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THE OFFICIAL JOURNAL OF THE OPHTHALMOLOGICAL SOCIETY OF PAKISTAN

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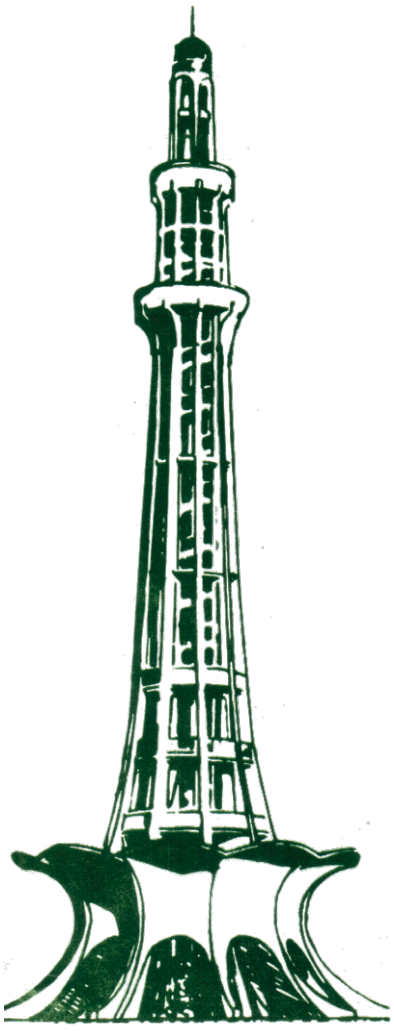

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THE OFFICIAL JOURNAL OF THE OPHTHALMOLOGICAL SOCIETY OF PAKISTAN

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Editorial

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

Burkitt's Lymphoma in Pakistan

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

The lead article by Zia Mohammad and M. Daud Khan in this issue (page 87) describes a rare occurrence of Burkitt's lymphoma in a Pakistani child. In 1987, the leading Pakistani expert on orbital tumors, Muhammad Munir-ul-Haq, F.P.A.M.S., did an analysis of 750 orbital tumors from his practice. There was not a single case of Burkitt's lymphoma in his report.[1] This brings to mind the Mayo Clinic survey of orbital tumors in the United States in which there was no case of this cancer among the consecutive 764 orbital tumors recorded in the 27-period 1948 through 1974.[2] Now, 200-300 cases of Burkitt's lymphoma are reported in the United States each year.[3] Surely, some cases of this tumor must have occurred in the United States in the period before Burkitt[4] drew attention to it in 1958. Obviously, these cases were either not recognized or were misclassified by the pathologists. No doubt with passage of time an increasing number of this malignancy will be recognized in Pakistan, an important reason why our ophthalmologists should become more familiar with its clinical behavior and effective management.

Although endemic to the tropical areas of Africa, particularly Uganda, Burkitt's lymphoma has been reported from all parts of the world, in the form of a non-endemic variety. Either variety may follow a similar pattern, but the endemic type predominantly occurs in younger children (mean age 8), affects males more than females (2:1 to 3:1 ratio), primarily involves face (55-75%), and invades the orbit more frequently (10-20%). On the other hand, the non-endemic variety occurs in older age group (mean age 12), affects sexes equally in patients older than 13, primarily starts in the abdomen in the majority of cases, and eventually involves the central nervous system in a higher percentage of cases (45%).[5]

When invading the orbit, the tumor usually arises as an osteolytic focus in the adjacent maxilla, but may rarely arise, like the case reported in this issue, in the ethmoidal sinus.[6] The vision is affected by direct invasion of the orbital tissues, causing retinal vascular tortuosity, eyelid edema, conjunctival chemosis, proptosis, and corneal anesthesia and exposure keratitis, which may eventually lead to panophthalmitis. A direct involvement of the structures of the globe may occur, but is rare.[7,8]

Burkitt's lymphoma also is the fastest growing human tumor, with a mean potential doubling time of 25.6 hours and an actual doubling time of 66 hours.[9] This underscores the need for an early diagnosis, because this cancer is also one of the few malignancies that are amenable to treatment when not very extensive, with a cure rate of 80% in patients in whom the tumor is confined to a single extra-abdominal site. The prognosis also heavily depends on the size of the tumor at the time of treatment. Hence, in some instances of extensive involvement, the bulk of the tumor may be surgically removed to reduce its size before chemotherapy. The most effective agent is cyclophosphamide in high doses. It may have to be combined with vincristine, methotrexate, and corticosteroids in some patients. The central nervous system involvement may also need craniospinal irradiation.

Burkitt's lymphoma, a B-cell and histologically clearly defined neoplasm, is also an interesting malignancy in that unlike the other cancers it is thought to be caused in some edemic instances by a specific infective agent, the Epstein-Barr (EB) virus (possibly in association with an insect vector), shows heritable predisposition in some cases, and is linked to a specific chromosomal abnormality, the translocation or exchange of a large segment of chromosome 8 with a small segment of chromosome 14. In the non-endemic Burkitt's lymphoma, it is not the EB virus but *human immunodeficiency virus* (HIV) that is thought to play an etiologic role. Hence, there is a higher incidence of non-endemic Burkitt's lymphoma in homosexuals and patients with acquired immunodeficiency syndrome (AIDS).[10]

This close relationship of Burkitt's lymphoma with a known exogenous infective agent has given rise to speculations that in the future a vaccine to prevent this tumor, and other virus-related cancers (e.g. nasopharyngeal carcinoma), may be produced.[11]

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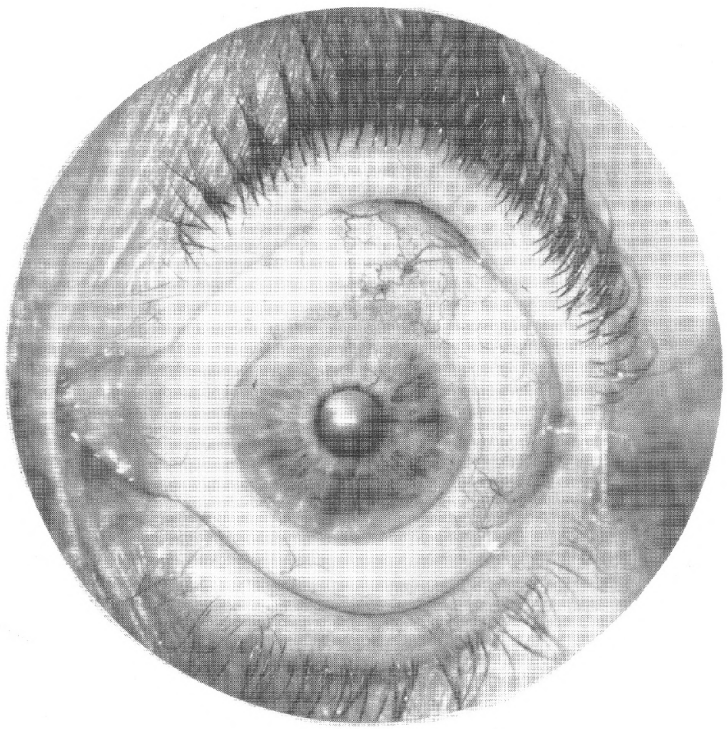
Camera Clinicals

In this section of THE JOURNAL, *photographic documentation of interesting and challenging observations are presented to the readers. They should make their diagnosis from the given information, and compare their conclusions with the exposition given on page 98.* -Editor



Figure

Figure: A 65-year-old woman came three years ago with complaint of difficulty in reading. She was suffering from chronic respiratory distress, and had been told by her doctor that her "blood was too thick." Her hemoglobin value varied between 16.5 gram/dl to 18.1 gram/dl and the hematocrit varied from 50 ml/dl to 55 ml/dl. Her internist had diagnosed her condition as secondary or stress polycythemia. She had also recovered from a recent congestive heart failure.

The eye examination at that time showed her visual acuity to be 20/40 (6/12) in the right eye and 20/100 (6/30) in the left eye with glasses. Her ocular fundi had the characteristic appearance and intense venous dilatation of polycythemia retinopathy. Fluorescein angiography demonstrated retinal pigment epithelial detachment near both maculae and an occult subretinal neovascularization inferior to the left fovea. Krypton laser photocoagulation did not improve her sight in the left eye. Another interesting ophthalmoscopic finding was the bilateral presence of a few small blot hemorrhages in the far peripheral retina.

About a year ago she started complaining of burning, excessive tearing, photophobia and foreign body sensation in her eyes. Her doctor's prescription of antibiotic/corticosteroid drops proved ineffective. On her recent visit to our office the interesting findings shown in the above Figure were noted in both eyes. The accurately diagnosed condition responded to its specific treatment.

Orbital Burkitt's Lymphoma Arising from the Ethmoidal Sinus

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

ABSTRACT: A five-year-old child developed a slowly progressive, painless, proptosis of the left eye. On histopathological examination of a biopsy specimen, it proved to be due to Burkitt's lymphoma of the orbit. The reports of this tumor's occurrence in Pakistan are very rare. The tumor arose from the ethmoidal sinus, eroded the medial and inferior walls of the orbit, and displaced the globe laterally and downwards. This is the second published report of an orbital Burkitt's lymphoma arising from the ethmoidal sinus without concurrent involvement of the maxillary sinus. The tumor responded well to radiotherapy. (Pakistan Journal of Ophthalmology 7:87-90, October, 1991).

Burkitt's lymphoma is a type of B-cell lymphocytic tumor that was first described in 1958 by Burkitt,[1] a British surgeon working in Africa at that time. This tumor may appear at any age, but in a vast majority of cases occurs in children, usually in the age group of 2-16.[2] Although it has been reported from all parts of the world, Burkitt's lymphoma is predominently found in tropical Africa. It is very rare in Pakistan, and in an analysis of 581 primary orbital tumors from the Institute of Ophthalmology, Lahore, Munir-ul-Haq[3] did not find any case of Burkitt's lymphoma. Burkitt's lymphoma is by far the commonest cause of proptosis in Uganda, and involves the orbit in 20% of children.[4] Sporadic non-African Burkitt's lymphoma (small noncleaved follicular cell lymphoma) is morphologically indistinguishable from the African endemic Burkitt's lymphoma.[5] In the non-African type (called American type lymphoma in the US), abdominal involvement is more common, while in the African type jaw is the most frequently affected structure. In 50% of the cases of Burkitt's lymphoma, there is a destructive lesion of the maxilla that erodes into the orbit causing proptosis. It usually remains confined to the orbit, but may rarely show intraocular involvement.[6]

We report here a case of a 5-year-old boy with unilateral proptosis, which proved to be due to Burkitt's lymphoma originiating from the ethmoidal sinus without any involvment of the maxillary sinus. It is a rare presentation of non-African Burkitt's lymphoma, and to our knowledge this is the second such documentation in the literature.[7]

Case Report

On June 13, 1991, the parents brought their 5-year-old boy with a history of bulging of his left eyeball for the previous 20 days. The swelling had increased gradually in size and was painless.

On eye examination, his visual acuity was 6/6 (20/20) in the right eye and 6/18 (20/60) in the left. A relative afferent defect was noted in the reaction of his left pupil. The anterior segment of each eye was otherwise normal. The patient's left eye had proptosis (Figures 1 and 2), with Hertel's exophthalmometric readings of 18 mm on the right and 21 mm on the left. There was no limitation of ocular movements. The proptosis was axial and was not reducible on palpation. There was no palpable discrete mass, no thrill, and no bruit.

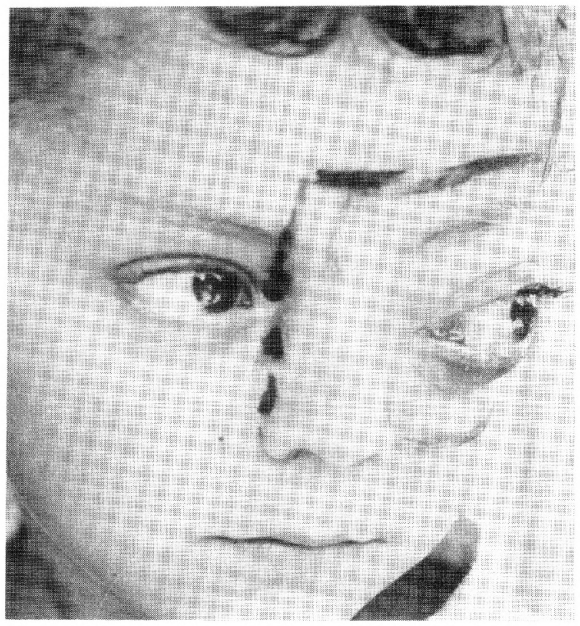


Figure 1 (Mohammad and Khan): Note the proptosis of the left eye.

From the Department of Ophthalmology, Postgraduate Medical Institute, Lady Reading Hospital, Peshawar, Pakistan.

Reprint request to Dr. Zia Mohammad, F.C.P.C. (Pak.), Eye Specialist, Clinic Opp. DHQ Hospital, Shamsi Road, P.O. Box 81, Mardan , Pakistan. Tel: (92-531)-4066 (Res.)

Figure 2 (Mohammad and Khan): Proptosis left eye.

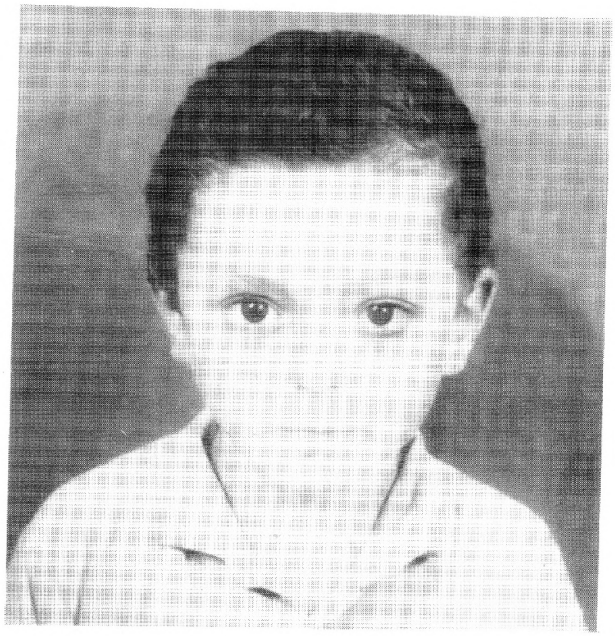


Figure 3 (Mohammad and Khan): The patient after radiotherapy.

The right ocular fundus was normal, while the left fundus revealed congestion of the optic disc. General physical examination was normal for the child's age.

Blood analysis showed a total white cell count of 10,900/cmm, with 57% neutrophils, 40% lymphocytes, 2% monocytes, and 1% eosinophils. Except for a mild hypochromia of the red blood cells, the peripheral smear was normal. No malarial parasites were seen. The blood hemoglobin was 10.8 G%. Bone marrow biopsy revealed normal morphology.

X-rays of the paranasal sinuses showed erosion of the orbit. X-rays of the chest was normal. CT scan showed soft tissue mass in the left nasal cavity, involving the spheno-ethmoidal sinuses and cribriform plate. It extended to the left orbit causing erosion of the bone in its medial wall. We decided to perform biopsy to study the cellular nature of the tumor.

HISTOPATHOLOGY: Grossly, during an incisional biopsy a chocolate colored fluid oozed out of the incision. Microscopic histopathological examination of the biopsied tissue showed characteristics of a malignant neoplasm. It was composed of round cells with scanty cytoplasm. The nuclei showed mitotic figures. Scattered between these cells were benign looking histiocytes, imparting the microscopic field the so called "starry sky" appearance. The diagnosis of a malignant lymphoma, morphologically resembling Burkitt's lymphoma was made on the basis of these histopathologic features.

During his stay in the hospital the proptosis slightly increased, with exophthalmometric reading of the left eye increasing to 23 mm. There also appeared a mild congestion of both the palpebral and the bulbar conjunctivae. The left globe became slightly more displaced laterally, and its movements developed slight restriction in all gazes. Fundus examination revealed blurring of the left optic disc margin. The visual acutiy in the left eye dropped to 6/60 (20/200). At this stage, on July 7, 1991, it was decided to employ radiotherapy, to which the tumor responded well. Within two to three weeks, the proptosis, conjunctival congestion and optic disc swelling in the left eye became significantly reduced. The last post-irradiation exophthalmometric readings, on August 6, 1991, were 18 mm on both sides (Figure 3).

Discussion

Burkitt's lymphoma is classified as a poorly differentiated lymphocytic lymphoma with very characteristic clinical and histopathologic manifestations.[6-8] On a review of the cancer and general medical literature, one realizes that it is predominently a disease of tropical Africa.[10-12] Nevertheless, since the time Burkitt first drew attention to this unusual tumor, it has been reported from all parts of the world.[13]

It occurs in all age groups but the majority of cases occur between the ages of 2-16 years.[2,6,14-17] Burkitt's lymphoma is by far the commonest cause of proptosis in children in Uganda.[4, 13]

Lesions affect the jaw, kidneys, ovaries, retero-peritneal lymph nodes, thyroid gland and testes. It can also affect superficial lymph nodes, liver and spleen though less often. In 50% of cases of Burkitt's lymphoma, the lesion erodes the maxilla and involves the orbit causing unilateral proptosis.[2,6,14-17]

In our case, the primary lesion originated in the ethmoidal sinus, eroding the medial wall of the orbit causing proptosis of the left eye. This is a very uncommon appearance of Burkitt's lymphoma, and to our knowledge in only one other report this tumor arose in the ethmoidal sinus and invaded the orbit in a 5-year-old girl.[13]

The poorly differentiated and highly mitotic lymphocytes, which have a basophilic staining property, are the dominant cell population. These lymphocytes usually contain cytoplasmic vacuoles that are filled with fat. The so characteristic "starry sky" appearance of histologic section of the tumor is due to the phagocytic histiocytes which are scattered among the sheets of lymphocytes.[18] The histopathologic features of the biopsy specimen from our case were very compatible with this histologic description.

The increased incidence of cases where malaria is hyperendemic suggests a link between malaria, EB virus and Burkitt's lymphoma. Thus etiologically Burkitt's lymphoma is thought to be caused by Epstein-Barr virus, although there is as yet no proof that this or any other virus is the cause of Burkitt's lymphoma. The E-B virus is ubiguitous and antibodies to it are present in the serum of many normal individuals. The immune response of the host is presumably so impaired by malaria or possibly other chronic infections that the B-cell response gets out of control and leads to lymphoma formation.[19]

Chromosomal abnormalities are believed to occur in almost all cases of Burkitt's lymphoma. In 90% of the patients, the chromosomal abnormality consists of translocation of genes from chromosome 8 to the long arm of chromosome 14.[13]

Burkitt's lymphoma is the most rapidly growing tumor reported in man, the history being seldom longer than 3 months.[4, 13] Biopsy specimens are often necrotic as the rapidly growing tumor outstrips the available nutritional supply. This rapid growth outstripping the nutrient supply may in some instances lead to spontaneous remission in the tumor.[13] In our patient the growth was equally rapid. From the biopsy incision, a chocolate-colored fluid oozed out probably due to a hemorrhagic necrosis in the tumor.

Orbital involvement in Burkitt's lymphoma most commonly follows from the maxillary sinus and/or jaw tumor.[13, 20] In our patient, however the primary tumor was in the ethmoidal sinus which had eroded the medial wall of the orbit, causing proptosis of the left eye. An otolaryngologist in consultation observed swelling of the nasal mucosa and middle turbinate to the extent of obscuring the inner view.

The tumor is extremely sensitive to chemotherapy and cure rates of more than 50% have been reported.[7,9] Cyclophosphamide, vincristin, methotrexate and prednisone prescribed in intermittent high dosage is the treatment of choice.[13, 21] Radiotherapy is also quite effective, and may be used in conjunction with chemotherapy.

The patient needs to be carefully evaluated for renal and hepatic functions before treatment, for the rapid destruction of tumor cells following treatment releases toxins which may prove fatal. The initial response to the treatment is usually good, with a rapid decrease in the the tumor's size. In our case, the proptosis tremendously decreased following radiotherapy.

Relapse in the first six months is a sign of bad prognosis. But it may still be successfully treated if managed aggressively. If there is a failure to achieve a complete response to the repeat therapy, the prognosis is grave.

Finally, the incidence of Burkitt's lymphoma in Pakistan is extremely low, and we could not find any published reports on its occurrence here. In one report on primary orbital tumors in Pakistan, published in 1985, no case of Burkitt's lymphoma was present.[3] We recently contacted the leading authority on orbital tumors in Pakistan, Professor Muhammad Muni-ul-Haq. He informs us that although he has seen two or three cases of Burkitt's lymphoma in Pakistanis in his career, he is also not aware of any published report of such cases.[21] This makes our present case report even more interesting and significant.

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Ophthalmic "Pastpourri"

The First Case of Orbital Hydatid Cyst in the United States

Over a century ago, in 1889, Dr. J.E. Weeks of New York reported the first American case of orbital hydatid cyst in a 22-year-old Austrian Jew who had been in the United States for five years, and who, wrote Dr. Weeks, "observes the precepts of the orthodox Jews in regard to his diet. Has no pet dog, nor is there any kept in the family with which he resides."

On eye examination, the patient had "a firm, elastic tumor" under the right upper lid, "situated in the orbit to the median side of its perpendicular meridian." The "elasticity and the indistinct sense of fluctuation imparted to the finger narrowed the diagnosis down to abscess or cystic tumor." A clear fluid and pus escaped when the cystic mass was opened by means of a Grafe knife. "On pressure, a little more pus escaped, accompanied with seven transparent vesicles, which proved to be the daughter cells of an echinococcus cyst." "No attempt was made to remove the wall of the mother cyst. The cavity was syringed with a bichloride solution 1/5,000, and the eye bandaged." Complete recovery without recurrence ensued in four weeks.

Dr. Weeks also gave the following diagnostic pointer to his readers: "If on puncture with an aspiration needle, some clear fluid is drawn off, found to be devoid of albumin, but rich in sodium chloride (nitrate-of-silver test), the diagnosis of hydatid cyst is positive, even without finding hooklets, since the contents of all other cystic tumors are albuminous."

Therapy, Then:

To achieve rapid recovery in cases where surgical excision of the cyst is not possible, Dr. Weeks advised that "insertion of a drainage-tube and the production of a mild process of suppuration should be resorted to."

Weeks, JE:
A case of epibulbar echinococcus, with a review of the literature on echinococcus cysts of the orbit.
Arch Ophthalmol 28:31-52, **1889.**

Therapy Today:

"Treatment (of orbital hydatid cyst) ideally is excision of the intact cyst . . . If complete excision is impossible, it is suggested that cyst should be drained and sterilized with formalin or alcohol (avoiding any spill into the orbit itself) and irrigated with saline, following which the wall can be removed."

Rootman, J:
Diseases of the Orbit. A Multidisciplinary Approach.
Philadelphia, J.B. Loppincott Company, **1988.**
158-159

Clinical Research

Extracapsular Cataract Extraction with Posterior Chamber Intraocular Lens Implantation at the Rawalpindi General Hospital

S. Imtiaz Ali, F.R.C.S.

ABSTRACT: Out of a total of 220 consecutive patients who underwent extracapsular cataract extraction with posterior chamber intraocular lens implantation at the Rawalpindi General Hospital, Rawalpindi Medical College, Rawalpindi, Pakistan, 135 (61.4%) patients did not keep their appointments for follow-up. This discouraging pattern is universal in Pakistan and makes clinical studies very difficult and less than ideal for drawing reliable conclusions. The following postoperative complications appeared in the 85 patients who did come for follow-up visits: anterior uveitis in 17 (20%) patients, after cataract (secondary cataract) in 11 (13.5%), glaucoma in 3 (3.5%), lens implant decentration in 3 (3.5%), and cystoid macular edema in 3 (3.5%). Intraopera-tively, posterior capsular rupture with or without vitreous loss occurred in three (3.5%) patients and the removal of anterior capsule was not complete in another three (3.5%). One female diabetic patient totally lost her sight due to postoperative rubeosis iridis and hyphema causing severe thrombotic (neovascular) glaucoma. One man (1.17%) developed bacterial endophthalmitis. The infection was successfully controlled, but the eye developed total corneal decompensation. A partially successful penetrating keratoplasty gave this patient a visual acuity of counting fingers. Of the 11 after cataract (posterior capsule opacification) cases, eight were treated with neodymium:YAG laser capsulotomy and three with surgical capsulotomy. Out of a total of 14 eyes which had traumatic cataract, 11 recovered a visual acuity of 6/36 (20/120) or better, two a visual acuity of 6/60 (20/200), and one only light perception. (Pakistan Journal of Ophthalmology 7:91-94, October, 1991.)

Extracapsular cataract extraction with intraocular lens implantation has become the procedure of choice for the management of cataract. New types of lenses are being introduced in an increasing number. Now the emphasis is on reducing postoperative astigmatism. However, the vexing problem of capsular opacification still eludes the researchers, and no measure has significantly reduced its incidence. Although neodymium: yttrium, aluminum, garnet (Nd:YAG) laser has made the treatment of posterior capsular opacification simple, it is not without complications. Moreover, the availability of Nd:YAG laser is not universal. Hence, there is a great need of some means to drastically reduce the incidence of opacification of the posterior capsule after extracapsular cataract surgery. Research on this aspect of lens implantation surgery has not as yet created any satisfactory solution. While economically prosperous and technologically advanced countries are in the search of ways to get perfect and lasting results from the modern techniques of pseudophakic surgery, the advancing nations are still struggling to make the very basic lens implantation operation practical for their people.

It is with this idea in mind that we decided to study the problems of the posterior chamber intraocular lens implantation in the Rawalpindi area of Pakistan. Our difficulties in carrying out this study and its outcome are the topic of this article.

Material and Methods

We had planned a prospective study designed to include 220 consecutive cases of posterior chamber intraocular lens implantation at the Rawalpindi General Hospital, Rawalpindi Medical College. Unfortunately, 135 (61.4%) of these patients never returned for follow-up visit. Hence, this report is based on the 85 patients who kept their follow-up appointments. These patients, 51 men and 34 women, varied in age from 5 to 85 years (mean age 50 years). Out of these 85 patients, nine had hypertension, seven had diabetes mellitus, and three had controlled

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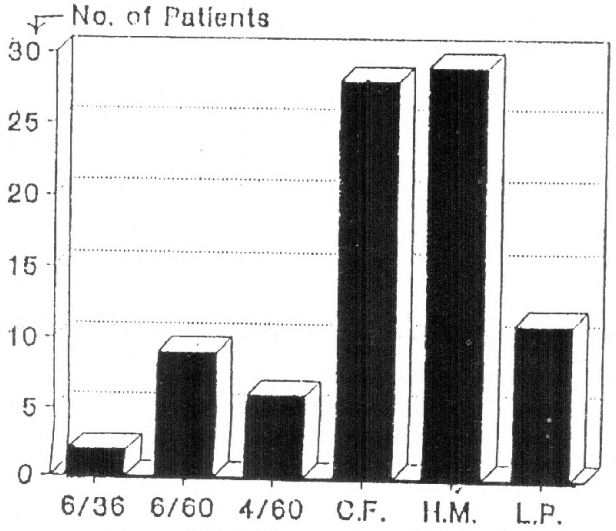

Figure 1 (Ali): Preoperative visual acuity

glaucoma. Out of the 85 patients, 14 had developed cataract following ocular trauma.

All patients included in this study had surgery and follow-up at the Rawalpindi General Hospital, Rawalpindi, from February 1989 to June 1991. The follow-up period varied from three-month to two-year, and follow-up visits were made every three months in the first year and every six months in the second year. The preoperative ocular examination included the evaluation of visual acuity, tonometry, slit lamp examination and ophthalmoscopy by direct and indirect methods. The patients with preexisting corneal opacities were not included in the study. All patients were screened for diabetes mellitus, hypertension and chest problems. The power of intraocular lens was calculated impirically in 75 eyes (88%), by SRK formula in four eyes (4.6%), and by clinical evaluation formula in six eyes (6.9%).

All patients received one-day preoperative course of antibiotic drops, mostly 0.5% chloramphenicol, every four hours in both eyes. The pupillary dilatation before the operation was achieved by tropicamide hydrochloride 1% and phenylepherine 10% drops. A mixture of equal amounts of 2% xylocaine and bupivacaine 0.5% solutions was used for facial akinesia, either O'Brien technique or van Lint technique, and the retrobulbar block. For additional sedation, we employed injection of a mixture of equal amounts of pentazocine (Talwin) and haloperadol (Serenace), (called LA Plus in our Department). The patients below 40 had surgery under general anesthesia. After a fornix-based conjunctival falp, a superior *ab externo* incision extending from 10 to 160 degrees from the horizontal was made through the partial thickness of the limbus. After a stab incision into the anterior chamber anterior capsulotomy was performed with a a 27-gauge insulin needle. The corneal section was then completed with corneal scissors. After delivery of the nucleus, the incision was closed temporarily and cortex aspirated by irrigation/aspiration manual technique using balanced salt solution or Ringer's lactate. The posterior chamber intraocular lens was inserted with McPherson forceps after reforming the anterior chamber with an air bubble or 2% methylcellulose.

Table 1
Final postoperative visual acuity (85 eyes)

Visual acuity	No. of eyes	Percentage
6/6 - 6/12	35	41%
6/18 - 6/24	19	22%
6/36 - 6/60	17	20%
Less than 6/60	14	17%

In more than 90% of the cases the implant was a plano-convex optic with modified J-loops of polypropylene at 10-degree anterior angulation. After the insertion of the implant pupil was constricted with either a minum of 4% pilocarpine or intracamaral injection of Miostat (0.04% carbachol). Whenever viscoelastic substance was used, it was aspirated and anterior chamber reformed with balanced salt solution at the end of procedure. The corneal wound was closed with six interrupted 10-O monofilament nylon sutures the knots of which were buried on the corneal side of the wound. Each patient received a subconjunctival injection of gentamicin 20 mg and methylprednisolone (Depo-Medrol) 20 mg at the conclusion of the operation.

Postoperative care included antibiotic/corticosteroid combination (usually Maxitrol) eyedrops four hourly , tropicamide hydrochloride 1% cycloplegic drops b.i.d., and beta-blocker antiglaucoma drops b.i.d. in patients with elevated intraocular pressure. Some patients also received 500 mg of acetazolamide (Diamox) orally for the first 2 to 5 postoperative days.

For the first few weeks the patients were seen at weekly intervals. The final refraction was done three months after the surgery. The corneal sutures that were felt to be responsible for astigmatism were removed with 27-gauge insulin needle. Those patient who needed laser capsulotomy had Nd:YAG laser capsulotomy done at the Pakistan Institute of Medical Sciences at Islamabad. The average number of pulses per patient was 20 (range between 10-50) with average power of of 4mJ. Nd:YAG laser posterior capsulotomy was done in eight patients. In other three patients capsulotomy was accomplished surgically. The preoperative visual acuity of 85 patients as shown in Figure 1, was light perception in 11 patients, hand movement perception in 29, counting fingers in 28 patients, and ranging from 6/60 to 6/36 in 17 patients. The average power of the intraocular lens was +20.5 diopters (range, between +18.0 diopters to 23.0 diopters).

Results

The postoperative visual acuity is shown in Table 1,

Table 2
Results in eyes with traumatic cataract (14 eyes)

No. of cases	Preoperative visual acuity	Postoperative visual acuity
1	Hand movements (HM)	6/6
1	HM	6/9
1	HM	6/12
5	Finger counting (FC)	6/18
3	Light perception or HM	6/36
2	FC or HM	6/60
1	Light perception (LP)	LP

Table 3
Postoperative period for post capsular opacification and method of capsulotomy (11 eyes, 13% of total of 85 eyes)

Postoperative period	Capsulotomy technique		Percentage (total 85 eyes)
	Surgical	Nd: YAG	
6 months	1 eye	4 eyes	6%
9-12 months	2 eyes	1 eye	3.5%
1-2 years	2 eyes	1 eye	3.5%

and as is clear from figures in it, the results were very encouraging. One patient ended up without light perception, because she developed rubeosis and thrombotic glaucoma as a result of uncontrolled diabetes.

The results in the 14 traumatic cases are shown in Table 2. In one patient the results were no better than the preoperative status. This patient had vitreous and retinal degeneration with detachment. The causes of decreased vision in other cases were corneal opacity or vitreoretinal pathology.

Visually significant posterior capsular opacification requiring capsulotomy occurred in 11 (13%) cases. The time of opacification and outcome of capsulotomy are shown in Table 3. (Five more patients have developed posterior capsular opacification since this study was concluded.) One patient had primary capsulotomy during the surgery.

Intraoperative inconsequential hyphema occurred in a few cases after iridectomy. Miosis of the pupil during surgery ocurred in some cases, and in them the lens was implanted by pulling the iris from its root with an iris forceps.

The postoperative complications and their incidence is shown in Table 4. The most significant of these was the early postoperative uveitis, which was very severe in five cases. One of which developed posterior synechiae and a fibrous membrane over the implant. In

Table 4
Postoperative and intraoperative complications (85 eyes)

Complication	No. of eyes
Uveitis	17 (20%)
After-cataract	11 (13%)
Glaucoma	3 (3.5%)
Incomplete anterior capsule removal	3 (3.5%)
IOL decentration	3 (3.5%)
Cystoid macular edema	3 (3.5%)
Posterior capsular rupture with or without vitreous loss.	3 (3.5%)
Endophthalmitis	1 (1.17%)

this patient, synechialysis and posterior capsulotomy were successfully done with a 27-gauge insulin needle. A large amount of retained cortex was the probable cause of this complication.

In two patients the glare caused by the lens decentration was nicely managed by pilocarpine drops.

One patient with uncontrolled diabetes developed rubeosis and hyphema with intractable neovascular glaucoma, due to which she lost sight in this eye. One young man recovered only light perception in his eye due to traumatic vitreoretinal degeneration with retinal detachment. Another male patient, who had posterior capsular ruptured with vitreous loss, developed endophthalmitis. Infection was controlled with intensive antibiotic therapy, but the eye developed decompensated cornea. After a penetrating keratoplasty, this patient recovered vision of only finger counting on account of a poor graft acceptance.

Comments

Duke-Elder,[1] writes about Ammar, an innovative Arab physician of the eleventh century, that he "inserted a hollow needle and sucked out the opaque material of soft cataract." Six hundred years later, in 1748, Jaques Daviel of France devised the planned extracapsular cataract extraction and published his results in 1753, which revolutionized the treatment of cataract forever.[1] Hence, extracapsular cataract removal is not a new idea. What has made it so attractive today, after a long reign of intracapsular technique, began in 1949, when Harold Ridley[2,3] implanted the first acrylic artificial lens in a human eye. Today, extracapsular cataract extraction with intraocular lens implantation is the favorite procedure of an overwhelming majority of the ocular surgeons around the globe.

Unfortunately, poor socioeconomic circumstances, a lack of public health education, limited transport facilities, and illiteracy make it difficult for many patients in Pakistan to follow doctor's instructions and

keep up with the follow-up care. This has plagued many of the clinical research projects in our country. Our study is a prime example of this. We hope that with the passage of time, circumstnaces will change for the better. But until that happens, we have no choice but to depend on the results of incomplete studies.

The results of our study surprised us in that such a high percentage of cases (20%) developed postoperative uveitis. One diabetic woman lost her eye to uveitis, glaucoma and hyphema (UGH syndrome). Other five patients with severe uveitis eventually responded to treartment. We believe that uveitis was mostly due to poor technique which left large amounts of cortex in these eyes. Some authors feel that removal of all of the cortex is not essential.[4] However, our experience dictates contrary to this view.

Posterior capsular opacification occurred in 13% of the eyes. Percival[5] recently reported that in eyes with triple glaucoma procedure with IOL implantation and in eyes with preoperatively having filtering procedure, the rate of capsular opacification was 20.4%, but otherwise it was only 1.75 to 6.8%, reaching a maximum of 12.9% in five years. We found the pars plana posterior capsulotomy technique of Lindstrom and Harris[6] to be very helpful when Nd:YAG laser is not available. Many studies are going on regarding the use of mitotic inhibitors and monoclonal antibodies to prevent posterior capsular opacification,[7,8] but their safe and effective use in human has still to come out.

One case of endophthalmitis (1.17%) in our 85 patients is very disappointing, because the currently accepted rate of endophthalmitis following cataract surgery is 0.02% to 0.1%. It is very clear that much more attention is required, on the part of both the surgeon and the patient, to avoid this terrible complication in Pakistan. In a recent report, wound problems, such as a small leak, exposed suture, etc. and vitreous related complications, such as vitreous loss, vitreous wick syndrome, etc. were identified as major factors contributing to development of endophthalmitis in pseudophakic eyes.[9]

Acknowledgements

Dr. B. A. Naeem, Registrar, Eye Unit, Rawalpindi General Hospital Rawalpindi, assisted in the completion of this study; Dr. Jehangir Akhtar, Assistant Professor, Ophthalmology Department, Pakistan Institute of Medical Sciences, Islamabad also helped. I am grateful to both.

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Ophthalmic "Pastpourri"

Intraocular Fluorescein, A Hundred Years Ago

Surprised by the conclusion of Pfluger, Schoeler and Uthoff that the fluorescein injected into the vitreous does not reappear in the aqueous, H. Gifford of Omaha reported the following experiment: "To test the question, therefore, the following experiment was made: September 17, 1890, small black rabbit, 3-4 gtt. of 10% solution of uranin (soluble ammonia salt of fluorescein) injected into each vitreous back of and below the centre. On withdrawing the needle about the same quantity of fluid flowed out under the conjunctiva. After 3/4-1 hour, vitreous strongly fluorescent, but no fluorescence for certain in the aqueous. On slight struggling of the rabbit, however, clouds of fluorescence could be seen to burst into the anterior chamber through the pupil in each eye."

Gifford, H: Further experiments on the lymph-streams as lymph-channels of the eye. **1892**

121-17192

Comparative Value of Iridectomy, Iridotomy, and No Iris Opening in Intraocular Lens Implantation Surgery

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ABSTRACT: Following a study of the complications, advantages, and disadvantages of an iris opening made during cataract surgery and intraocular lens implantation, I have conclude that an iris opening to create a communication between the anterior and posterior chambers in addition to the pupil adds to the safety of extracapsular cataract extraction with posterior chamber intraocular lens implantation. Comparing the results of an iridectomy, scissors iridotomy, no iris opening, and transfixation iridotmy as described here shows that an iris opening is much more advantageous than no opening at all, and that the last mentioned method is easy, relatively harmless, and prevents a number of complications seen either with no iris opening, or with a large iridectomy. (Pakistan Journal of Ophthalmology 7:95-97, October, 1991).

No iridectomy or not large enough iridotomy could lead to pupillary block glaucoma with or without internal prolapse of the iris (iris bombe) in eyes with anterior chamber angle-fixated and iris supported lenses.[1-5]

The increased intraocular pressure may push the intraocular lens against the cornea causing rapid corneal decompensation. Even posterior chamber lenses, especially the iridociliary sulcus-fixated ones, could get pushed out of place by increased intraocular pressure caused by pupillary block.[6-8] Such pressure may also lead to "captive iris syndrome" with one edge, or both edges, or whole of the optic of intraocular lens becoming displaced in front of the iris through the pupil. The lenses so displaced cannot be permanently reposited without an iridectomy.

Iridectomy, however, has its own problems.[9-10] If performed before the implantation of the intraocular lens, the lens can get caught in it leading to iris tears, iridodialysis, hemorrhage, difficulty in placement of the intraocular lens, and exposure of the peripheral vitreous during the surgery. Hence, most experts advise doing iridectomy as the last step of procedure.

The use of posterior chamber intraocular lenses has led some surgeons to regard iridectomy as unnecessary,[11-13] or even dangerous.[14,15] I reviewed the literature on iridectomy, and conducted a study to evaluate the advantages and complications of various types of surgical openings in the iris, including a newly devised technique of iridotomy.

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Material and Method

This study was conducted in two parts. In one part, 1,092 consecutive cases, 352 with scissors iridectomy, 290 with scissors iridotomy, and 450 without any iris opening were reviewed for any early and late complications.

Following this review, 254 consecutive patients were subjected to creation of an iris opening using the below described new technique. These patients were then also studied for the early and late complications in the same fashion as the cases in the first part of the study. In an iridectomy, a small piece of iris was cut out, and in an iridotomy the iris was simply incised. The patency of the opening was routinely ascertained with a spatula in all cases. All iris surgery was performed at the end of the implantation when the lens implant was securely in place, the capsule remnants had been removed, and the wound secured with one or more interrupted sutures. There were no diabetics among the patients.

TECHNIQUE: After the lens implant is securely in position, the pupil is constricted with a miotic. The cataract wound is not sutured and the chamber is left filled with Healon. A knife-needle (I use Grieshaber-Haab discision knife No. 681.05) is passed through the limbal wound parallel to the iris. At a suitable point th iris is engaged by the tip of the knife and penetrated. If a direct penetration does not happen and a roll of iris tissue develops at the tip of the knife, the knife is pushed through to the other side of this roll, achieving a double penetration of the iris. These openings are about half-way between the fully constricted pupil and the base of the iris (Figure 1). Alternatively, the knife may be introduced through a prelimbal stab incision after the cataract incision is closed. This helps avoid accidental cutting of the sutures of the corneal incision by the discision knife. It is most important that the

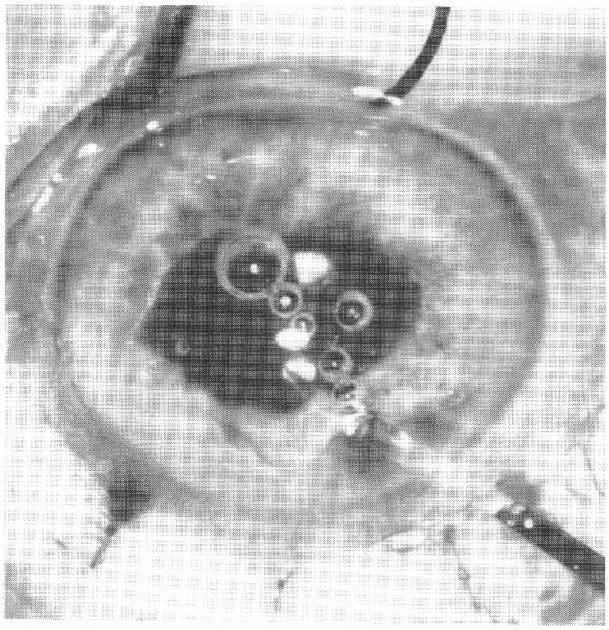

Figure 1 (Alpar): The Grieshaber-Haab discision knife No. 687.05 is introduced through the closed cataract incision to engage and perforate the iris midway between its bases and the pupil. Care must be taken to avoid entangling and cutting the cataract incision sutures. To sidestep this problem the discision knife may be introduced through a prelimbal stab incision. The anterior chamber is still filled with HealonR.

knife remains parallel with the iris surface and does not puncture the capsule or the vitreous face. In effect, this procedure is greatly similar to the "transfixio iridis" (transfixion of the iris), an operation which was used in the past to treat the "iris bombe."

Results

The major early complication in the review group was hemorrhage from the iris surgery. In the iridectomy group it occurred during the operation or on the first postoperative day in 80 (14.3%) of the cases, in the scissors iridotomy group in 21 (7.23%) cases, and in the new technique groupit only in one patient (0.39%). In the late complications, peripheral anterior synechiae was the most common. It was present in 123 (34.75%) cases of iridectomy, 50 (17.1%) cases with scissors iridotomy, two (0.78%) cases in the new technique group, and in 12 (2.66%) cases in the no iridectomy group. Captive iris developed in one (0.28%) patient with iridectomy, in two (0.68%) patients with iridotomy, no case in the new technique group, and in 14 (3.1%) in the no iridectomy group. The late peaking of the pupil without adhesions to the capsule appeared in two (0.56%) patients with iridectomy, in two (0.68%) patients with irodotomy, and one (0.39%) patient in the new technique group. In the patients who had no iris opening, pupillary block glaucoma ocurred in 16 (3.54%) patients, whereas it was not seen in any patient with basal iridectomy or transfixation iridotomy.

Discussion

Iridectomy performed during cataract surgery may be accompanied with certain complications, some of which are minor, but others may be more serious. These complications include:

1. The iris tissue necrosis and may be somewhat increased rate of iritis due to crushing of the tissues by forceps.
2. In some instances, the iridectomy may remain incomplete due to the pigmentary layer's remaining intact. This may necessitate a second operation, or application of laser, argon or YAG, to open the pigment epithelial layer in the iridectomized area.
3. A too large an iridectomy may turn out to be a simple cosmetic blemish, or be a source of glare, or may rarely be a cause of monocular diplopia, or it can result in the displacement of intraocular lens implant elements by their engagement or rotation into the iris opening. The anterior chamber angle-fixed lens can rotate ito the iridectomy and dislocate. The posterior chamber lens haptic can do the same, leading to implant-corneal endothelial touch.
4. Updrawn pupil, filtering scar, chronic irits, etc. may result from one or both arms of the iridectomy becoming incorporated into the wound.
5. An intraoperative hemorrhage from the incised iris, which sometimes is not easy to stop, may require intraocular cauterization. Often the hemorrhage may recur the next day or during the night. Such hemorrhages though mostly benign, may in rare instances lead to elevated intraocular pressure, iritis, increased corneal swelling, pupillary disfiguration, or adhesions inside the eye. Even if no disastrous consequences occur, the healing process may be slowed.
6. A vitreous loss may occur through the iridectomy, especially if the incision is done before the delivery of the lens. Such vitreous loss through the interruption of the zonules, however, does not seem to be as dangerous as vitreous loss through the pupil and can be managed through the iris opening without disrupting the posterior capsule.

Conversely, not making an iris opening can also lead to various problems:[16]

1. Peripheral anterior synechiae occur more readily in the non-iridectomized eye in the event there is a small wound leak. In such leaks, the iris sweeps into the faulty area of the wound, forming peripheral anterior synechia and sealing the leak at the same time. The iridectomy helps maintain the pressure in the anterior and the posterior chambers same, and a small and slow leak will not offset the pressure balance between anterior chamber and posterior chamber sufficiently to allow the iris to prolapse through the site of the leak. Since the leak lowers the intraocular pressure, the wound edges can adapt and a spontaneous correction of the leak can occur rapidly.

2. In the absence of an iridectomy, the adhesions between iris and capsular flap,[17-18] or between the iris and the intraocular lens, may lead to a late pupillary block.

3. Pupillary block may develop even with posterior chamber lenses, especially if they are not placed into the capsular bag and/or if they are not angled. In such cases, captive iris syndrome develops. An attempt to reposition such displaced lenses invariably fails. Even if it appears to be successful "on the table", within a few days the implant dislocates again. An iridectomy acts as prophylaxis against such problems.

4. In the rare cases of malignant glaucoma, the anterior hyaloid membrane can be disrupted with a Nd:YAG laser through the peripheral opening of the iridectomy. Vitreous entering the anterior chamber through the iridectomy as a result of such therapy does not seem to cause any dangerous adverse changes.

5. The cortex at 12 o'clock can be evacuated with ease through the iris opening and the anterior capsular flap placed over the superior edge of the IOL.

I have concluded from this study that by doing iridotomy the tranfixation technique described here, most of the advantages of a conventional iridectomy can be acquired with minimum of its complications in patients who undergo extracapsular cataract extraction with posterior chamber intraocular implantation.

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Ophthalmic "Pastpourri"

First IOL Implantation in a Pakistani

In **1953,** Harold Ridley, the father of intraocular lens implantation surgery, implanted the artificial lens he had designed in the eye of a Pakistani railway officer, who traveled to England for this purpose. In December, 1989, this patient appeared at a meeting of the Ophthalmological Society of Pakistan at Lahore. He was still seeing 20/30 (6/9) from the eye with Ridley's implant, and stated that he never had any problem with his eye since the operation.

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

Superior Limbic Kerato-conjunctivitis in Secondary Polycythemia

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

ABSTRACT: A 65-year-old woman complained of burning, light sensitivity, tearing and foreign body sensation in her eyes. She had been suffering from chronic obstructive pulmonary disease for many years, and her hemoglobin level varyied from 16.5 grams/dl to 18.1 grams/dl and hematocrit from 50 ml/dl to 55 ml/dl. In addition to bilateral retinal venous congestion and far peripheral blot hemorrhages, she developed the characteristic superior limbic keratoconjunctivitis. This case is unusual, because the patients with respiratory distress syndromes usually develop a generalized passive congestion of the conjunctiva. After the treatment with antibiotic-steroid drops had failed, this patient's symptoms dramatically responded to swabbing of the involved conjunctiva with cotton-tipped applicators soaked in 0.5% silver nitrate solution. (Pakistan Journal of Ophthalmology 7:86, 98, October, 1991.)

The Figure on page 86 shows that the conjunctival blood vessels in the superior bulbar conjunctiva are dilated. This inflammation also extended into the upper palpebral conjunctiva. Fluorescein staining showed punctate areas limited to the upper cornea adjacent to these dilated vessels. The findings were bilateral. The condition had been unsuccessfully treated by the patient's family physician. The application of a cotton-tipped applicator soaked in silver nitrate 0.5% solution cleared the symptoms and redness. All these findings point to the diagnosis of superior limbic keratoconjunctivitis as described by Theodore.[1]

The etiology of superior limbic keratoconjunctivitis is not clear.[2] Therefore, no definite treatment is available. Medical measures include, in addition to the above mentioned 0.5% silver nitrate solution, 10 to 20% solution of acetylcysteine three to five times a day or 4% solution of cromolyn every three hours. Surgical techniques include scraping of the superior bulbar conjunctiva, cryotherapy of the same site, pressure patching for a week, or recession (or resection) of the 2 to 5 mm strip of conjunctiva and the Tenon's capsule from 10 to 2 o'clock meridian. The last method may lead to scleral melt in the exposed area.[2]

Superior limbic keratoconjunctivitis is sometimes associated with other ocular conditions, such as dry eye syndrome, pseudoptosis, chronic blepharitis, etc.[2] These conditions should also be looked after accordingly.

An association of superior limbic keratoconjunctivitis with thyrotoxicosis has been previously considered.[3] However, any report of this condition in patients with polycythemia or chronic pulmonary obstructive disease has not, to our best knowledge, appeared in print.

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The ocular changes in primary or secondary polycythemia that have been so far reported include intense engorgement of the conjunctival, episcleral, and uveal vessels, marked venous stasis in the retina with occasional retinal hemorrhages and at times retinal venous obstruction, sometimes hemorrhagic glaucoma, and papilledema.[4,5] When present all these conditions are less severe in secondary polycythemia than in primary polycythemia. Rarely, hemangiomatous tumors in the posterior cerebral fossa may be the cause of the secondary polycythemia.[5] Vitreous hemorrhage[6] or optic disc swelling[7] rarely develops in patients with chronic respiratory distress conditions, such as sleep apnea.

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Book Reviews

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

WALSH AND HOYT'S CLINICAL NEURO-OPHTHALMOLOGY, 4th Edition, Volume 4, by Neil R. Miller, 1991. Williams and Wilkins, 428 East Preston Street, Baltimore, Maryland 21202, USA. 897 full-sized pages, 43-page index, illustrated, clothbound. Price, US $150.00.

A decade ago, the late Professor Frank B. Walsh, the father-author of *Clinical Neuro-Ophthalmology* , asked Miller to undertake revision of the 3rd edition of this great classic of ophthalmic literature. The third edition was a product of combined efforts of two giants of neuroophthalmology, Professor Walsh himself and Professor William F. Hoyt. The invitation to revise their masterpiece was like asking a modern playwright to revise a Shakespeare. Professor Miller felt honored, but the stature and the sanctity of the task also made him "distressed". Nonetheless, he accepted the challenge on the condition that Dr. Hoyt would review the manuscript. The results prove that not only was Walsh an astute observer of clinical manifestations of diseases, he also was equally deft at perceiving signs of greatness in the young healers around him. Both ophthalmology and those who engage in its practice are fortunate that Miller accepted this invitation.

This volume equals its three predecessors in its superior quality and contents. One difference being that in this volume many more neuroimaging illustrations are employed to provide up-to-date and more understandable depictions of clinical features. It is entitled "Vascular Lesions and Circulatory Disorders of the Nervous System," and covers almost every imaginable disorder of these systems which might even remotely affect the eye. Matching the standard established by the previous volumes of this prodigious undertaking, this volume is an awesome complication which is most extensively referenced from the earliest to the latest literature. The index spans 52 pages and serves more than adequately, containing diverse entries from "Abdominal migraine" to "Zygomatico-orbital artery." It even denotes the pages with figures by italicizing the page numbers and the pages with tables by adding "t" to the page number.

The contents of the book are divided into following seven chapters: Anatomy and Physiology of the Cerebral Vascular System, Aneurysms, Carotid-Cavernous Sinus Fistulas, Cerebrovascular Disease, Migraine, Vasculitis, and Venous Occlusive Disease. The topics are so numerous and discussions so extensive in each of these chapters that the book makes not only an authoritative and guiding source for the neuro-ophthalmologist but also a useful text for any physician interested in any ocular disorder. It is a magnificent reference source for the research minded as well. Though content coverage in each chapter is truly imposing, this reviewer was particularly impressed by the chapters on migraine, carotid-cavernous fistulas, and vasculitis. They are so comprehensive, well-written, and thoroughly referenced that each of them can independently serve as an exhaustive monogram.

The very lucid writing is so easy to follow that even those whose first language is not English will find the book appealing. The printing and paper are also excellent. Hence, this reviewer is of the opinion that no medical library should be without it, and the individuals with budget restrictions must place this volume at the top of their shopping list. This reviewer doubts that Miller's monumental accomplishment will ever become outdated, at least not in the present generation's lifetime. He recommends it to all physicians most enthusiastically. ❐

A COLOUR ATLAS OF LENS IMPLANTATION, edited by Peirs Percival, 1991. Wolfe Publishing Ltd, 2-16 Torrington Place, London WC1E 7LT. Large-sized (12½ inch x 10 inch) sized 318 pages, clothbound, illustrated with colored (mostly) and black and white figures, index, table of contents. Price, UK £ 110.00.

How popular intraocular lens implantation has become since the introduction, due to the advances in production technology, of safe and effective artificial lenses can be measured by the fact that in 1972 only about one thousand lens implantations were done world wide. A decade later, this annual figure had multiplied to a thousand-fold, and it is estimated that by 1992, nearly ten million lens implantation procedures will be performed each year in the world. This popularity of lens implantation, among surgeons and patients alike, has made it imperative for all ophthalmologists to become thoroughly familiar with every aspect and variation of this modern procedure. Hence, the need and timeliness of high quality books on this subject is quite apparent.

There are many books in the market on lens implantation. The purpose of this atlas, according to its Editor, is "to explain in pictorial form the options now available, the reasoning behind change and the management of problems and complications." The book is intended for "experts wishing to enhance their skills with the very latest information . . , as well as a guide for those just beginning in this new and exciting field."

In addition to a preface by the father of lens implantation, Mr. Harold Ridley, 55 international authorities on the lens implantation surgery, 11 from England, 25 from the United States, two each from Canada, France, Ireland, Israel, and Japan, and one each from Australia, Belgium, Federal Republic of Germany, Finland, Hungary, Kenya, The Netherlands,

Philippines, and Singapore, have contributed to the *Colour Atlas*. Their contributions have made the book a scholarly treatise on the subject, and not a mere pictorial rendition of various surgical steps and techniques.

The contents of the *Colour Atlas* are divided into eight sections under the headings of Extraction of Cataract, Lens Implantation and Postoperative Care, Preoperative Assessment, Special Cases: Precautions and Modifications to Technique, Histopathology, Management of Complications, The Posterior Capsule, and Epilogue. The last section contains discussion of cataract surgery in Asia, the problems in developing countries, and low cost surgery.

The book is beautifully printed on a high quality paper. The color illustrations and explanatory line drawing are of excellent quality and crisply demonstrate the points being made in the accompanying text. The text though concise is packed with important information. The contents are well up-to-date and each chapter provides at its end a short list of most significant and current "further readings." The writing is lucid and to the point. However, what makes this book of exceptional quality is its technical contents. In addition to nicely discussing the well-known and more common aspects of lens implantation surgery, the book provides impressive treatment of important but less frequently discussed topics, such as ophthalmic irrigation solutions, less familiar intraocular lens styles (the Lobster Claw Lens, for example), thrilling future possibilities in cataract surgery (laser phakoablation, for instance), etc.

This is one of the most impressive and informative publications on intraocular lens implant surgery, and deserves to be on the book shelves of all the lens implant surgeons and all the medical libraries. ❐

COLOR ATLAS/TEXT OF OPHTHALMIC PARASITOLOGY. By B.H. Kean, Tsieh Sun, and Robert M. Ellsworth, 1991. Igaku-Shion Medical Publishers, Inc., One Madison Avenue, New York, NY 10010. Full-sized 233 pages, colored and black 7 white illustrations, hardcover, index. Price, US $110.00.

All other evaluatory remarks aside, the very idea of bringing out a publication devoted entirely to parasitology related to ocular diseases is highly commendable and well-timed. This book is the result of combined labors of an expert in Tropical Medicine and Public Health (Professor Kean), a clinical pathologist (Professor Sun), and a highly respected ophthalmologist (Professor Ellsworth), all of Cornell University Medical College of New York. The very stimulus that led the senior author 30 years ago "to write a definitive study of the parasites of the human eye" signifies its need as well. Professor Kean states in the preface that the inspiration came from "the papers by Helena Wilder at the Army Medical Museum (now the Armed Forces Institute of Pathology), who made the final pathologic diagnosis of toxocariasis and toxoplasmosis on eyes that had been enucleated for syphilis, tuberculosis, and other mistaken diseases." Unfortunately, Professor Kean did not publish his book for reasons of his own. Enter Professor Sun, who accepted a recent offer by Professor Kean to rearrange and rewrite his unpublished manuscript for the present book. Since the intention was to write parasitology for ophthalmologists and not for parasitologists, input from an experienced ophthalmologist was essential. Professor Ellsworth filled that role.

The contents of *Atlas/Text* are divided into five parts, which are preceded by a one-page introduction and brief chapter on "Anatomy and Physiology of the Eye." The Part 1 contains discussions of protozoan diseases including toxoplasmosis, terramebiasis, entamebiasis, malaria, giardiasis, leishmaniasis, trypanosomiasis, pneumocystosis and microsporidiosis. Nematode infestations, toxocariasis, onchocerciasis, loiasis, filariasis, dracunculiasis, thelaziasis, gnathostomiasis, angiostrongyliasis, and trichinosis are the subject of Part II. Diseases caused by cestodes including cysticercosis, echnococcosis, coenurosis, and sparganosis are discussed in Part III. Part IV deals with two diseases caused by trematodes, schistsomiasis and paragonimiasis. The arthropod disease ophthalmomyiasis constitutes the bulk of Part V. Each chapter has a sufficient number of important and current references.

Discussion of each entity begins with epidemiology, then successively sheds light on parasitology, clinical features, pathology, and laboratory diagnosis, and concludes with treatment. Although adequate, these discussions are either too brief or deficient in some areas. For instance, in the chapter on toxoplasmosis the role of clindamycin, periocular corticosteroids, and photocoagulation is summarily dismissed in a single sentence. The authors emphasize that toxoplasmosis has been observed in only one member each of three sets of twins to support higher frequency of acquired retinochoroiditis, but do not mention the recent well-documented reports of congenital toxoplasmosis in living siblings. They also reject toxoplasmosis as a cause of anterior uveitis, retinal vasculitis, or geographical choroiditis, without clarifying that either severe anterior segment inflammation or retinal vasculitis may accompany the typical recurrent, proven toxoplasmic chorioretinitis. This reviewer has also seen at least two patients with geographic chorioretinal lesions due to recurrent toxoplasmosis. More detailed discussions in treatment sections would have definitely made this book more useful to the clinicians. It is hoped that this aspect will receive serious consideration at the time of revision for the second edition.

A drawback in the arrangement of contents is that all the related figures are lumped together at the end of each chapter. This makes referring to them during reading of the text very inconvenient, and because of

this these figures will not give the reader their intended benefit. This reviewer also sorely missed the markers to point out the features of the histopathologic figures. For instance, out of 10 histopathologic figures on pages 28 and 29 only one has an arrow pointing to its critical feature. The authors should not forget that only a very small percentage of their intended readers has significant background in histopathology. Unfortunately, the reproduction of almost all of the borrowed figures is less than satisfactory. This reviewer found the sketched lifecycles of most of the discussed parasites, appended at the end of chapters, most helpful. All in all this is a timely and valuable publication, which all ophthalmologists will find highly useful.❐

EYE TRAUMA, by Bradford J. Shingleton, Peter S. Hersh, and Kenneth R. Kenyon, 1991. Mosby-Year Book, Inc., 11830 Westline Industrial Drive, St. Louis, MO 63146. Clothbound, 427 pages, 526 black and white illustrations, 70 color illustrations, index. Price, UK £70.00.

This latest of the recent publications on ocular trauma is a result of the efforts of three editors, two associate editors, and 52 authors (including the editors). The objectives of the editors were to "provide a broad and definitive source of information relating to ocular injury" and "to provide practical, hands-on information to the practitioner, whether in office, emergency room, or operating suite." The intended readership of the *Eye Trauma* is the general ophthalmologist, subspecialist, ophthalmologist-in-training, and any other physician who is interested in injuries of the eyes. The book attempts to meet these vast objectives in a beautifully printed and very handy volume of 427 pages. The text is divided into six parts, Evaluation and Surgical Preparation of the Eye Trauma Patient, Anterior Segment Trauma, Posterior Segment Trauma, Orbital and Adnexal Trauma, Neuroophthalmic Trauma, and Injury Prevention and Medicolegal Aspects of Eye Trauma. Each part is further subdivided into many chapters that contain much more diverse material than is apparent from the above titles of the book sections.

The chapters that stand out include Chapter 2 on "Diagnostic imaging of ocular and orbital trauma," in which the author guides the reader very clearly and concisely, adding here and there innovative hints that are helpful in examining the traumatized eye. Chapter 5 on "Evaluation and management strategies for the pediatric eye trauma patient" include useful discussion of birth trauma, self-inflicted injuries in retarded children, and very important subject of child abuse. It is very astute of the editors to include a short chapter on "Pathophysiology of anterior segment wound healing," and another on techniques of suturing corneal lacerations. The chapter on "Traumatic endophthalmitis" is well-written, and the authors here have wisely included tables on infecting organisms, recommended drug therapy, including a separate table on therapy of fungal endophthalmitis. There is one drawback in this chapter that no step by step instructions are given on the preparation of antibiotic solutions for intravitreal injections from the standard commercial products. Addition of such a table in the second edition would definitely make this chapter more useful. The chapters on "Epidemiology and prevention, "Medicolegal considerations," and "Guidelines for tetanus prophylaxis" in the last section are very practical additions.

Thera are a few minor aspects which could have been presented in a more useful manner. For instance, the large number of cross-references scattered throughout the book are indicated by chapter numbers only. It would have been more convenient and less time consuming for the reader if the page numbers had been employed instead. Also, in view of the fact that many of the color illustrations could have been equally effective in black and white, it would have been better if in their stead some other figures, such as Fig. 23-2 of Gram staining on page 144, were reproduced in color. On the same page, the legend of Fig.23-1 includes the words "displaying extreme eyelid edema," but the eyelids have been entirely cropped out of the reproduced illustration.

A few omissions in the text are exemplified by the discussion on dislocated lens. Although sufficient details of complications and management of the intact dislocated lens into the vitreous are provided, nothing is included on the dislocation of the nucleus or a cortical chunk into the vitreous, which, one would agree, is a relatively more important topic in today's extracapsular cataract surgery environment. On page 245 the unit of intravitreal dose of dexamethasone is missing in the table on drug therapy of traumatic endophthalmitis, obviously a typographical error.

One of the few errors of commission is on page 127, where the authors state without citing any reference that phacoanaphylactic uveitis was first described by Verhoeff, no doubt a true giant of ocular pathology in his own right. Nevertheless, the truth remains that a Dutch ophthalmologist from Amsterdam, named Manuel Straub, described in great detail this entity in,1919, three years before the paper by Verhoeff titled "Endophthalmitis Phacoanaphylactica" appeared in print. (See *American Journal of Ophthalmology*, 48:463-472, 1959.) The statement on page 160 that the "perpendicular central incisions (of the cornea) open spontaneously under the influence of normal intraocular pressure" needs further clarification in that this is true only if such openings are longer than 2-3 mm. The central corneal penetrations smaller than this length have a tendency to self-seal. This may not be true of the peripheral lacerations, which may show persistent or delayed leaking when not spontaneouly plugged by the iris tissue soon after the injury.

These few minor points in this critique should not detract one from many good points of this book, which surely has ample usefulness for its intended readers. ❐

Abstracts From Elsewhere

Edited by Khalid J. Awan, FPAMS

New England Journal of Medicine

ANALYSIS OF AQUEOUS HUMOR IN OCULAR TOXOPLASMOSIS, AP Brezin, CE Eqwuagu, C Silveira, P Thulliez, MC Martins, RM Mahdi, R Belfort, Jr., RB Nussenblatt. The authors report the results of their use of the polymerase chain reaction to identify *Tosoplasma gondii* in the aqueous humor of patients with presumed ocular toxoplasmosis. Currently, the diagnosis of ocular toxoplasmosis is based clinically on the observation of a necrotizing lesion in the fundus. However, in cases of atypical retinitis or when the fundus is masked by vitreal inflammation, aqueous-humor analysis may be used as a diagnostic tool.

Using the polymerase chain reaction, they detected *T. gondii* DNA in the aqueous humor of three patients during two independent amplifications of the B1 gene fragment. The amplified product was detected by Southern blot analysis with an oligonucleotide internal to the amplified fragment. No other method has been reported to date that can identify the parasite in human aqueous humor. No association between local antibody production and detection of the parasite DNA in the aqueous humor was found. Their data also supports the concept that proliferation of the parasite might not be the only factor leading to the inflammatory response observed during ocular toxoplasmosis. *(N Engl J Med 324:699, March, 1991.) Reprint requests to Robert B. Nussenblatt, M.D., National Eye Institute, Bethesda, M.D. 20892.*

NITROGLYCERIN TO TREAT ACUTE LOSS OF VISION. JM Ritter. The author and his colleagues have studied the effects of nitroglycerin and suspensions of human vascular smooth-muscle cells on platelet aggregation and found that specific amounts of these two agents were without effect when added singly, but profoundly inhibited aggregation when added in combination. This effect was prevented by hemoglobin, a potent inhibitor of nitric oxide (endothelium-derived relaxing factor), suggesting that inhibition of platelet aggregation by glyceryl trinitrate in the presence of vascular muscle is due to the generation of nitric oxide. It seems possible that nitroglycerin is metabolized to nitric oxide by vascular smooth muscle in retinal vessels, which activates guanylate cyclase in vascular smooth muscle and in platelets, causing relaxation of blood vessels and inhibition of platelet adhesion and aggregation, respectively. This combination of actions could be uniquely effective in inhibiting thrombosis in vivo and might underlie the successful treatment of retinal-artery thrombosis. *(N Engl J Med 324:997, March, 1991.) Reprint requests to James M. Ritter, F.R.C.P., Guy's Hospital, London SE1 9RT, United Kingdom.*

TOTAL RECOVERY OF VISUAL FUNCTION AFTER TREATMENT FOR CEREBRAL CYSTICERCOSIS. KJ Propert, RO Dillman, M Green. The authors discuss that brain cysticercosis is now treated effectively with either albendazole or praziquantel, but most studies demonstrating the efficacy of cysticidal drugs have been based on objective evidence from imaging studies of the disappearance of cysts after cysticidal therapy. Even with this evidence, some authors have doubts about the real benefits of cysticidal therapy when weighed against the possibility of adverse reactions in the host due to the acute inflammation triggered by the destruction of the parasites. The authors treated, with albendazole, a 43-year-old woman with three large cysticerci at the base of the brain and loss of sight. Albendazole (15 mg per kilogram of body weight daily for eight days) was started. In addition, dexamethasone (8 mg every eight hours) was started. In addition, dexamethasone (8 mg every eight hours) was administered intravenously. A notable improvement of visual function occurred a few days after the beginning of therapy. At the end of treatment, her bilateral visual acuity was 20/20 with normal visual fields, and magnetic resonance imaging documented the successful destruction of cysticerci. *(N Engl J Med 324:1137-1139, April, 1991.) Reprint requests to National Institute of Neurology and Neurosurgery of Mexico, 14410 Mexico 22, D.F.*

THERAPEUTIC USES OF BOTULINUM TOXIN. J Jankovic, MF Brin. The authors talk of botulinum toxin type A (hereafter referred to as botulinum toxin), one of the most lethal biologic toxins, has been found to be of therapeutic value in the treatment of a variety of neurologic and ophthalmologic disorders. The Food and Drug Administration recently approved botulinum toxin (Oculinum as a therapeutic agent in patients with

strabismus, blepharospasm, and other facial-nerve disorders, including hemifacial spasm.

The neurotoxic component of botulinum toxin has a molecular weight of only 150,000, but the toxin forms a complex with nontoxic proteins and hemagglutinin. The toxin exerts its paralytic action by rapidly and strongly binding to presynaptic cholinergic-nerve terminals. The toxin is then internalized and ultimately inhibits the exocytosis of acetylcholine by decreasing the frequency of acetylcholine release. The treatment of muscle with botulinum toxin results in an accelerated loss of junctional acetylcholine receptors. Paralysis and a nearly complete decline of miniature end-plate potentials occur within a few hours after the injection of botulinum toxin. The delay in the onset of clinical effect may be related in part to the spontaneous release of acetylcholine. The muscle becomes functionally denervated, atrophies, and develops extrajunctional acetylcholine receptors. Within two days after muscle exposure to the toxin, the axon terminal begins to sprout, and the proliferating branches then form new synaptic contacts on the adjacent muscle fibers.

Botulinum toxin was initially used to weaken extraocular muscles. The toxin was then introduced for the treatment of strabismus as an alternative to conventional incisional surgery. The injections are performed under electromyographic guidance with a Teflon-coated needle to ensure accurate placement in the extraocular muscle. Most patients are treated in the office with topical anesthesia; children under seven years of age may require light ketamine anesthesia and restraint. Follow-up studies up to five years after the injection show that 85 percent of the patients available for reassessment had satisfactory improvement. Side effects, including partial ptosis and secondary vertical deviations, are usually transient and do not result in amblyopia. Others present a less optimistic outcome for infantile esotropia with only 33 percent of the patients being adequately controlled with botulinum toxin.

Botulinum toxin has been injected into extraocular muscles to treat comitant strabismus, vertical strabismus, lateral rectus palsy, nystagmus, and dysthyroid myopathy.Other chief therapeutic use is for the treatment of focal dystonias, writer's cramp and retrocollis, and blepharospasm.

Blepharospasm is a form of focal dystonia manifected by intermittent or sustained closure of the eyes due to involuntary contractions of the orbicularis oculi. It is often accompanied by spasms of facial, oromandibular, pharyngeal, laryngeal, and neck muscles (cranial-cervical dystonia or Meige's syndrome). The severity of symptoms may range from increased blinking to functional blindness as a result of sustained, sometimes painful, forceful closure of the eyelids. Medications such as clonazepam, lorazepam, baclofen, and trihexyphenidyl provide symptomatic benefit in up to a third of these patients.

The beneficial effects of injections of botulinum toxin in the treatment of blepharospasm have been demonstrated in one controlled and several open trials. There is moderate-to-marked symptomatic and functional improvement in 70 to 90 percent of the treated patients. The time between the injection and the onset of improvement was about two to five days, and the benefits lasted an average of 3 1/2 months. There was no demonstrable decline in efficacy after repeated injections. The most common adverse effects, all self-limited, included ptosis, blurred vision, diplopia, local pain and swelling, entropion, and increased tearing. Because of its proved safety and efficacy, botulinum toxin is now considered a primary form of therapy for blepharospasm.

Ptosis induced by injecting botulinum toxin into the levator palpebrae superioris has been used to protect the cornea and to treat eyelid entropion, intractable orbicularis myokymia, and other eyelid disorders.

Other conditions treated by Botulinum injection are cervical dystonia, oromandibular dystonia, spasmodic dysphonia, tremors, and hemifacial spasm.

Chemical denervation with botulinum toxin is now considered by many the treatment of choice of blepharospasm, cervical dystonia (torticollis), laryngeal dystonia (spasmodic dysphonia), certain task-specific dystonias (occupational cramps such as writer's cramp), and hemifacial spasm. The therapy may also be useful in patients with other forms of dystonia and in those with certain focal repetitive involuntary movements, such as tremor, tics, segmental myoclonus, and other hyperkinetic movement disorders. Motor dysfunction due to abnormally increased muscle tone, such as spasticity, may also be ameliorated by treatment with botulinum toxin.

It is hardly necessary to emphasize that injections of botulinum toxin should be administered only by clinicians thoroughly knowledgeable about the physiologic as well as the clinical effects of the toxin. Familiarity with local anatomy is necessary to ensure proper and safe administration. In addition to the required skills in the technique of administration, the clinicians should be experienced in the recognition and management of the various disorders for which treatment with this potent drug is planned. Finally, it is a prudent clinical practice to inform patients about the alternative therapies, limited duration of benefit, possibility of poor or no response, potential complicaitons, and relative paucity of information on the effects of long-term treatment with botulinum toxin. *(N Engl J Med 324:1186-1193, April, 1991.) Reprint requests to Joseph Jankovic, M.D., at the Movement Disorders Clinic, Department of Neurology, Baylor College of Medicine, 6550 Fannin 1801, Houston, TX 77030.*

LISCH NODULES IN NEUROFIBROMATOSIS TYPE 1. ME Lubs, MS Bauer, ME Formas, B Djokic. The authors stated that

Lisch nodules are malanocytic hamartomas that appear as well-defined, dome-shaped elevations projecting from the surface of the iris and are clear to yellow or brown. Lisch nodules appear to be found only in patients with peripheral neurofibromatosis (neurofibromatosis type 1, or von Recklinghausen's disease), an autosomal disorder with a prevalence of 1 in 3500, and are the most common clinical feature of neurofibromatosis 1 in adults.

The diagnosis of neurofibromatosis 1 is often difficult in children, who have multiple cafe au lait spots but no other symptoms or signs. This study was designed to determine the prevalence of Lisch nodules in patients with neurofibromatosis 1 and to assess their usefulness in the diagnosis of the disorder.

The authors found these nodules in 100 percent of the patients over the age of 20 who had neurofibromatosis 1. No association was found between Lisch nodules and the overall clinical severity of neurofibromatosis 1 in these patients. Multiple Lisch nodules, unlike cafe au lait spots and neurofibromas, are specific for neurofibromatosis 1. They have not been found in patients with central neruofibromatosis, except in one case. Patients with segmental neurofibromatosis, which is due to a somatic mutation of the neurofibromatosis 1 gene, may have Lisch nodules.

The observation that all patients above the age of 20 who had neurofibromatosis 1 also had Lisch nodules is important for genetic counseling, since it permits a distinction to be made between minimally afffected and unaffected parents. All apparently unaffected parents (who constitute the parents of half the affected children) and all children whose status is in question should therefore have a thorough ophthalmologic examination as part of their evaluation so that appropriate genetic counseling can be provided. If the diagnosis is in doubt and a child has no Lisch nodules, the examination should be repeated periodically. It is particularly useful since Lisch nodules often appear before neurofibromas. *(N Engl J Med 324:1264-1266, May, 1991.) Reprint requests to Milsen S. Bauer, M.D., at Miami Children's Hospital, Miami, FL 33155.*

CLINICAL AND LABORATORY FINDINGS IN THE OCULOCEREBRORENAL SYNDROME OF LOWE, WITH SPECIAL REFERENCE TO GROWTH AND RENAL FUNCTION. LR Charnas, I Bernardini, D Rader, JM Hoeg, WA Gahl. The authors investigated growth, renal function, and serum chemistry values in patients with the oculocerebrorenal syndrome to determine the natural history of the disorder and its heterogeneity with respect to these characteristics.

Renal glomerular deterioration is slowly progressive in the oculocerebrorenal syndrome. Renal tubular dysfunction begins early and persists; most patients require alkalinization therapy, and many benefit from supplemental potassium, phosphate, calcium, or carnitine. Serum enzyme elevations suggest muscle involvement n the oculocerebrorenal syndrome. *(N Engl J Med 324:1318-25, May, 1991.) Rerint requests to William A. Gahl, M.D., Ph.D., at the Human Genetics Branch, NICHD, NIH, Bldg. 10, Rm. 9S242, 9000 Rockville Pike, Bethesda, M.D. 20892.*

AIR-BAG KERATITIS. HJ Ingraham, HD Perry, ED Donnenfeld. The authors report ocular injury in two patients , one a two-year-old child, from bursting of an automobile air-bag. The eyes had erythema of the facial skin and eyelids and moderate conjunctival injection. Both corneas showed focal clouding, with large, interpalpebral epithelial defects. The tear pH was 8.5 to 9.0 in both eyes. Double eversion of the eyelids and sweeping of the conjunctival fornices yielded a small amount of particulate foreign material. Each eye was irrigated with balanced salt solution, followed by 3 liters of Ringer's solution, with use of an irrigating scleral lens. The pH immediately after the 30 minutes after irrigation was 7.5 in both eyes. One month later, the child's vision returned to 20/40 in the right eye and 20/30 in the left, with residual, small, superficial corneal scars. The child is being monitored closely by a pediatric ophthalmologist for the possible development of amblyopia.

An automobile air bag is a rubberized nylon bag that inflates on spark ignition of sodium azide and yields nitrogen gas, ash, and a small amount of sodium hydroxide. These products most likely were responsible for the ocular injuries and facial burns sustained by the patients. We have subsequently examined a second patient who sustained chemical burns to the face and eyelids, without keratitis, after an air bag inflated and burst. The authors were surprised to see a chemical keratitis in this setting, and we hope their report will alert others to this potentially serious and vision-threatening entity. *(N Engl J Med 324:1600, May, 1991.) Reprint requests to Herbert J. Ingraham, M.D., North Shore University Hospital, Manhasset, NY 11030.*

SUCCESSFUL TREATMENT WITH HYDROXYUREA OF OCULAR INVOLVEMENT IN CHRONIC MYELOMONOCYTIC LEUKEMIA. PG Wolff-Korman, GC Hazenfratz, B Heinrich. The authors report an 85-year-old man with myelomonocytic leukemia. He had intraocular infiltration and exudative retinal detachment that resolved rapidly after chemotherapy with hydroxyurea. He received a dose of 1 G daily for several weeks, and then was kept on a maintenance dose for eight months. *(N Engl J Med 325:209, August, 1991.) Reprint requests to Universtiy Eye Hospital, 8 Minich 2, Germany.*

TREATMENT OF MIGRAINE ATTACKS WITH SUMATRIPTAN. The Subcutaneous Sumatriptan International Study Group. The

authors studied 639 patients with migraine attacks in a randomized, double-blind, placebo-controlled, parallel-group clinical trial. They assessed the effect of subcutaneous injections of 6 or 8 mg of sumatriptan or placebo on the severity of headache and associated migraine symptoms 30, 60, and 120 minutes after treatment. Patients who were not free of pain after 60 minutes subsequently received placebo if they had initially received placebo or 8 mg of sumatriptan, and 6 mg of sumatriptan.

After 60 minutes, the severity of headache was decreased in 72 percent of the 422 patients given 6 mg of sumatriptan, 79 percent of the 109 patients given 8 mg of sumatriptan, and 25 percent of the 105 patients given placebo. The authors conclude that a single 6-mg dose of sumatriptan given subcutaneously is a highly effective, rapid-acting, and well-tolerated treatment for migraine attacks. The administration of a second dose 60 minutes later to patients not responding well to an initial dose affords little additional benefit. *(N Engl J Med 1991; 325:316-21.) Reprint requests to Michel D. Ferrari, M.D., at the Department of Neurology, University Hospital, P.O. Box 9600, 2300 R C Leiden, the Netherlands.*

TREATMENT OF ACUTE CLUSTER HEADACHE WITH SUMATRIPTAN. The Sumatriptan Cluster Headache Study Group. The authors conducted a randomized, double-blind, placebo-controlled crossover study to assess the efficacy and tolerability of sumatriptan in 49 patients with cluster headache. The patients received, in random order, a subcutaneous injection of 6 mg of sumatriptan for one cluster-headache attack and placebo for another attack. The results for the two attacks could be fully evaluated for 39 patients. A response to treatment was defined as complete or almost complete relief of headache (no pain or mild pain) within 15 minutes after the injection.

In the 39 patients, the severity of headache decreased in 74 percent of the attacks within 15 minutes of treatment with sumatriptan, as compared with 26 percent of the attacks for which placebo was given. Thirty-six percent of the patients were free of pain within 10 minutes after the administration of sumatriptan, as compared with 3 percent after placebo; by 15 minutes these numbers had increased to 46 percent of the patients required oxygen as an additional treatment 15 minutes after receiving sumatriptan, as compared with 49 percent of those who received placebo. The severity of functional disability and the incidence of ipsilateral conjunctival injection also decreased more in response to sumatriptan than placebo. Sumatriptan was well tolerated, and there were no serious adverse events.

Sumatriptan is an effective and well-tolerated treatment for acute attacks of cluster headache. *(N Engl J Med 1991; 325:322-6.) Reprint requests to Karl Ekbom, M.D., at the Department of Neurology, Soder Hospital, S-11883 Stockholm, Sweden.*

ERYTHROPOIETIN AND VISUAL HALLUCINATIONS. H Steinberg. The author draws attention to the observation during the past year of five patients on hemodialysis who had visual hallucinations while taking erythropoietin. None of these patients had any evidence of delirium or psychosis, and there was no other medication or physical condition that could account for the visual hallucinations. There were no auditory hallucinations, disorientation, or cognitive impairments.

The hallucinations were of objects moving in front of the patient, commonly people and sometimes cartoon figures on a wall, or animals or birds. The patients were puzzled and curious but not terrified by the hallucinations, although one woman was afraid to look at the ceiling because she saw "black marks that move(d) and twinkle(d) like electricity." One man described waking up at night thinking that he was working in his job as a plumber. He saw himself fixing a faucet with a wrench, but his vision disappeared when his wife called to him and asked him what he was doing.

These hallucinations occurred after the patients had received erythropoietin for 2 to 13 months at doses ranging from 2000 units once a week to 4000 units three times a week. Four patients stopped taking erythropoietin, and the visual hallucinations disappeared within one to three weeks. All four patients later resumed erythropoietin therapy, and two had recurrences within three to four weeks, which disappeared after the erythropoietin was again stopped. Ther fifth patient was treated with perphenazine but continued to take erythropoietin; the hallunications disappeared and did not recur after treatment with perphenazine was stopped.

If the hallucinations are truly due to the erythropoietin, the cause is unclear. Further confirmation of visual hallucinations due to erythropoietin would be necessary to verify this as an important side effect and to explore the possible central nervous system mechanisms of the drug. *(N Engl J Med 325:285, July, 1991.) Reprint requests to Herbert Steinberg, M.D., Long Island Jewish Medical Center, New Hyde Park, NY 11042.*

EYE INJURIES. BJ Shingleton. The author reviews the evaluation of the injured eye and present guidelines for early identification and treatment of chemical burn, ruptured globe, and hyphema.

Chemical injury is the only eye problem for which emergency treatment is begun before visual acutiy is measured. Irrigation is the treatment of choice, to remove the offending chemical and neutralize the surface pH. Patients who call a physician's office or emergency department for advice about direct contact with a chemical at home or work should be instructed

to begin irrigation immediately. When they arrive at the physician's office or emergency department, more irrigation should be given at once. It is impossible to overirrigate a chemically burned eye. Intravenous tubing (with the eyelids retracted by hand) is the most effective way to deliver the irrigating solution to the ocular surface. There is no need to search for neutralizing solutions; any nontoxic solution will suffice. Irrigation should be continued for at least 10 minutes, and the pH should then be checked. Any particulate matter should be removed.

After irrigation, topical steroids, ascorbate, citrate, antibiotics, and cycloplegic agents to minimize inflammation and the risk of infection should be started. Systemic ascorbate is commonly used, and elevated intraocular pressure must be treated, if present. Patching, and sometimes the application of a contact lens, is required to facilitate epithelialization of the corneal surface. In the early phase of repair (days 7 to 21) epithelialization is the primary goal. It may be aided by topical lubrication and temporary or permanent surgical closure of the lid. Topical corticosteroids are oftern replaced with progestational steroids to minimize the lysis of collagen in the corneal stroma. In the late phase of healing (after day 21), all efforts are directed toward maintaining epithelialization and minimizing vascularization in the visual axis. If epithelialization is impaired, conjunctival transplantation or the grafting of mucous membrane may be required. Once the ocular surface has been stable for an extended period, penetrating keratoplasty (corneal transplantation) is occasionally undertaken to restore a clear cornea.

Ruptured Globe (Corneal Laceration) may present a tricky situation, because upto 20 percent of patients with ruptured globes do not have readily apparent signs of perforation. Vision may remain excellent, and the most important clues to occult rupture may be found in what the patient was doing at the time of the injury. The ovaling or tenting of the pupil into a pear shape may be an important sign. Patients with hyphema must be considered to have a ruptured glove until proved otherwise.

If a ruptured globe is suspected or confirmed during examination, a metal shield should be placed over the involved eye to protect it. Minor manipulation of a ruptured glove may exacerbate an already serious problem. Patching is not recommended. Broad-spectrum intravenous antibiotics with gram-positive and gram-negative coverage should be given. Topical antibiotics are generally avoided before surgery for fear of incorporating concentrated antibiotic into the eye. Radiography and CT scanning are indicated to detect intraocular foreign bodies.

The application of a soft contact lens may be enough to treat a corneal laceration with a formed anterior chamber. Tissue adhesive (cyanoacrylate glue) is occasionally used. If there is any question about the stability of the wound or the patient's degree of compliance, suturing with 10-0 nylon is indicated. The placement of sutures should be guided by rules designed to minimize postoperative astigmatism.

When uveal tissue (iris, ciliary body, or choroid) is incarcerated in the laceration, the uveal tissue should be repositioned if at all possible. Only devitalized tissue that has been outside the eye for an extended period should be excised. The lens and vitreous may need to be removed with automated cutting and aspiration equipment. Rarely, corneal or scleral tissue is lost, and lamellar-inlay grafting can be performed with donor tissue. Full-thickness penetrating keratoplasty or patch grafts with sclera from an eye bank may be needed to restore the integrity of the glove in rare cases. After surgery most patients with routine corneal lacerations receive three to five days of intravenous antibiotics and extended treatment with topical antibiotics and cycloplegic agents. Days to months after primary repair of the laceration, new techniques involving conjunctival grafting, keratoplasty, iris surgery, and automated cutting and aspiration of membranes may be used to reconstruct the anterior segment. Rehabilitation of the posterior segment has been aided by the development of bimanual, intraocular retina - vitreous instruments and the use of intraocular fluids, oils, gases, and antibiotics.

If the visual potential is nil, enucleation of the injured eye must be considered. With enucleation, the development of a painful and cosmetically unacceptable eye can usually be avoided. In addition, surgical removal within two weeks after primary repair greatly reduces the risk of sympathetic ophthalmia, which occurs in fewer than 2 accidental injuries per 1000. The cause is unclear but appears to be based on an autoimmune mechanism. Systemic therapy with prednisone and immunosuppressive agents greatly improves the rate of success once sympathetic ophthalmia is present.

Twenty-five to 35 percent of patients with hyphema also have other eye damage. Rebleeding, glaucoma, and staining of the cornea with blood are three of the most important complications. Since even the smallest of hyphemas may rebleed and lead to further problems, it is important that hyphema be detected on initial examination. Approximately one third of patients with hyphema have associated glaucoma, corneal damage, cataract, choroidal rupture, or posterior-segment bleeding. The eyes with hyphema should be protected with a metal shield and the globe treated as ruptured until proved otherwise.

Salicylates and nonsteroidal anti-inflammatory agents are avoided because they prolong the bleeding time and may exacerbate bleeding. Cycloplegic agents, such as 1 percent atropine, are commonly used to minimize inflammation and enhance observation of the posterior segment. Topical steroids reduce

inflammation and systemic steroids may reduce the rate of rebleeding. Antifibrinolytic agents (aminocaproic acid and tranexamic acid) have been documented by some authors to reduce rebleeding. The side effects, however, which include nausea, vomiting, and postural hypotension, are not innocuous. Given the relatively low incidence of severe complications, even in those in whom there is rebleeding, a clear role for antifibrinolytic agents in patients with hyphema has not been fully established.

Surgical intervention is indicated in the early period after injury in cases of persistently elevated intraocular pressure, staining of the cornea with blood due to stromal retention of products of red-cell degradation, and prolonged clots. Paracentesis with a washing out of blood from the anterior chamber is the simplest and safest surgical procedure. Late sequelae reflecting damage to the cornea, lens, filtration angle, and retina may make long-term medical therapy and additional surgery necessary.

Patients with sickle cell disease and sickle cell trait are predisposed to the sickling of red cells in the anterior chamber. Rigid sickle cells are less able to leave the eye through normal pathways of drainage. Hypoxia, acidosis, and hypercapnia develop in the anterior chamber, leading to increased sickling. The intraocular pressure rises, and damage to the optic nerve may result. The threshold for surgical intervention in such patients should be low, in order to break this vicious circle and remove sickled cells from the anterior chamber.

Seventy-five percent of patients with hyphema end up with large hyphemas, patients with episodes of rebleeding, and children may have poorer prognoses. Persistent visual reduction is most commonly due to associated damage in the posterior segment.

The National Society for the Prevention of blindness estimates that more than 2 1/2 million eye injuries occur each year in the United States. There are 423 acute hospital-treated eye injuries per 100,000 residents, and 30,000 admissions per year are directly attributable to eye injury in the United States. Fortunately, 95 percent of the injuries are categorized as minor, but even these injuries were associated with a temporary loss of vision, the need for topical medications and follow-up care, and time lost from work or school.

Most eye injuries are preventable. Effective and cosmetically acceptable eye protection is available for work, home, and sports activities. Polycarbonate lenses and frames provide the best protection. Industrial goggles with side shields are important in the workplace. Glass lenses, contact lenses, ordinary plastic lenses, and lensless eye guards do not give adequate protection. *(N Engl J Med 325:408-413, August, 1991.) Reprint requests to Bradford J. Shingleton, M.D., at 50 Staniford St., Boston, MA 02114.)*

A CONTROLLED TRIAL COMPARING VIDARABINE WITH ACYCLOVIR IN NEONATAL HERPES SIMPLEX VIRUS INFECTION. R Whitley, et al., and The National Institute of Allergy and Infectious Diseases Collaborative Antiviral Study Group. Despite the use of vidarabine, herpes simplex virus (HSV) infection in neonates continues to be a disease of high morbidity and mortality. The researchers undertook a controlled trial comparing vidarabine with acyclovir for the treatment of neonatal HSV infection.

In this multicenter, randomized, blinded study there were no differences in outcome between vidarabine and acyclovir in the treatment of neonatal HSV infection. The study lacked statistical power to determine whether there were sizable differences within the sub-groups of those with localized HSV, encephalitis, or disseminated disease. *(N Engl J Med 1991; 324:444-9.) Reprint requests to Richard Whatley, M.D., Departments of Pediatrics, University of Alabama at Birmingham, Birmingham, AL.*

GIANT-CELL ARTERITIS. R. Stern. The author a rheumatologist, objects to the statemtent that "10 to 18 percent of patients presenting with the syndrome of polymyalgia rheumatica and giant-cell arteritis prove to have an underlying malignant tumor," made in June 7, 1991, issue of the Journal. According to most reviews of this subject, there does not appear to be an increased association between either polymyalgia rheumatica or temporal arteritis and malignant conditions. In fact, when malignant tumors do occur, as they can in any population, there is no indication that any particular type is prevalent. He refutes that "polymyalgia rheumatica is rarely associated with malignance."

This is not to say that patients with malignant tumors cannot present with a myalgic picture similar to polymyalgia rheumatica. The classic picture of polymyalgia rheumatica (proximal myalgias, responsiveness to steroids, and an elevated erythrocyte sedimentation rate) is nonspecific, and perhaps it would be best to consider such patients as having a myalgic syndrome. The most common cause of such a syndrome is probably polymyalgia rheumatica - temporal arteritis complex, but other conditions may also be responsible, including various myopathies, rheumatoid arthritis, and rarely, malignant conditions. Goth polymyalgia rheumatica and temporal arteritis can present with anorexia and weight loss as their predominant feature in a picture that has been described as "malignoid." Lastly, there are case reports of malignant tumors presenting as temporal arteritis, but isolated case reports do not imply an association. *(N Engl J Med 324:496-497, 1990.) Reprint requests to Richard Stern, M.D., Hospital for Special Surgery, New York, NY 10021.*

Editor's note: There are reports of cancer in patients with

giant-cell arteritis, though only in a small percentage of cases. It appears safer to stay alert to the possibility that the patients with polymyalgia rheumatica may rarely have or subsequently develop cancer.

ADVANCED GLYCOSYLATION END PRODUCTS IN PATIENTS WITH DIABETIC NEPHROPATHY. Z Makita, S Radoff, EJ Rayfield, Z Yang, E Skolnik, V Delaney, EA Friedman, A Cerami, and H Vlassara. The authors state that glucose reacts nonenzymatically with proteins in vivo, chemically forming covalently attached glucose-addition products

and cross-links between proteins. The excessive accumulation of rearranged late-glucose-addition products, or advanced glycosylation end products (AGEs), is believed to contribute to the chronic complications of diabetes mellitus.

AGEs accumulate at a faster-than-normal rate in arteries and the circulation of patients with diabetes; the increase in circulating AGE peptides parallels the severity of renal functional impairment in diabetic nephropathy. *(N Engl J Med 1991; 325:836-42.) Reprint requests to Helen Vlassara, M.D., at the Pickower Institute for Medical Research, 350 Community Ave., Manhasset, NY 11030.*

TRANSIENT MONOCULAR BLINDNESS CAUSED BY VASOSPASM. SK Bruger, RF Saul, JB Selhort, SE Thruston. The authors describe five patients with transient visual loss due to clinically observable vasospasm. In two patients, one 59-year-old man and the other a 78-year-old woman, vasospasm was photographically recorded and in the other three it was ophthalmoscopically observed.

Of six previously reported cases of transient monocular visual loss with retinal vosospasm, two occurred in teenagers and three in middle-aged patients. Their five patients were older. This confirms the suggestion that nonembolic transient visual symptoms are not restricted to the young and healthy. These previously described stereotypical episodes of monocular blindness were also brief, lasting no longer than five minutes in five of the six patients. As in authors' patients, three of these patients had 1 to 12 such episodes per day. Four of the six patients also had a diffuse or constricting loss of a visual field.

Two of their patients with vasospastic transient monocular blindness had pathologically confirmed temporal arteritis. Vasospastic transient monocular blindness has also been reported in patients with pathologically proved periarteritis nodosa, or cosinophilic vasculitis.

The visual loss may also be associated with a migrainous phenomenon. Many observers also regard transient monocular losses of vision in healthy children or young adults as benign events. However, these episodes in children may result from vasospasm. Temporary vasospasm offers a plausible and attractive explanation for complicated migraine and transient cerebral ischemic attacks. *(N Engl J Med 325:870-873, September, 1991.) Reprint requests to Jon B. Thurston, M.D., Department of Neurology, St. Louis University, Box 3635, Vista at Grand St., St. Louis, MO 63110-02250, U.S.A.*

MYASTHENIA GRAVIS UNMASKED BY COCAINE ABUSE. J Berciano, A Oterino, M Rebollo, J Pascual. The authors describe the case of a 24-year-old woman prostitute with myasthenia gravis whose clinical picture was unmasked by her cocaine use.

There is no doubt that cocaine first unmasked and then exacerbated myasthenia gravis. Symptoms indicative of impairment of motor-nerve axons or neuromuscular transmission are not included among the recognized acute neurologic consequences of cocaine. However, a critical reduction in the number of acetylcholine receptors per neuromuscular junction, as characteristically occurs in myasthenia gravis, probably renders the peripheral motor system more susceptive to the drug, leading to the episodic weakness observed in our patient. *(N Engl J Med 325:892, September, 1991.) Reprint requests to Jose Berciano, M.D., University Hospital, "Marques de Valdecilla", 39008 Santander, Spain.*

GLIOBLASTOMA IN NEW ENGLAND OPHTHALMOLOGISTS. R Bakshi, AM Ducatman, FH Hochberg. Authors bring attention to the curious occurrence of histopathlogically confirmed intracranial glioblastoma multiforme (grade VI astrocytoma) in four members of the New England Ophthalmological Society and an ophthalmic nurse between 1985 to 1990. This is a much higher than the expected glioblastoma incidence in the general population, based on which there should have been only 0.21 case in the Society membership averaging 554 during this period. The authors suggest that because viruses and exposure to industrial chemicals and monoionizing electromagnetic fields have been implicated as etiologic factors for glioblastomas, this disease cluster among ophthalmologists may be due to exposure to an environmental factor in the workplace. *N Engl J Med 324:1440-1441, May 16, 1991. For inquiries: Fred H. Hochberg, M.D., Massachusetts General Hospital, Boston, MA 02114.*

CLOMIPRAMINE IN THE TREATMENT OF SELF-MUTILATING BEHAVIOR. JF Lipinski, Jr. Referring to thereport of Swedo, et al. on effectiveness of clomipramine in trichotillomania, the author suggests that this drug may be also useful in other repititive self-mutilating behaviors listed under the "stereotype/habit disorder" and "borderline personality disorder" sharing the pathophysiologic features of obsessive compulsive disorders. *N Engl J Med 324:1441, May 16, 1991. Inquiries to: Joseph F. Lipinski, Jr, M.D., McLean Hospital, Belmont, MA 02178.*

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IN THE NAME OF ALLAH, THE BENEFICENT, THE MERCIFUL

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Whoever therefore sees,
Does so for himself;
And whoever remains blind,
Does so to his own loss.
Holy Quran 6:105

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Pakistan Academy of Medical Sciences

CONVOCATION '91 AND CONFERENCE '91, December 16, 1992, LAHORE

The Pakistan Academy of Medical Sciences will hold its Convocation '91 on **Monday, December 16, 1991** at 10 a.m. at the **Postgraduate Medical Institute, Lahore. President of the Islamic Republic of Pakistan** is expected to deliver the Convocation Address.

The **PAMS Convocation '91** will be followed by a **Conference on "Public Health Education in Pakistan."** There will be a reading of the 1991 **Pakistan Academy of Medical Sciences Lecture (Oration)** by a very eminent scientist before the discussions on the Public Health Education 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, 1991**, to the addresses given with this announcement.

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For further details contact: **Professor Mohammad Daud Khan, F.P.A.M.S., Chairman, Organizing Committee, XV Congress, Department of Ophthalmology, Lady Reading Hospital, Peshawar, Pakistan. Tel: (92-521) 811-857; 812-857(Res.); FAX: (92-521) 218-124,*OR* Dr. Shad Mohammad Tel:212-402.**