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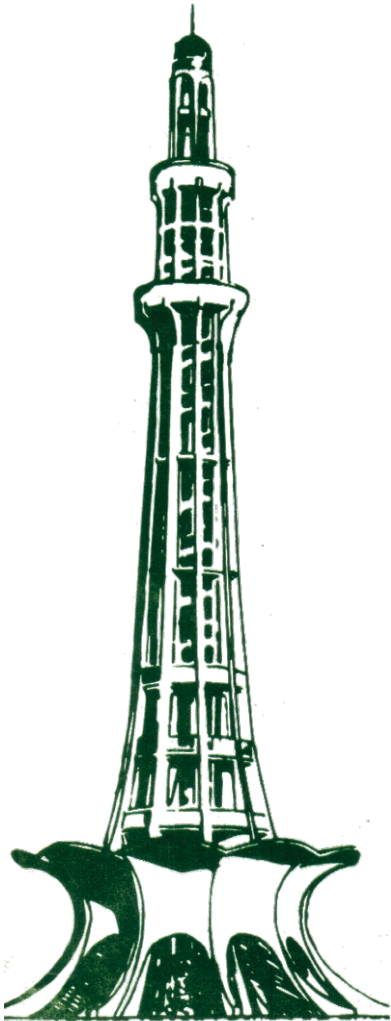
PAKISTAN JOURNAL OF OPHTHALMOLOGY

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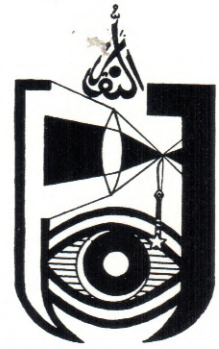
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Tuberculosis of the Conjunctiva, Eyelid, and Periocular Skin

Khadim Ullah Kakakhel, M.B., B.S., D.D.V.
Shad Mohammad, F.R.C.S.

ABSTRACT: Over a period of 4 years, we saw 10 cases of tuberculosis involving conjunctiva, eyelids, and periocular skin. The presentation was usually in the form of conjunctival and lid ulcers and lid edema. Only one case presented as a tumorlike granuloma of the conjunctiva. The investigations included complete dermatologic and ophthalmologic examinations, culture, histologic study and guinea pig inoculation of the biopsy material, tuberculin skin test, chest x-ray, and routine blood workup. Discussion includes comments on diagnosis and treatment. Primary tuberculosis was commonly seen in under 20-year-olds. Unlike the previous reports, more males were affected than females. (Pakistan Journal of Ophthalmology 4:73-77, 1988.)

Tuberculosis (TB) is less common in developed countries and its incidence in developing countries is also declining. Nonetheless, tuberculosis is quite common in Pakistan where the mortality rate has been estimated at 200 per 100,000, and 0.5% of the population has been shown to be sputum positive. However decrease in the incidence of TB in Pakistan has been reported in recent years. Also, it is being reported that the incidence of tuberculosis is actually on the rise in the United States (written communication from H. Bruce Ostler, M.D., Francis I. Proctor Foundation, San Francisco, U.S.A.). Tuberculosis of the conjunctiva, eyelids, and other parts of the eye is very rare, even in areas where tuberculosis is more common. That way little has been published about the ocular and periocular tuberculosis in the past two decades had induced us to present our experience with the tuberculosis of the extraocular structures and adnexal skin.

Materials and Methods

During a study on tuberculosis of the skin at the District Headquarters Teaching Hospital, Abbottabad, Pakistan from 1982-1984, three cases involving the lid and periocular skin were noted. Seven more cases of tuberculosis involving the eye directly or indirectly were observed in the next two years. In each case, a detailed

history was taken. Examination of the skin was done by a dermatologist, and eyes were examined by an ophthalmic surgeon. All patients were given a tuberculin skin test, and their lesions were biopsied for histopathology. The biopsy material was also used for culture and guinea pig inoculation. Histological sections were studied under hematoxylin and eosin and acid fast staining. Chest x-ray and routine blood and urine tests were performed in all patients. Special investigations were done in some cases to rule out the involvement of other organs. Classification into various types of skin and mucosal tuberculosis was done by correlating clinical, histological and immunological characteristics. As a principle, we followed the criteria listed in Table 1 to make the diagnosis.

Table 1
Criteria for diagnosis of tuberculosis

- A. Absolute Criteria
 - 1. Positive culture
 - 2. Positive guinea pig inoculation
- B. Relative Criteria
 - 1. Suggestive history and clinical signs
 - 2. Presence of active proven TB elsewhere in the body
 - 3. Presence of acid fast bacilli in the lesion
 - 4. Characteristic histopathology
 - 5. Positive reaction to tuberculin
 - 6. Successful response to antitubercular therapy

From the Department of Dermatology (Dr. Kakakhel) and Ophthalmology (Dr. Mohammad), Ayub Medical College, Abbottabad, Pakistan. Send reprint requests and other inquiries to Dr. Kakakhel at the above address.



Figure 1 (Kakakhel, Mohammad): Right eye. Primary tuberculous ulcer of skin and of the conjunctiva showing tumorlike mass.



Figure 2 (Kakakhel, Mohammad): Left eye. Orificial tuberculosis of the eyelids and adjacent skin.



Figure 3 (Kakakhel, Mohammad): Left eye. Orificial tuberculosis of the upper and lower eyelids.



Figure 4 (Kakakhel, Mohammad): Left eye. Scarring and ectropion resulting from orificial tuberculosis.



Figure 5 (Kakakhel, Mohammad): Left eye. Lupus vulgaris causing ectropion of the lower eyelid.

Results

Many lesions of various types of tuberculosis involving the eye were seen (Figures 1-5). The results of the investigations are summarized in Table 2. The affected structures and the type of tuberculosis in each case are listed in Table 3. The treatment and outcome of the treatment are summarized in Table 4. In five out of a total of 10 cases, tuberculosis of the eye was of primary type. In the remaining five cases of secondary tuberculosis, three belonged to orificial tuberculosis and two belonged to lupus vulgaris.

Discussion

Tuberculosis is caused by *Mycobacterium tuberculosis* of the human, bovine and very rarely the

Kakakhel and Mohammad • TUBERCULOSIS OF THE EYE

Table 2
Results of Investigations

Case No.	Tuberculin Test*	Culture	Guinea Pig Inoculation	Histopathology
1	Positive	Positive	Positive	Typical tuberculous granuloma. Bacilli not seen.
2	Negative	Positive	Positive	Ulcer with mixed neutrophilic and lymphocytic infiltrate. No granuloma. Bacilli seen.
3	Positive	Positive	Positive	Ulcer with nonspecific infiltrate in upper dermis. Tuberculoid granulomas in deep dermis. Bacilli seen.
4	Positive	Positive	Positive	Ulcer with nonspecific infiltrate in upper dermis. Tuberculous granulomas in deep dermis. Bacilli seen.
5	Positive	Positive	Negative	Ulcer with mixed infiltrate. Some tuberculoid granulomas seen. Bacilli demonstrated.
6	Positive	Positive	Positive	Epidermal hypertrophy, tuberculoid granulomas, no caseation. Bacilli not seen.
7	Positive	Negative	Positive	Epidermal hypertrophy, hyperkeratosis, tuberculous granulomas with slight caseation necrosis.
8	Negative	Positive	Positive	Ulcer, mixed neutrophilic and lymphocytic infiltrate, no granulomas. Bacilli seen.
9	Positive	Positive	Positive	Ulcer, mixed neutrophilic and lymphocytic infiltrate, tuberculous granulomas with caseation. Bacilli seen.
10	Positive	Positive	Positive	Ulcer, mixed infiltrate, tuberculoid granulomas in depth with caseation necrosis. Bacilli seen.

* A highly antigenic tuberculin, 0.1 ml containing 5 T.U. of standard P.P.D. was injected intradermally (Mantoux). The appearance of a wheal of 5 mm or more disregarding the surrounding erythema in 72 hours was considered positive.

Table 3
Structures Involved and Type of Tuberculosis

Case No.	Age/Sex	Site of Lesion	Type of TB	Coexisting TB Lesion
1	13 yrs/male	Palpebral conjunctiva, upper eyelid and temporal skin	Primary (granuloma)	Preauricular lymphadenitis
2	10 yrs/male	Tarsal conjunctiva	Primary ulcer	Nil
3	18 yrs/female	Lower eyelid + skin	Orificial TB	Abdominal TB
4	65 yrs/female	Upper eyelid + cheek	Orificial TB	Pulmonary TB
5	13 yrs/male	Lower eyelid + facial skin	Orificial TB	Pulmonary TB
6	48 yrs/male	Nose, face, lower eyelid	Lupus vulgaris	Nil
7	36 yrs/female	Nose, face, lower eyelid	Lupus vulgaris	Healed pulmonary TB
8	6 yrs/male	Lower eyelid, tarsal conjunctiva	Primary ulcer	Nil
9	8 yrs/male	Bulbar conjunctiva + preauricular lymphadenitis	Primary ulcer	Nil
10	16 yrs/female	Upper eyelid	Primary ulcer	Tuberculous lymphadenitis + scrofuloderma neck + preauricular lymphadenitis

Table 4
Treatment and its Outcome

Case No.	Treatment	Results after 6 Months Treatment
1	Excisional biopsy of conjunctival lesion + systemic anti-TB therapy	Complete healing with slight lid scarring
2	Systemic anti-TB therapy	Complete healing
3	Suturing of skin + syst. anti-TB ther.	Complete healing
4	Systemic anti-TB therapy	Patient lost to follow-up
5	Systemic anti-TB therapy	Scarring of face + ectropion
6	Systemic anti-TB therapy	Mild scarring of nose + ectropion
7	Systemic anti-TB therapy	Mild scarring of nose and face + ectropion
8	Systemic anti-TB therapy.	Complete healing
9	Systemic anti-TB therapy	Complete healing
10	Systemic anti-TB therapy	Complete healing

avian type.¹⁻² The eye of the child may be more susceptible to the bovine strain as suggested by Locatcher-Khorazo and Seegal,³ although more data are needed to fully establish this. A typical mycobacteria may cause identical lesions, separable only by culture characteristics. The route of infection for primary lesions is by direct inoculation from infected individuals, dust, fomites, and fingers of the patient transmitting the infected material. Other important routes, especially in cases of secondary tuberculosis are direct extension from skin, mouth, and nasal lesions and endogenous or metastatic spread through blood stream.^{3,5,6} Conjunctival tuberculosis occurs in the form of ulcerative, nodular, hypertrophic papillary and polypoid lesions.^{3,4,7,8,9} The patients present with conjunctival irritation and discharge. The swelling, ulcer, and tumor of the lid may be noted. The regional lymphadenitis is a common occurrence with primary tuberculosis of the eye and periocular skin.⁷⁻⁹

Primary tuberculosis is common in patients under 20 years of age, but occurrence above 20 years of age has been reported.^{10,11} The incidence in females is reported to be twice as common as in males.^{4,6} Our patients with primary tuberculosis were younger than 20 years of age, but more males were affected than females. Symptomatic pulmonary tuberculosis was present in Cases 4 and 5. Case 3 low grade fever, chronic diarrhoea and loss of weight. Abdominal tuberculosis was confirmed in her by investigations. Evidence of healed pulmonary TB was seen in Case 7. She had received anti-TB treatment in the past. Supporating lymphadenitis in the neck with fever was present in case 10.

Orificial tuberculosis is the tuberculous infection of the mucosa or the skin adjoining orifices in a patient with advanced internal tuberculosis. It is usually an auto-inoculation, resulting from direct inoculation or lymphatic or haematogenous spread. The shallow, painful, and poorly healing ulcers are characteristic, and syphilitic, carcinomatous and aphthous ulcers should be excluded.

Lupus vulgaris may rarely involve lids and conjunctiva. It usually is secondary tuberculosis, but may be primary at times. We observed that the ectropion of the eyelid was a common complication of lupus vulgaris.

The criteria for diagnosis of tuberculosis are summarized in Table 1. Eversion of the lid is a must, even if anesthesia is required to do it, when conjunctival symptoms, lid swelling, lid ulceration and preauricular lymphadenitis are present without an apparent cause.

Culture and guinea pig inoculation should be undertaken in all suspected cases. The bovine strain is most easily distinguished from the human strain by its

high pathogenicity for the rabbit, which is resistant to the human strain.³ Histologically, bacilli may be seen in primary cases, but are difficult to see in secondary cases. Histology shows nonspecific infiltrate in the early cases, but typical tuberculous or atypical tuberculoid granulomas in cases older than 8 weeks.^{12,13}

In our Case 6, histopathologic picture could have been confused with sarcoidosis due to the absence of caseation; however, positive culture and guinea pig inoculation for tuberculosis bacilli, positive tuberculin test, and favorable response to anti-tuberculosis therapy confirmed the diagnosis of tuberculosis. At times diagnosis is difficult even with the use of all the criteria listed in Table 1. The establishment of diagnosis then rests on exclusion. Differential diagnosis includes sarcoidosis, trachoma, other causes of conjunctivitis, such as leptotrichosis, tularemia, cat scratch fever, syphilis, diphtheria, Parinaud's syndrome, episcleritis, simple granulation tumor, foreign body granuloma, and squamous cell and basal cell carcinomas.^{4,11,14,15,16} Although also rare, it appears from many recent reports that the intraocular tuberculosis is more common than the external involvement of the eye.¹⁷⁻²¹

The treatment, as recommended by the American College of Chest Physicians, for non-pulmonary tuberculosis is a 9-month course of isoniazid and rifampicin, usually supplemented by a 2-3 months of initial phase of one additional drug, usually ethambutol, streptomycin or pyrazinamide. Further details are available elsewhere.^{22,23} The routine treatment of tuberculosis in Pakistan consists of a 9-18 month course of multiple drug regimens. Commonly, isoniazid (INH) and ethambutol are combined with rifampicin or streptomycin in the first 3 months. Thiacetazone and pyrazinamide are also used occasionally. Local treatment for ocular tuberculosis, such as cycloplegia and antibiotic covered corticosteroids for the uveal inflammation, protective measures, such as antibiotic and lubricating ointments and surgical repair for ectropion, etc. must also be instituted where indicated.

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Acknowledgement: The authors thank Mrs. Janet Gschnitzer for her assistance.



Ophthalmic "Pastpourri"

"An Epoch-making Addition"

"Although there were no epoch-making additions to ophthalmic science during the past year the Editor wishes to draw the attention of his readers to at least one notable contribution to general diagnosis through the ocular apparatus. This is the ophthalmo-tuberculin and typhoid diagnosis test of Calmette, an adaptation to the eye of von Pirquet's cutaneous inoculation."

Casey Wood of Chicago - 1908
In Introduction to "The Practical
Medicine Series," Vol. 3.

"For the ophthalmic diagnosis the tuberculin, diluted about ten times, is dropped into the conjunctival sac. When performed on a tuberculous patient congestion is manifest in from three to five hours, the conjunctiva and caruncle become edematous and a certain amount of fibrinous secretion accumulates in the lower cul-de-sac. Within about 24 hours the reaction has disappeared." C. Wood -



Camera Clinicals

In this section of The Journal, photographic documentation of interesting and challenging observations are presented to the readers. They should make their diagnoses from the given information and compare these with the expositions given on pages 91 and 92 -Editor.



Figure 1



Figure 2



Figure 3

Figures 1, 2, and 3: A 30-year-old woman saw her ophthalmologist with left ocular pain and headache of four-day duration. There was also present edema of the eyelids and conjunctiva. The visual acuity was 20/30 (6/9) in each eye. Because of slight ptosis and restriction in eye movement, the diagnosis of orbital cellulitis was made and the patient treated with intensive intravenous antibiotic therapy without much improvement for four weeks (Figure 1). When laboratory workup showed normal blood count, normal erythrocyte sedimentation rate, negative blood culture,

normal x-rays for chest and paranasal sinuses, she was referred to us. Now her visual acuity was 20/20 (6/6), OD and 20/40 (6/12), OS. There was no afferent pupillary defect. She had severe limitation of the eye movements and proptosis of 5mm on the left (Figure 1). Other ocular findings were left redness and chemosis of the conjunctiva with dilated vessels in upper temporal quadrant, intact corneal sensitivity, normal slit lamp examination, and hyperemic optic disc. The computerized tomographic (CT) scanning proved most helpful in making the diagnosis. The

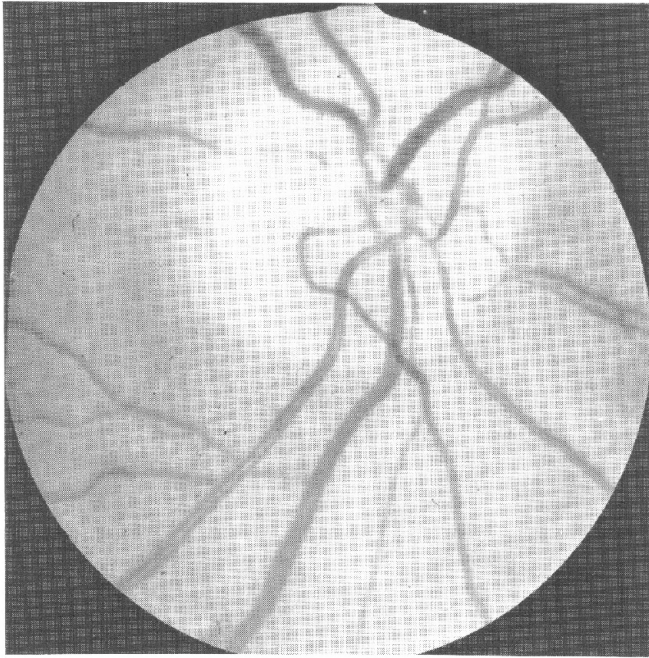


Figure 4

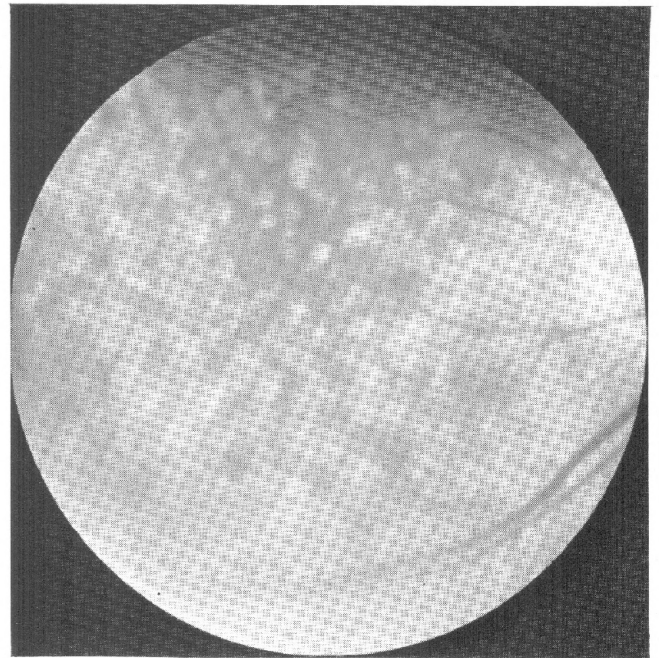


Figure 6



Figure 5

coronal CT section (Figure 2) and transverse CT section (Figure 3) both showed an intraconal mass.

Figure 4: A 58-year-old woman requested eye examination because she could not read well with her glasses. The eye examination showed visual acuity of 20/20 (6/6) in both eyes for distance with corrective lenses. Her add was changed, which she found satisfactory. External eye examination, extraocular muscle functions, pupillary

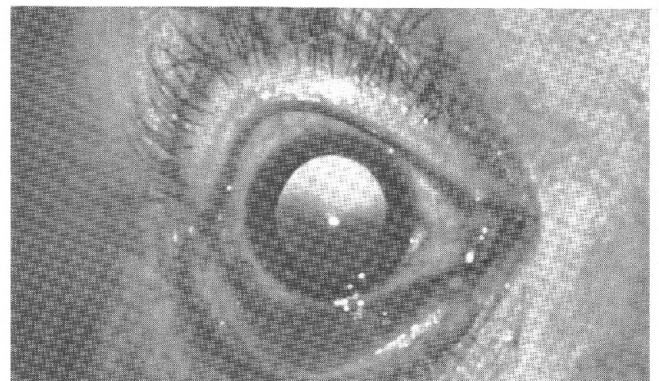


Figure 7

reactions, intraocular pressure, and slit lamp examination were normal. On ophthalmoscopic examination very interesting findings (Figure 4) were noted. Fluorescein angiography was normal.

Figure 5, 6, and 7: A 41-year-old man (Figure 5) complained of redness and irritation of his eyes. External examination showed right esotropia and conjunctivitis. On slit lamp examination the corneas, the anterior chambers, and the lenses were clear. The best corrected visual acuity was counting fingers in OD and 20/80 (6/24) in OS with "E" chart. Ophthalmoscopic examination bilaterally revealed the unexpected findings shown in Figure 6. No opacity of the lenses was noted even on fundus reflex after pupillary dilation (Figure 7). His refractive error was $-200 + 50 \times 90$, OD and $-650 + 175 \times 80$, OS. An antibiotic-corticosteroid topical medication was prescribed and the mother of patient instructed on how to apply it.



Extracapsular Cataract Extraction with Intraocular Lens Implantation in Pakistanis

Samina Jahangir, F.C.P.S.(P.)
Wasif M. Kadri, F.R.C.S.

ABSTRACT: We studied extracapsular cataract extraction with posterior chamber intraocular lens implantation in 40 Pakistani patients, 26 men and 14 women, ranging in age from under 10 to over 80. Seven (17%) patients were known diabetics. Four of the young patients had traumatic cataract. Preoperative visual acuity was 6/60 (20/200) or less in 38 patients and 6/36 (20/120) in two. Postoperative visual acuity was 6/12 (20/40) or better in 34 patients, 6/18 (20/60) in four, 6/24 (20/80) in one, and 6/60 (20/200) in one after one to 15 months of followup. Postoperative complications included iridocyclitis in 6 patients, glaucoma in one, and corneal edema in one, and all responded to medical treatment. Healon was used to reform the anterior chamber before insertion of the lens implant in at least 37 cases. Only one patient developed astigmatism of higher than two diopters. (Pakistan Journal of Ophthalmology 4:80-82, 1988.)

Modern extracapsular cataract extraction differs from its counterpart old technique in that the removal of cortex from behind the iris is accomplished in a closed chamber using a system of infusion and aspiration. If the cortex behind the iris has not been adequately removed, serious complications may develop. The basic procedure includes an opening in the anterior capsule, removal of the nucleus, and aspiration of the cortex capsule under the microscope. Properly performed posterior chamber lens implantation has produced excellent visual results worldwide. We conducted a study to learn its usefulness in Pakistani patients.

Materials and Methods

This study of 40 randomly selected patients, 26 (65%) men and 14 (35%) women, took 15 months. Twenty-four (55%) of the patients were over 50 and 16 under 50, two being children under 10. The indication for surgery was a cataract in older patients with no other ocular pathology and a unilateral traumatic cataract in children and young adults. A thorough eye examination, relevant systemic investigations, axial biometry, and keratometry were routinely performed. The power of the intraocular lens implant was calculated by SRK formula¹ of $P = A - BL -$

CK; where P is the implant power, L the axial length in millimeters, and K the corneal power in diopters, B is 2.5 and C is 0.9. (The constant A varied with implant design and the manufacturer.)

Patients received chloromycetin eye drops t.i.d. preoperatively. Pupils were dilated with 1% tropicamide and 10% phenylephrine. Surgery was performed under general anesthesia, using Zeiss operating microscope. An 11 mm half thickness limbal incision was employed. Anterior capsulectomy was done in a close anterior chamber with a 25 gauge hypodermic needle attached to infusion line. Nucleus was removed by expression, by applying pressure at 6 and 12 o'clock meridians. Cortex was irrigated and aspirated with balanced salt solution (BSS) using McIntyre canula. The anterior chamber was reformed with Healon, which was also injected behind the iris. One loop of lens was placed at 6 o'clock and the other at 12 o'clock behind the iris. Healon was removed with double barrel canula and the pupil constricted with acetylcholine chloride (Miochol). The incision was closed with 10-0 monofilament nylon continuous shoelace sutures. Postoperatively the patients received chloromycetin and Maxitrol eye drops t.i.d., and 1% tropicamide if uveal reaction was seen.

Seven (17%) patients had diabetes, and one each hypertension, joint disease, depression and trachoma. Four (10%) had traumatic cataract, and three (7.5%) had IOL implanted elsewhere. Thirty-eight (95%) of the patients had

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visual acuity of 20/120 (6/36) or less. The followup period ranged from one to 15 months. All of the implants were of posterior chamber type, with J loop design in 95 % and modified J loop in 5%. The lens power ranged from 15 to 23 diopters. The manufacturers of implants were Allergan in 30 (75%) cases, Pharmacia in four (10%), Domilens in two (5%), Cilco in two (5%), Vision Care in one (2.5%), and Mediconzept in one (2.5%).

Results

In 11 (29%) patients the final visual acuity was 6/6 (20/20) in 16 (40%), 6/9 (20/30) in seven (17.5%), 6/12 (20/40) in four (10%), 6/18 (20/60) in one (2.5%), 6/24 (20/80) in one (2.5%), and 6/60 (20/200) in one (2.5%) (Figure). The postoperative complications (Table) included anterior uveitis in six (15%) patients, glaucoma in one (2.5%), and corneal edema in one (2.5%).

Table
Postoperative Complications

Complication	No. of Cases
Anterior uveitis	6 (15%)
Thickening of posterior capsule	2 (5%)
Glaucoma	1 (2.5%)
Corneal edema	1 (2.5%)
Astigmatism (over 2 diopters)	1 (2.5%)
Subretinal neovascular membrane	1 (2.5%)

One patient developed subretinal neovascular membrane (SRNVM) 15 months after surgery and has a visual acuity of 6/24 (20/80). One patient with traumatic cataract revealed a macular scar after surgery, and has visual acuity of 6/60 (20/200). In the remaining three cases of trauma the vision improved to 6/6 (20/20) postoperatively. Six (15%) subjects developed uveitis postoperatively. In four patients the residual lens matter was responsible for postoperative inflammation which disappeared over a period of three months with medication and absorption of lens material. Two (5%) patients have developed slight thickening of the posterior capsule, and their vision is 6/9 (20/30). The patient who developed glaucoma is a diabetic, and his intraocular pressure is in control with medication. Moderate corneal oedema developed in one case and resolved latter. Posterior capsular rupture during operation occurred in one case, and an anterior chamber implant was used in him. One patient had high postoperative astigmatism of -4Dx180. His visual acuity is 6/12 (20/40). None of the remaining 39 cases has astigmatism of more than two diopters. In six cases of uveitis the manufacturers of implants were Allergan

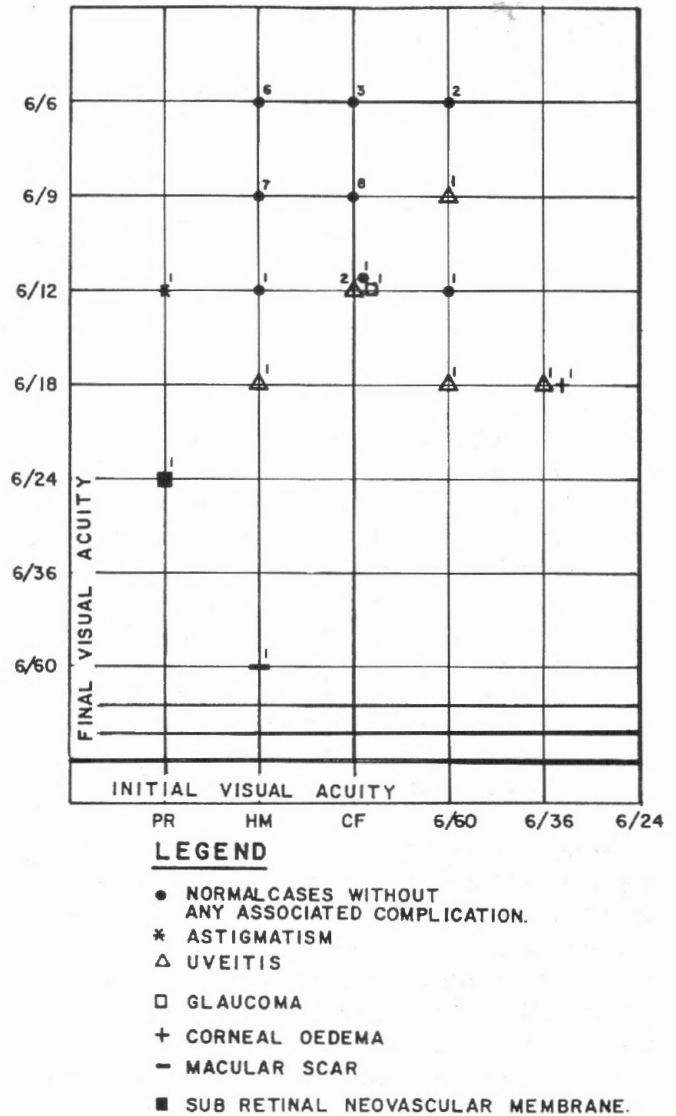


Figure (Jahangir, Kadri): The distribution of complications and initial and final visual acuity.

in 3 (out of 30), Domilens in 1 (out of two), Vision Care in 1 (out of one), and Mediconzept in 1 (out of one).

Discussion

In November, 1949, Harold Ridley² was the first surgeon to insert an intraocular lens implant after cataract extraction. By 1985, two million intraocular implants had been inserted.³ Early history of intraocular lens implant was characterized by a happy optimism, but in the mid-1950's it turned almost disastrous. The personal error was assigned a lesser role in this failure of lens implants, and it was suggested that the implant material was the cause of many complications. However, polymethylmethacrylate, which has been used since Ridley's first implantation, so far remains unmatched by other materials.¹ The main reasons

for the extensive use of posterior chamber implant are its superior postsurgical results and a lesser number of complications. The corneal endothelial damage is minimal, the view of fundus is better, the pupil may be dilated and is freely mobile and the appearance is cosmetically better.⁴

The early postoperative problems include corneal edema,^{5,6,7,8} hyphema, iridocyclitis, endophthalmitis,¹³ glaucoma⁹ pupil distortion, residual anterior capsule and lens matter,¹⁰ and displacement of the implant.¹¹ If the surgery is properly performed, these complications are significantly reduced. A major late complication is the thickening and opacification of posterior capsule, and there is no way to avoid it. A primary posterior capsulotomy may be performed at the time of surgery to circumvent it. Other late complications are retinal detachment,¹² cystoid macular edema,¹³ and postoperative astigmatism.^{14,15,16}

The blinding complications of lens implantation can be minimized by demanding intraocular lenses of proven superiority in design and quality, and by a strict adherence to uncompromising surgical technique, careful patient selection, and proper followup care.

We strongly feel that visualization with an operating microscope is mandatory during surgery. A controlled drip of balanced salt solution on to the operative field without pooling maintains optimum clarity of the cornea. It also prevents blood in the field from clotting, allowing visualization of bleeders themselves for control by cautery. The surgeon must be sufficiently trained to handle not only the routine lens implantation but also the complications which may arise during surgery. The implant system is a very sensitive one. Small deviation in implant design, manufacturing, sterilization, or surgical technique may lead to serious long-term complications. Drews¹⁷ has aptly quoted Binkhorst: "Errors of technique or judgement will be punished."

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Ophthalmic "Pastpourri"

The First Contact with a "Contact-Lens"

"I have succeeded in excluding the defective cornea from all dioptric influence by a small glass shell, and thus, without narrowing the field of vision or the field of fixation, have increased the minimum acuteness of vision from 1/30 to 1/6. As a matter of fact, I could not advise the possessor of this eye to use the remedy devised by me, and which I call "a contact lens," continually, because the fellow of her imperfect eye was approximately normal... The "contact-lens" consists of a thin glass shell, bounded by concentric and parallel spherical segments. It is placed upon the eye, and the interspace between it and the eyeball is filled with a liquid having the same refractive index as the cornea."

Fick, AE, of Zurich - 1887
216-1788



Migrating Bee Stings in the Cornea*

Khalid J. Awan, F.P.A.M.S.
Patricia W. Smith, M.D.

ABSTRACT: Two retained lancets of a bee sting in the left cornea of a 43-year-old truck driver gradually migrated from their original site during a 9-month followup. One lancet extruded leaving behind a small fibrotic scar, and the other migrated halfway into the anterior chamber without causing any reaction. In another patient, a seven-year-old boy, the retained bee stinger in the right cornea developed a disc-shaped chronic low-grade inflammatory reaction around it. After eight months, one end of the lancet became extruded and it was removed under a slit lamp. The eyes of both patients remained free of any serious inflammation, and recovered 20/20 (6/6) vision. Discussion includes comments on anatomy, pharmacology, toxicity, ocular morbidity, and therapy of bee stings of the eye and surrounding areas. (Pakistan Journal of Ophthalmology 4:83-86, 1988).

Although bee stings of the face and eyes are common, the direct involvement of the cornea is rare.¹⁻⁴ Depending on the susceptibility of the patient and the virulence of the venom of the insect, the sequelae may vary from only an insignificant corneal scar to a total blindness. We report two cases of corneal bee stings in whom the lancets in the cornea caused no severe reaction for many months. Eventually, the lancets migrated from their original sites, leading to a spontaneous extrusion of two lancets and halfway projection into the anterior chamber of the third without inciting any reaction there.

Case Reports

Case 1: On May 26, 1987, a 43-year-old truck driver was hit by a flying insect in his left eye. Immediate pain, visual loss and swelling of the eyelids ensued. The patient was initially treated with topical steroids and antibiotics at another facility. This treatment brought about gradual relief of pain and moderate improvement in vision. The patient then transferred to the Department of Ophthalmology at the University of Virginia.

The eye examination at our clinic showed visual acuity of 20/20 (6/6) OD, and 20/70 (6/21) OS. The eyelid edema

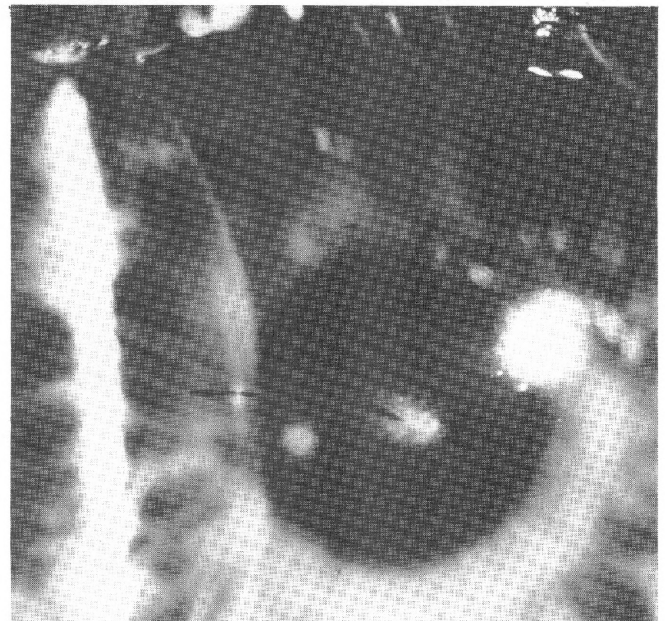


Figure 1 (Awan and Smith): Case 1. Left eye. One of the sting lancets has spontaneously extruded, leaving behind a small scar. The other lancet is deep in the cornea and projecting into the anterior chamber.

had completely resolved. Biomicroscopic examination showed some changes of guttata in the central cornea on the right. The left cornea had mild stromal edema in the area of pupillary axis. Two stingers were present in the cornea, one in the anterior stroma just past Bowman's membrane and the other in the deeper stroma near Descemet's membrane, which the lancet had not perforated (Figure 1). There were no

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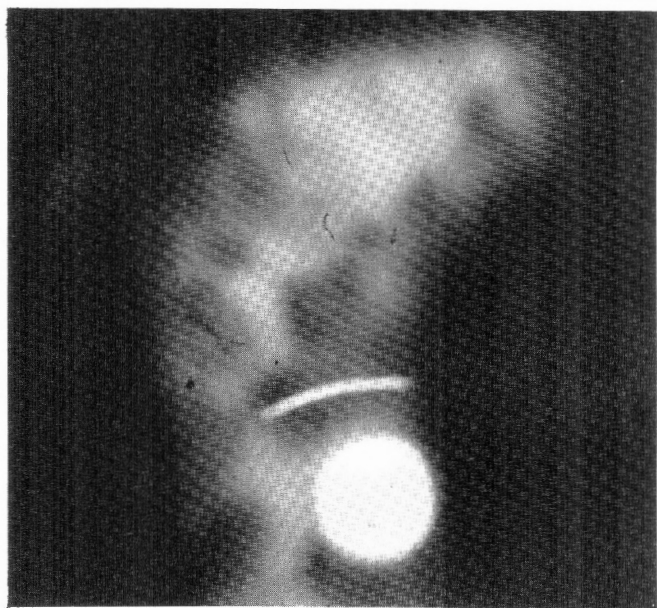


Figure 2 (Awan and Smith): Case 2. Right eye. Photobiomicrograph. Note the disc-shaped area of reaction in the cornea surrounding the stinger, eight months following initial bee sting. The anterior end has already become extruded by migrating.

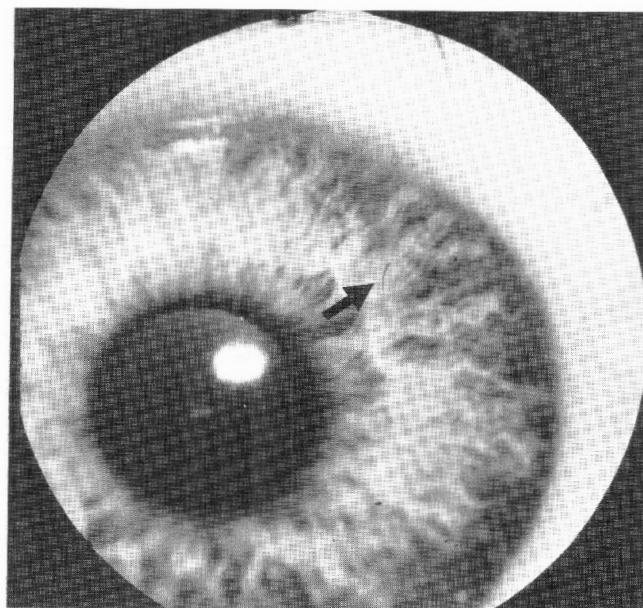


Figure 3 (Awan): Case 2. Right eye. Direct view of the bee sting in the cornea (Arrow).

keratic precipitates on the endothelium and the anterior chamber was free of any reaction. On gonioscopy, the angles were clear. The medications were tapered as the patient became more comfortable and as the visual acuity improved. On his recent followup visit on February 5, 1988, the anterior corneal stinger had disappeared, leaving behind a small fibrotic scar in the superficial stroma. However, the posterior barb had migrated posteriorly and projected halfway into the anterior chamber. No inflammation was present in any of the ocular structures and the visual acuity in the left eye had improved to 20/25 (6/7.5).

Case 2: A 7-year-old boy was seen with intermittent complaint of watering and irritation of his right eye. Eight months previously he had been stung by a bee in his right eye. The pain and swelling of the eye had subsided in a few days, so the parents never sought any medical help.

Eye examination showed the visual acuity of 20/50 (6/15) in the right eye and 20/20 (6/6) in the left. The right eye was moderately injected and tearing. On slit lamp examination, a rounded area of stromal reaction was seen in the right mid-central cornea. No reaction was present in the anterior chamber. In the middle of the stromal reaction, a black linear foreign body with a slightly extruded end was noted under a slit lamp (Figures 2 and 3). It was easily removed with forceps, and found to be a bee sting, which had migrated anteriorly. The patient was treated with topical antibiotic drops for a few days with good recovery and final visual acuity of 20/20 (6/6) in both eyes.

Discussion

The ocular morbidity of a bee sting depends on the allergens and toxins contained in its venom and the degree of

the patient's idiosyncratic reaction to them. The bee venom contains the toxins apamin, formic acid, and melittin, and the principal allergen phospholipase A.^{5,6} Apamin causes immediate pain by acting on the neural junctions, and melittin, the main toxin, releases serotonin from the thrombocytes and histamine and other pharmacologic mediators from the mast cells. The resulting Type 1 hypersensitivity response is mediated by IgE. Hence, the corneal sting may cause no more than a minimal foreign body sensation in individuals resistant or immune to these substances; however, very severe changes, even in distant structures, in others may result in blindness or the loss of an eye.^{1,2,3,7} The various pathologic changes between these two extremes that have been reported are given in the Table.

Young⁴ published the first extensive review of the subject in English. A more recent report on corneal bee-sting included three patients, all of whom eventually recovered full vision.³ The injurious effects of a bee sting of the eye or around it may be of three types: mechanical trauma, local toxic reaction, and remote indirect damage due probably to hypersensitivity or anaphylactoid response.^{1,4,8} Hence, cataract may appear even when there is no direct injury to the lens, and bee stings of the head and face have been reported to cause papillitis, papilledema, and optic atrophy.^{1,2,8} In one report the patient developed necrosis and hemorrhages of the central retina from remote effects of a presumed bee sting of the cornea.⁷ In this case the eye had to be enucleated on the ninth day due to severe endophthalmitis after a corneal insect bite. Goldstein, Rucker, and Klass⁸ noted papillitis after bee sting of the head, and Walsh and Hoyt² described two patients, one with external ophthalmoplegia and the other with optic atrophy, following

Table
Local and Remote Ocular Changes
From Bee-Sting

Eyelid edema, erythema, gangrene^{1-4,10}
 Conjunctival hyperemia and chemosis¹⁻⁴
 Corneal ulcer, stromal keratitis, and necrosis^{1-4,11}
 Corneal scarring with vascularization¹⁻⁴
 Recurrent erosion¹
 Bullous keratopathy¹
 Corneal perforation^{2,4}
 Iridocyclitis, hypopyon¹⁻⁴
 Endophthalmitis^{1,7}
 Iridoplegia³
 Accommodation paralysis⁹
 External ophthalmoplegia^{2,9}
 Iris depigmentation^{2,4}
 Glaucoma^{2,4}
 Anterior polar cataract^{1,4}
 Retinal necrosis⁷
 Papillitis^{2,8}
 Optic atrophy^{2,8}
 Visual field contraction⁹
 Orbital osteoperiostitis¹⁰
 Ophthalmia nodosa¹

a bee sting of the eyelid. Szeghy, Papai, and Vas⁹ reported a case of corneoscleral bee sting which resulted in partial and irreversible depigmentation of the iris, diffuse vitreous haze, paralysis of accommodation, paresis of the lateral rectus, and concentric narrowing of the visual fields.

Anatomical studies have established that the stinger of a bee contains two barbed lancets or darts. Ordinarily, only one lancet is propelled at the time of the insect bite, but both darts may be used to stab simultaneously in some instances.¹² This may have happened in our Case 1, who had two lancets burried in his cornea. The bee-sting may be distinguished from other insect bites by the fact that only the honey-bee leaves a stinger in the tissues.¹³ The venom pouch with poison injecting apparatus remain attached to the stinger (Figure 4), allowing repeated injections of venom into the wound by repeated contractions of the pouch. An attempt to remove the stinger by grasping its fleshy outer end may compress the venom pouch, causing injection of more venom into the ocular tissues with resultant severe aggravation of inflammation.¹⁴ Hence, great care should be taken to avoid compression of the fleshy part of sting during its removal. The retained stinger usually remains inert after the acute reaction to the venom has subsided, even if the lancet has penetrated into the anterior chamber.^{1-4,15} However, in some instances chronic inflammation resembling ophthalmia nodosa due to the retained sting in the cornea has been recorded.¹ In such cases surgical removal of the sting may become necessary. Ophthalmia nodosa and keratoconjunctivitis nodosa characteristically develop in

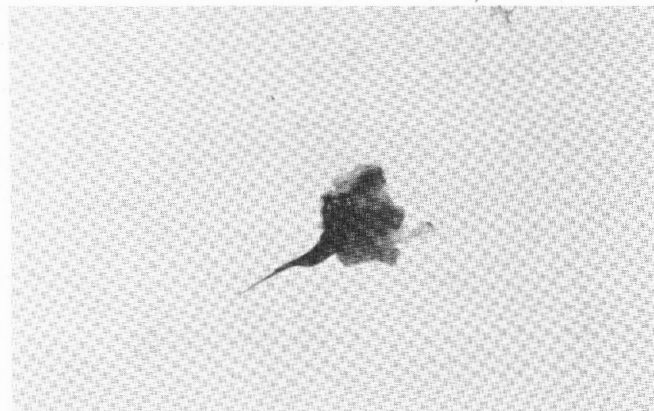


Figure 4 (Awan and Smith): A bee sting with its venom injecting apparatus.

response to caterpillar hair or other insect parts.^{16,17} Our Case 1 belongs to the category where retained sting remains inert, and Case 2 to the category in which it leads to chronic inflammation. When no reaction is seen in response to a sting in the cornea or anterior chamber, it need not be removed.^{1,2,15} We followed this course of action in Case 1. In Case 2, persistent reaction and extrusion of one end of the sting necessitated its removal, which was easily accomplished under a biomicroscope.

At times, the sting in the eyelid may remain buried in the tissues of the eyelid for many days, months, or even years after the subsidence of the acute reaction, only to gradually work its way deeper through the tarsus to damage the cornea by jabbing on it.¹⁸⁻²⁰ Simple removal of the lancet after everting the lid may suffice to cure this problem. The gradual migratory movement of the lancets may be due to their barbed structure. The sting's migration in the tissues is determined by the direction of barbs on it. It has been postulated by some authors that the migration of a caterpillar hair in the cornea is caused by the pressure exerted on it by accumulation of surrounding inflammatory cells.²¹ A similar mechanism may be at work with a bee sting. In our Case 1, one of the lancets migrated inward to enter the anterior chamber, whereas the other lancet in this case, and the one in Case 2 extruded by migrating outward.

In the management of the bee-sting of the cornea, the treatment should be directed at mechanical trauma, toxic and allergic reaction to the venom, and any complications. The sting should be removed without grasping the venom injecting apparatus. Cold compresses, 1:1000 epinephrine drops, intravenous antihistamines, and intensive topical and, if required, systemic corticosteroids will control the allergic manifestations in most instances. Cycloplegics and other treatment for iridocyclitis, cataract extraction for serious enough lens opacities, antibiotics to prevent or treat any infection, antiglaucoma medication for secondary glaucoma, sufficient followup to discover any late complications, may be necessary.^{13,22}

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Ophthalmic "Pastpourri"

A Trachoma "Tale"

"In shape and position, the trachoma-coccus greatly resembles the staphylococcus pyogenes, from which it differs (a) by its greater size, (b) lesser mobility, (c) greater tenacity of the cultures, which liquefy gelatine but slowly. The trachoma-cocci are sometimes arranged in chains."

Schmidt, E: The micro-organism of trachoma and some other mycotic diseases of the conjunctiva. Inaug. Diss.

- 1887

117-11188



Unusual Presentation of Sebaceous Carcinoma of the Eyelid*

Faiz Muhammad Halepota, F.C.P.S.(P.)
Sher Mohammad Shaikh, M. Phil. (Path.)

ABSTRACT: A clinically benign looking elongated and bulky tumor of the left lower eyelid in a 60-year old Pakistani man proved to be a sebaceous carcinoma on histopathologic examination. It is a most unusual presentation of this malignant neoplasm. Fortunately, the lesion was limited to the excisional biopsy specimen, and no metastases appeared during a one-year followup. (Pakistan Journal of Ophthalmology 4:87-89, 1988.)

Sebaceous carcinoma of the eyelid is rare but one of the more malignant lesions. Its incidence over the years have undergone very interesting variations, from total denial of its existence to the utterly erroneous claim of all meibomian neoplasms being carcinomatous.¹ The latest incidence has been reported to be 0.67% to 1% of all malignant eyelid tumors.^{2,3} The neoplasm usually presents as persistent chalazion or meibomianitis.¹⁻¹⁰ However, very exceptionally it may present as a cutaneous horn,^{5,11} or a diffuse eczematoid thickening.¹² We report here another unusual presentation of sebaceous carcinoma of the eyelid, as an elongated smooth mass that clinically appeared benign.

Case Report

A 60-year-old farmer was admitted on May 6, 1987 to Chandka Medical College Hospital, Larkana with a pedunculated growth, hanging from outer part of the left lower eyelid. He gave a history of a direct blunt injury to the left lower orbit ten years ago. Following this trauma a small growth erupted on the lower lid. It gradually enlarged to attain the size of a small egg (Figure 1). A few weeks before his admission, the tumor ulcerated, leading to a blood stained discharge. The preauricular and submandibular glands were not palpable. X-ray of the chest was normal. On eye examination, the visual acuity was reduced to counting fingers at one meter distance in both eyes due to bilateral advanced cataracts (cataract nigra). The views of the optic fundi were totally obscured. The intraocular pressure was



Figure 1 (Halepota and Shaikh): Preoperative photograph showing an elongated bulky tumor hanging from the temporal half of the left lower lid. The free extremity of the tumor is ulcerated and blood stained discharge.

within normal limits. A surgical excision of the tumor under local anesthesia was performed. The lid margin was split at gray line and the base of the growth was carefully dissected. A 4 mm wide collar of healthy skin was also removed around the base of tumor to ensure total removal of the tumor-bearing area. Tarsal plate was left intact as the tumor did not involve it. The skin defect was covered by a full thickness free skin graft taken from the mastoid region.

The graft took very well and the patient was pleased with the result. The patient was seen in followup on June 16, 1987 and November 19, 1987. No evidence of local recurrence and lymph node enlargement was noted on one-month, 6-month and one year followup.

Histopathological examination grossly showed an oval nodule of firm, gray brown tissue measuring about 6 cms in circumference. It was covered with skin showing an area of ulceration. The cut surface showed gray white areas interspersed with dark brown areas. Microscopically the tumor tissue consisted of sheets of epithelial cells containing abundant foamy and vacuolated cytoplasm

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*This paper was presented at the 11th Congress of the Ophthalmological Society of Pakistan at Peshawar, February 18-20, 1988.

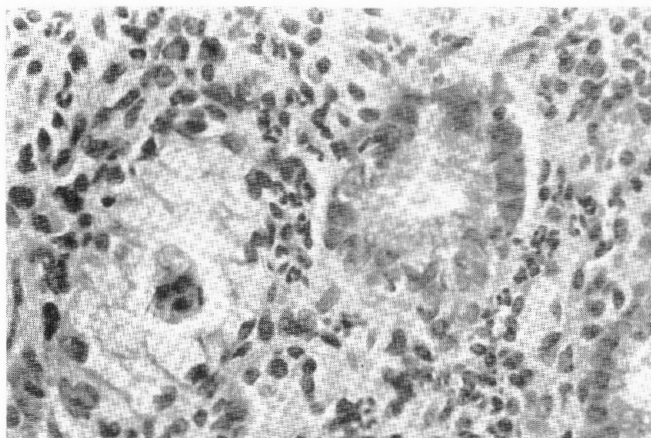


Figure 2 (Halepota and Shaikh): Histopathologically the tumor shows glandular pattern of malignant cells surrounded by chronic inflammatory cells (hematoxylin and eosin, X400).

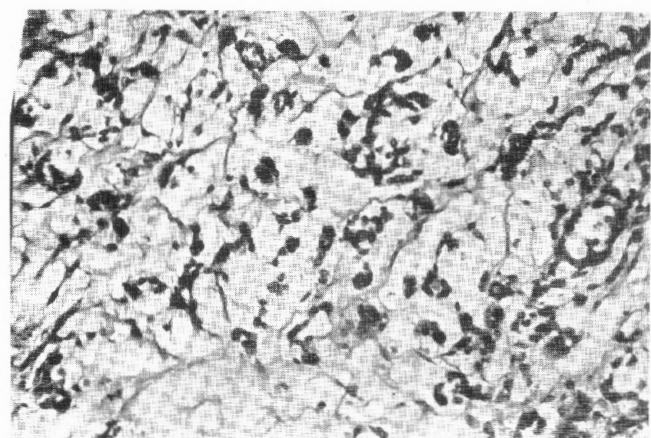


Figure 3 (Halepota and Shaikh): In another histologic view the tumor shows sheets of malignant epithelial cells. Cytoplasm has foamy and vacuolated appearance, suggestive of sebaceous material (hematoxylin and eosin X100).

suggestive of sebaceous material. At places the cells showed glandular pattern of arrangement. The neoplastic cells contained large round to oval hyperchromatic vesicular nuclei with prominent nucleoli (Figures 2 to 4). The histopathologic diagnosis was sebaceous carcinoma of the eyelid.

Discussion

Sebaceous carcinoma of the eyelid usually occurs in old age; however, younger age group may occasionally be affected.¹⁰ The upper lid is involved more often than the lower.⁵ The clinical features are variable, and it may simulate basal cell carcinoma, chalazion, blepheroconjunctivitis, eczematous thickening, diffuse carcinoma, papillary lesion or an orbital tumor.¹⁻¹² Because of this and its rare occurrence, there are many errors in diagnosis and treatment of this neoplasm.^{1,2,5,8} These tumors arise in normal skin without any apparent etiological factors. Reese⁶ described three patterns of tumor growth: an external bulky fungating mass, an invasion eroding the lid

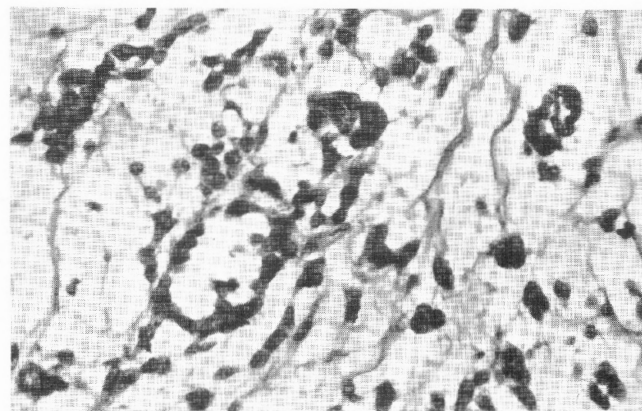


Figure 4 (Halepota and Shaikh): Tumor cell nuclei are hyperchromatic and depict pleomorphism, (hematoxylin and eosin, X 400).

margin without producing appreciable tumor mass, and a mass penetrating deep structures of the orbit including bones.

Sebaceous carcinoma may arise from any of the sebaceous glands of the lid, the glands of Zeis, the sebaceous glands associated with the hair follicles, or the meibomian glands. Sebaceous carcinoma of the lid is a rare but a very serious tumor.¹⁻¹² Boniuk and Zimmarman⁵ reported an incidence of 30% death rate after five years, and a tendency to metastasize early. The growth on the eyelid in our patient presented as a bulky pedunculated tumor mass, which was ulcerated and bleeding at the free end. The tumor had attained quite a large size before its excision. The peculiar features of this tumor are its history of long existence, large size, its mainly outward growth, non-infiltration of the deeper structures of the lid, easy excision, well defined capsule, and much delayed ulceration at the free end. These features gave an impression of a benign tumor. These findings further assert that errors in diagnosis may be easily made.^{4,5} To our knowledge, only two other cases presented as an elongated mass (cutaneous horn) on the eyelid.^{2,9}

We postulate that initially the lesion in this case was an adenoma which later underwent malignant change within the confines of a well developed capsule. This assumption is consistent with observation of other authors, who contend that malignant proliferations in adenomas are frequent and some adenomas of sebaceous glands may change into adenocarcinomas.^{3,4} Generally, the tumor grows in the direction of least resistance and in the lid the thin and loose skin has least of it to offer when compared to the rigid tarsal plate. This may explain non-invasion of the deeper lid tissues in our case. Late ulceration and necrosis could be attributed to compromising of the blood supply, a feature common to large-sized both benign and malignant neoplasms. Since in this case the growth was localized and encapsulated without infiltration of the lid, the possibility of any metastases was felt to be remote. The foci of sebaceous differentiation in squamous and basal cell carcinomas are not

uncommon; also, poorly differentiated sebaceous carcinoma may simulate squamous cell carcinoma.¹ However, the serial sections of the tumor in our patient showed neither squamous nor basal cell carcinomatous areas. Another interesting aspect of sebaceous carcinoma of meibomian gland is that at occasions it may be multicentric.¹³ Trauma as a precipitating cause for ocular malignancy has been considered in some reports.¹⁴ We cannot say with certainty whether the trauma sustained by our patient prior to the appearance of his neoplasm did or did not play any role in the development of the tumor.

As regards the origin of the tumor, in most instances it is not possible from histological material and clinical information to determine the exact site of origin of tumor.¹ However, non-invasion of the tarsal plate by carcinoma definitely concludes that the tumor arose either from Zeis glands or sebaceous glands associate with fine hair follicles of the lid, and not from meibomian glands.

In the treatment of lesions suspected to be sebaceous carcinomas, a bold and wide surgical excision is recommended by most authorities.^{1-3, 6-9} Some authors advise that all tissues under the abnormal appearing conjunctiva should be removed and checked histopathological to confirm total excision of the tumor. However, even this approach may fail to eradicate the pagetoid spread of sebaceous carcinoma.¹⁵

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Ophthalmic "Pastpourri"

Hemorrhage Horror

The author observed "in a haemophilous child aged 9 months, a hemorrhage from the conjunctiva of the left upper lid, which could not be permanently stilled, and finally caused the death of the child."

H. Schmidt-Rimpler - 1888
117-24388



Pakistan Academy of Medical Sciences

Convocation 1988 and Conference on
"Hospitals in Pakistan"
December 22, 1988 at Karachi

The Pakistan Academy of Medical Sciences will hold its Convocation 1988 on December 22, 1988 at 10 a.m. at The Agha Khan University in Karachi. President of the Islamic Republic of Pakistan, Gen. Mohammad Zia-ul-Haq, will deliver the Convocation 1988 address.

The PAMS Convocation 1988 will be followed by a Conference on "Hospitals in Pakistan." There will be a reading of the Pakistan Academy of Medical Sciences Oration by a very eminent scientist before the discussions on Hospitals in Pakistan. The PAMS Oration carries the distinction of the title of PAMS Professor for the lecturer. There will be no other reading or presentation of papers during the conference discussions. However, all the participants will be given copies of all the written papers that are received by the Chairman of the Organizing Committee. All interested scholars are invited to send their papers before November 15, 1988, to the addresses given with this announcement.

Pakistan Academy of Medical Sciences Junior Award and Gold Medal is given annually to a Pakistani professional holding the position of Assistant Professor or under in any of the medical and biomedical fields for writing the most outstanding original research paper during the previous year. The PAMS Junior Award and Gold Medal are intended to stimulate interest in research and writing. In addition to a Gold Medal, the recipient is awarded a bursary of Rs. 10,000.00. A committee of experts appointed by The Academy evaluates the entries and decides on the most deserving paper. All interested are invited to submit their entries to Professor Najib Khan, FPAMS, before September 30, 1988.

Address: Professor Najib Khan, FPAMS

Chairman, Organizing Committee, PAMS Convocation 1988

Said Clinic

I.I. Chundrigar Road

Karachi, Pakistan Tel: 214841



OPHTHALMOLOGICAL SOCIETY OF PAKISTAN

XIII Congress at Karachi
February 23-25, 1989

The XII Congress of the Ophthalmological Society of Pakistan will be held on February 23-25, 1989 in Karachi. Speakers and participants are cordially invited from all parts of the world. Anyone interested in making a presentation should send the abstract(s) of his paper(s) to the Chairman, Organizing Committee, Dr. Jamshed H. Wania, F.A.C.S. The program of the XII Congress into following sections:

(1) **Pediatric Ophthalmology:** a. Genetically transmitted diseases and parental counselling; b. Strabismus and its Management; c. Management of Congenital Cataracts.

(2) **Neuro-Ophthalmology:** a. Diagnosis and Medical Therapy of Neuro-Ophthalmic Disorders; b. Role of an Ophthalmologist in assisting Neurosurgical Department with specific reference to "CAT SCANNING."

(3) **Lacrimal Drainage System:** a. Problems in Children; b. Problems in Adults; c. Recent Trends in Surgical Approach.

(4) **Management of Diabetic Ophthalmic Problems.**

(5) **Toxicity of Ophthalmic Drugs.**

(6) **Principles of Ultrasonography and its Ophthalmic Applications.**

In addition to these topic, free papers on Surgical and Medical Aspects of Ophthalmology will be welcome. Last Pre-Registrations date is December 31, 1988. For further details contact: Dr. Jamshed H. Wania, F.A.C.S., Chairman, Organizing Committee, XII Congress, Room 1, Anklesaria Nursing Home, Karachi, Pakistan.



Figures 1 and 2

Computerized Tomography in Diagnosis of Idiopathic Inflammatory Pseudotumor of the Orbit

Rizwan A. Cheema, FRCS

ABSTRACT: A 30-year-old woman presented with pain, proptosis and external ophthalmoplegia of the left eye. Although routine investigations were normal, computerized tomographic scanning suggested a diagnosis of non-specific and idiopathic inflammatory pseudotumor of the orbit. The patient was successfully treated with oral corticosteroids. (Pakistan Journal of Ophthalmology 4:78, 79, and 91, 1988). Reprint requests to Rizwan A. Cheema, F.R.C.S., Cheema Hospital, Daska, Pakistan.

The term "idiopathic inflammatory pseudotumor" or IIP encompasses a wide spectrum of non-specific inflammatory conditions which simulate orbital neoplasm. Most authorities now do not include specific inflammatory and space-occupying lesions in this category, and reserve the term pseudotumor only for those cases where no systemic or local cause of inflammation can be found. An immune mechanism occurring in the orbit may play a part, and there have been suggestions that certain types of IIPs are seen more frequently in patients with autoimmune disorders.¹ The disease has also been associated with chronic sinusitis and upper respiratory tract infections.^{2,3} The histological features show a wide spectrum, from reactive lymphoid hyperplasia and atypical lymphoid hyperplasia consisting of predominantly mature lymphocytes, plasma cells, histiocytes and lymphoplasmacytoid bodies at one extreme to a non-specific polymorphous infiltrate of inflammatory cells consisting of polymorphs, lymphocytes, plasma cells and macrophages with a varying amount of fibrosis depending on the chronicity of the process at the other. The idiopathic inflammatory pseudotumor may be diffuse or localized with inflammatory process targeting a specific orbital tissue such as myositis, dacryoadenitis, periscleritis, or perineuritis.⁴

The disease is more common in the middle age, but can affect children as well as those in late 60's and 70's. There is no sex predilection and the disease is usually unilateral. Local pain and proptosis are cardinal features, but associated constitutional symptoms may be present. There is engorgement of vessels with swelling of the lids and conjunctivitis. The features vary depending on the structures involved. There may be a varying amount of ophthalmoplegia and the involvement of posterior orbit and cavernous sinus may give rise to orbital apex syndrome. It may also be associated with trigeminal nerve involvement, uveitis, papillitis, optic neuropathy and exudative retinal detachment. The involvement of extraocular muscles may

cause engorgement of vessels over the involved muscle and restrict its function. The involvement of lacrimal gland can cause pain and tenderness in the upper outer quadrant of the orbit. Despite treatment, the disease may have a chronic recurrent or progressive course with visual loss. Laboratory evaluation is usually not rewarding, and the diagnosis may rest entirely on history, examination, ultrasonography and computerized tomography scan findings. The advent of new generation CT scanners have helped enormously in the differential diagnosis of the orbital disease. The role of fine needle aspiration biopsy is controversial.

In most instances, the disease responds to systemic corticosteroids. Some authorities feel that diagnosis should be reconsidered if there is no favorable response to systemic corticosteroids within 24-48 hours. This patient was started on systemic prednisolone 80 mg on alternate days. Her headache and pain subsided within 24 hours and proptosis improved over next few days, completely clearing up in three months. High doses of prednisolone, 80 mg per day with breakfast, should be given for three weeks and then gradually tapered off. Recurrent cases may require a low maintenance dose.⁵ Chronic cases resistant to steroids may need supervoltage radiation. Surgery has no role in the treatment of idiopathic inflammatory pseudotumor. Immunosuppressive agents, such as chlorambucil, have recently been used alone or in combination with other modalities in resistant cases.

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Figures 4: Anomalous Retinal Veno-Venous Crossing

ABSTRACT: A 58-year-old woman had an anomalous veno-venous crossing of her left retina in which the inferior temporal vein coursed over the inferior nasal vein near the optic disc. Visual functions and fluorescein angiography were normal. Veno-venous crossing in the retina is regarded as a very rare anomaly, but in my opinion it is not that uncommon. (*Pakistan Journal of Ophthalmology* 4:79 and 92, July, 1988.) Reprint requests to Khalid J. Awan, F.P.A.M.S., 1921 Park Avenue, SW, Norton, Virginia 24273 USA.

It was thought in the past that veno-venous or arterio-arterial crossings never occur in the retina.¹ However, more recent reports have shown that such anomalies do occur and, in fact, are not uncommon.^{2,3} These non-pathologic veno-venous or arterio-arterial crossings always occur on or near the optic disc, which proves my previous hypothesis that they result when tissue of Bergmeister's papilla is absorbed, and the vessels in it lie flat in an irregular fashion, leading to falling across each other of some vessels of

identical nature.³ These anomalies are benign, and must be differentiated from neovascularization.

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Figures 5, 6, and 7: Macular Drusen (Doyme's) and the Absence of Lenticular Opacities in Adult Down's Syndrome

ABSTRACT: A 41-year-old man with Down's syndrome with right esotropia had no lenticular opacities, but had bilateral extensive macular drusen of Doyme's honeycomb type. This apparently rare association has not been previously reported. The patient's vision was affected by amblyopia in OD and by myopic degeneration in OS. Interestingly, the lenses of this 41-year-old mongoloid had not developed any opacities. (*Pakistan Journal of Ophthalmology* 4:79 and 92, July, 1988.) Reprint requests to Khalid J. Awan, F.P.A.M.S., 1921 Park Avenue, SW, Norton, Virginia 24273 USA.

Dominantly inherited drusen with late macular dystrophy are a type of senile macular degeneration which is also called Doyme's honeycombed or Tay's central guttate choroiditis.¹ It may be familial and inherited as an autosomal dominant trait, but the role of heredity is uncertain in most patients.¹ The earliest sign is appearance of multiple rounded drusen in macular area in the third decade of life. Most patients retain excellent vision into their sixties, and some may never lose sight.^{1,2,3} In many patients loss of sight in one eye is never followed by its loss in the other. Fluorescein angiography reveals more drusen than are visible on ophthalmoscopy, and when vision is disturbed, it may uncover serous detachments of the retinal pigment epithelium. No treatment is known for this condition. This type of macular changes are very rare in patients with Down's syndrome (Mongolism).

Usually, the patients with Down's syndrome develop at least some lenticular opacities before they reach 40. It is interesting that in this patient lenses have remained clear at age 41. The association of Doyme's honeycombed macular dystrophy with Down's syndrome is interesting and unusual, and has not been documented before.

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Diagnostic Pitfalls of Pseudoexfoliation Syndrome*

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

ABSTRACT: A 60-year-old Pakistani engineer had a normal eye examination by an American ophthalmologist in 1983. In 1984, I examined his eyes and found intraocular pressures of 26 mm Hg, OD and 21 mm Hg, OS. The cup/disc ratio was higher in OD without any visual field changes. In 1985, a British ophthalmologist diagnosed primary open-angle glaucoma. In 1986, a Pakistani ophthalmologist who agreed with this diagnosis started medical therapy. Later, a Mexican ophthalmologist advised discontinuation of antiglaucoma drops. In 1987, he again consulted me when the ophthalmologist in Pakistan recommended surgery on the right eye. On examination, I discovered the typical changes of pseudoexfoliation syndrome in the right eye. This case illustrates: that pseudoexfoliation is easily missed when even highly trained examiners do not have a high index of suspicion, that elevated intraocular pressure may precede the clinical manifestation of pseudoexfoliation, that primary open-angle glaucoma may coexist with pseudoexfoliation, that all cases of unilateral glaucoma should be examined biomicroscopically with dilated pupils on each visit for pseudoexfoliation, and that in addition to the recently reported pigmentary changes in the iris sphincter area, the eyes with unilateral hypertension, more advanced cataract, a poorer pupillary dilation, and a greater pigmentary dispersion during pupillary dilation should be considered pseudoexfoliation syndrome suspects. (*Pakistan Journal of Ophthalmology* 4:93-94, July, 1988.)

Pseudoexfoliation syndrome is a degenerative exudative condition wherein fibrillo-granular material accumulates in the tissues of the anterior segment of the eye, particularly on the anterior lens capsule, the zonules, pupillary border, and the trabecular meshwork. It is often accompanied by raised intraocular pressure which may or may not induce glaucomatous cupping and visual field changes. Recently, Streeten, Prince, Ritch et al¹ pointed out pigmentary changes which may help in the diagnosis of this entity before the actual appearance of biomicroscopically visible dustlike and flaky deposits of pseudoexfoliative material. Pseudoexfoliation syndrome is found worldwide. However, its presence may be easily overlooked even by a well-trained ophthalmologist who is not specifically looking for it.

Case Report: A 60-year-old Pakistani engineer had eye examination by an American ophthalmologist in 1983. Other than a change in his prescription for glasses, no abnormality was detected. A year later, he came to see me,

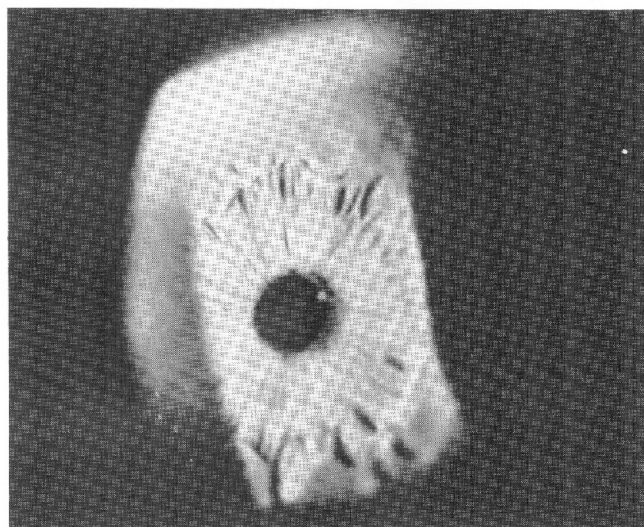


Figure 1 (Awan): Right eye. Biomicrograph. Note one white speck of exfoliative material on the pupillary margin. Also, note atrophic pupillary ruff.

and I found his intraocular pressure elevated to 26 mm Hg in OD and 21 mm Hg in OS. The optic cup on the right was also larger, but visual fields on a tangent screen were normal. I advised the patient to have his intraocular pressure and optic discs evaluated periodically to make sure no visual

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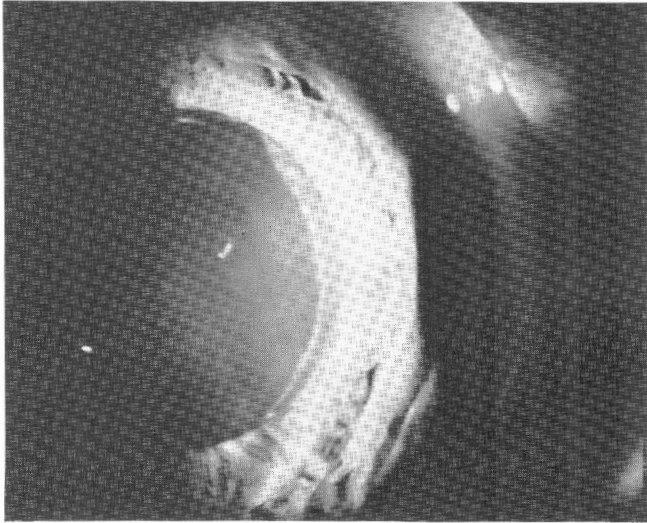


Figure 2 (Awan): Right eye. Biomicrograph. Note the typical exfoliative material deposits on the anterior lens capsule, which became noticeable only after dilating the pupil.

damage occurred. In 1985, the patient visited an ophthalmologist in England, who told the patient of his having open angle glaucoma and the need for its treatment. Next year, the patient saw an ophthalmologist in Pakistan who concurred with the opinion of the British ophthalmologist, and started the patient on timolol 0.25% drops in the right eye. Later, he changed the treatment to 0.5% timolol in both eyes. Over the next many months, first pilocarpine 2% and then pilocarpine 4% were also added. When glaucoma did not appear to respond, acetazolamide 250 mg daily was added. Eventually, the Pakistani ophthalmologist advised surgery in the right eye. In July 1987, the patient came to the United States to seek my opinion. On biomicroscopic examination, I noted a tiny white spot on the pupillary border of the right eye (Figure 1) I decided to do the examination by dilating the pupils once again. The right pupil dilated less than the left. There were greater pigment dispersion and cataractous changes in the right eye. The most interesting, however, was the finding of typical pseudoexfoliation deposits on the anterior lens capsule (Figure 2). The patient also informed me that he had seen a Mexican ophthalmologist who had advised him to discontinue the glaucoma drops. The left eye had no changes compatible with pseudoexfoliation syndrome, although its intraocular pressure had been recorded in lower twenties at least on three occasions.

Comments

Pseudoexfoliation is being reported with increasing frequency in eastern countries, such as Pakistan,² Iran,³ Syria,⁴ Japan,⁵ etc. The accurate diagnosis of pseudoexfoliation is important. Secondary exfoliative glaucoma does not respond as well to medical therapy

including currently popular timolol, as primary open angle glaucoma does.¹ Also, cataract surgery on eyes with exfoliation syndrome has higher incidence of complications like posterior capsular rupture,⁶ zonular rupture,⁷ and intraocular bleeding.^{6,7} The eyes with exfoliation syndrome have greater incidence of cataract formation and their pupils dilate less well.^{1,2,5} This Case demonstrates that unless the examiner has a high index of suspicion even the highly trained ophthalmologists may overlook the diagnosis of pseudoexfoliation syndrome. This Case also demonstrates that intraocular pressure elevation may precede the actual appearance of pseudoexfoliation. It may also be argued that this is a case of open-angle glaucoma in whom pseudoexfoliation is merely an incidental change. This Case appears also to lend support to the concept that pseudoexfoliation is merely another manifestation of degenerative changes that are responsible for glaucoma.⁸ Nonetheless, it is hard to deny that in many cases exfoliation is indeed directly responsible for a secondary type of glaucoma.^{1-5,8}

Prince, Streeten, Ritch, et al¹ consider pupillary ruff defects, iris sphincter tranillumination, whorl-like particulate pigment deposits on the iris sphincter, the anterior iris surface, and trabecular meshwork as helpful signs for an early diagnosis of pseudoexfoliation syndrome.⁹ I think if these are seen in an eye with greater cataractous changes and lesser pupillary dilation, a most careful slit lamp examination is warranted to rule out pseudoexfoliation.

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Say: "Are the blind and the seeing man equal, or are the darkness and the light equal?"
Holy Quran 13:16

Pseudoexfoliation Syndrome

Robert Ritch, M.D.

One concept presented to us early in medical school was that the world was divided into lumpers and splitters. Lumpers, adherents of the "final common pathway" approach, were oriented toward treatment of specific signs and symptoms. Splitters, on the other hand, tended to be more inquisitive, and believed that further subdivision of diseases and the elucidation of the etiology of disorders at more fundamental levels would lead both to more specific modes of therapy at earlier points of the disease pathway and to more effective methods of disease prevention. Recent history has justified the value of splitting, and the rapid expansion in the past decade of our knowledge in such fields as immunology, molecular genetics, and neurobiology has been accompanied by increasingly rapid application of scientific discoveries to medical care.

Glaucoma is a group of diseases with great potential for further splitting. Less than a half century has passed since the development of gonioscopy allowed accurate differentiation of angle-closure from open-angle glaucomas. Numerous secondary open-angle glaucomas have been described, and primary open-angle glaucoma (POAG) itself is potentially biochemically heterogeneous. As our knowledge of differences in prognosis and responses to treatment between various types of glaucoma continues to be refined, their recognition becomes increasingly important for the institution of proper patient care.

Pseudoexfoliation syndrome (PXS) is the most common of the secondary open-angle glaucomas, and is much more prevalent worldwide than generally perceived.^{1-3,5,9,10,12,29,31,32} From the therapeutic standpoint, it has previously been considered little different from primary open angle glaucoma, thus lessening the impetus for diagnostic accuracy. Its presence is often overlooked clinically, as is so clearly illustrated by Awan's report on pages 93 and 94 of this issue of The Journal. Reported frequencies vary, partly on the basis of case selection and thoroughness of examination. It has been reported to occur in 0% to 29% of the general population over age 60, and in 1.4% to 52% of glaucoma populations.

Pseudoexfoliation syndrome appears to be quite common in Pakistan. Khanzada¹⁶ reported the findings in 696 patients with pseudoexfoliation and found variable prevalences in different tribes and geographic areas, the disease being more common in the mountains than in lower regions. However, the total number of persons examined in each group was not mentioned. Because our knowledge of the prevalence of pseudoexfoliation is so confused, it would be well worthwhile to perform a prospective epidemiologic survey, examining, say, 100 persons in each decade over age 40 in each group in order to determine whether a true difference exists, either on an ethnic or geographic basis. The results should surely be interesting. If a true difference were found, further studies might be carried out to determine the most important risk factors, whether environmental, dietary, or genetic, predisposing to the development of pseudoexfoliation.

Glaucoma occurs more frequently in eyes with pseudoexfoliation than in those without it. In 100 sequential patients in whom pseudoexfoliation was detected as an incidental finding during routine examination, Kozart and Yanoff¹⁷ found 78% to have normal intraocular pressure, 15% to have ocular hypertension, and 7% to have glaucoma. In addition, ocular hypertensives are more likely to develop glaucomatous damage when pseudoexfoliation is present. The 5 and 10-year cumulative probabilities of initially non-glaucomatous eyes with pseudoexfoliation developing glaucoma have been reported as $5.3 \pm 0.1\%$ and $15.4 \pm 2.0\%$, respectively.¹⁵

Primary open angle glaucoma is a bilateral disorder, while pseudoexfoliation with glaucoma frequently occurs unilaterally. This does not, however, rule out the "two hit" theory; i.e., in a patient predisposed to open-angle glaucoma, the appearance of pseudoexfoliation further decompensates the trabecular meshwork. However, patients with pseudoexfoliation responded to corticosteroid testing similarly to the normal population in three studies.^{13,19,30} The 5 and 10-year cumulative probabilities of unilateral pseudoexfoliation becoming bilateral have been reported to be $6.8 \pm 0.3\%$ and $16.8\% \pm 2.2\%$, respectively.¹⁵

Mechanisms proposed to account for the development of glaucoma include primary dysfunction of the trabecular endothelium, outflow obstruction by pigment and/or pseudoexfoliation material (PXM), and/or concomitant

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primary open angle glaucoma.^{11,22,34} Ultrastructural studies of trabecular meshwork in pseudoexfoliation have been inconclusive, and there has been no definitive correlation between the amount of pseudoexfoliation material in the meshwork and severity of glaucoma. Pseudoexfoliation appears to be related to degeneration of elastic fibers in the eye.²⁸ Some investigators have reported a correlation between the severity of glaucoma and the degree of meshwork pigmentation, but this has not been quantitated histologically and patients may have increased pigmentation and normal intraocular pressure. Nevertheless, we find this explanation an appealing one which requires further investigation.

Elevated intraocular pressure, when associated with pseudoexfoliation, usually responds less well to medical therapy and more often requires laser or surgical intervention than does primary open angle glaucoma. Several investigators have found pseudoexfoliation with glaucoma to respond less well to topical timolol therapy. Pseudoexfoliation responds well to argon laser trabeculoplasty, but sudden, late rises in intraocular pressure are seen in some 20% of patients within two years.^{19,24}

The lens is also involved in eyes with pseudoexfoliation.^{7,8,25} Khanzada¹⁶ reported that 70% of his cases had cataracts. It has been our impression that patients with unilateral pseudoexfoliation and unilateral cataract always have the cataract in the eye with pseudoexfoliation. A prospective study to confirm this on an objective basis would be worthwhile.

Lens subluxation is not uncommon, and pseudoexfoliation appears to be a significant risk factor for intraoperative zonular breaks, capsular rupture, and vitreous loss.^{6,14,23,27,33} Spontaneous lens subluxation occurs in as many as 16% of patients with pseudoexfoliation.¹⁸ The importance of preoperative evaluation for weakened zonules in patients with pseudoexfoliation should be stressed.

The diagnosis of pseudoexfoliation syndrome is made by observation of pseudoexfoliation material on the anterior lens capsule and/or pupillary margin. However, there are other clinical signs which have been largely overlooked clinically and which should alert the examiner to search carefully for pseudoexfoliation on slit lamp examination after pupillary dilation.^{4,21,26} These are related to the loss of pigment from the iris pigment epithelium of the sphincter region and its reaccumulation in the outflow pathway. Liberation of iris pigment is evidenced by loss of the pupillary ruff, iris sphincter transillumination defects, and pigment dispersion in the anterior chamber after pupillary dilation. Pigment particles are deposited on the corneal endothelium, anterior iris surface, Schwalbe's line, and trabecular meshwork. Because the mechanism by which glaucoma occurs in pseudoexfoliation may be fundamentally different from that in primary open angle glaucoma, there is

significant potential for the development of alternative methods of prevention and treatment as our understanding of the pathogenesis of this disorder progresses.

In a recent study, we categorized a group of patients as "pseudoexfoliation suspects."²² These patients demonstrated varying degrees of involvement with respect to pigment-related secondary signs of pseudoexfoliation, without evidence of clinical pseudoexfoliation in either eye. These signs included: pupillary ruff defects, iris sphincter transillumination, particulate pigment deposition on the iris surface, increased trabecular meshwork pigmentation, and anterior chamber pigment dispersion after pupillary dilation. In 9 of 26 eyes (34.6%), pseudoexfoliation material was identified in specimens of bulbar conjunctiva examined by transmission electron microscopy. These data suggest that pseudoexfoliation is present in a much greater proportion of the aging population than previously believed and raise a number of interesting questions.

What is the role of pseudoexfoliation in the pathophysiology of glaucoma in these early cases? Is it pathogenetic, and inciting factor in combination with a susceptible outflow pathway, or merely an innocent bystander? The potential importance of pigment dispersion in pathogenesis takes on enhanced meaning. If pseudoexfoliation material can occur in conjunctiva in the absence of clinically visible pseudoexfoliation material on the anterior lens surface, it may also be present in trabecular meshwork and perhaps affect outflow resistance. Would inhibition of pigment liberation, as with miotic therapy, decrease the incidence of glaucoma?

The fact that pseudoexfoliation material may be a pathogenetic factor in many patients diagnosed as having primary open angle glaucoma has great bearing on laboratory investigations of primary open angle glaucoma. Admixture of specimens from eyes with pseudoexfoliation in biochemical and tissue culture studies may lead to anomalous results. At the clinical level, earlier and more accurate diagnosis of exfoliation syndrome may lead to more definitive separation of exfoliation syndrome from primary open angle glaucoma in terms of ocular and systemic correlates and pharmacologic responses. Further research into pseudoexfoliation syndrome and other secondary glaucomas, as well as primary open angle glaucoma, is needed to elucidate many of our unanswered questions.

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Ophthalmic "Pastpourri"

"Pterocephalic Edema"

"Dr. Traquair... has introduced the term "pterocephalic edema," to indicate an edema of the disk due to increased intracranial tension, and thus distinguish it from a disk-edema dependent upon some other cause, for instance, a local optic nerve disease. It will be interesting to observe whether this term secures a permanent place in neuro-ophthalmological literature."

de Schweinitz, GE - 1928

In review on HM Traquair's "An Introduction to Clinical Perimetry."

157-10528



EYE INJURIES. An Illustrated Guide. By Elizabeth M. Eagling and Michael J. Roper-Hall, Philadelphia, J.B. Lippincott Company, 1986. Indexed, hardcover, large size, multiple color figures on each page, 172 pages, U.S. \$67.50.

This book, which is "primarily written for ophthalmologists in training," is the result of the efforts of two consultant ophthalmologists of Birmingham, U.K., and deals with "the early management of ophthalmic injuries." The book departs from the conventional style of serial numbering of pages, and each section has its own independent pagination, giving the book the format of a collection of 10 pamphlets, namely; In the Accident Department, Lacerations of the Face, Eyelids, and Lacrimal Drainage System, Orbital Injuries, Burns to the Eye and Periocular Tissues, Blunt Injuries of the Eye, Penetrating Eye Injuries, The Management of Complicated Injuries: Introduction, The Management of Anterior Segment Complications, The Management of Posterior Segment Complications, and The Management of Late Extraocular Complications.

The right column of each page carries exquisitely beautiful clinical photographs and occasional high quality photomicrographs, and the left column is devoted to written instructions, which at many places are aided by very clear line drawings to better elucidate the pertinent features of the corresponding actual photographs. The duplicate reproductions of several figures, such as 1.1 and 6.37, 1.10 and 6.42, 1.2 and 3.34R, 6.43 and 9.13, 6.63 and 9.16, 6.58 and 9.15 appear unnecessary, despite their excellent quality. There are many figures representing the same feature of preretinal foreign body (Figures 1.1, 6.37, 6.43, 6.57, 6.58, 9.13, and 9.14).

The reconstruction of eyelid from rotational forehead skin flap, cosmetic surgery for severe skin burns of the eyelids, complicated vitrectomy techniques in severely traumatized eyes, etc. perhaps are too esoteric for a book intended for "ophthalmologists in training," but may have appeal for more experienced practitioners. The treatment of lacrimal and orbital trauma limited to author's own approach obviously cannot bring out the different views on these aspects of ocular trauma. Some of the opinions, such as lensectomy as "the treatment of choice" for traumatic cataracts in "younger age group" are certainly not based on universal concepts. This reviewer has very successfully managed such cataracts in younger age group by standard aspiration techniques.

These critical comments do not, however, reduce the value of this beautifully produced book for the trainees, who will find it acceptable and attractive source of learning.

-KJA

PRIMARY EYE CARE IN DEVELOPING NATIONS. By Larry Schwab, Oxford, Oxford University

Press, 1987. Index, 203 pages, soft cover, pocket size, \$15.00.

The author of this pocket size book is a highly trained ophthalmologist with over 10 years of experiences in Ethiopia, Kenya, Malawi, and Zimbabwe. A quote from the preface perhaps best describes the motivation behind this book: "More than 40 million people are blind today, and nearly 75 percent suffer from conditions which are either preventable or curable. Two-thirds of the world's population live in developing nations where most of the blindness is found. These statistics tell a story of the needless tragedy of millions of sightless human beings trapped in a world of poverty and despair."

This easy-to-read 200-page paper back book, written in simple English, has an abundance of drawings and photographs. It is intended for medical assistants, clinical officers, nurses, and general physicians involved in eye care in developing nations.

After reviewing structure and function of the eye (Chapter 1) and diagnostic methods (Chapter 2), The author allocates a chapter to each of the six major blinding eye conditions: cataract, blinding infection, malnutrition and external diseases, glaucoma, eye injuries, and refractive errors. The most interesting chapters are those on related technology (Chapter 10), and on public health ophthalmology (Chapter 11). Chapter 10 is a gold mine of practical ideas and suggestions with relevant illustrations on how to make anything from pinholes to locally constructed cryoextractors.

The most useful chapter is perhaps the one on public health ophthalmology (P.H.O.), a system that integrates prevention of disease with its cure, and is directed at the community rather than exclusively at the individual patient. Most blinding eye diseases can be prevented by public health measures. The author concludes this chapter with a short note on the organization and delivery of eye care, and on conducting surveys.

Six appendices provide practical information on essential ophthalmic drugs and how to use them (Appendix B), on basic surgical instruments for cataract and glaucoma surgery and how to take care of them (Appendix C), on surgical guidelines for ophthalmic medical auxiliaries including surgical indications and pre-operative evaluation (Appendix D), a list of resource organizations (Appendix E), a classification of blindness (Appendix F), a selected bibliography (Appendix G), and a substantial glossary. My suggestion would be to supplement Appendix A (diagnosis of the red eye) with colored photographs.

In summary, this book is valuable to those interested in primary eye care, public health ophthalmology, international ophthalmology, or in the prevention of blindness in the developing regions of the world.

-Reviewed by Jean-Paul Heldt, MD

BOOK REVIEWS

MANAGEMENT OF OCULAR, ORBITAL, AND ADNEXAL TRAUMA. Edited by Thomas C. Spoor and Frank A. Nesi. New York, Raven Press, 1988. Hardcover, 445 pages including index, large number of black and white figures including photographs, tomographs, photomicrographs, and line drawings. Price USA \$88.50.

In addition to the Editors, two dozen other experts have contributed to this book, which is beautifully printed on a very high quality paper. Its multiauthorship apparently reflects the opinion of the Editors that the "successful management of ocular, orbital, and adnexal trauma requires a multidisciplinary approach." The authors have established background in fields of neuroradiology, plastic surgery, otolaryngology and various subspecialties of ophthalmology.

The text is divided into three sections which are further subdivided into many chapters. The Section I on Ocular Trauma includes chapters on The Ruptured Globe: Primary Care, Anterior Segment Trauma, Corneal Trauma, Management of Traumatic Hyphema, Management of Retinal and Vitreous Injuries, The Role of Vitreoretinal Surgery in Ocular Trauma, Endophthalmitis, Antibiotic Usage, Pediatric Ocular Trauma-Amblyopia as a Special Consideration, and Ultrasonography in Ocular Trauma. The Section II on Orbital Trauma contains chapters on Neuro-ophthalmologic Manifestations of Trauma, Radiology of Orbital Trauma, Penetrating Orbital Injuries, Maxillofacial Fractures, Management of Blow-Out Fractures, Orbital Infections, and Orbital Hemorrhage. Eyelid Trauma is the topic of Section III, and is discussed under headings of Basic Management of Soft Tissue Injury, Management of Injuries to the Ocular Adnexa, and Eyelid Burns.

Like many other multiauthored texts, this book also suffers from the unevenness in the quality of writing and the contents of different chapters. However, when taken as a whole, this book is undoubtedly a very useful one. Some chapters, such as the one on maxillofacial fractures, appear redundant for ophthalmologists; whereas, the chapters like the one on antibiotic usage are too concise to be useful in practice or to hold the interest of a reader. The chapter on pediatric ocular trauma does not even consider the management of superficial corneal foreign bodies in children. This unfortunately seems to be a universal shortcoming of books on ocular trauma. The duplication of some material in more than one chapter may prove distracting. Hence, a reader interested in the management of blow-out fracture may become totally disappointed if he seeks information on this in Chapter 14, without realizing that the topic is excellently covered in the next chapter. Some of the chapters are so well-written and are so informative that I would not hesitate to purchase this book just to read those. The chapters on Management of Traumatic Hyphema, Endophthalmitis, and Management of Blow-Out Fractures are of such high quality

that I feel most comfortable in recommending the purchase of this book by anyone interested in ocular trauma.

-KJA

CLINICAL OPHTHALMOLOGY. Edited by Sir Stephen Miller. Bristol, John Wright, IOP Publishing Limited, 1987. Hardcover with dustcover, 578 pages plus index, Illustrated with black and white figures, 17 color plates. Price UKL 65.00.

This book came about as a result of John Wright's request to the Editor to compile a book to help candidates for "postgraduate diplomas and higher degrees" overcome "poor technique in writing answers" and to become knowledgeable about "the particular interests of the examiners." This naturally limits the audience of this very nicely produced book. The Editor was not unaware of the disappointments which may be expected in achieving of such sharply-defined objectives. Also, the personal biases of the super experts on various sub-specialties who are invited to write cannot be avoided from becoming a hindrance to the overall educational value of such a work. The book is strictly intended to fill "a gap in the literature for postgraduates" and the basic and elementary aspects are not given any detail. In completing the task, the Editor also sought the advice of ten most outstanding and international leaders of ophthalmology, including the Consultant Editor of THE JOURNAL, Professor Blodi. The book has 50 contributors, mostly British, but there also are a few from the United States (3), Switzerland (1), Hong Kong (1), and Jamaica (1). The book is divided into 23 chapters on Physiology, Pharmacology and Toxicology, Methods of Examination, External Diseases, The Corneas, Scleritis, The Uveal Tract, The Retina, The Vitreous and its Disorders, The Lens, Glaucoma, The Visual Pathways, The Management of Ocular Tumors, Ocular Injuries, The Lids: Diseases and Treatment, The Lacrimal Drainage Apparatus, Concomitant and Incomitant Squint, Strabismus, Paediatric Ophthalmology, Hereditary Diseases, Immunology and the Eye, Systemic Diseases and the Eye, The Management of Visual Disability, and The Epidemiology of Blindness. Each chapter is supplied with a list of current references at its end.

The writing is variable in style and selection of contents from chapter to chapter. Despite their conciseness, some of the chapters are excellent, conveying great amount of conceptual insight and technical understanding on their perusal, such as chapters on retinal detachment surgery, cataract and lens implant surgery, external diseases, electrodiagnosis, ultrasonography, biochemistry of the eye, scleritis, diseases of the optic nerve, the lids and lacrimal system, and immunology. There too are several deficiencies, such as the chapter on glaucoma providing no discussion on pseudoexfoliation of the lens, and the pseudoexfoliation syndrome having no listing in the index. Many other terms are also missing from the index. In many instances, some of

BOOK REVIEWS

the familiar terms have been replaced by the authors' personal favorite but less known terms, such as "centroserous chorioretinopathy" for the idiopathic central serous chorioretinopathy. This makes it quite difficult to find intended material if one is not familiar with the terms the authors of this book use. It would have been helpful to supply at least in parentheses various other terms, and to also include them in the index. The color plates are excellent clinical material and are produced beautifully; however, one finds it annoying to see them abruptly inserted in the middle of the chapter on glaucoma. This reviewer hopes a more suitable place would be found for them in the future editions.

These remarks do not in any substantial way diminish the great value this book has for its intended audience. I recommend that all candidates aspiring for a postgraduate degree from the British institutions for higher education read this book at least once before appearing in examination.

-KJA

ATLAS OF CLINICAL OPHTHALMOLOGY.

By D.J. Spalton, R.A. Hitchings, and P.A. Hunter. Philadelphia, J.B. Lippincott Company, 1984. Hardcover, 430 pages, index, 1,400 illustrations (1,200 in color) which include photographs, artwork, line drawings, photomicrographs, photobiomicrographs, and histologic sections, extra-large size (10" x 12 1/8"). Price US \$115.00

By any standard this is an extraordinary publication. It is printed with the exquisite beauty on a very high grade glossy paper. The contents consist of 20 independent chapters, or as described by the publishers "Volumes." Each volume covers a specific topic which include Methods of Ocular Examination, The Eyelids, The Conjunctiva: Diseases and Tumors, Infections of the Outer Eye, Allergic Eye Disease: Episcleritis and Scleritis, The Cornea, Primary Glaucoma, Secondary Glaucoma, The Uveal Tract, Intraocular Inflammation, The Lens, Vitreous and Vitreo-Retinal Disorders, The Retina: Normal Anatomy and Physical Signs, The Retina: Vascular Diseases I, Retina: Vascular Diseases II, The Retina: Macular Diseases and Retinal Dystrophies, The Optic Disc, Strabismus, Neuro-Ophthalmology, and The Orbital and Lacrimal System. The format is typical of a quality atlas: each disease or entity is discussed in the left hand column and very high quality clinical color photographs, photomicrographs (where felt necessary), photobiomicrographs, fluorescein angiographs, ultrasonographs, color artwork, and x-rays or computerized tomographs are given in the adjoining portion of the right column to illustrate the features of the disease. In numerous areas a labelled excellent line-sketches depicting the accompanying photographs are also added in the left column to enhance the understanding of the features depicted in the photographs. This plan has left the photographs unmutated

by arrows and markers, preserving the exquisite beauty of the figures.

The contents are concise but highly informative. The goal of the authors was to provide "an introduction to clinical ophthalmology" in the form of "an illustrated reference work." I am pleased to say that the book is an outstanding and rare example of achieving this aim. The book is one of the best teaching tools I have ever come across. Particularly in Pakistan, it will be most sought after book in the libraries of medical schools and ophthalmology departments. Considering what has gone into preparation of this book, price is also very reasonable by modern standards. The publisher also has produced "A Slide Atlas of Ophthalmology" that contains 1,175 slides of figures used in this book. These slides are divided into 20 sets of about 60 slides each which correspond to 20 chapters of this book. This "Slide Atlas" should be in every teaching ophthalmic center, particularly in Pakistan.

The late Dr. Henkind wrote in the Foreword of this book: "This volume should be a required reading for those who know little of ophthalmology and also for those who think they know everything. For the former it will be a foundation stone of inestimable value and for the latter, a touchstone useful for teaching colleagues and patients the nuances of the eye in health and disease." I completely agree with him, and urge every student and scholar of ophthalmology to own this splendid storehouse of ophthalmic knowledge.

-KJA

THE PHYSICIAN AS TEACHER. By Thomas L. Schwenk and Neal Whitman. Baltimore, Williams & Wilkins, 1987. Softcover, pocket-sized, 203 pages including index. Price US \$25.00.

In a newspaper interview, I recently emphasized that every physician has a dual responsibility of treating patients and of sharing what he learns from this with his colleagues. Any physician who is good in the former, but ignores the latter, meets only half of his responsibility as a healer. Moreover, a physician's very calling demands him to be a good teacher, whether he is a professor in a renowned institution, or a country doctor in a remote little town. "The Physician as Teacher" is a much needed tool for every physician or a would-be physician.

The book is a manual that sort of step by step presents the principles and techniques of establishing a fruitful teacher-student relationship. Part One of the book explains the whys and whats of effective teaching, and Part Two deals with teaching under different settings.

I hope someone in Pakistan gets the permission of the publisher to print it in Pakistan and sell at a more affordable price.

-KJA



American Journal of Ophthalmology

A REASSESSMENT OF INFANTILE ESOTROPIA. G.K. von Noorden. Essential infantile esotropia, an early acquired and not a congenital condition, may be helped by congenital factors in its development between the ages of 3 and 6 months. It is to be distinguished from other forms of esotropia with an onset between birth and the first six months of life. The cause remains unknown, but advances in our knowledge are expected from the rapidly emerging discipline of infant psychophysics. A clear distinction must be made between normal, subnormal, and anomalous forms of binocular cooperation in analyzing treatment results. A complete restoration of normal binocular function is rarely, if ever, achieved. However, anomalous binocular cooperation has many functional advantages over suppression or diplopia and should not be disturbed by overzealous treatment. Subnormal binocular vision, microtropia, and a residual small angle heterotropia are acceptable results of surgical therapy. The author studied 358 surgically treated patients with a documented onset of essential infantile esotropia before age 6 months and found that subnormal binocular vision was present in 71 (20%), a microtropia in 25 (7%), and a small angle esotropia or exotropia in 140 (39%) of the patients. Surgical alignment before completion of the second year of life improved the chances for an optimal treatment result. However, surgical treatment after the age of 2 or even 4 years did not preclude the development of binocularity on a subnormal or anomalous basis in many patients. (*Am J Ophthalmol* 105:1-10, January, 1988.) Reprint requests to G.K. von Noorden, M.D., Ophthalmology Service, Texas Children's Hospital, Box 20269, Houston, TX 77225.

ROLE OF THE VITREOUS IN BRANCH RETINAL VEIN OCCLUSION. M Kado, CL Trempe. The authors analyzed the vitreous findings in 62 patients (62 eyes) with major branch retinal vein occlusion to determine whether vitreous examination was useful in predicting the development of retinal or disk neovascularization, or both. In 18 eyes with no or partial posterior vitreous detachment and large areas of capillary nonperfusion (5

disk diameters or more), ten eyes (55.6%) eventually developed neovascularization. Only three of the remaining 44 eyes (6.8%) developed neovascularization. The probability of this development was greater in eyes with no or partial posterior vitreous detachment at the initial vitreous examination ($P = .0177$, Cox's regression analysis), and in those with large areas of nonperfusion ($P = .0097$, Cox's regression analysis). (*Am J Ophthalmol* 105:20-24, January, 1988) Reprint requests to Library, Eye Research Institute, 20 Stanford St., Boston, MA 02114.

VITREOUS SURGERY FOR HEMORRHAGIC AND FIBROUS COMPLICATIONS OF AGE-RELATED MACULAR DEGENERATION. E de Juan, Jr, R Machemer. The authors applied vitreous surgical techniques in the treatment of hemorrhagic and fibrous complications of choroidal neovascular membranes by removing subretinal scars or hemorrhage, or both, in four patients. The surgical goals were achieved in all patients with visual acuity improvement in three. The major complication was recurrent detachment association with large retinotomies. (*Am J Ophthalmol* 105:25-29, January, 1988). Reprint requests to Eugene de Juan, Jr., M.D. Duke University Eye Center, Box 3802-200, Durham, NC 27710.

THE SURGICAL TREATMENT OF BLEPHAROPTOSIS IN OCULOMOTOR NERVE PALSY. TJ Malone, A Nerad. The authors reviewed retrospectively 170 cases of congenital and acquired oculomotor nerve palsy to determine the effectiveness of surgery in the treatment of blepharoptosis in oculomotor nerve palsy. Twenty patients had had surgical repair of the blepharoptosis. Patients with congenital oculomotor palsy were more often selected for blepharoptosis surgery than patients with acquired blepharoptosis because of the absence of diplopia caused by suppression in congenital cases. There are complete recovery in 72 of 109 patients (66%) with acquired oculomotor palsy. Results were judged with regard to functional improvement and cosmetic improvement. Primary functional disability was relieved by lifting the eyelid above the pupil in ten of 12 patients (83%). Primary cosmetic disability was relieved in six of six patients. Corneal complications occurred in six of 20 patients (30%) who were treated surgically. (*Am J Ophthalmol* 105:57-64, January, 1988). Reprint requests to Jeffrey A. Nerad, M.D., Department of Ophthalmology, University of Iowa Hospitals and Clinics, Iowa City, IA 52242.

LINEAR SUBCUTANEOUS FAT ATROPHY AFTER CORTICOSTEROID INJECTION OF PERIOCCULAR HEMANGIOMAS. PJ Droste, D Ellis, N Sondhi, M. Helveston. The authors report two children who developed evidence of subcutaneous fat atrophy after corticosteroid injection of periocular hemangiomas. The atrophy appeared at the site of injection and followed the expected course of lymphatic channels to the

vicinity of regional lymph nodes. (*Am J Ophthalmol* 105:65-69 January, 1988). Reprint requests to Forrest D. Ellis, M.D., 702 Rotary Circle, Indianapolis, IN 46223.

SOLAR RETINOPATHY IN PERSONS ON RELIGIOUS PILGRIMAGE. GC Cangelosi, DA Newsome. Six children in Medjugorje, Yugoslavia reported witnessing an apparition of Mary, the Mother of Jesus. This event has stimulated worldwide pilgrimages to Medjugorje to witness the phenomena associated with the apparition, such as spinning, jumping, and pulsating movements of the sun. The sun has also been reported to emit various colors, and is occasionally covered by a dark disk. Anecdotal reports of prolonged sungazing without noticeable visual effects have appeared in the local media of Medjugorje. The authors examined three patients who developed various degrees of solar retinopathy after sungazing during a pilgrimage to Medjugorje. Two of our three patients recovered vision fully, but the third had what appeared to be, after nine months of follow-up, permanent visual loss (Figure). All patients were prescribed 200 mg of ibuprofen twice daily and 1% prednisolone acetate four times daily for periods of one to four months. One patient also received 40 mg of oral corticosteroids for one week. (*Am J Ophthalmol* 105:95-96, 1988). Inquiries to Glen C. Cangelosi, M.D., 4201 Frenchman St., New Orleans, LA 70122.

EXCIMER LASER KERATECTOMY FOR CORRECTION OF ASTIGMATISM. T Seiler, T Bende, J Wollensak, S Trokel. The authors treated 13 eyes of 12 patients with excimer laser surgery for correction of astigmatism using linear corneal T-excision. All eyes were followed up for a minimum of three months. We used a newly developed delivery system and special contact masks to deliver the 193-nm excimer light. Astigmatic corrections of up to 4.16 diopters were obtained. The actual corrections corresponded well with the intended values as predicted by a biomechanical theory. The refractive change over time was different than that observed after knife incisions, suggesting different repair mechanisms. An epithelial plug filling the whole T-excision persisted for over one year in all eyes. (*Am J Ophthalmol* 1105:117-124, February, 1988). Reprint requests to Theo Seiler, M.D., Universitätsaugenlinik im Klinikum Charlottenburg, Spandauer Damm 130, 1000 Berlin 19, West German.

LOW-CONTRAST LETTER CHARTS TO DETECT SUBTLE NEUROPATHIES. MD Drucker, J Savino, C Sergott, M Bosley, NJ Schatz, PS Kubilis. The authors performed visual contrast sensitivity testing with Regan's low-contrast letter charts on 30 patients with optic neuropathies and good Snellen visual acuity. Results of this testing were compared with color vision and the presence of an afferent pupillary defect. Regan's low-contrast letter charts were found to be 93% sensitive for detecting subtle optic neuropathies, with the apparent false-negative findings being readily

explainable. Color vision testing was abnormal in only 49% of eyes with known optic neuropathies. (*Am J Ophthalmol* 105:141-145, February, 1988). Reprint requests to Peter J. Savino, M.D., Wills Eye Hospital, 9th & Walnut Sts., Philadelphia, PA 19107.

SUSTAINED REDUCTION OF INTRAOCULAR PRESSURE IN HUMANS WITH THE CALCIUM CHANNEL BLOCKER VERAPAMIL. MB Abelson, CM Gilbert, LM Smith. The authors investigated the effect of the calcium channel blocker verapamil on intraocular pressure in human volunteers. In the initial trial, 15 subjects with untreated ocular hypertension were tested. After a baseline measurement was obtained with applanation tonometry, a 40- μ l drop of verapamil, 1.25 mg/ml, was instilled in one eye. After 30 minutes, a second reading was taken. In a subsequent trial of 12 untreated ocular hypertensive subjects, the duration of action was determined using the same dose and method of delivery. Results showed that verapamil elicited a mean \pm S.E.M. change in intraocular pressure of -3.8 ± 0.900 mm Hg in the treated eye, and -1.6 ± 0.400 mm Hg in the untreated eye. This reduction was statistically different in both eyes (treated eye, $P = .007$; untreated eye, $P = .005$). This decrease in intraocular pressure remained statistically significant when compared to predrug baseline values for up to ten hours. (*Am J Ophthalmol* 105:155-159, February 1988). Reprint requests to Mark B. Abelson, M.D., Eye Research Institute, 20 Staniford St., Boston, MA 02114.

NUCLEAR SCLEROSIS AFTER VITRECTOMY FOR IDIOPATHIC EPIRETINAL MEMBRANES. S de Bustros, T Thompson, RG Michels, C Enger, TA Rice, MB Glaser. The authors performed a retrospective analysis of lens changes occurring after vitrectomy for idiopathic epiretinal membranes causing macular pucker on 75 consecutive phakic eyes. Thirty-five eyes (47%) had appearance or progression of nuclear sclerosis. There were no changes in anterior subcapsular cataracts and three eyes (4%) had minimal changes in posterior subcapsular cataracts. Based on univariate analysis, presence of preoperative nuclear sclerosis, length of follow-up period, and surgeon were found to be associated with the occurrence or progression of nuclear sclerosis. Differences between surgeons were largely accounted for by the difference in proportions with preoperative nuclear sclerosis, and differing lengths of follow-up. Kaplan-Meier life table analysis performed on 53 eyes with examination of fellow eyes and longer follow-up showed significantly more nuclear sclerosis in the operated on eye when compared to the fellow eye (log-rank test, $P < .0001$). (*Am J Ophthalmol* 105:160-164, February, 1988). Reprint requests to Serge de Bustros, M.D., Maumenee 115, 600 N. Wolfe St., Baltimore, MD 21205.

PATHOGENESIS AND THERAPY OF TRACTION DETACHMENT IN VARIOUS RETINAL VASCULAR DISEASES. R Machemer, JM Williams, Sr. The authors examined six patients with retinal vasculopathies (two cases each of angiomatosis retinae [von Hippel-Lindau disease], exudative vitreoretinopathy, and Coats' disease), who developed preretinal and vitreal membranes, retinal traction detachment, and exudation under the retina. The membranes consisted of a core of collagen of various diameter fibres usually covered on both sides by cells that displayed glial cell characteristics. The retina reattached spontaneously and exudates disappeared when traction was eliminated by vitreous surgery. Membrane formation did reoccur after vitrectomy if the vessels continued to leak, leading again to traction and subretinal exudation. Treatment consisted of surgical removal of vitreal and preretinal membranes and destruction of leaking vessels. (*Am J Ophthalmol* 105:170-181, February 1988). Reprint requests to Robert Machemer, M.D., Duke University Eye Center, Box 3802, Durham, NC 27710.

BACILLUS KERATITIS ASSOCIATED WITH CONTAMINATED CONTACT LENS CARE SYSTEMS. PB Donzis, BJ Mondino, BA Weissman. The authors examined two soft contact lens wearers who developed keratitis associated with *Bacillus* contamination of their contact lens care systems. Patient 1 developed a corneal ulcer caused by *B. subtilis*, and Patient 2 demonstrated multiple, diffuse, punctate corneal epithelial opacities associated with *B. cereus* contamination in the contact lens and lens case compartment. The contact lens cases of both patients contained *Bacillus* spores that survived multiple heat disinfection treatments. Three different contact lens chemical disinfection systems used of the minimum recommended time failed to kill the *Bacillus* organisms. (*Am J Ophthalmol* 105:195-197, February, 1988). Reprint requests to Bartly J. Mondino, M.D., Jules Stein Eye Institute, 800 Westwood Plaza, Los Angeles, CA 90024.

ALDOSE REDUCTASE INHIBITOR (CT-112) EYEDROPS FOR DIABETIC CORNEAL EPITHELIOPATHY. Y Ohashi, M Matsuda, H Hosotani, Y Tano, I Ishimoto, M Fukuda, R Manabe. The authors treated two diabetic patients with corneal epithelial disorder that resisted conventional medical therapy with topical CT-112 (5-[3-ethoxy-4-pentyloxyphenyl]-2,4-thiazolidinedione), a newly synthesized aldose reductase inhibitor. One patient had developed recurrent corneal erosion after vitrectomy and the other had spontaneously developed superficial punctate keratopathy. The corneal lesion in each patient responded to topical CT-112 in two to four weeks and was almost cleared within two months. A similar corneal lesion recurred in both patients soon after CT-112 was discontinued, but it disappeared again when the drug was resumed. (*Am J Ophthalmol* 105:233-

238, March, 1988). Reprint requests to Yuichi Ohashi, M.D., Division of Ophthalmology, Kansai Rosai Hospital, 3-1-69 Inabaso, Nagasaki, Hyogo 660, Japan.

FORTIFIED ANTIBIOTIC OINTMENT IN BACTERIAL KERATITIS. RA Hyndiuk, DN Skorich, SD Davis, LD Sarff, K Divine, E Burd. The authors conducted experiments to determine the effectiveness of fortified antibiotic ointment in the treatment of *Pseudomonas* keratitis in rabbits. We evaluated gentamicin ointment (3, 10, 20, and 40 mg/g), gentamicin solution (3 and 10 mg/ml), and placebo, each given every 30 minutes. We also examined the effectiveness of fortified ointment given in extended treatment intervals. In short-term trials, commercial-strength gentamicin solution (3 mg/ml) was therapeutically superior ($P < .001$) to commercial-strength gentamicin ointment (3 mg/g) in reducing corneal bacterial colony counts. No significant difference in antimicrobial effect was noted between fortified gentamicin ointment and fortified gentamicin solution at 30-minute treatment intervals. Fortified gentamicin ointment reduced colony counts even at extended treatment intervals of up to four hours in a severe keratitis model. (*Am J Ophthalmol* 105:239-243, March, 1988). Reprint requests to Robert Hyndiuk, M.D., Eye Institute, 4th floor, 8700 W. Wisconsin Ave., Milwaukee, WI 53226.

USE OF SPLIT-THICKNESS DERMAL GRAFT IN THE SURGICAL TREATMENT OF CORNEAL AND SCLERAL DEFECTS. JA Mauriello, Jr, PM Fiore, S Pokorny, DJ Cinotti. The authors used split-thickness dermal grafts for the surgical treatment of corneal and scleral perforations in two patients and obtained excellent results. Patient 1 had severe lye burns and bilateral corneal perforations and Patient 2 had scleromalacia perforans. The dermal graft self-epithelializes and, thus, does not need to be covered by conjunctiva; is supple, without the bulkiness of other materials, particularly cartilage and periosteum; is hearty and flourishes on avascular surfaces such as cornea and sclera; has good tensile strength; and is autogenous. (*Am J Ophthalmol* 105:244-247, March, 1988). Reprint requests to Joseph A. Mauriello, Jr., M.D., 15 S. Ninth St., Newark, NJ 07107.

LONG-TERM CORNEAL ENDOTHELIAL CHANGES AFTER INTRAOCULAR LENS IMPLANTATION. M Matsuda, K Miyake, M Inaba. The authors studied the morphologic characteristics of the corneal endothelium in a series of patients who had undergone phacoemulsification with intraocular lens implantation performed by one surgeon. Specular microscopy and computer-assisted morphometry were performed preoperatively and three years after surgery. Nineteen eyes that received posterior chamber lenses with intracapsular fixation had a mean endothelial cell loss of 18.1%, without any significant change in cell size (polymegethism) or shape variability (pleomorphism). Implantation of anterior

chamber lenses with the posterior capsule left intact (18 eyes) caused a similar degree of cell loss (23.5%) but caused marked polymegathism and pleomorphism of the cells. Endothelial cell loss (28.5%) and morphologic changes were greatest in five eyes that received anterior chamber lenses because of a rupture of the posterior capsule. (*Am J Ophthalmol* 105:248-252, March, 1988). Reprint requests to Mamoru Matsuda, M.D., Department of Ophthalmology, Osaka University Medical School, 1-1-50 Fukushima, Fukushima-ku, Osaka 553, Japan.

PREDICTORS OF SCLERAL RUPTURE AND THE ROLE OF VITRECTOMY IN SEVERE BLUNT OCULAR TRAUMA. SR Russell, KR Olsen, JC Folk. The authors reviewed retrospectively 40 eyes that had received blunt trauma and had been explored for scleral rupture. Twenty-nine eyes had scleral rupture. Of these 29, ten had ruptures seen preoperatively. Nineteen had occult ruptures. The preoperative findings predictive of scleral rupture were a visual acuity of light perception or no light perception, an intraocular pressure of less than 10 mm Hg, hyphema, and chemosis. Of the 29 ruptures, 27 involved the superior hemisphere and 25 involved the anterior hemisphere of the globe. Ten of 29 eyes (34%) with scleral rupture and eight of 11 eyes (73%) without rupture achieved a final visual acuity of 5/200 or better over an average follow-up period of 6.7 months. Factors prognostic of ambulatory vision for eyes with ruptured and intact globes included an initial visual acuity of 5/200 or better, absence of scleral rupture, and a rupture length of less than 11 mm in eyes with ruptures. The vitrectomized eyes also had a better result, suggesting that early pars plana vitrectomy is of benefit in selected rupture cases. (*Am J Ophthalmol* 105:253-257, March, 1988). Reprint requests to Karl R. Olsen, M.D., Bascom Palmer Eye Institute, P.O. Box 016880, Miami, FL 33101.

ENLARGEMENT OF THE BLIND SPOT CAUSED BY PAPILLEDEMA. JJ Corbett, DM Jacobson, RC Mauer, HS Thompson. Blind spot enlargement in papilledema has been attributed to either mechanical disruption of the integrity of the peripapillary percipient elements by the swollen optic disk or to the Stiles-Crawford effect. The authors investigated the possibility that blind spot enlargement in papilledema is caused, at least in part, by a refractive scotoma due to peripapillary hyperopia. They reduced the enlarged blind spot in a patient with focal peripapillary hyperopia, without papilledema, to near normal size by using progressively stronger plus lenses. Similarly, with the addition of plus sphere, they reduced the size of the blind spot in five of six patients with papilledema, but in none of our normal subjects. (*Am J Ophthalmol* 105:261-265, March, 1988). Reprint requests to James J. Corbett, M.D., Department of Neurology, University of Iowa Hospitals and Clinics, Iowa City, IA 52242.

THE PATHOGENESIS OF TEARS OF THE RETINAL PIGMENT EPITHELIUM. EL Chuang, AC Bird. The authors compared drusen in the fellow eye of patients with unilateral retinal pigment epithelial tears with those in an age- and sex-matched group of patients with unilateral primary neovascular disciform lesions. In the fellow eye of patients with tears, drusen were more confluent and manifested less fluorescence on angiography than in the comparison group. These observations are in accord with the concept that Bruch's membrane represents a significant barrier to fluid flow, and that fluid beneath detached pigment epithelium is derived in part or wholly from the pigment epithelium. (*Am J Ophthalmol* 105:285-290, March, 1988). Reprint requests to Elaine L. Chuang, M.D., Bascom Palmer Eye Institute, 900 N.W. 17th St., Miami, FL 33134.

A LONG-TERM FOLLOW-UP STUDY OF LASER COAGULATION OF NEOVASCULAR MEMBRANES IN ANGIOID STREAKS. O Gelissen, F Hendrikse, AF Deutman. The authors treated 30 eyes of 24 patients with angioid streaks and neovascular membranes using light coagulation. Of 30 eyes, 16 showed either ameliorated or unchanged visual acuity. Twelve of the remaining 14 eyes retained a visual acuity of 20/200 or better. In 11 patients, the fellow untreated eye showed central macular degeneration with loss of central vision. The follow-up period ranged from two months to 16 years (mean, 3.4 years). (*Am J Ophthalmol* 105:299-303, March, 1988). Reprint requests to F. Hendrikse, M.D., Institute of Ophthalmology, University of Nijmegen, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands.

NEGATIVE ANTIBODY RESPONSE TO LONG-TERM TREATMENT OF FACIAL SPASM WITH BOTULINUM TOXIN. RS Gonnering. Over a period of 163 weeks, the author administered 223 injections of botulinum A toxin to 38 patients with facial spasm syndromes. The maximum cumulative toxin dose was 553 units, the maximum number of injections in any given patient was 16, and the maximum dosage of any given injection was 52.5 units. Sera from these patients showed no antibody production when measured with a standard mouse lethality bioassay. (*Am J Ophthalmol* 105:313-315, March, 1988). Reprint requests to Russell S. Gonnering, M.D., 2600 N. Mayfair Rd., Milwaukee, WI 53226.

IMAGING OF CEREBRAL BLOOD FLOW AND METABOLISM IN AMBLYOPIA BY POSITRON EMISSION TOMOGRAPHY. JL Demer, GK von Noorden, ND Volkow, KL Gould. The authors used positron emission tomography to study monocular visual activation of various brain regions in four amblyopic and two normally sighted adults. Imaging of relative cerebral blood flow using the tracer H₂¹⁵O

showed reduced activation of primary visual cortex by the amblyopic as compared with the sound eye. Imaging of relative cerebral glucose metabolism using the tracer [^{18}F]-2-deoxyglucose showed equal activation of primary visual cortex by either eye in the control subject, but reduced activation of primary and accessory visual cortex by the amblyopic as compared with the sound eye in two amblyopic subjects. Relative glucose metabolism was consistently higher in the frontal and temporal lobes contralateral to the viewing eye, both in normal and amblyopic subjects. (*Am J Ophthalmol* 105:337-347, April, 1988). Reprint requests to Joseph L. Demer, M.D., Cullen Eye Institute, Baylor College of Medicine, 6501 Fannin St., NC-200 Houston, TX 77030.

PENETRATING KERATOPLASTY FOR PSEUDOPHAKIC BULLOUS KERATOPATHY AFTER EXTRACAPSULAR CATARACT EXTRACTION. SB Koenig, RO Schultz. The authors studied 17 women who underwent simple penetrating keratoplasty for pseudophakic bullous keratopathy after extracapsular cataract extraction. Corneal edema occurred an average of eight months after cataract surgery (range, zero to 32 months). Of 17 corneal grafts, 16 (94%) have remained clear during an average follow-up period of 14 months (range, two to 32 months). One eye had a nonimmunologic graft failure. Of 16 eyes with clear grafts, 14 (87%) achieved a visual acuity of 20/40 or better after surgery. Two eyes with a visual acuity of 20/50 and 20/200 had opacified posterior capsules and one demonstrated age-related macular degeneration. One eye with a postoperative visual acuity of 20/40 demonstrated cystoid macular edema. Fifteen of 17 contralateral eyes showed slit-lamp evidence of endothelial dystrophy. (*Am J Ophthalmol* 105:348-353, April, 1988). Reprint requests to Steven B. Koenig, M.D., 8700 W. Wisconsin Ave., Milwaukee, WI 53226.

MICROBIAL KERATITIS ASSOCIATED WITH CONTAMINATED OCULAR MEDICATIONS. OD Schein, PJ Wasson, SA Boruchoff, KR Kenyon. The authors studied seven cases of severe gram-negative microbial keratitis associated with the use of contaminated topical ocular medications. Five cases involved *Pseudomonas aeruginosa*, one involved *Serratia marcescens*, and one involved *Proteus mirabilis*. In each case the same organism was cultured from corneal scrapings and from the medication. Either prednisolone acetate (one case) or timolol maleate (seven cases) was implicated in all instances. (*Am J Ophthalmol* 105:361-365, April, 1988). Reprint requests to Oliver D. Schein, M.D., Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston, MA 02114.

OCULAR INVOLVEMENT IN MYCOTIC SINUSITIS CAUSED BY BIPOLARIS. WM Jay, RW Bradsher, B LeMay, N Snyderman, EJ

Angtuaco. The authors examined two patients with unilateral ophthalmologic findings secondary to pansinusitis caused by *Bipolaris*. Both patients were healthy young men. One patient had a gradual visual loss, whereas the other showed proptosis. Surgical debridement was the primary treatment in both patients. One patient received antifungal therapy, whereas the other was cured with surgery alone. (*Am J Ophthalmol* 105:366-370, April, 1988). Reprint requests to Walter M. Jay, M.D., Department of Ophthalmology, University of Arkansas for Medical Sciences, Mail Slot #523, 4301 W. Markham, Little Rock, AR 72205.

VITRECTOMY FOR IMPENDING IDIOPATHIC MACULAR HOLES. WE Smiddy, RG Michels, BM Glaser, S de Bustros. The authors performed pars plana vitrectomy in 15 patients with physical changes and visual loss believed to indicate impending macular hole formation. Twelve (80%) of 15 eyes have not progressed to macular holes during a minimum follow-up period of 15 months (average, 26 months). In these 12 patients final vision was within one line of the preoperative vision in four eyes, improved two or more lines in five eyes, and was two lines worse in three eyes. Decreased vision in all three eyes was the result of progressive lenticular nuclear sclerosis. A thin sheet of cortical vitreous fibers was sometimes found intraoperatively on the surface of the retina, despite an apparent complete posterior vitreous detachment. (*Am J Ophthalmol* 105:371-376, April, 1988). Reprint requests to Ronald G. Michels, M.D., Maumenee 127, 600 Wolfe St., Baltimore, MD 21205.

PERIPHERAL RETINAL CRYOPEXY FOR SUBTOTAL VITREOUS HEMORRHAGE. WH Ross, MJ Gottner. The authors used peripheral retinal cryopexy to treat 20 eyes in 15 patients with subtotal vitreous hemorrhage secondary to proliferative diabetic retinopathy. In 18 eyes, complete panretinal photocoagulation had been performed before the subtotal vitreous hemorrhage and the subsequent cryopexy. The length of follow-up averaged 16 months. The vitreous hemorrhage completely cleared in 11 eyes (55%) and partially cleared in six eyes (30%). Visual acuity after treatment improved in 13 eyes (65%), remained unchanged in six eyes (30%), and decreased in one eye (5%). (*Am J Ophthalmol* 105:377-382, April, 1988). Reprint requests to William H. Ross, M.D., Retina Service, Department of Ophthalmology, St. Paul's Hospital, 1081 Burrard St., Vancouver, BC, Canada V6Z 1Y6.

EFFECTS OF ARGON LASER IRIDOTOMY ON THE CORNEAL ENDOTHELIUM. WC Panek, DA Lee, RE Christensen. The authors studied 14 eyes of 14 patients undergoing argon laser peripheral iridotomy for occludable (narrow) anterior chamber angles. Preoperative and postoperative pachymetry and corneal endothelial cell counts by specular microscopy were obtained centrally and in both the treated and nontreated superior

quadrants. No significant differences were found between preoperative and postoperative measurements of corneal thickness and endothelial cell counts in any area. (*Am J Ophthalmol* 105:395-397, April, 1988). Reprint requests to David A. Lee, M.D., Glaucoma Division, Jules Stein Eye Institute, 800 Westwood Plaza, Los Angeles, CA 90024-1771.

EFFECTS OF YELLOW FILTER GLASSES ON THE RESULTS OF PHOTOPIC AND SCOTOPIC PHOTOMETRY. EA Aarnisalo. The author measured the luminosity of a white surface with seven different yellow filter glasses and a photometer with two different sensitivities, which resembled the photopic and scotopic sensitivity of a Commission Internationale d'Eclairage standard observer. When measured with filters GG 400, GG 420, GG 435, and GG 455, there was a small and almost equal reduction in the relative photopic and scotopic luminosities. Measured with filters GG 475, GG 495, and OG 515, there was a more marked reduction in the scotopic luminosities than in the photopic luminosities. (*Am J Ophthalmol* 105:408-411, April, 1988). Reprint requests to Eero A. Aarnisalo, M.D., Department of Ophthalmology, Satakunta Central Hospital, SF-28500, Pori, Finland.

OPTIC NERVE SWELLING SECONDARY TO THE OBSTRUCTIVE SLEEP APNEA SYNDROME. FA Bucci, Jr., GB Krohel. The obstructive sleep apnea syndrome is characterized by an interruption of normal breathing during sleep secondary to airway obstruction below the nasopharynx. The diagnosis is confirmed by polysomnography, which documents period of apnea during sleep. The authors recently treated a 46-year-old man with obstructive sleep apnea syndrome who developed secondary disk edema. The disk edema resolved after therapeutic tracheostomy. (*Am J Ophthalmol* 105:428-430, April, 1988). Inquiries to Gregory B. Krohel, M.D., Department of Ophthalmology, Albany Medical College, Albany, NY 12208.

MONOCULAR DIPLOPIA ACCOMPANYING ORDINARY REFRACTIVE ERRORS. P Coffeen, DL Guyton. The authors discovered that monocular diplopia could be induced in nine (82%) of 11 normal eyes with ordinary spherical or astigmatic defocus of the retinal image. Possible mechanisms responsible for this effect include retinal processing, diffraction effects, and spherical aberration. By employing geometric blur circle theory and using a simple optical model to photography the effect, we concluded that monocular diplopia in the setting of ordinary refractive error is secondary to relatively minor optical irregularity such as spherical aberration. Contour enhancement properties of the retina probably accentuate this effect. Ordinary refractive error should therefore not be overlooked or discounted in patients with monocular diplopia. (*Am J Ophthalmol* 105:451-459, May, 1988).

Reprint requests to David L. Guyton, M.D., Wilmer B1-35, Johns Hopkins Hospital, Baltimore, MD 21205.

A CLINICAL INDEX FOR PREDICTING VISUAL ACUITY AFTER CATARACT SURGERY. MJ Graney, WB Applegate, ST Miller, JT Elam, JM Freeman, TO Wood, TC Gettlefinger. The authors developed a clinical index for predicting postoperative visual acuity of cataract patients and cross-validated it using data from 182 patients aged 70 years and older. The index consisted of four statistically combined indicators: age, preoperative visual acuity, frequency of reading, and comorbidity. Validation of the index included comparisons to two standard technical instruments for measurement of retinal visual acuity. For the clinical index, 72% of predictions were accurate within one Snellen line of postoperative visual acuity compared to 37% using a laser interferometer and 33% using a potential acuity meter. Testing of the clinical index's external validity using data from 111 patients in a different ophthalmology clinic disclosed 61% of predictions accurate within one Snellen line. (*Am J Ophthalmol* 105:460-465, May, 1988). Reprint requests to Marshall J. Graney, Ph.D., 340 Library/Nursing Building, 877 Madison Ave., Memphis, TN 38163.

INCREASED INTRAOCULAR PRESSURE IN THE IMMEDIATE POSTOPERATIVE PERIOD AFTER EXTRACAPSULAR CATARACT EXTRACTION. JG Gross, DR Meyer, AL Robin, AA Filar, JS Kelley. The authors evaluated the immediate intraocular pressure increase after extracapsular cataract surgery in 58 eyes. Two to three hours postoperatively, 34 eyes (59%) developed an intraocular pressure greater than or equal to 25 mm Hg. We found an intraocular pressure of at least 40 mm Hg in seven eyes. Mean intraocular pressure two to three hours postoperatively was 8.9 ± 9.8 mm Hg greater than the preoperative intraocular pressure. The use of intraocular sodium hyaluronate had no apparent effect on intraocular pressure. (*Am J Ophthalmol* 105:466-469, May, 1988). Reprint requests to Alan L. Robin, M.D., Wilmer Institute, Maumenee Bldg., Room B-117, Johns Hopkins Hospital, 600 N. Wolfe St., Baltimore, MD 21205.

CLINICAL FINDINGS AND COMMON SYMPTOMS IN RETINITIS PIGMENTOSA. JR Heckenlively, SL Yoser, LH Friedman, JJ Oversier. The authors performed data analysis in a prospective study of clinical symptoms and findings in 500 patients with retinitis pigmentosa. The symptoms and findings in these patients met the usual definitions of the disease. At initial examination the patients were questioned in a standardized manner; symptoms and associated health problems were reviewed. Some patients were unable to answer all of the questions. Of the patients 274 (55%) were men and 226 (45%) were women, with a race distribution of 21 (4%) black, 47 (9%) Hispanic, 26 (5%) Oriental, three

(1%) American Indian, and 403 (81%) white. Sixty-nine patients reported no symptoms of night blindness and 116 patients claimed no visual field changes; 90 stated that they saw better at dusk. The most common problem noted by 263 (53.3%) was headaches, 31 on a daily basis, 42 at least weekly, 124 infrequently, and the remainder nonspecifically. Numbness or tingling, mainly in extremities, was reported by 99 patients. The second most common problem affecting 170 patients (34.6%) was light flashes; since eight patients had retinal detachments, light flashes cannot be totally discounted. Of 143 patients who had been pregnant, 14 had visual changes. (*Am J Ophthalmol* 105:504-511, May, 1988). Reprint requests to John R. Heckenlively, M.D., Jules Stein Eye Institute, 800 Westwood Plaza, Los Angeles, CA 90024.

PRESUMED AUTOIMMUNE CORNEAL ENDOTHELIOPATHY. RH Paul. The author reviewed 20 previously published cases of presumed autoimmune corneal endotheliopathy. The disease appeared clinically with stromal edema and a slowly migrating line of keratic precipitates. All patients had acute stromal edema and keratic precipitates, and decreased visual acuity. Anterior chamber cells were noted in 11 patients. Inflammatory processes, such as pars planitis and iritis, and intraocular lens implantation were present in 13 patients. (*Am J Ophthalmol* 105:519-522, May, 1988). Reprint requests to Randal H. Paul, M.D., Department of Ophthalmology, Duke University Medical Center, Box 3802, Durham, NC 27710.

VITAMIN A EYEDROPS FOR SUPERIOR LIMBIC KERATOCONJUNCTIVITIS. Y Ohashi, H Watanabe, S Kinoshita, H Hosotani, M Umemoto, R Manabe. The authors treated 12 patients with superior limbic keratoconjunctivitis with topical vitamin A (retinol palmitate) eyedrops. After a follow-up period of at least three months, this therapy was found to be effective, to a varying extent, in ten patients (83%). Superior limbic keratoconjunctivitis lesions did not recur in these patients as long as topical application was continued. (*Am J Ophthalmol* 105:523-527, May, 1988). Reprint requests to Yuichi Ohashi, M.D., Division of Ophthalmology, Kansai Rosai Hospital, 3-1-69 Inabaso, Amagasaki, Hyogo 660, Japan.

BILATERAL ANTERIOR UVEITIS AND INTERSTITIAL NEPHRITIS. JT Rosenbaum. The author reports on five patients had bilateral anterior uveitis associated with renal disease. Interstitial nephritis was histologically confirmed in three cases. Fatigue, anorexia, abdominal pain, weight loss, and anemia were common systemic complaints or findings. In each case, the uveitis was anterior, eventually bilateral, and associated with minimal visual impairment. Complications of the uveitis included increased intraocular pressure, synechiae, keratic precipitates, macular edema, and intraretinal hemorrhage as

well as cells in the anterior vitreous humor and an exudate over the pars plana. Sjogren's syndrome, systemic lupus erythematosus, sarcoid, and syphilis were excluded as diagnoses that could explain the association of renal and uveal disease. (*Am J Ophthalmol* 105:534-537, May, 1988). Reprint requests to James T. Rosenbaum, M.D., Department of Ophthalmology, L329A, Oregon Health Sciences University, Portland, OR 97201.

QUANTITATION OF TUMOR SEEDING FROM FINE NEEDLE ASPIRATION OF OCULAR MELANOMAS. BJ Glasgow, HH Brown, AM Zargoza, RY Foos. The authors performed 25 fine needle (30 gauge) aspirations in eyes enucleated for the clinical diagnosis of melanoma. Cytologic preparations were evaluated for adequacy of material, and needle tracts were evaluated for tumor implantation. A scleral marking method was used to identify all needle tracts. The number of tumor cells in tracts of direct transcleral aspirates was compared to those in tracts of indirect aspirates that traversed the anterior chamber of vitreous. Cellular material obtained with 30-gauge needles was sufficient for the diagnosis of malignant melanoma in all but one case. While 14 of 21 (67%) of all fine needle aspiration tracts and eight of 15 (53%) of indirect tracts contained tumor cells, the number of tumor cells was less than that associated with tumor growth in experimental models. Indirect aspirate tracts contained significantly fewer cells than tracts of direct aspirates ($P < .001$). (*Am J Ophthalmol* 105:538-546, May, 1988). Reprint requests to Robert Y. Foos, M.D., Jules Stein Eye Institute, 800 Westwood Plaza, Los Angeles, CA 90024.

MATERNAL ORBITAL HEMATOMA ASSOCIATED WITH LABOR. DM Jacobson, K Itani, KB Digre, KC Ossoinig, MW Varner. The authors examined two women with orbital hematomas that occurred during labor. Both women developed sudden diplopia, proptosis, and orbital pain. The location of the hematoma was confirmed by orbital echography and computed tomography. The patients were observed without surgical intervention. Neither patient developed clinical or echographic signs of compressive optic neuropathy. Clinical resolution occurred during the following two weeks. Serial standardized orbital echographic examinations documented resolution of the hematomas. (*Am J Ophthalmol* 105:547-553, May, 1988). Reprint requests to Daniel M. Jacobson, M.D., Neuro-ophthalmology Unit (4F), Marshfield Clinic, 1000 N. Oak Ave., Marshfield, WI 54449.

عید مبارک



Scholarship Schedules

International Society for Ophthalmic Ultrasound

August 28 - September 2, 1988

The International Society for Ophthalmic Ultrasound will hold its meeting Aug. 28-Sept. 2, 1988, in Iguazu Falls, Argentina. For further information, write Prof. Dr. Roberto Sampaolesi, Parana 1239, 1^o Piso, 1018 Buenos Aires, Argentina.

Eye Hospital Rotterdam: Third Course Silicone Oil in Vitreoretinal Surgery September 12 and 13, 1988

The Eye Hospital Rotterdam will present the Third Course, Silicone Oil in Vitreoretinal Surgery, Sept. 12 and 13, 1988, in Rotterdam, The Netherlands. For further information, write Dorine Verhoeven, Course Secretary, Eye Hospital Rotterdam Schiedamsevest 180, 3000 LM Rotterdam, The Netherlands.

American Academy of Ophthalmology: 1988 Annual Meeting October 8-12, 1988

The Annual Meeting of the American Academy of Ophthalmology will be held Oct. 8-12, 1988, in Las Vegas, Nevada. For further information, write Meetings Department, American Academy of Ophthalmology, P.O. Box 7424, San Francisco, CA 94120.

Fifth International Retinitis Pigmentosa Congress November 4-7, 1988

The Fifth International Retinitis Pigmentosa Congress will be held Nov. 4-7, 1988, in Melbourne, Australia. For further information, write Leonie Kelleher, 46A Oxley Road, Hawthorn, Victoria 3122, Australia.

Hong Kong Ophthalmological Society: Hong Kong Clinical Ophthalmological Symposium December 2-4, 1988

The Hong Kong Ophthalmological Society will sponsor the Hong Kong Clinical Ophthalmological Symposium, Dec. 2-4, 1988, in Hong Kong. For further information, write Patrick C.P. Ho, M.D., Chairman, c/o Secretariat Office, Exhibition and Convention Division, Room 810-814, Wing On Plaza, 62 Mody Road, Tsimshatsui East, Kowloon, Hong Kong.

Intraocular Lens Convention January 4-5, 1989

The Eye Research Centre will present an Intraocular Lens Convention, Jan. 4-5, 1989. For further information, write

Dr. Chander Neroor, Director, Dr. Agarwal's Eye Institute, 13, Cathedral Road, Madras 600 086, India.

International Society for Optical Engineering: Laser Imaging and Photoeffects in Ophthalmology January 15-20, 1989

The International Society for Optical Engineering will hold a symposium, Medical Applications of Lasers and Optics, Jan. 15-20, 1989, in Los Angeles, California. Abstracts for papers should be submitted by July 11, 1988, and the due date for manuscripts is Dec. 19, 1988. For further information write, SPIE Technical Program Committee: OE/LASE '89 Medicine, P.O. Box 10, Bellingham, WA 98227.

XXVI International Congress of Ophthalmology March 18-24, 1990

The XXVI International Congress of Ophthalmology is scheduled for March 18-24, 1990, in Singapore. Arthur S.M. Lim, Singapore, is President of the Congress. For further information, write The Congress Secretariat, XXVI International Congress of Ophthalmology, c/o Department of Ophthalmology, National University Hospital, Lower Kent Ridge Rd., Singapore 0511.

Sixteenth Annual Congress of the Society of Ophthalmology: German Democratic Republic April 23-28, 1990

The Sixteenth Annual Congress of the Society of Ophthalmology of the German Democratic Republic will be held April 23-28, 1990, in Rostock, West Germany. For further information, write The Congress Secretariat, XVI International Congress 1990, Eye Clinic of the Wilhelm-Pieck-University, Doberanerstr. 140, 2400 Rostock, West Germany.

XII Congress of the Ophthalmological Society of Pakistan February 23-25, 1989

For details see page 90 of this issue.

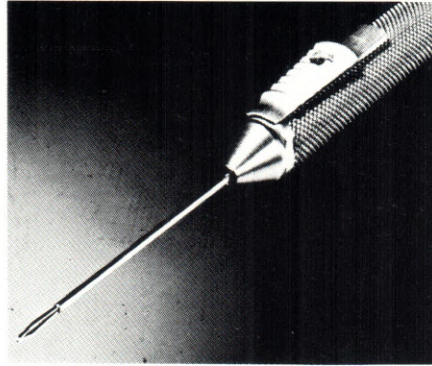
PAMS Convocation 1988 and PAMS Conference on "Hospitals in Pakistan" December 22, 1988

For details see page 90 of this issue.



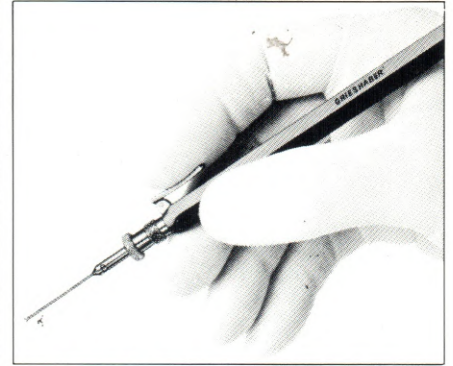
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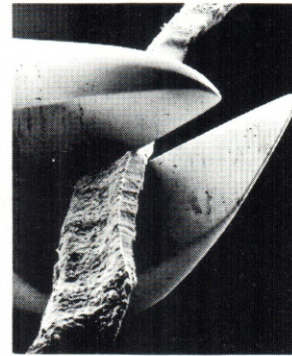
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B. For Books:

1. Newell, FW: Ophthalmology: Principles and Concepts. 6th ed., St. Louis. C.V. Mosby Company, 1986, p 73.
2. Duke-Elder, S, and Leigh, AG: Diseases of the Outer Eye. Cornea and Sclera. In Duke-Elder, S (ed): System of Ophthalmology, vol. 8, pt. 2. St. Louis, C.V. Mosby Company, 1965, pp 110-114.

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