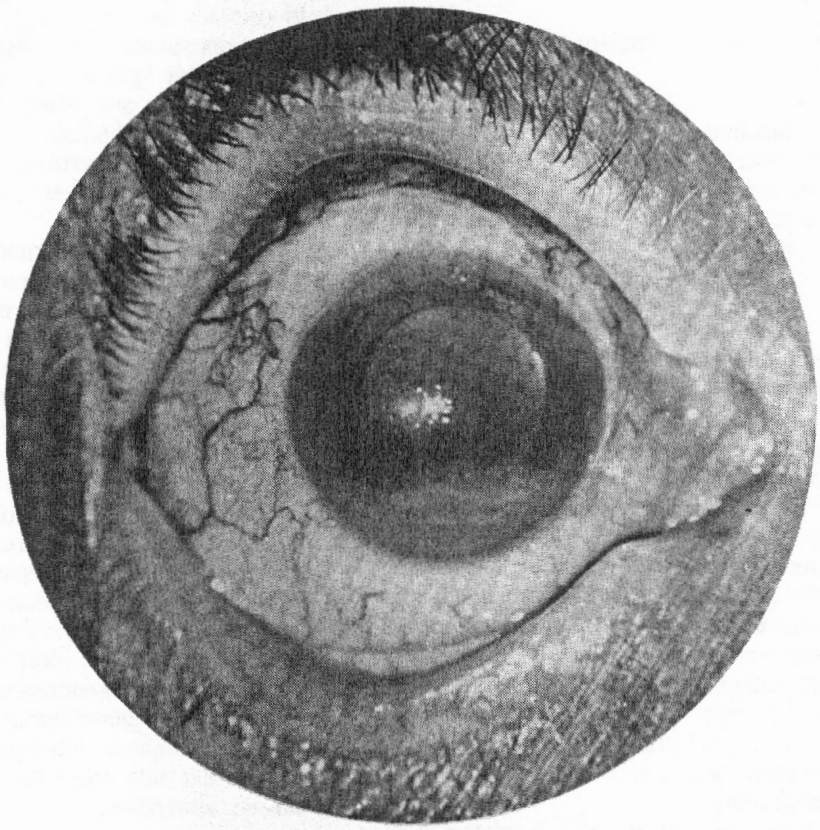

Figure 1

Figure 1: A 59-year-old man suffered blunt trauma to his right eye. Eye examination showed that the injured eye had iridodialysis at 1 o'clock position with vitreous prolapsing into the anterior chamber. When a small hyphema and traumatic iridocyclitis cleared up in a few days, the examination through a dilated pupil showed traumatic cataract of the lens. The visual acuity of the right eye was 20/200, and 20/20 in the left eye. When eye became quiet after medical therapy, cataract extraction and repair of iridodialysis were advised. Successful anterior vitrectomy, cataract extraction with intraocular lens implantation, and repair of iridodialysis were performed. However, one interesting complication was detected at the time of first postoperative visit. Treatment for it was immediately started. However, in about a week the clinical picture shown in Figure 1 developed. Further intensive medical treatment and surgical intervention with pars plana vitrectomy took care of the complication. The final visual acuity in the right eye, a year after the last operation, is 20/30.

Incidence of Ocular Complications of Diabetes Mellitus in Pakistan

Zia Mohammad, M.C.P.S., Mohammad Daud Khan, F.P.A.M.S.

ABSTRACT: We studied 150 patients, 122 with non-insulin-dependent diabetes mellitus (NIDDM) and 28 with insulin-dependent diabetes mellitus (IDDM), to determine the incidence of retinal and non-retinal ocular manifestations of diabetes mellitus in population visiting the Department of Ophthalmology, Lady Reading Hospital, Postgraduate Medical Institute, Peshawar. Corneal sensitivity was reduced in 62.3% of the NIDDM patients and in 35.7% of IDDM patients. Mature cataract or aphakia was present in 25.4% of the NIDDM and 14.2% of the IDDM patients. We found true diabetic cataract in only one patient, an 11-year old girl with IDDM in diabetic coma. Chronic open-angle glaucoma or ocular hypertension was present in 9% of the NIDDM patients but in none of the IDDM patients.

Background diabetic retinopathy (BDR) occurred in 15% of the NIDDM and 11% of the IDDM patients with a diabetes duration of less than 5 years. With a duration of 5-10 years, this figure rose to 18% in the NIDDM group and to 28.5% in the IDDM group. The frequency increased to 66.6% in the NIDDM group and to 33.3% in IDDM group beyond 10 years of disease duration.

Proliferative diabetic retinopathy (PDR) or preproliferative diabetic retinopathy (PPDR) was present in 5% of the NIDDM patients and in none of the IDDM patients with disease duration of less than 5 years. In the group with disease duration of 5-10 years, PDR or PPDR was present in 26% of the NIDDM patients, 14.2% of the IDDM patients. When duration of diabetes went beyond 10 years, this figure rose to 33.3% in both NIDDM and IDDM groups.

Diabetic maculopathy was present in 11.6% of the patients with NIDDM and in no patient with IDDM with diabetes duration of less than 5 years. This figure increased to 20% in the NIDDM group and to 28% in IDDM group with a duration of 5-10 years. In patients with diabetes of more than 10-year duration, maculopathy was present in 25% of the patients in the NIDDM group and none in the IDDM group. (Pakistan Journal of Ophthalmology 8:7-11, January, 1992.)

Diabetes mellitus affects nearly all tissues of the eye. Although some effects are mild or temporary with little visual disability, a significant loss of vision can occur when patients develop more serious ocular complications, such as diabetic retinopathy, macular edema, etc. The problems range from simple error of refraction to a total loss of vision.[1,2] Diabetic retinopathy is the most common cause of blindness in the working age group of diabetics in England and the United States.[3] Recent reports from Pakistan suggest that the frequency of diabetic retinopathy in diabetics seen in the eye clinics varies from 26% to 50%.[4,5]

Photocoagulation of the diseased retina may prevent, or delay, blindness from diabetic retinopathy, provided the retinopathy is detected at a manageable stage. Unfortunately, the benefits of photocoagulation are significantly diminished when the retinal changes are advanced. Early detection of diabetic retinopathy by regular eye examinations is thus very important.[6]

In the study presented here, we examined 150 diabetic patients to analyze ocular findings in them, and, hence, assess the incidence of retinal and non-retinal ocular complications of diabetes mellitus in our population.

Material and Methods

A total of 150 patients, 122 with non-insulin-dependent diabetes mellitus (NIDDM) and 28 with insulin-dependent diabetes (IDDM), underwent eye

From the Department of Ophthalmology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, Pakistan.
Reprint requests to Dr. Zia Mohammad, P.O. Box No. 81, Mardan, Pakistan.

Table 1

Duration of diabetes among NIDDM patients

Duration	Males	Females	Total
Less than 5 years	23	37	60
5-10 years	17	33	50
More than 10 years	6	6	12
Total	46	76	122

Table 2

Duration of diabetes among IDDM patients

Duration	Male	Female	Total
Less than 5 years	15	3	18
5-10 years	6	1	7
More than 10 years	2	1	3
Total	23	5	28

examination at the Department of Ophthalmology, Lady Reading Hospital, Postgraduate Medical Institute, Peshawar for cataloging of all non-neuromuscologenic ocular complications of diabetes mellitus.

All patients received a complete ophthalmic examination, including visual acuity determination, slit lamp examination of the anterior segment, tonometry, and funduscopy. In a few selected patients with elevated intraocular pressure gonioscopy was performed to assess the status of the angle of anterior chamber. The fundi were examined by direct as well as indirect ophthalmoscope. Corneal sensitivity was tested with a cotton ball rolled into a thin filament.

According to the duration of diabetes mellitus, the patients were divided into three groups: Group A , the patients with diabetes of five or less years duration; Group B, the patients with diabetes of up to 10 years; and Group C, the patients with diabetes of over 10 years duration. (Tables 1 and 2)

Results

Out of a total of 150 patients examined, 122 (48 men and 74 women) had NIDDM and 28 (23 men and five women) had IDDM. The NIDDM patients ranged in age between 30-80 years and those with IDDM between 12-60 years.

A positive family history for diabetes mellitus was present in 45.9% of the patients with NIDDM in 14.2% of the patients with IDDM.

Corneal sensitivity was reduced in 62.3% of NIDDM patients and 35.7% of IDDM patients.

Cataracts in the form of posterior subcapsular lens opacities was present in 26.2% of NIDDM patients. Mature cataracts or aphakia was noted in 25.4% of NIDDM patients and 14.2% of IDDM patients. True (classical) diabetic cataract was present in only one patient, an 11-year old girl who was in diabetic coma from IDDM.

Chronic open-angle glaucoma or ocular hypertension was present in 9% of NIDDM patients.

Background diabetic retinopathy (BDR) was present in 15% of NIDDM patients in Group A . In NIDDM patients in Group B this figure rose to 18%. In Group C-NIDDM, two-thirds of the patients (66.6%) had BDR. In the IDDM Group A patients, 11% showed BDR. In IDDM Group B this figure climbed to 28.5%, and in Group C it rose to 33.3%. (Tables 3 and 4).

Diabetic maculopathy or macular edema was present in 11.66% of NIDDM Group A, 20% in Group B, and 25% in Group C. In IDDM patients, it was not present in anyone in Group A and Group C, but in two out of seven (28%) patients in Group B. (Tables 5 and 6)

Preproliferative diabetic retinopathy (PPDR) or proliferative diabetic retinopathy (PDR) was present in 5% of NIDDM patients in Group A, 26% in Group B, and in one-third of them (33.3%) in Group C. In the IDDM patients, it was present in 14.2% of patients in Group B and in 33.3% in Group C. No one in Group A had PPDR or PDR. (Tables 7 and 8)

Discussion

Until the discovery of insulin, a young diabetic was fortunate to survive two years after the time of diagnosis.[7] For the last 50 years, the prognosis for

Table 3

BDR in NIDDM patients

Duration	No. Patients	Male	Female	Total	Percentage
Less than 5 years	60	6	3	9	15%
5-10 years	50	4	5	9	18%
More than 10 years	12	3	5	8	66.6%

Table 4

BDR In IDDM patients

Duration	No. Patients	Male	Female	Total	Percentage
Less than 5 years	18	2	-	2	11%
5-10 years	7	2	-	2	28.5%
More than 10 years	3	-	1	1	33.3%

Table 5

Diabetic maculopathy in NIDDM patients

Duration	No. Patients	Male	Female	Total	Percentage
Less than 5 years	60	1	6	7	11.6%
5-10 years	50	3	7	10	20%
More than 10 years	12	1	2	3	25%

Table 6

Diabetic maculopathy in IDDM patients

Duration	No. Patients	Male	Female	Total	Percentage
Less than 5 years	18	-	-	0	0%
5-10 years	7	1	1	2	28%
More than 10 years	3	-	-	0	0%

Table 7

PPDR and PDR in NIDDM patients

Duration	No. Patients	Male	Female	Total	Percentage
Less than 5 years	60	0	3	3	5%
5-10 years	50	5	8	13	26%
More than 10 years	12	4	-	4	33.3%

Table 8

PPDR and PDR in IDDM patients

Duration	No. Patients	Male	Female	Total	Percentage
Less than 5 years	18	-	-	0	0%
5-10 years	7	1	-	1	14.2%
More than 10 years	3	1	-	1	33.3%

diabetes mellitus has been favorable, but the debilitating complications of diabetes of extended duration have become a major threat to both the quality and the length of life for a diabetic.

Diabetic retinopathy is the major cause of blindness in diabetics in the developed countries, and the accelerated cataract formation is the more prevalent diabetic complication in the underdeveloped countries.

In the outer eye, diabetic changes are minimal and the visible lesions take years to develop.[8] The functional abnormalities however, may be detected long before anatomical changes are evident.

Corneal abnormalities in diabetics include increased corneal thickness, epithelial alterations, decreased tear production, and reduced corneal sensativity.[9,10] In our series, corneal sensitivity was reduced in 62.3% of the NIDDM patients and in 35.7% of the IDDM patients. Schultz and associates[9] have, however, reported reduced sensitivity in 18% of their diabetic patients. This variation might be due to different methods of testing in our and their studies.

The evidence about the association of diabetes and cataract is a conflicting one. Some studies[11] have shown that senile cataract extraction is more common in diabetics than in non-diabetics. Conversely, Waite and Beetham[12] have reported no difference in the incidence of cataract development in diabetic and non-diabetic populations. In our study, mature cataract or aphakia was present in 25.4% of the NIDDM patients and in 14.2% of the IDDM patients. Many observers[11,13,14] have reported a cataract prevalence rate varying from 6% to 45% with increasing age in diabetics. An increase in the incidence with increasing age was noted in both of our groups. The true diabetic cataract was found in only one patient, an 11-year old girl with IDDM, and who was in diabetic coma. Her lenticular opacities disappeared on normalization of her blood glucose level.

Primary open-angle glaucoma bears some relationship to diabetes mellitus. We detected an increase in the intraocular pressure in 9% of our patients. Other writers have reported that nearly 13% of patients attending glaucoma clinics have diabetes.[11] Our results are also comparable to these workers.

Background diabetic retinopathy (BDR)

It has been well established that retinopathy is the most serious and most debilitating ocular complication of diabetes mellitus. It generally becomes more prevalent as the duration of diabetes increases.[2,15] We found a similar relationship between increased frequency and increase in the duration of diabetes.

Background diabetic retinopathy was found in 15% of NIDDM patients and in 11% of IDDM patients in Group A. This figure rose to 18% in NIDDM patients and to 28.5% in IDDM patients in Group B. In Group C, the incidence was 66.6% in NIDDM patients and 33.3% in IDDM. This figure is in agreement with that reported in the Wisconsin epidemiologic study for diabetic retinopathy (WESDR).[15,16] In the WESDR,[15] the incidence of retinopathy was 24% in those having the disease for less than 3 years. With 20 or more years BDR was present in 60% of patients.

Diabetic maculopathy

Diabetic maculopathy is the greatest single cause of visual impairment in diabetic patients.[17] In our patients diabetic maculopathy was found in 11.6% of NIDDM patients at diagnosis and below 5 years duration. After 5-10 years duration this figure was 20%. In those NIDDM patients having the disease for more than 10 years this figure was 25%. In the IDDM group 28% had maculopathy after 5 to 10 years duration. These findings are consistent with those of WESDR.[18] In the WESDR,[18] this figure was 21% in those with a duration of 20 years or more. In this study maculopathy was rarely found during the first 9 years after diagnosis in the juvenile onset IDDM and more frequently in the NIDDM subjects shortly after diagnosis.

Proliferative diabetic retinopathy (PDR)

Proliferative stage of diabetic retinopathy begins with the appearance of abnormal blood vessels, usually found as neovascularization on the optic disc (NVD) or as neovascularization elsewhere (NVE) in the vicinity of retinal vessels. According the Klein et al,[15,18] PDR develops in 40% of NIDDM patients treated with insulin, but in only 20% of those on oral agents and diet only. It reaches up to 60% in IDDM patients after 20 or more years of duration of diabetes.

In the present study 5% of NIDDM subjects with less than 5 years duration of diabetes were found to have proliferative lesions. With 5-10 years duration 26% were found to have PDR and 33.3% of those having the disease for more than 10 years suffered from PDR. In the IDDM group, on the other hand, no proliferative lesions were detected in those with less than 5 years duration of diabetes. With a duration between 5-10 years, 14.2% had proliferative diabetic retinopathy, while such lesions were found in 33.3% of those IDDM patients who had diabetes for more than 10 years.

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Ophthalmic "Pastpourri"

Glaucoma *Déjàvu*

One repeatedly hears about the incredible explosion of ophthalmic technology and knowledge that has taken place over the past two decades. This greased lightning of progress has changed things with such a speed and brought so many new options in the practice of ophthalmology that most of the diagnostic and therapeutic modalities of just a few years ago have become obsolete in many parts of the world. It may interest both the doctors and the government of our country to check out how much our masses have benefitted from this staggering progress around the globe. The following observation made 35 years ago by an American ophthalmologist who then visited our part of the world could serve as a sobering starting point to measure our own pace of progress:

"Glaucoma surgery was second in frequency and, again, bilateral operations were done at the same time. ...Because of the previously mentioned economic and social conditions, medical therapy was not feasible in most instances. Patients could not afford miotics, even if they were available, and could not be relied upon to use them daily. Therefore, the indication for surgery would consist merely of making the diagnosis of primary glaucoma."

Richard A. Ellis:
Ophthalmic surgery in India.
Am J Ophthalmol 44:762, **1957.**

Camera Clinicals

In this section of THE JOURNAL, *photographic documentation of interesting and challenging observations are persented to the readers. They should make their diagnosis from the given information, and compare their conclusions with the exposition given on page 18.* -Editor.

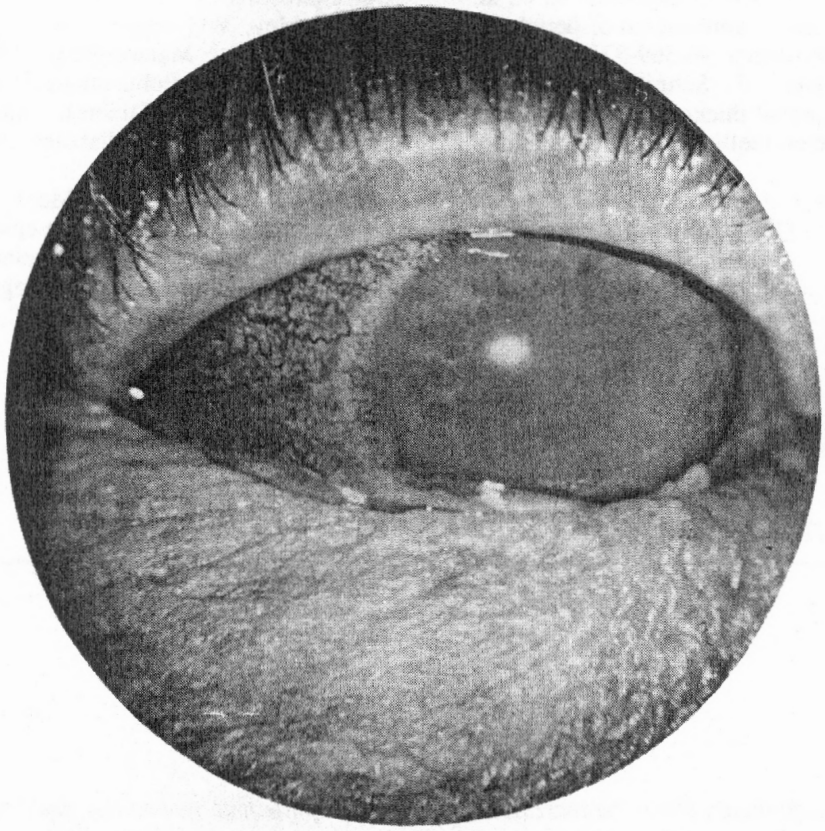

Figure 2

Figure 1: A 69-year-old woman complained of recurrent episodes of pain, redness, and watering in her right eye for over a year. Each episode would last for a couple of weeks. On several occasions it was diagnosed as conjunctivitis and treated with topical antibiotic-steroid combination drops either by her family physician or an emergency room doctor. The overwhelming characteristic of her attacks was constant foreign body sensation. Although other attacks had responded to the above treatment, the present episode became steadily worse, and at the time of visit to the ophthalmologist, the sight in the eye had also significantly decreased. The other eye did not have any sort of signs or symptoms.

On eye examination her visual acuity was reduced to CF (counting fingers) in the right eye. In the left eye it was 20/20. The findings shown in Figure 2 were striking, with intense redness of the conjunctiva and clouding of the cornea. A closer examination revealed an area of loss of epithelium in the lower half of the cornea. There was copious white discharge. The cloudiness of the cornea precluded any meaningful evaluation of the anterior chamber on slit lamp. However, the cause of eye problem was easily detectable. The patient was placed on topical antibiotics and other relevant therapy, to which the condition responded very nicely. However, to prevent further similar episodes, the patient underwent a surgical procedure.

Management of Persistent Retinal Detachment without Repeat Surgery*

Khalid J. Awan, F.P.A.M.S.

ABSTRACT: In selected cases of persistent retinal detachment following a scleral buckling procedure, a postoperative non-invasive manipulation by herein described new technique may be helpful in its successful resolution. The separated retina is held in a forced contact with the buckle for two to three minutes by scleral depression throughout the extent of the buckle under a 360° perilimbal subconjunctival or retrobulbar anesthesia with 0.75% bupivacaine (Marcaine). The technique involves three repititions of this maneuver in a single procedure. A supplemental retrobulbar injection of bupivacaine at the conclusion of procedure ensures immobility of the eyeball for several hours following this manipulation. The patient receives post-manipulation binocular patching and bedrest for 24 hours. This technique was successful in reattaching the retina in two aphakic and five phakic eyes. (Pakistan Journal of Ophthalmology 8:13-16, January, 1992.)

The persistence or recurrence of retinal detachment necessitates reoperations in 5-15% of the patients who undergo a surgical procedure for the reattachment of a detached retina.[1] If the absorption of subretinal fluid does not take place within four to five postoperative days, a defect in the operative technique or in the subretinal fluid absorption mechanism is the usual cause of failure.[2-4] In the absence of any reason for a slower absorption of the subretinal fluid, a residual vitreous traction, inadequate indentation of sclera by a correctly located buckle, a poorly placed buckle, a partially closed hole, a missed retinal hole, a radial retinal fold, proliferative vitreoretinopathy, etc. may be responsible for a postoperative persistence of detachment.[2-7] In cases where the mechanism of subretinal fluid absorption is not defective, and where no evidence of residual vitreous traction or proliferative vitreoretinopathy is present, photocoagulation around the faulty breaks,[1,2,5,8,9] releasing of the subretinal fluid,[2] an intravitreal air or gas injection,[2,10] replacing the lost volume by physiologic solution injection,[2] etc. have been suggested. I herein describe a new non-invasive approach to manage persistent retinal detachment in carefully selected cases.

Technique

Following local anesthesia, either by a 360° perilimbal subconjunctival or by a retrobulbar injection of 0.75% bupivacaine solution, the areas of detached retina suspected of harboring any open or partially sealed holes are brought in forced contact with the buckle by scleral depression. The scleral depressor is held in the desired position for 2-3 minutes, and the maneuver is repeated three times at each selected site in a single procedure. At the conclusion of procedure, a supplemental retrobulbar injection of 0.75% bupivacaine is given. A binocular patching is applied after bathing the eye with a topical antibiotic ophthalmic solution, and the patient is kept on bedrest for the next 24 hours.

This technique proved successful in reattaching the retina within 24 hours in seven carefully selected patients, four men and three women, ranging from 35 to 74 years in age. Five of the eyes were phakic and two were aphakic.

Case Reports

CASE 1: A 47-year-old man underwent a segmental scleral buckling procedure with drainage of the subretinal fluid for a rhegmatogenous retinal detachment with a single superior temporal horseshoe tear in his left eye. The detachment was nearly three weeks old and involved macula and inferotemporal retina from 1 o'clock to 8 o'clock positions (Figure 1). The retina was flat on the operating table and the buckle appeared to nicely cover the retinal hole. However, an accumulation of some subretinal fluid was noticed on the first postoperative day. On third postoperative day, the detachment had reached the preoperative extent with a convex ballooning of detached retina. Indirect ophthalmoscopy showed that the posterior edge of the hole was not in contact with the buckle. Bilateral eye patching and bedrest were

From the Department of Ophthalmology, University of Virginia School of Medicine, Charlottesville, Virginia (USA), and Awan Ophthalmology Clinic, Norton, Virginia.

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Reprint requests to Khalid J. Awan, M.D., F.P.A.M.S., 1921 Park Avenue, SW, Norton, Virginia 24273 USA.

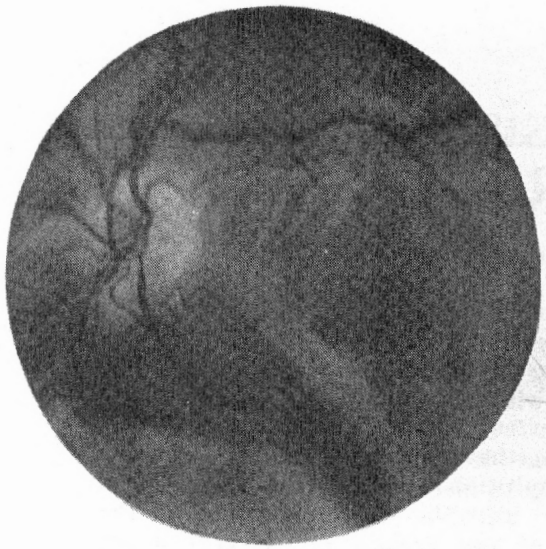

Figure 1 (Awan): Case 1. Left eye. Three-week old rhegmatogenous retinal detachment with macular involvement.

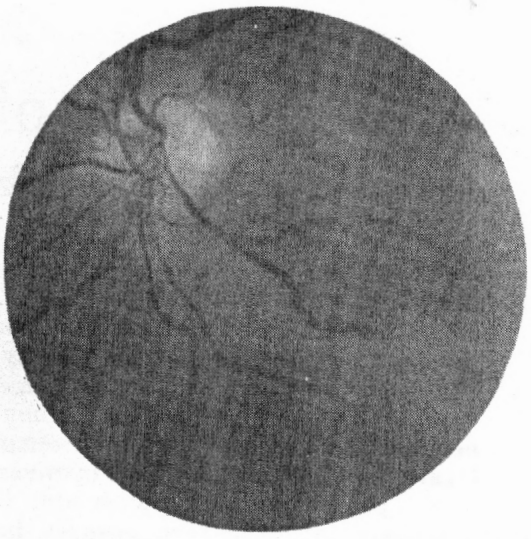

Figure 3 (Awan): Case 1. Left eye. On the sixth day after buckling (24 hours after the extended scleral depression), the retina is fully reattached The haze in the right lower corner is an artifact.

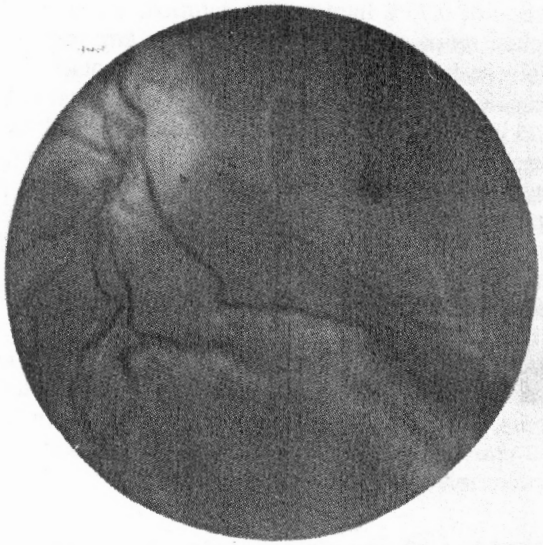

Figure 2 (Awan): Casel. Left eye. One day after the buckling procedure, tte retina has re-detached to the preoperative extent, although it was flat on the operating table.

continued until the fifth postoperative day, with no improvement (Figure 2). On sixth postoperative day, the above described technique of forced contact of detached retina to its treated bed was applied under local anesthesia. Twenty-four hours afterward, the subretinal fluid had completely absorbed and the retina had become fully reattached (Figure 3), and has remained attached for the last many years with a final corrected visual acuity of 20/30.

CASE 2: A 71-year-old man developed loss of sight in his left eye. He had undergone a successful intracapsular cataract extraction in that eye nearly a decade ago with good visual outcome. His present complaint, which was worse in the mornings and improved as the day went on, began about three weeks before his visit. The examination showed a detachment of the inferior half of the retina with edema of the macular area. An indirect ophthalmoscopic examination uncovered three round holes surrounded by an atrophic patch in the inferonasal retina. He underwent a 360° encircling episcleral band with cryoretinopexy and subretinal fluid drainage. Although the retina was found perfectly flat and the holes were properly positioned on the buckle at the conclusion of the operation, it had redetached to the preoperative extent the next morning (Figure 4).

The examination with indirect ophthalmoscope showed that the preoperatively recorded retinal holes appeared well-treated and properly positioned. It was decided to keep the patient under observation. The bilateral patching and bedrest for four more days failed to bring about any improvement. A detailed indirect ophthalmoscopic examination was performed on the fifth postoperative day, and although no other definite holes were detected, an area of atrophy with a questionable round hole was found at the 6 o'clock position in the area of posterior half of the crest of buckle. The patient was given a retrobulbar block, and the suspicious area of the retina was brought in contact with the buckle by using the scleral depression technique as described above. To be sure, the scleral depression manipulation was done all around the encircling buckle. At the conclusion of the procedure, the patient received a supplemental retrobulbar injection of a 0.75% solution of bupivacaine and a

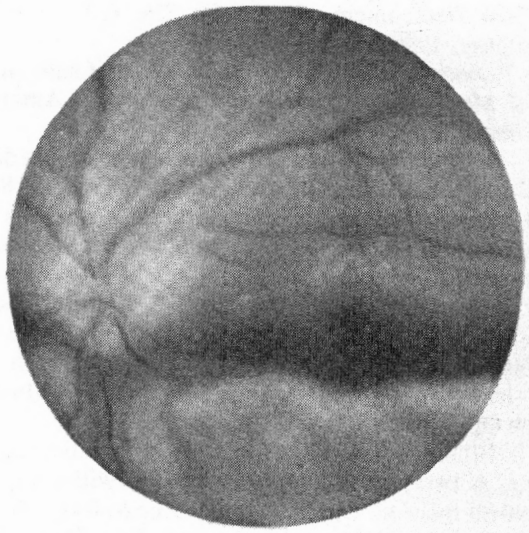

Figure 4 (Awan): Case 2. Left eye. On the first postoperative day following the scleral buckling procedure, the retina has become re-detached to its preoperative extent, although it was totally flat on the operating table.

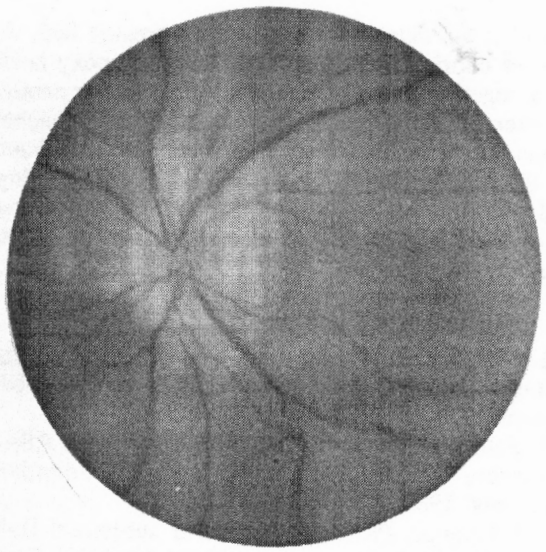

Figure 5 (Awan): Case 2, Left eye. On the sixth post-buckling procedure day (24 hours after the extended scleral depression manipulation), the retina has become completely reattached.

binocular patching. The patient remained on bedrest for the following 24 hours.

The retina had completely reattached the next morning, and it has remained so for the last several years (Figure 5). The eye currently has an aphakic corrected visual acuity of 20/50+.

Discussion

Following a scleral buckling procedure, even when the subretinal fluid is only partially drained, the detached retina becomes rapidly settled within 24 to 48 hours in otherwise healthy eyes.[3,4,5,11,12] Nonetheless, the absorption of subretinal fluid may be very slow, taking weeks or even months, in some patients with faulty subretinal fluid absorption mechanism, abnormally viscous subretinal fluid, or some local pathologic process, such as residual vitreous retraction, proliferative vitreoretinopathy, et cetra.

The retinal pigment epithelium is significantly responsible for the removal of subretinal fluid by metabolic transport of ions and the fluid absorption associated with it. Any abnormality affecting its function delays the fluid absorption.[5] The oncotic pressure difference between the choriocapillaris and the subretinal space and hydrostatic force of the intraocular pressure pushing the detached retina against the pigment epithelium are also involved in the removal of the subretinal fluid. The eyes with inflammatory or effusive phenomena also have slow subretinal fluid absorption. Hence, the postoperative settling of the detached retina takes a much longer time in eyes with retinal pigment epithelial atrophy, choroidal detachment, choroidal effusion, uveal inflammation, degenerative changes at the level of choriocapillaris and Bruch's membrane, and bullous retinoschisis. Although it is felt that the duration of retinal detachment, age of the patient, high myopia, and senile choroidopathy also influence the absorption of subretinal fluid,[3,7] some studies do not support this view.[12] The viscosity of the subretinal fluid also plays a role in the rate of absorption of the subretinal fluid. In long-standing detachments, the protein content of the subretinal fluid increases, making the fluid more viscous and higher in osmolarity. This disrupts the normal fluid uptake by the retinal pigmentary epithelial cells, delaying the absorption of fluid. Robertson[6] pointed out that the peculiar subretinal precipitates he recently described also portend a slow absorption of the subretinal fluid.

The patients with a defective subretinal fluid absorption mechanism, are not suitable for the technique described in this paper. Similarly, the patients with a missed retinal tear, the treated holes that are more than 3 mm away from the buckle, a grossly poor placement of the buckle, an obvious residual vitreous traction, any signs of proliferative vitreoretinopathy, or the ones showing a steady day-to-day improvement are also poor candidates. After a postoperative observation of four to five days, the patients with a worsening and convex-contoured persistent detachment are selected for this technique, provided they have an open hole within 3 mm of a well-placed buckle, a low but correctly located buckle, a partially closed but on the buckle hole, a "fishmouthing" tear on a radial fold, or a persistent detachment the cause for which cannot be attributed to any of the above discussed factors.

The exact mechanism of how this technique works is not clear. I feel that after an extended forced contact

between the detached retina and its treated bed, the thicker chorioretinal exudation from cryopexy holds them together to seal the retinal tear, just long enough to interrupt the seepage of fluid under the detached retina. This situation, kept undisturbed for many hours by the immobility of the eyeball, bilateral patching, and bedrest, leads to rapid absorption of the subretinal fluid with subsequent resolution of the persistent detachment.

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Ophthalmic "Pastpourri"

MYOPIC VIEW, OLD AND NEW

A Century Ago:

Edward Jackson, of Jackson's Cross-Cylider fame and who later became the renowned Editor-in-Chief of the American Journal of Ophthalmology, emphatically recommended that " the constant wearing of corrective lenses, no matter what their strength, with careful attention to other aspects of ocular hygiene, checks promptly and permanently the advance of myopia in majority of cases."

- Jackson, E: The full correction of ametropia. J Am Med Ass September 2, **1891.**

Today:

In 1970, Sir Stewart Duke-Elder commented on Jackson's above view about myopia and wrote in his famous System of Ophthalmology: "...such insistence is unjustified in simple myopia and its value is questionable even in higher degrees associated with degenerative changes.

- System of Ophthalmology, vol. 5, St. Louis, C.V. Mosby Company, **1970.**

Figure 1

Posterior Dislocation of the Nucleus During Extracapsular Cataract Surgery

Khalid J. Awan, F.P.A.M.S.

ABSTRACT: A 59-year-old man underwent anterior vitrectomy and extracapsular cataract extraction with posterior chamber intraocular lens implantation for a traumatic cataract in his right eye. Although procedure went well, on the first postoperative visit a large fragment of nucleus was noted in the vitreous. The patient refused further surgical intervention so soon after the recent eye operation. He was placed on topical cycloplegics, corticosteroids, and antibiotics, and systemic steroids. A week following the cataract operation the patient developed a pseudohypopyon of lens material floating forward from the vitreous. In another four weeks the intraocular pressure increased to 35 mm Hg. An anterior chamber irrigation cleared the pseudohypopyon and normalized the intraocular pressure. However, both reappeared in another two weeks. The successful removal of nuclear fragment by uncomplicated pars plana vitrectomy recovered the eye, which has visual acuity of 20/30 a year after the last surgery. (Pakistan Journal of Ophthalmology 8:6, 17, January, 1992.)

This dislocation of the nucleus or its fragments into the vitreous during intracapsular and extracapsular cataract extraction has been reported, but fortunately is not a common event.[1] However, the complication is more common (0.4% to 4.0%) during phacoemulsification.[2]

Small lens fragments in the vitreous may not cause any problem for sight or the eye, and topical corticosteroids might be all that is required. In most instances, these small particles will become spontaneously absorbed.[3] However, large particles of nuclear material may lead to many undesirable complications with loss of sight or even of the eye itself.[1] The more important of these complications are phacogenic glaucoma and lens-induced uveitis. Hence, the presence of large amount of cortical material in the vitreous, persistent uveitis, or glaucoma non-responsive to medical therapy are indications for surgical intervention. In other cases on proper medical therapy, a delay in operative measures may be acceptable.

In those patients who need surgical removal of lens material from the vitreous, referral to a vitreoretinal surgeon for pars plana vitrectomy is indicated. However, if such a facility is not available, the patient should be placed on his belly, and the nuclear fragment allowed to drop into the anterior chamber, from whereit can be removed through a limbal route. A Stryker frame may be a helpful tool in this maneuver.[4] It must be remembered, however, that despite a successful removal of the nucleus dislocated into vitreous even with the most sophisticated modern method, the final outcome may not be so satisfying. In one recent report on the subject, seven of 13 patients in whom nucleus was removed from vitreous by modern pars plana technique regained visual acuity of only 20/80 or less.[2] In another report, the authors used high-density vitreous substitute (fluorosilicone oil) to remove the dislocated lens by making it float.[5]

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Figure 2

Cloudy Cornea with Ulceration, an Unusual Complication of Entropion

Khalid J. Awan, F.P.A.M.S.

ABSTRACT: A 69-year-old Caucasian American woman had episodes of inflamed painful eye, which were treated as conjunctivitis by her physicians for nearly a year. The last episode did not respond to their treatment. The consultant ophthalmologist made the diagnosis of senile entropion of the right lower eyelid. The irritation from the eyelashes had caused most severe reaction in the conjunctiva, marked cloudiness of cornea, and the loss of corneal epithelium in the lower half of the cornea. Topical antibiotics and taping of the lid to the cheek soon cleared the corneal infiltrate and the conjunctival inflammation. The surgical repair corrected the entropion. To see such a severe complication of entropion in an advanced country is very unusual. The curricula for emergency room doctors and family physicians should include more about clinical features of common eye diseases. (Pakistan Journal of Ophthalmology 8:12, 18, January, 1992.)

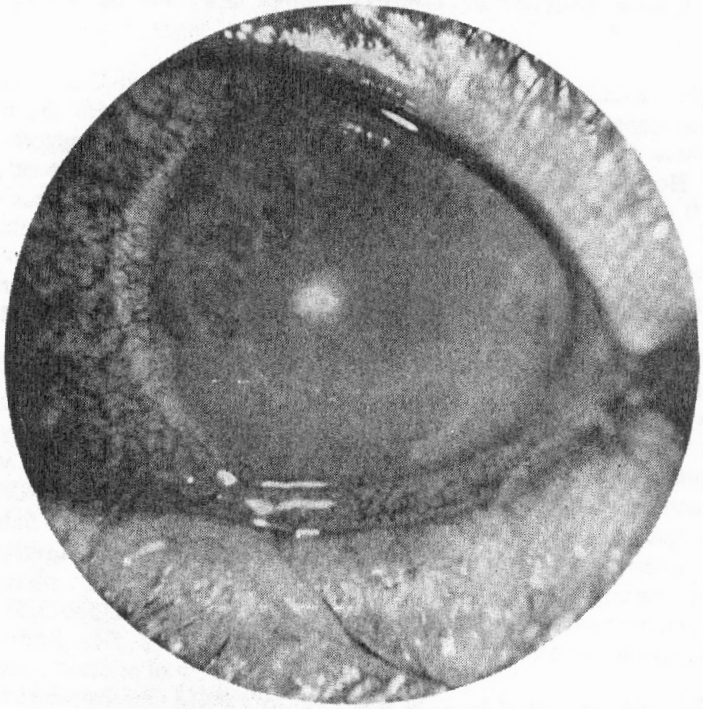

Figure 3

The rolling in of the lower eyelid due to an involutional entropion in this elderly lady is obvious in Figure 2 on page 12. Figure 3 on this page clearly illustrates the epithelial defect caused by the mechanical injury from eyelashes jabbing against the globe. Inflammation that caused cloudiness of cornea was in probably due to mechanical trauma rather than due to infection. The same seems true for the epithelial defect. Both of these rapidly cleared after the eyelid was taped to the cheek. Apparently, antibiotic drops prescribed by her doctor kept the patient's eye from getting infected. This case demonstrates that even minor problems may lead to serious,even permanent, loss of sight if neglected, or mismanaged.

Book Reviews

Edited by Khalid J. Awan, F.P.A.M.S.

UVEITIS. A Clinical Approach to Diagnosis, and Mangement, Second Edition, by Ronald E. Smith, Robert A. Nozik, 1989. Williams & Wilkins, 428 East Preston Street, Baltimore, MD 21202, U.S.A. 266 pages, 12-page index, hardcover. Price, US $83.95.

"What we need here most is a plan on how to diagnose and treat uveitis without having to resort to a battery of expensive and sophisticated tests, most of which are not available to us anyway." I was told some time back by Professor Murad Ali, Chairman, Department of Ophthalmology, Khyber Medical College, Peshawar, where I was doing my visiting professor stint under the auspices of the United Nations Development Programme for transfer of technology to the developing countries. I had mentioned to him an article titled "A Clinical Approach to Uveitis Diagnosis" by Drs. Santos and Nozik that was published in THE JOURNAL in October 1986, and which had become popular with the residents of several institutions. Hence, to review this book was a dual pleasure. Firstly because this book serves an urgent need, and secondly because reviewing this book has made the reviewer himself a better clinician in the field of uveitis.

The book is divided into three sections. Section One contains chapters on clinical history, terminology, classification, signs, goals of management, laboratory testing, and the nonspecific treatment of uveitis. Chapter 6 of this section, "Quick Reference Chart for Uveitis Diagnosis and Management" should be read over and over by the residents; better yet, they should commit it to memory, something the great value of which will come to their mind on innumerable occasions throughout their career. This chart is not just a diagnostic tool, it also is an excellent framework to further build on.

Section Two deals with "Special Clinical Problems in Uveitis." In addition to the discussion of the clinical manifestations directly related to uveitis, choroiditis, and retinitis, this section enhances its practical value by devoting very concise but separate writeups on such topics as "Endophthalmitis," "Retinal Detachment and Uveitis," "Cystoid Macular Edema and Uveitis," "Surgery in Uveitis Patients," etc.

Section Three deals with individual specific entities in separate chapters. The up-to-datedness of this section is made obvious by inclusion of chapters on such recently defined conditions as birdshot choroidopathy, acute retinal necrosis, acquired immunodeficiency syndrome, etc. Another interesting chapter is on relationship of uveitis and gastrointestinal disease.

There are scattered "Selected References" throughout the book, each listing plenty of references on the related topic. However, this reviewer would have preferred if they were also cited in the text. At the beginning of the book a useful chapter on definitions of most relevent terms in uveitis is included. However, this dictionary style alphabetized presentation contains only 39 entries, and some of the oft-used terms, such as pars planitis, are missing from the list. This reviewer also found some interesting typographical errors, such as "fungus" for fundus (page 103), "that" for than (page 104), etc. in this most appealing and reassuringly made up book. The printing and writing are excellent. The conciseness of the book is another attractive feature. First naming the form of uveitis and then meshing it with the most likely diagnosis technique of Drs. Smith and Nozik is a very helpful idea that every reader will find highly useful in taking care of uveitis patients. This reviewer highly recommends this book to clinicians and trainees alike. It belongs on the shelves of all medical libraries in Pakistan and elsewhere. Professor Murad, here is the answer to your wish.

ATLAS OF OCULAR MOTILITY, by Leonard B. Nelson, Robert A. Catalano, 1989. W.B. Saunders Company, Harcourt Brace Javonovich, Inc., The Curtis Center, Independence Square West, Philadelphia, PA 19106-3399, 228 pages, 4-page index, illustrated, hardcover. Price, US $85.

Axes, planes, laws, vergences, ductions, hypers, hypos, esos, exos, phorias, tropias, synergists, antagonists, recessions, resections, there is so much to confuse the poor resident coming for the first time to the pediatric ophthalmology rotation. Want to make someone lose his sanity? Just ask him to make a mental picture of a few of the terms mentioned above. Hence, this reviewer was so delighted when he first saw von Noorden and Maumenee's *Atlas of Strabismus* almost twenty-five years ago during his early training years. The fact is that extraocular muscle functions and strabismus cannot be adequately taught without some kind of sketches and drawings.

This beautifully produced publication is prepared on the established style of modern atlases, giving concise text on the left-hand page and the figures that go with it on the opposite right-hand page. The writing is easily understandable, and the drawings and photographs clearly illustrate the written text.

The contents are divided into 13 chapters titled Anatomic Relationships, Anatomy of Extraocular Muscles, Physiology of Ocular Muscles, Sensory Physiology and Pathology, Sensory Adaptations to Strabismus, Tests of the Sensory Status, Introduction

to Strabismus, Esodeviations, A and V Patterns, Cyclovertical Deviations, Monofixational Syndrome, and Syndromes and Special Forms of Strabismus. This last chapter includes descriptions and photographic demonstrations of Duane's retraction syndrome, all three types, Brown's syndrome, cyclic strabismus, Mobius's syndrome, double elevator palsy, thyroid myopathy, blowout fractures of the orbit with extraocular muscle entrapment, Marcus Gunn's syndrome, generalized fibrosis syndrome, chronic progressive external ophthalmoplegia, Parinaud's syndrome, and internuclear ophthalmoplegia, all of which are presented with lucid text and easily understood drawings and photographs. I found another chapter, 4th on sensory physiology and pathology to be very impressive, and it also includes techniques of the assessment of visual acuity in infants and preverbal childern. At the end of the book are given "Selected References" for each of these chapters. The remark by Dr. Robison Harley in the foreword that the Atlas is "characterized by the excellence of diagrams supplemented by lucid descriptions" will find support from all quarters.

This publication though similar to *Atlas of Strabismus* in format and approach, it is more detailed and employs more photographs of actual patients to illusrate each entity. This obviously makes it a more useful tool for learning and reviewing the strabismus problems.

The *Atlas* will serve, as the authors say in the preface, " as a helpful teaching and review manual for those interested in ocular motility." This reviewer strongly recommends it for the residents. In fact, ophthalmologists at all levels of training and expertise will be well-armed against problems of motility if they combine the owning of Nelson and Catalano's *Atlas* with that of Kenneth W. Wright's book *Color Atlas of Ophthalmology Surgery: Strabismus*, which was reviewed in the April 1991 issue of THE JOURNAL.

HANDBOOK OF OCULAR DRUG THERAPY AND OCULAR SIDE EFFECTS OF SYSTEMIC DRUGS, by Deborah Pavan-Langston, Edmund C. Dunkel, 1991. Little, Brown and Company, 34 Beacon Street, Boston MA 02108, U.S.A. 464 pages, 65-page two part index, paperback. Price, US $32.50.

This useful paperback is, according to the authors, actually two books in one. The first part is titled the "Ocular Drug Therapy," and the second the "Ocular Side Effects of Systemic Drugs." The information is presented in text outlines as well as in the form oftables for "rapid retrievel of data on an extensive list of drugs."

The contents are divided into 30 chapters. The first 10 belonging to the first part include Pharmacokinetic of Ophthalmic Drug Administration, Anti-Allergy Agents and Ocular Decongestants, Antibiotics, Antifungal Drugs, Antioparasitic Drugs, Corticosteroid, Immunosuppressive Agents, and Nonsteroidal Anti-inflammatory Drugs, Local Anesthetics, and Mydriatics and Cycloplegics. The remaining 20 chapters are on "Ocular Side Effects of Systemic Drugs" and include Alcoholism Antagonists, Analgesics and Antiarthritics, Anesthetics, Antiallergy Drugs: Antihistamines, Anti-infectives, Antineoplastic Agents, Cardiovascular Drugs, Central Nervous System Agents, Dermatologic Agents, Diuretics and Osmotics, gastrointestinal Agents, Heavy Metal Chelators, Hematologic Agents, Hormonal Agents, Immunosuppressants, Neuromuscular Agents, Solvents, Vaccines, and Vitamins.

The information in this book appears on the surface to resemble that found in *Physician's Desk Reference*, but actully is much more extensive and selective. The book will be of greatest benefit to the trainees. However, even seasoned practitioner will occasionally benefit from it when faced with a drug he never uses or uses only occasionally.

The writing is easily understandable, but a larger point size of the print would have been more helpful. However, the printing is excellent.

At the end of the book there are two extensives indices, a 37-page one alphabetical one and the other 38-page one on proprietary and generic names, both of which are naturally very essential for such a text. The book will prove a very useful device for residents and trainees.

CLINICAL TESTS OF VISION, by Lars Frisen, 1990. Raven Press, Ltd., 1185 Avenue of the Americas, New York, New York 10036, U.S.A. 212 pages, 8-page index, paperback. Price, US $71.00.

"A good verbal description of a visual scenery requires a rich language." This opening sentence of the preface to *Clinical Tests of Vision* readily conveys the quality of presentation this book makes. Add to it the clinical and experimental expertise of Dr. Frisen, and you have an excellent book, which makes a definitive guide for evaluation of eye patients. The book presents a comprehensive scheme of testing visual impairment in a clinical setting.

This book is divided into the following chapters: The Foundations of Clinical Vision Tests, Measuring Vision, Visual Acuity, Metamorphopsia and Dysmetropsia, Color Vision, The Foundations of Visual Field Testing, The Cornerstones of Perimetric Strategy, Visual Field Screening, Selecting Equipment for Visual Field Defects: General Principles, Interpretation of Visual Field Defects: Up to the Optic Nerve, Interpretation of Visual Field Defects: The Visual Pathways, and Test Cases. Out of the total 204 pages of text in this book, 150 are devoted the testing of visual fields.

The book departs from the standard writing of technical ophthalmic texts, using an informal style, which makes the book a joy to read as well. *-KJA*

Abstracts from Elsewhere

Edited by Khalid J. Awan, F.P.A.M.S.

Archives of Ophthalmology

MULTIPLE-DOSE EFFICACY COMPARISON OF THE TWO TOPICAL CARBONIC ANHYDRASE INHIBITORS SEZOLAMIDE AND MK-927, A Brown, EA Lippa, F Gunning, C Benichou, P Lesure, D Sirbat, J Royer, J Flament, C Clineschmidt, D Panebianco, A Buntinx, F Brunner-Ferber, JL George, E Greve. The authors compared the multiple-dose twice-daily efficacy of the topical carbonic anhydrase inhibitor MK-927, a racemic compound, with that of its pharmacologically more active S-enantiomer in a four-center, double-masked, randomized, placebo-controlled, parallel study of 1.8% sezolamide hydrochloride (MK-417), 2% MK-927, and placebo, given twice daily to 48 patients with bilateral primary open angle glaucoma or ocular hypertension and morning intraocular pressure greater than 24 mm Hg in both eyes following washout of ocular hypotensive medications. Paralled 10-hour modified diurnal curves were performed before the study and on day 14, with 4-hour curves on days 1 and 4. Both compounds demonstrated significant lowering of intraocular pressure at 8 a.m., 12 hours following the evening dose, and through 10 and 6 hours following the 8 a.m. dose for sezolamide and MK-927 (-13.5% and -9.6%); peak effect occurred 2 hours after dose administration and was -19.4% and -19.2% for sezolamide and MK-927, respectively. Morning trough (evening) activity as measured by mean percent change in intraocular pressure from prestudy was -9.2% for sezolamide and -11.1% for MK=927, respectively. From 2 hours after dose administration, sezolamide consistently demonstrated a slightly greater decrease in intraocular pressure than MK-927; however, these differences were not statistically significant. *(Arch Ophthalmol. 1991;109:50-53) Reprint requests to Clinical Research, Merck Sharp & Dohme Research Laboratories, West Point, PA 19486 (Dr Lippa).*

CHRONIC POSTOPERATIVE ENDOPHTHALMITIS ASSOCIATED WITH *ACTINOMYCES* SPECIES, TJ Roussel, ER Olson, T Rice, D Meisler, G Hall, D Miller. The authors isolated *Actinomyces* species, gram-positive, non-spore-forming anaerobic bacilli were isolated from intraocular fluid obtained from four otherwise healthy patients with a delayed onset of postoperative endophthalmitis. One patient had a mixed anaerobic infection with recovery of both *Actinomyces israelii* and *Propionibacterium acnes*. In all four patients early postoperative visual acuity was good but was eventually markedly reduced by intraocular inflammation that was first observed between 21 days and 4 months following uneventful extracapsular cataract extraction and posterior chamber intraocular lens implantation. Inflammation was characterized by anterior segment and vitreous cellular debris in all cases. All eyes responded to therapy that included intraocular, topical, and systemic antibiotics as well as pars plana vitrectomy and partial iridectomy. These cases further illustrate the need for microbiologic investigation, including anaerobic cultures, in all cases of chronic postoperative inflammation following extracapsular cataract extraction, regardless of the time of onset. *(Arch Ophthalmol. 1991;109:60-62) Reprint requests to 329 W 40th St, Scottsbluff, NE 69361 (Dr Roussel).*

THE RELATIONSHIP OF VISUAL ACUITY, REFRACTIVE ERROR, AND PUPIL SIZE AFTER RADIAL KERATOTOMY. JT Holladay, MJ Lynn, GO Waring III, M Gemmill, GC Keehn, B Fielding. The authors undertook a study to better define the relationship between residual refractive error, uncorrected visual acuity, and pupil diameter, by comparing 42 eyes that had an eight-incision radial keratotomy according to the Prospective Evaluation of Radial Keratotomy Study protocol with 42 matched control eyes. The parameters measured were best corrected visual acuity, uncorrected visual acuity, and the change in cycloplegic refraction with enlarging pupil diameter. The best corrected visual acuity was 20/16 in both the radial kertatotomy and control groups, but the varibility (SD) was higher in the radial keratotomy group. The average uncorrected visual acuity was 0.35 (35%) better in the radial keratotomy group, but the variability was 1.77 times higher. Change in refraction with dilatation occurred in 9% of the controls and 36% of the radial keratotomy patients, indicating a significant difference (P=.002). The change in refraction with dilation in the eyes with radial keratotomy was almost equally split between a hyperopic change (17%) and a myopic change (18%), which was much different than in the control eyes, only 2% of which changed in a hyperopic direction and 7% in a myopic direction. The radial keratotomy patients with a myopic change had the best uncorrected visual acuity, indicating that positive spherical aberration yielded the best aspherical surface for uncorrected visual acuity. *(Arch Ophthalmol. 1991;109:70-76) Reprint requests to Hermann Eye*

Center, Hermann Hospital, 6411 Fannin, Houston, TX 77030 (Dr Holladay).

CLINICALLY DETECTABLE NERVE FIBER ATROPHY PRECEDES THE ONSET OF GLAUCOMATOUS FIELD LOSS. A Sommer, J Katz, HA Quigley, NR Miller, AL Robin, RC Richter, KA Witt. The authors performed standardized perimetery and nerve fiber layer and color fundus photography annually on 1344 eyes with elevated intraocular pressures. In 83 eyes, glaucomatous field defects developed that met rigid criteria on manual kinetic and suprathreshold static perimetery. Individual nerve fiber layer photographs were read by two masked observers. The more sensitive of the two identified nerve fiber layer defects in 88% of readable photographs at the time field loss first occurred; 60% (6/10) of eyes already had nerve fiber layer defects 6 years before field loss. In contrast, the nerve fiber layer was considered abnormal in only 11% (3/27) of normal eyes and 26% (84/327) of hypertensive eyes. The location of nerve fiber layer and yield defects closely corresponded, but nerve fiber layer loss was generally more wide-spread. Examiner experience and severity of optic nerve damage influenced results. Mild focal defects were more readily recognized than more severe diffuse atrophy. Nerve fiber layer defects expanded with time, often by the development and coalescence of adjacent areas of damage. *(Arch Ophthalmol. 1991;109:77-83) Reprint requests to Wilmer 120, The Johns Hopkins Hospital, 600 N Wolfe St, Baltimore, MD 21205 (Dr Sommer).*

OPTIC NERVE HEAD AND NERVE FIBER LAYER IN ALZHEIMER'S DISEASE, CS Tsai, R Ritch, B Schwartz, SS Lee, NR Miller, T Chi, FY Hsieh. The authors studied (1) the differences in the retinal nerve fiber layer between 26 patients with Alzheimer's disease and 30 age- and race-matched normal controls with use of blue-light high-resolution photography, (2) the differences in disc pallor between 30 patients with Alzheimer's disease and 32 controls with use of a boundary-tracking program and fundus photographs, and (3) the topographic disc variables between 26 patients with Alzheimer's disease and 36 controls with use of an optic nerve head analyzer. A higher proportion of patients with Alzheimer's disease had detectable nerve fiber damage as seen by red-free photography compared with controls. Although the pallor area-to-disc area ratio was not significantly different between patients with Alzheimer's disease and controls, the patients with higher pallor area-to-disc area ratios had higher Alzheimer's Disease Assessment Scale (ADAS) scores and longer durations of disease. Patients had an increased cup-to-disc ratio and cup volume and decreased disc rim area compared with controls. These variables also correlated significantly with ADAS scores and the duration of disease. The correlation among the optic nerve head changes and the ADAS scores in patients with Alzheimer's disease suggests a potential role for optic nerve head analysis in monitoring the progression of Alzheimer's disease and in assessing the effectiveness of any treatments developed. *(Arch Ophthalmol. 1991;109:199-204) Reprint requests to the Glaucoma Service, The New York Eye and Ear Infirmary, New York, NY 10003 (Dr Ritch).*

LOCULATED FLUID. A PREVIOUSLY UNDESCRIBED FLUORESCEIN ANGIOGRAPHIC FINDING IN CHOROIDAL NEOVASCULARIZATION ASSOCIATED WITH MACULAR DEGENERATION. NM Bressler, SB Bressler, J Alexander, N Javornik, SL Fine, RP Murphy, the Macular Photocoagulation Study Reading Center. The researchers participating in the Foveal Photocoagulation Study, a component of the Macular Photocoagulation Study, evaluated whether laser treatment can reduce the risk of severe visual loss in eyes with well-defined choroidal neovascular membranes associated with macular degeneration that extend through the foveal center. On one third of the 554 baseline angiograms of study patients enrolled in and whose eyes were graded in the study as of January 31, 1990, the Reading Center staff has noted an unusual pattern of hyperfluorescence in the late-transit frames that has not been described previously. This pattern, which we call "loculated fluid," consists of a well-demarcated area of hyperfluorescence that appears to represent pooling of fluorescein in a compartmentalized space anterior to the choroidal neovascular leakage. Although the loculated fluid may conform to a pattern of typical cystoid macular edema, it can also pool within an area deep to the sensory retina in a shape that does not bear any resemblance to cystoid macular edema. This pattern is important to recognize because it (1) should no be confused with the angiographic pattern or extent of choroidal neovascularization and (2) should be differentiated from a serious detachment or tear of the retinal pigment epithelium. *(Arch Ophthalmol. 1991;109:211-215) Reprint requests to 550 N Broadway, Suite 900, Baltimore, MD 21205 (Dr N. M. Bressler).*

LARGE RECTUS MUSCLE RECESSIONS FOR THE TREATMENT OF CONGENITAL NYSTAGMUS, GK Noorden, DT Sprunger. The authors discovered that retroequatorial recessions of the horizontal rectus muscles 10 to 12 mm behind their insertions reduced the amplitude of manifest congenital nystagmus in three patients. Modest improvement of visual acuity occurred in two patients. In a third patient with periodic alternating nystagmus, a compensatory head turn was eliminated by shifting the neutral zone of the nystagmus to the primary position of gaze. In spite of large recessions of the muscle insertions, none of the patients had a functionally significant postoperative

limitation of ocular motility. *(Arch Ophthalmol. 1991;109:221-224) Reprint requests to Ophthalmology Service, Texas Children's Hospital, Box 20269, Houston, TX 77225 (Dr von Noorden).*

LOW-DOSE ASPIRIN AND RISKS OF CATARACT IN A RANDOMIZED TRIAL OF US PHYSICIANS, JM Seddon, WG Christen, JE Manson, JE Buring, RD Sperduto, CH Hennekens. The authors state that observational studies have raised the question of a possible benefit of aspirin on the development of cataract. The Physicians' Health Study, a randomized double-masked placebo-controlled trail among 22,071 male physicians, aged 40 to 84 years, provided the opportunity to collect information about whether low-dose aspirin therapy (325 mg on alternate days) affects the development or extraction of cataract. There were 173 age-related cataracts among those physicians assigned to aspirin therapy and 180 among those given placebo (relative risk, 0.95; 95% confidence interval, 0.74 to 1.22). Cataract extractions were less frequent in the aspirin than in the placebo group, but this difference was not statistically significant (relative risk, 0.80; 95% confidence interval, 0.56 to 1.15). Among younger men (aged 40 to 59 years), the relative risks were 0.62 (95% confidence interval, 0.40 to 0.94) for cataract development and 0.67 (95% confidence interval, 0.38 to 1.31) for cataract extraction. These randomized trial data tend to exclude any large benefit of aspirin. While the overall findings concerning cataract development seem to be null, the data on extraction of age-related cataract, while not statistically significant, cannot exclude a possible small to moderate benefit of alternate-day aspirin therapy on the extraction of age-related cataract. *(Arch Ophthalmol. 1991;109:252-255) Reprint requests to 55 Pond Ave, Brookline, MA 02146 (Dr Hennekens).*

A PROSPECTIVE STUDY OF ACUTE CENTRAL RETINAL ARTERY OBSTRUCTION. JS Duker, A Sivalingam, GC Brown, R Reber. The authors conducted a prospective study to determine the incidence of ocular neovascularization following acute central retinal artery obstruction. Only patients initially evaluated within 7 days of visual loss were eligible. Any patient with preexisting ocular neobascularization or clinical evidence of the ocular ischemic syndrome noted at the initial evaluation was excluded. During the 18-month study, 33 consecutive patients were enrolled. Six patients subsequently develo0ed neovascularization of the iris, an incidence of 18.2%. In these six patients, neovascularization of the iris appeared as early as 12 days to as late as 15 weeks following the artery obstructions. Five of the six patients (15.2% of the total) later developed neovascular glaucoma. Another patietn in this series developed neovascularization of the optic disc without neovascularization of the iris, an incidence of 3.0%. Only two of the seven patients with ocular neovascularization had ipsilateral hemodynamically significant carotid artery disease as determined by noninvasive carotid artery testing. This study confirms results of previous retrospective studies that the incidence of ocualr neovascularization after central retinal artery obstruction is higher than commonly thought. It also shows that, in the majority of cases, carotid artery disease is not responsible for eth neovascularization seen after central retinal artery obstruction. *(Arch Ophthalmol. 1991;109:339-342) Reprint requests to Eye Researc Institute Library, 20 Staniford St, Boston, MA 02114 (Dr Duker).*

ORBITAL DECOMPRESSION IN GRAVES' DISEASE. THE PREDICTABILITY OF REDUCTION OF PROPTOSIS, WB Wilson, WF Manke. The authors studied 42 orbits of 23 patients with Graves' disease who had proptosis secondary to the orbitopathy of their disease and had undergone decompressive operations. The patients were evaluated preoperatively and underwent computed tomography. They were also examined frequently during the first postoperative year and the computed tomography was repeated at 6 months. Three variables correlated well with the reduction in proptosis: the percentage of increase in orbital volume after surgery, the absolute increase in orbital volume, and the degree of orbital "stiffness" as measured preoperatively. The first two variables have an inverse correlation with the third. We refer to the loss of resilience of orbital tissues and the increase in adherence between tissue planes, collectively, as orbital "stiffness." While other factors, such as the size of the anterior opening of the orbit, the resistance of the lid diaphragm, and pressure changes within the orbit, may affect the resultant reduction in proptosis, we did not attempt to measure these factors. (Arch Ophthalmol. 1991;109:343-345) *Reprint requests to 850 E Harvard (535), Denver, CO 80210 (Dr Wilson).*

CLINICAL USE OF THE 193-NM EXCIMER LASER IN THE TREATMENT OF CORNEAL SCARS, NA Sher, RA Bowers, RW Zabel, JM Frantz, RA Eiferman, DC Brown, JJ Rowsey, P Parker, V Chen RL Lindstrom. The authors performed a phototherapeutic keratectomy using a 193-nm excimer laser at four centers on 33 sighted patients with corneal opacity and/or irregular astigmatism. Pathologic conditions included anterior stromal and superficial scarring from postinfectious and posttraumatic causes, including inactive herpes simplex virus, anterior corneal dystrophies, recurrent erosions, granular dystrophy, and band keratopathy. Most patients received peribulbar anesthesia and underwent removal of the epithelium prior to laser ablation. A majority of patients had a reduction in the amount to corneal scarring and approximately half had improved visual acuity. No intraocular reaction or changes in endothelial counts were seen, and some patients

avoided the need for penetrating keratoplasty. Reepithelialization usually occurred within 4 or 5 days and we noted no significant scarring secondary to use of the laser. It was difficult to eliminate preexisting irregular astigmatism despite the use of surface modulators, such as methylcellulose. A hyperopic shift secondary to corneal flattening was encountered in approximately 50% of the patients. A combination of myopic ablation, followed immediately by a secondary hyperopic steepening, may minimize this refractive change. The 193-nm excimer laser is an effective new tool in the treatment of selected patients with superficial corneal opacity from a variety of conditions. *(Arch Ophthalmol. 1991;109:491-498) Reprint requests to the Phillips Eye Institute, 2215 Park Ave, Minneapolis, MN 55404 (Dr Sher).*

FULMINANT PSEUDOMONAL KERATITIS AND SCLERITIS IN HUMAN IMMUNODEFICIENCY VIRUS-INFECTED PATIENTS, M Nanda, SC Pflugfelder, S Holland. The authors conclude that patients with human immunodeficiency virus infection are predisposed to fungal, parasitic, and viral infections. Bacterial infection can also be seen, although ocular bacterial infections have not been reported in patients with acquired immunodeficiency syndrome until recently. We present two cases of *Psuedomonas* corneoscleritis and one case of *Pseudomonas* keratitis in patients with human immunodeficiency virus infection that failed to respond to antibiotic treatment. Predisposing factors included extended-wear soft contact lens use in one patient and exposure secondary to Bell's palsy in another patient. All three patients had neutropenia that may have contributed to their poor response to treatment. Enucleation was required to treat two patients with overwhelming infection. Enucleation has been rarely required for treatment of corneoscleritis in immunocompetent patients treated at our institution. *Pseudomonas* keratitis in human immunodeficiency virus-infected patients represents a serious ocular infection requiring early diagnosis and aggressive treatment. *(Arch Ophthalmol. 1991;109:503-505) Reprint requests to Bascom Palmer Eye Institute, Department of Ophthalmology, PO Box 016880, Miami, FL 33101 (Dr Pflugfelder).*

LATE-ONSET ELEVATION IN INTRAOCULAR PRESSURE AFTER NEODYMIUM-YAG LASER POSTERIOR CAPSULOTOMY, S Fourman, J Apisson. The authors retrospectively studied the long-term change in intraocular pressure after neodymium-YAG laser posterior capsulotomy. Uncomplicated extracapsular cataract extraction with posterior chamber intraocular lens implantation was performed in 446 consecutive normal eyes (401 patients), 237 (53%) of which subsequently underwent uncomplicated neodymium-YAG laser posterior capsulotomy. Late-onset elevation in intraocular pressure was seen in three eyes (1.4%) after cataract surgery alone (mean follow-up, 2.7±1.7 years) and in 14 eyes (5.9%) after cataract and laser surgeries (mean follow-up, 3.0±1.1 years). This difference was statistically insignificant (P<.03). These results suggest that, in addition to immediate changes in intraocular pressure, neodymium-YAG laser posterior capsulotomy may be associated with an increase in intraocular pressure long after the laser surgery, even in normal eyes without obvious postoperative complications. *(Arch Ophthalmol. 1991;109:511-513) Reprint requests to SUNY at Stony Brook, HSC L-2, Room 152, Stony Brook, NY 11794-8123 (Dr Fourman).*

DOPPLER ULTRASONOGRAPHY OF THE OPHTHALMIC AND CENTRAL RETINAL VESSELS, RF Guthoff, RW Berger, P Winkler, K Helmke, LC Chumbley. The authors did B-Scan-guided, pulsed Doppler ultrasonography of central retinal vessels and ophthalmic arteries on 72 normal subjects. They performed the following average peak flow velocities: central retinal artery, 9.5±3.1 cm/s; central retinal vein, 5.7±1.5 cm/s; and ophthalmic artery, 31.6±9.0 cm/s. Doppler shifts in the central retinal vessels were absent at intraocular pressures above 80 mm Hg. Ophthalmic artery systolic, middiastolic, and end-diastolic velocities significantly declined as a function of age. The technique is independent of the status of the ocular media, is noninvasive, requires little time, and causes minimal discomfort. *(Arch Ophthalmol. 1991;109:532-536) Reprint requests to Hamburg University Eye Hospital, Eppendorf, Martini Str 52, 2000 Hamburg 20, Federal Republic of Germany (Dr Guthoff).*

VITREOUS SURGERY FOR IDIOPATHIC MACULAR HOLES, KN Kelly, RT Wendel. The authors performed modern vitrectomy techniques to evaluate two questions regarding idiopathic macular holes: (1) Is it possible to reattach the retina around the macular hole? (2) If it is reattached, will the patient's central vision improve? In 30 (58%) of 52 patients, they were able to reattach successfully the detached macula with their surgical procedure. In 22 (73%) of the 30 patients in whom the macula was successfully reattached, there was an improvement in visual acuity of two lines or better. In the 22 patients in whom reattachment of the macular hole was not obtained, there was no significant improvement in visual acuity. Thus, the overall success rate for improved vision postoperatively was 42% (22/52). Complications related to surgery were observed in eight patients (15%) early in our experience with this procedure and included increase in the size of the macular hole, mottling of the retinal pigmented epithelium, and a vascular occlusion. Our clinical observations indicate that the treatment of macular holes by vitrectomy may offer some promise for this otherwise untreatable condition. In patients in

whom reattachment was successful, the technique used appeared to allow for clinically significant improvements in visual acuity. However, additional work on increasing surgical success and minimizing surgical complications, as well as a further understanding of the mechanism of retinal reattachment, is required before widespread use of this procedure for treating macular holes. *(Arch Ophthalmol. 1991;109:654-659) Reprint requests to 3939 J St, 106, Sacramento, CA 95819 (Dr Wendel).*

NONARTERITIC ANTERIOR ISCHEMIC OPTIC NEUROPATHY AND INTRAOCULAR PRESSURE, JW Kalenak, GS Kosmorsky, EJ Rockwood. The authors note that several reports have suggested that nonarteritic anterior ischemic optic neuropathy (AION) may be related to increased intraocular pressure. We reviewed the records of 45 patients aged 48 through 86 years with nonarteritic AION (10 patients had bilateral AION) for intraocular pressure measurements and the diagnosis of glaucoma or suspected glaucoma. This group was compared with 45 age-and sex-matched patients with normal eye examination results. The mean±SD intraocular pressures were 16.3±3.3 mm Hg for 45 eyes of the 45 patients with AION and 16.1±2.8 mm Hg for 45 eyes of the 45 control patients (paired *t* test, P=.70). Among patients with unilateral AION, intraocular pressure was not greater in the involved eye than in the uninvolved eye. Three patients with AION had a previous diagnosis of glaucoma, while three of the control patients were being followed up with suspected glaucoma. We found no evidence in our series to support the hypothesis that AION is associated with increased intraocular pressure. *(Arch Ophthalmol. 1991;109:660-661) Reprint requests to Department of Ophthalmology, The Cleveland Clinic Foundation, One Clinic Center, Cleveland, OH 44195-5024 (Dr Kalenak).*

OPTIC NERVE SHEATH DECOMPRESSION FOR NONARTERITIC ISCHEMIC OPTIC NEUROPATHY IMPROVES MULTIPLE VISUAL FUNCTION MEASUREMENTS, SE Kelman, MJ Elman. The authors performed the optic nerve sheath decompression in seven patients with nonarteritic anterior ischemic optic neuropathy. Visual function was evaluated by measurement of visual acuity with standardized Early Treatment Diabetic Retinopathy Study charts, color vision testing, quantization of relative afferent pupillary defects with neutral-dessity filters, and Goldmann and Humphrey perimetry. Visual acuity improved markedly in all patients (at least doubling of the visual angle); the peripheral visual field expanded by at least 20^0 (as measured by Goldmann perimetry) in six patients. Three patients also experienced marked improvement in color vision, relative afferent pupillary defect, and foveal sensitivity. Our experience supports the possible beneficial effect of optic nerve sheath decompression in patients with nonarteritic anterior ischemic optic neuropathy. *(Arch Ophthalmol. 1991;109:667-671) Reprint requests to the Neuro-Ophthalmology Service, Department of Ophthalmology, University of Maryland Hospital, 22 S Greene St, Baltimore, Maryland 21201 USA (Dr Kelman).*

PERFORATING (THROUGH-AND-THROUGH) INJURIES OF THE GLOBE. SURGICAL RESULTS WITH VITRECTOMY, DF Martin, TA Meredith, TM Topping, P Sternberg, HJ Kaplan. The authors treated 51 eyes of 48 patients with perforating (through-and-through) injuries of the glove with vitrectomy during a 12-year period. Functional success was obtained in 32 eyes (63%), anatomic success was obtained in nine eyes (17%), and treatment failed in 10 eyes (20%). In 16 eyes (32%), 20/20 to 20/100 visual acuity was obtained; in 17 eyes (33%), 20/200 to 5/200 visual acuity was obtained; and in 18 eyes (35%), less than 5/200 visual acuity was obtained. The mechanism of injury was an important prognostic indicator of final visual outcome. Eight (62%) of 13 eyes that sustained knife or nail injuries achieved a final visual acuity of 20/50 or better, while only six (16%) with missile injuries achieved a similar level of acuity. Final visual outcome correlated well with the state of the macula and was not predicted by preoperative visual acuity. Despite improvement in surgical techniques and instrumentation, no trend toward improved visual outcomes was identified during the 12-year period. *(Arch Ophthalmol. 1991;109:951-956) Reprint requests to The Wilmer Institute, The Johns Hopkins Hospital, 600 N Wolfe St, Baltimore, MD 21205 (Dr Meredith).*

CONGENITAL CYSTIC EYE WITH MULTIPLE OCULAR AND INTRACRANIAL ANOMALIES, LR Pasquale, N Romayananda, J Kubacki, MH Johnson, GH Chan. The authors describe a newborn with congenital cystic eye, contralateral persistent hyperplastic primary vitreous, and cerebrocutaneous abnormalities. The cerebrocutaneous abnormalities consisted of agenesis of the corpus callousum, midbrain deformity, malformed sphenoid bone, right upper eyelid coloboma, and a left periocular hamartoma. The results of karyotype analysis of the patient and his parents were normal. The association of congenital cystic eye with contralateral persistent hyperplastic primary vitreous has not been previously reported, to our knowledge. Although no unifying diagnosis exists for the collection of anomalies demonstrated in this patient, the term cranial ectodermopathy broadly classifies most of the defects. *(Arch Ophthalmol. 1991;109:985-987) Reprint requests to the Wilmer Eye Institute, 600 N Wolfe St, Maumenee B-110, Baltimore, MD 21205 (Dr Pasquale).*

NATIONAL OUTCOMES OF CATARACT EXTRACTION. ENDOPHTHALMITIS FOLLOWING INPATIENT SURGERY, JC Javitt, S Vitale, JK Canner, DA Street, H Krakauer, AM McBean, A Sommer. The authors analyzed the likelihood of re-hospitalization for endophthalmitis in 338,141 Medicare beneficiaries over age 65 years who were admitted to US hospitals for cataract extraction in 1984. This cohort represents approximately one half of all persons who underwent cataract extraction under the medicare program in 1984. Extracapsular extraction was performed in 195,587 (58%) of cases, intracapsular cataract extraction in 99,971 (30%), and phacoemulsification in 28,474 (8%). The risk of rehospitalization for endophthalmitis in the year following surgery was 0.17% for intracapsular cataract extraction compared with 0.12% for extracapsular extraction or phacoemulsification (P<.002). The risk of endophthalmitis at 1 month was higher for extracapsular extraction or phacoemulsification (0.11% vs 0.085%), although the difference did not reach statistical significance. Cataract surgery accompanied by anterior vitrectomy increased the 1-month risk of rehospitalization for endophthalmitis to 0.41%, more than a four-fold increase over that for cataract surgery alone (0.09%; P<.05). The rates of endophthalmitis at 1 year were 0.58% and 0.13%, respectively, for cataract surgery with anterior vitrectomy and cataract surgery alone (P<.0001). No significant differences in the rate of rehospitalization for endophthalmitis were observed based on the use of an intraocular lens, age, or race. Endophthalmitis within 1 year of surgery was 1.2 times more frequent in men than in women (0.16% vs 0.13%; P=.03). Overall, the likelihood of postoperative endophthalmitis from a national sample is consistent with case series previously reported. *(Arch Ophthalmol. 1991;109:1085-1089) Reprint requests to Center for Sight, 3800 Reservoir Rd, Washington, DC 20007 (Dr Javitt).*

EXERCISE TRAINING REDUCES INTRAOCULAR PRESSURE AMONG SUBJECTS SUSPECTED OF HAVING GLAUCOMA, MS Passo, L Goldberg, DL Elliot, EM Van Buskirk. The authors noted that the effects of exercise conditioning on elevated intraocular pressure has not been previously described among sedentary individuals. They prospectively observed intraocular pressure for nine sedentary subjects suspected of having glaucoma before and after 3 months of aerobic exercise training. Mean (±SEM) aerobic capacity, as assessed by maximal oxygen uptake, increased 6.3 ± 1.6 mL kg^{-1} min^{-1} (30%) (P<.02). Mean intraocular pressure decreased 4.6+0.4 mm Hg (20%) (P<.001) at the end of the conditioning period. With cessation of exercise and subsequent detraining, intraocular pressure returned to elevated preconditioning levels by 3 weeks. Regular aerobic exercise in associated with a reduction in elevated intraocular pressure and may represent an effective nonpharmacologic intervention for patients suspected of having glaucoma. *(Arch Ophthalmol. 1991;109:1096-1098) Reprint requests to 112-P, Department of Veterans Affairs Medical Center, 3710 SW US Veterans Hospital Rd, Portland, OR 97207 (Dr Passo).*

SUBFOVEAL NEOVASCULAR LESIONS IN AGE-RELATED MACULAR DEGENER-ATION. GUIDELINES FOR EVALUATION AND TREATMENT IN THE MACULAR PHOTOCOAGULATION STUDY, Macular Photocoagulation Study Group. The authors noted that the Macular Photocoagulation Study (MPS) gives guidelines for interpreting angiograms of eyes with subfoveal choroidal neovascularization (CNV) secondary to age-related macular degeneration and for treating these lesions to assist ophthalmologists in applying the results of the MPS clinical trails of laser treatment. The MPS criteria for treatment of subfoveal neovascular lesions require the following conditions: (1) the presence of classic CNV, (2) well demarcated lesion boundaries, and (3) size less than or equal to 3.5 disc areas (if no previous treatment of CNV in the macula was performed). In subfoveal recurrent CNV, size had to be such that after treatment of the recurrence, the final treatment scar (prior treatment scar and newly treated area) would be no larger than 6 disc areas and would spare some retina within 1500 μm of the center of the foveal vascular zone. Treatment of all classic and occult CNV and areas in which the boundaries of CNV may be obscured is recommended, as is treatment extending at least 100 μm beyond the peripheral boundaries of the lesion. In subfoveal recurrent CNV, treatment should also extend at least 300 μm into the previous treatment scar and cover any feeder vessels. The desired end point for the intensity of the laser burns is a uniformly white lesion. *(Arch Ophthalmol. 1991;109:1242-1257) Reprint requests to Macular Photocoagulation Study Reading Center, 550 N Broadway, Ninth Floor, Baltimore, MD 21205 (Dr N. Bressler).*

FUNGAL ENDOPHTHALMITIS. AN EXPERIMENTAL STUDY WITH A REVIEW OF 17 HUMAN OCULAR CASES, TW McGuire, JD Bullock, JD Bullock, BL Elder, JW Funkhouser. The authors report that *Psuedallescheria boydii* is, an opportunistic fungus that is histologically indistinguishable from *Aspergillus fumigatus*, may cause endophthalmitis, orbital cellulitis, and corneal ulceration and is, thus, important to the ophthalmologist. A clinical review of 17 patients with *P boydii* ophthalmic infections is presented. In addition, animal models of endophthalmitides caused by *A fumigatus* and *P boydii* were created and compared. Dutch-Belted rabbits used

for the experimental models were immunosuppressed with methylprednisolone acetate (Depomedrol). Exogenous and endogenous models of *P boydii* endophthalmitis were created in immunocompetent and immunosuppressed animals. An exogenous model of *A fumigatus* endophthalmitis was created in immunocompetent and immunosuppressed animals for comparison. The clinical and histopathologic features of *A fumigatus* and *P boydii* endophthalmitis are indistinguishable in the immunocompentent and immunosuppressed exogenously infected rabbits. Endogenous *P boydii* endophthalmitis has a similar fundus appearance to that caused by Nocardia asteroides. *(Arch Ophthalmol. 1991;109:1289-1296) Reprint requests to Plumwood Bldg, Suite 250, 5 Plumwood Rd, Dayton, OH 45409 (Dr Bullock).*

MOLTENO IMPLANTS AS A TREATMENT FOR REFRACTORY GLAUCOMA IN BLACK PATIENTS, J Freedman, B Rubin. The authors treated 82 black patients with refractory glaucoma with a single-plate Molteno implant inserted in a single-stage procedure. A successful outcome (intraocular pressure ≤21 mm Hg with or without adjunctive medical therapy) was achieved in 72% of the patients with a mean follow-up of 30 months. Success was achieved in 23 (73%) of the 31 patients with open angle glaucoma, 20 (83%) of the 24 patients with either aphakic or pseudophakic glaucoma, 12 (67%) of the 18 patients with neovascular glaucoma, four (80%) of the five patients with uveitic glaucoma, and two (50%) of the four patients with congenital glaucoma. All but four patients required additional medical therapy. Visual acuities remained the same or improved in 21 (68%) of the 31 patients with open angle glaucoma, 11 (61%) of the 18 with neovascular glaucoma, 19 (79%) of the 24 patients with aphakic/pseudophakic glaucoma, three (75%) of the four patients with congenital glaucoma, and four (80%) of the five patients with uveitic glaucoma. Complications included hyphema (18%), "kissing" choroidal effusion (6%), blocked tube (8%), flat anterior chamber (12%), cataracts (5%), Tenon's cyst (encapsulated bleb) (17%), uveitis (7%), phthisis bulbi (5%), and erosion of the silicone tube (1%). *(Arch Ophthalmol. 1991;109:1417-1420) Reprint requests to Long Island College Hospital, Department of Ophthalmology, 340 Henry St, Brooklyn, NY 11201 (Dr Freedman).*

MANAGEMENT OF OCULAR PENETRATION FROM INJECTION OF LOCAL ANESTHESIA PRECEDING CATARACT SURGERY, JS Rinkoff, BH Doft, LA Lobes. The authors herein describe 12 patients who suffered penetration or perforation of the globe during injection of a local anesthetic before cataract surgery. Minimum follow-up was 6 months. Six eyes had a final visual acuity of 20/50 or better and an attached retina. Four eyes had an attached retina with a visual acuity of 20/80 to 2/200. Two eyes were anatomic failures because of a recurrent retinal detachment complicated by proliferative vitreoretinopathy. These cases show that retinal penetrations without retinal detachment may be treated effectively with photocoagulation. Vitreous surgery is recommended when the retinal penetration is associated with a retinal detachment. Eyes with a dense vitreous hemorrhage and a suspicion of a penetrating injury should either be followed up closely with echography or should undergo vitreous surgery since the extent of the injury cannot be determined. *(Arch Ophthalmol. 1991;109:1421-1425) Reprint requests to Retina Vitreous Consultants, Suite 500, 3501 Forbes Ave, Pittsburgh, PA 15213 (Dr Rinkoff).*

CORNEAL WOUND HEALING AFTER 193-NM EXCIMER LASER KERATECTOMY, WCS Wu, WJ Stark, WR Green. The authors performed, as part of a human trial of phototherapeutic keratectomy, anterior keratectomy using excimer laser 193-nm ablation on patients with superficial corneal opacities. We examined the ultrastructural changes in the corneas of four patients who underwent penetrating keratoplasty 6 to 15 months after excimer laser ablation. The four cases included macular dystrophy, recurrent keratoconus in a graft, and corneal scarring (two eyes). Light microscopy showed epithelial thickening, absence of Bowman's layer, and superficial stromal scarring in the area of ablation. Ultrastructural study showed that the epithelial basement membrane had focal discontinuities. At the margin of the ablation superficial collagen bundles terminated abruptly in a steplike configuration. The anterior stroma was scarred, with loss of lamellar structure and an increase in number of fibrocytes. The laser-induced scar was 10 to 15 μm thick in the two eyes without a stromal scar before laser ablation. The stroma underlying the scarred areas and that in the untreated area appeared normal. Linear collagenlike fibers were present in the posterior aspect of Descemet's membrane. Laser-induced scarring may be an important factor in limiting visual improvement in patients undergoing phototherapeutic keratectomy. *(Arch Ophthalmol. 1991;109:1426-1432) Reprint requests to Eye Pathology Laboratory, The Johns Hopkins Hospital, 600 N Wolfe St, Baltimore, MD 21205 (Dr Green).*

THE USE OF THE 193-NM EXCIMER LASER FOR MYOPIC PHOTOREFRACTIVE KERATECTOMY IN SIGHTED EYES. A MULTICENTER STUDY, NA Sher, V Chen, RA Bowers, JM Frantz, DC Brown, R Eiferman, SS Lane, P Parker, C Ostrov, D Doughman, E Carpel, R Zabel, T Gothard, RL Lindstrom. The authors report on photorefractive keratectomy performed at three centers using the 193-nm excimer laser on 31 sighted myopic eyes. Preoperative refractive errors (spherical

equivalent) ranged from -12.00 to -4.00 diopters (D) (mean, -6.49 ±1.75D). Peribulbar anesthesia, a 5.2- to 0.6-mm beam diameter, and topical corticosteroids were used for up to 6 months after surgery. The epithelium healed within 3 to 4 days, and all patients returned to their best corrected visual acuity within 1 line of their preoperative acuity. There was minimal subepithelial reticular haze, peaking at 3 weeks and diminishing over the next 3 to 4 months, which was not felt to be visually significant. At 6 months, the average residual refractive error was -1.85+2.5 D. Sixty-eight percent of eyes were corrected within 2 D and 55% within 1 D of attempted correction. There was no significant change in astigmatism, contrast sensitivity, corneal sensation, or endothelial cell counts. This preliminary work shows that photorefractive keratectomy has promise in the reduction of moderate myopia. *(Arch Ophthalmol. 1991;109:1525-1530) Reprint requests to Phillips Eye Institute, 2215 park Ave, Minneapolis, MN 55404 (Dr Sher).*

SURFACE ULTRASTRUCTURE AFTER EXCIMER LASER ABLATION. EXPANDING VS CONTRACTING APERTURES, A Sinbawy, PJ McDonnell, H Moreira. The authors report on excimer laser corneal ablation (photorefractive keratectomy) for myopia using a diaphragm delivery system on eye bank and animal eyes by either progressively expanding or contracting the iris aperture. Use of an expanding aperture, in which the final ablations cover the entire treatment zone, produced a pseudomembrane over the zone that had few discontinuities. Ultrastructural examination of the corneas ablated using a progressively contracting aperture, however, revealed numerous discontinuities in the surface pseudomembrane. Use of an expanding iris aperture to perform ablations to correct myopia may be preferable as it produces a psuedomembrane with few discontinuities. Clinical studies of surface healing in humans are necessary to determine the clinical relevance of improved continuity of the surface pseudomembrane. *(Arch Ophthalmol. 1991;109:1531-1533) Reprint requests to Doheny Eye Institute, 1355 San Pable St, Los Angeles, CA 90033 (Dr McDonnell).*

PROGRESSIVE ENLARGEMENT OF LASER SCARS FOLLOWING GRID LASER PHOTOCOAGULATION FOR DIFFUSE DIABETIC MACULAR EDEMA, H Schatz, D Madeira, R McDonald, RN Johnson. The authors state that laser photocoagulation in a grid pattern is effective in many eyes in resolving diffuse diabetic macular edema and stabilizing vision. They retrospectively studied 203 eyes of 203 patients, all of whom had diabetic macular edema treated with grid laser photocoagulation. During the follow-up period, 11 of these 203 eyes developed enlargement of the laser scars that progressed into the central fovea. Each of the 11 eyes experienced loss of vision, and in five, the visual loss was significant. Five of these eyes also developed a retinal pigment hyperplastic scar. After the laser treatment, but before the scars enlarged, the visual acuity of these eyes was unchanged from that before treatment. But after the atrophic scars enlarged into the central fovea, visual acuity in one eye decreased from 20/40 to 20/100, and the other 10 eyes had visual acuities of 20/200 or worse. *(Arch Ophthalmol. 1991;109:1549-1551) Reprint requests to 1 Daniel Burnham Ct, Suite 210, San Francisco, CA 94109 (Dr Schatz).*

INFECTIOUS ENDOPHTHALMITIS FOLLOWING CATARACT SURGERY, KG Stonecipher, VC Parmley, H Jensen, JJ Rowsey. The authors report that within the past 6 months, three cases of infectious endophthalmitis following sutureless cataract surgery have been referred to us. Two of these cases followed uncomplicated sutureless phacoemulsification with intraocular lens implantation. One case was complicated by a postoperative hyphema with additional surgery for clot removal 2 days following the initial procedure. *(Arch Ophthalmol. 1991;109:11562-1563) Reprint requests to the Dean McGee Eye Institute, 608 Stanton L. Young Blvd, Oklahoma City, OK 73104 (Dr Jensen).*

INFLUENCES OF CORTICOSTEROID ON EXPERIMENTALLY INDUCED KERATOMYCOSIS, DM O'Day, WA Ray, WS Head, RD Robinson, TE Williams. The authors assessed the effect of corticosteroid on the establishment of experimentally induced keratomycoses in rabbits injected subconjunctivally with triamcinolone acetonide on two successive days before inoculation with *Candida albicans, Aspergillus fumigatus,* or *Fusarium solanae.* Whereas isolate recovery rates declined steadily in normal control corneas, they remained stable over 15 days in corticosteroid-treated corneas. Clinically, inflammation was equivalent (*A fumigatus* and *F solanae*) or significantly less (*C albicans*; P=.001) until the 10th day. At 15 days, inflammation in corticosteroid-treated corneas was significantly worse in animals infected with *A fumigatus* (P=.003) or *F solanae* (P=.02). Inflammatory signs correlated inconsistently with late recovery. Pathogenicity of the infecting organism appears to be important in determining the degree to which cortiosteroid is able to mask clinical signs of infection while enhancing fungal replication. *(Arch Ophthalmol. 1991;109:1601-1604) Reprint requests to the Department of Ophthalmology, D-5217, Vanderbilt University Medical Center, Nashville, TN 37232-2540 (Dr O'Day).* - *KJA*

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To you have come signs from your Lord;
Whoever therefore sees,
Does so for himself;
And whoever remains blind,
Does so to his own loss.
Holy Quran 6:105

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