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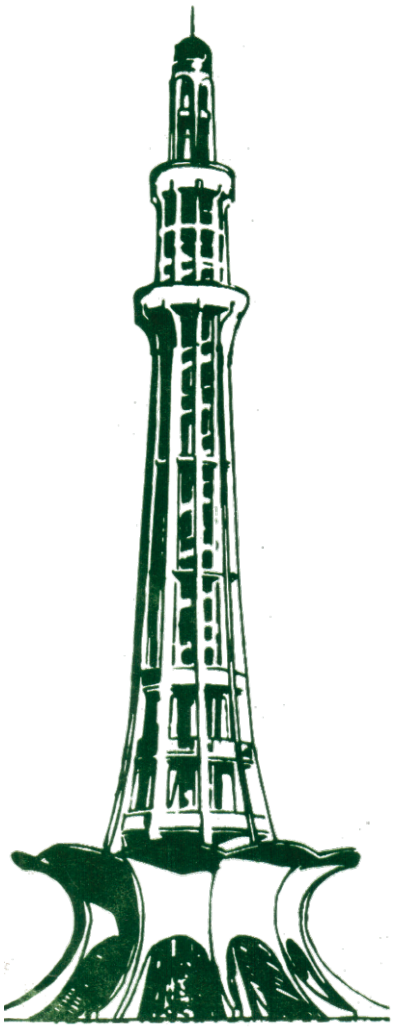

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IN THE NAME OF ALLAH, THE BENEFICENT, THE MERCIFUL

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Originial Article

Glaucoma in Punjab: Experience in a Teaching Hospital

Riaz A. Mahju, F.R.C.S.

ABSTRACT: Glaucoma afflicted 10% of all the patients who visited the ophthalmology clinic of Punjab Medical College, Faisalabad. Out of 2,234 glaucoma patients, 368 required hospitalization. Among the hospitalized patients, 106 had simple (open-angle) glaucoma; 79 had narrow-angle glaucoma; 13 had absolute glaucoma; two had congenital glaucoma; and 168 had secondary glaucoma. The largest number of patients fell in the age group of 51-60 years for simple glaucoma and 41-50 years for narrow-angle glaucoma. Secondary glaucoma developed due to hypermature cataract in 100 patients; anterior dislocation of lens in four patients; posterior dislocation of lens in 15 patients, ten of whom had *couching* done in remote areas; uveitis in 15 patients, in eight of whom it was tuberculous; complications of aphakia in 13 patients; old trauma with adherent leukoma in 7 patients; traumatic hyphema in 4 patients; vitreous hemorrhage from Eales's disease in 4 patients; and invasion of anterior chamber by retinoblastoma in 6 patients. The ratio of narrow-angle glaucoma to open angle glaucoma was 1:1.34. An intensified public education, better training of physicians, and improved availability of therapeutic agents is greatly needed for a successful fight against glaucoma in Pakistan. (Pak. J. Ophthalmol: 1: 53-59, 1985).

A chronic inflammatory condition of the eye with raised tension is how At-Tabari, an Arabian physician, first described glaucoma in the 10th century. About three centuries later, another Muslim Shamas-ud-Din called it "migraine of the eye" or "headache of the pupil" and described it as a pain in the eye with hemicrania, dullness of humors, and dilation of the pupil followed by cataract formation.[1] It was not until another three hundred years went by that the disease was first recognized in Europe by Richard Banister, an astute English oculist who was also the author of the first book on ophthalmology in English.[1] Certain blindness remained the ultimate consequence of glaucoma for another two hundred fifty years when iridectomy (von Graefe, 1857)[1] and medical treatment with pilocarpine (A. Weber, 1877)[2] were introduced. Today, although precise understanding of the fundamental process causing glaucoma still eludes researchers, knowledge of its clinical and therapeutic aspects has advanced to a level sufficient to halt glaucoma's deleterious effects on sight in an overwhelming majority of patients. The modern advances in diagnostic techniques and therapeutics have significantly improved the management of glaucoma the world over in the last decade.

The purpose of this paper is to present our experience with glaucoma at the District Headquarters Hospital, Punjab Medical College, Faisalabad (previously called Lyallpur), Pakistan in an effort to bring attention to the various professional and socioeconomic problems we face in our fight against the blindness caused by glaucoma in Punjab, Pakistan.

MATERIAL AND METHODS

Out of 2,234 glaucoma patients seen in the outpatient clinic of the Department of Ophthalmology, Punjab Medical College, Faisalabad, Pakistan, I examined the records of all 368 patients who required admission to the hospital for treatment. These patients were grouped according to the type of glaucoma. Each group was subdivided according to sex, age, treatment given, and ultimate outcome.

To confirm the diagnosis of glaucoma, the majority of these patients underwent: 1. hand held applanation tonometry, 2. gonioscopy, 3. visual field examination, and 4. evaluation of optic disc cupping. An intraocular tension over 20 mm Hg was regarded as suspicious in

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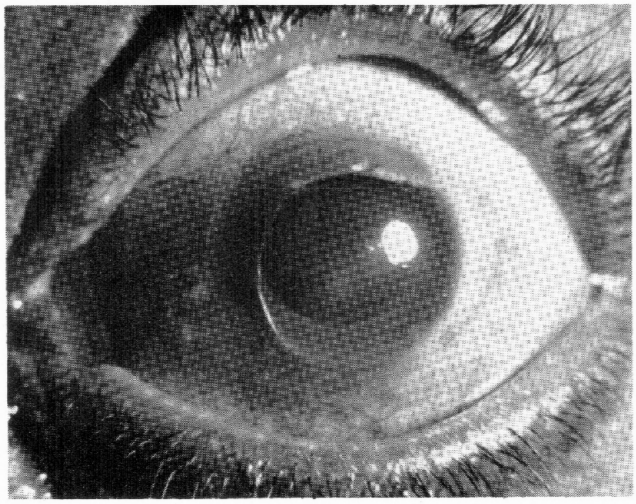

Figure 1. (Mahju): Traumatic anterior dislocation of the lens from a fist blow to the eye. (Courtesy of Khalid J. Awan, M.D.)

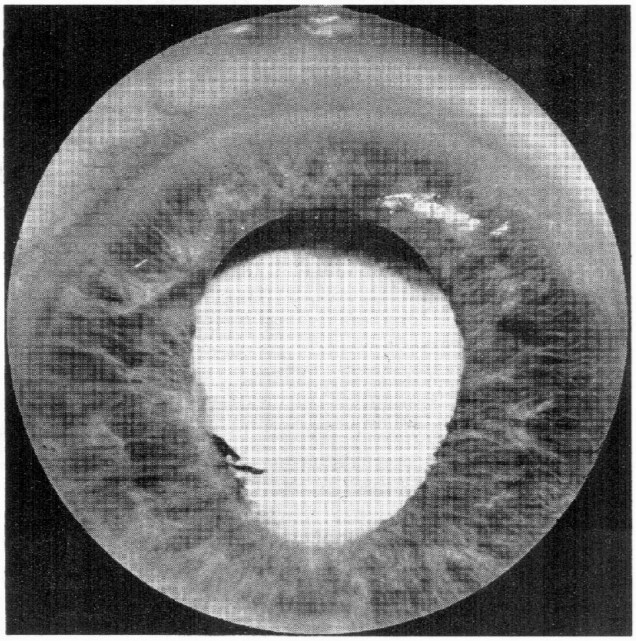

Figure 2. (Mahju): Posterior subluxation of hypermature cataract. (Courtesy of Khalid J. Awan, M.D.)

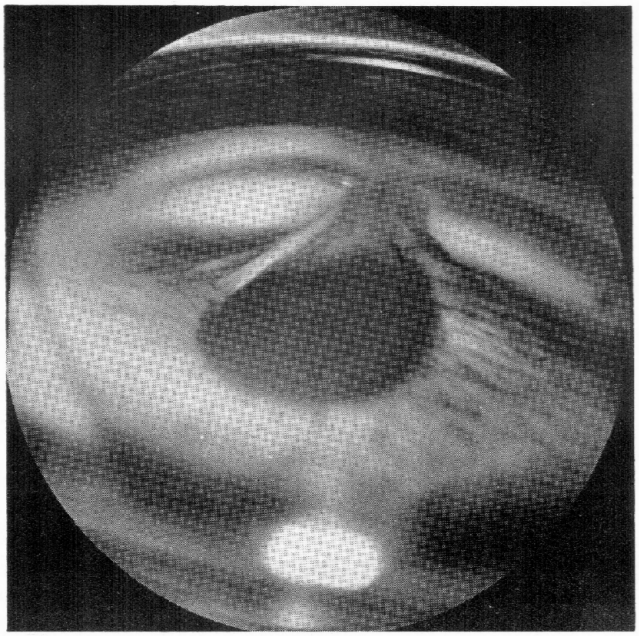

Figure 3. (Mahju): Adherent leukoma from an old perforation of the cornea. Gonioscopic view. (Courtesy of Khalid J. Awan, M.D.)

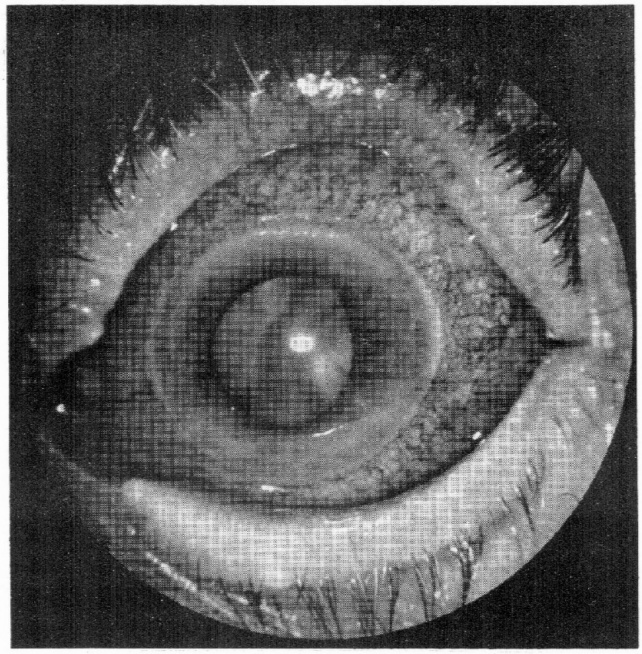

Figure 4. (Mahju): Acute angle closure glaucoma from an intumescent cataract in a 67-year-old woman. Although attack was three-day old, an intracpasular cataract extraction with sector iridectomy cured it. (Courtesy of Khalid J. Awan, M.D.)

patients with other wise normal findings. An intraocular pressure of 24 mm Hg with at least one other parameter showing findings compatible with glaucoma was considered diagnostic of glaucoma. The optic discs were evaluated by the method of cup/disc ratio[3] on direct ophthalmoscopy. In doubtful cases, progressive increase in cupping on repeated examinations helped in making diagnosis. The visual fields were plotted on a tangent screen at one meter distance. The grading system described by Scheie[4] was employed in evaluation of anterior chamber angle on gonioscopy (the narrowest angle was Grade I and the widest Grade IV).

The medical treatment consisted of miotics and carbonic anhydrase inhibitors. The surgical procedures, other than simple peripheral iridectomy for angle closure glaucoma, included iridencleisis and Scheie's operation.[5] Our techniques of iridencleisis and Scheie's operation are outlined here:

IRIDENCLEISIS: After reflection of the limbal-based flap of the conjunctiva and Tenon's capsule, a scratch incision was made with a Bard-Parker blade at the imbus at 12 o'clock position. The incision was deepened by repeated cuts till the anterior chamber

was entered. The iris usually prolapsed through the opening of the wound with the gushing out of the aqueous. It was grasped in an iris forceps and pulled out to bring the pupillary margin out of the wound. This was manually accomplished in eyes where the iris failed to spontaneously prolapse. A meridional cut extending the full width of the iris was made. The cut ends were pulled apart and the pillars of the iris were incarcerated, one in each end of the wound, under the flap of conjunctiva and Tenon's capsule. The conjunctiva and Tenon's capsule were closed with chromic absorbable stitches.

SCHEIE'S OPERATION: A conjunctival incision was made in the conjunctiva and Tenon's capsule about 7 mm behind the limbus. An 8 mm wide flap of these structures was reflected on to the cornea and the limbus delineated. A scratch incision was made within one mm of the limbus about 5 mm in length along the circumference of the limbus. The site of the scratch was treated with cautery, and the wound deepened with the blade edge. This alternate application of cautery and sharp deepening of the wound was carried out till the anterior chamber was entered. The posterior lip of the wound was cauterized more than the anterior one. A peripheral iridectomy was done immediately behind the wound. The conjunctiva and the Tenon's capsule were closed together with a running absorbable suture. An attempt to reform the anterior chamber at the conclusion of the procedure by a non-forced injection of saline under the flap was made in some of the cases.

In 35 patients the procedure was Scheie's scelrocautery and in 15 patients it was iridencleisis. We arbitrarily decided to do iridencleisis in patients who had intraocular tension in lower thirties or less, and Scheie's operation in those who had intraocular pressure in upper thirties or higher. An intraocular pressure of below 20 without any supplemental medication was regarded as successful surgical control. Of the 70 patients with acute congestive attack who were treated with peripheral iridectomy, seven needed filteration procedure at a later date during this study. Cyclodiathermy was done in some patients with absolute glaucoma with painful eyes who refused enucleation. Injections of 90% alcohol were employed in some.

RESULTS

Table 1 shows the distribution of 368 patients with glaucoma according to the type of glaucoma. Out of 106 patients with open angle chronic simple glaucoma, 56 were men and 50 were women. Age distribution of these is given in Table 2. Narrow angle glaucoma was affecting 79 patients, of whom 33 were men and 46 were women, making this disorder appreciably commoner in women of Punjab. The age distribution is shown in Table 3. Table 4 shows the types of secondary glaucoma seen in the 168 patients. Interestingly, a majority of these patients had glaucoma that was secondary to lens disturbances hence, one hundred cases were due to a hypermature cataract; four were due to traumatic anterior dislocation of the lens (Figure 1); and 15 patients had glaucoma due to posterior dislocation of the lens (Figure 2). Ten of these patients had posterior dislocation as a result of couching that had been done by quacks in remote villages. Thirteen patients had developed glaucoma from pupillary block or extensive peripheral synechia following cataract operation (aphakic glaucoma). Fifteen patients had glaucoma with iridocyclitis, eight of whom were found to have tuberculosis (elevated E.S.R., typical lung involvement on chest X-rays, and positive tuberculin test). Seven patients had glaucoma from adherent leucomas from previous corneal perforations (Figure 3). Six patients had glaucoma from anterior chamber

TABLE 1
Distribution According to Type of Glaucoma

(Total cases, 368)

Simple (Open-Angle) Glaucoma	106
Narrow-Angle Glaucoma	79
Secondary Glaucoma	168
Congenital Glaucoma	2
Absolute Glaucoma	13

TABLE 2
Age Distribution in Simple Glaucoma

(Total patients, 106)

21-30	8
31-40	18
41-50	25
51-60	38
61-70	15
71-80	3

TABLE 3
Age Distribution in Narrow-Angle Glaucoma

(Total patients, 79)

21-30	6
31-40	17
41-50	24
51-60	20
61-70	7
71-80	4

TABLE 4
Types of Secondary Glaucoma

(Total cases, 168)

Cataract Related	100
Anterior Lens Dislocation	4
Posterior Lens Dislocation*	15
Aphakia	13
Iridocyclitis**	15
Adherent Leukoma	7
Vitreous Hemorrhage	4
Traumatic Hyphema	4
Retinoblastoma	6

* 10 cases were from previous couching
** 8 patients had tuberculosis

invasion by retinoblastoma. Four patients were young children with glaucoma due to traumatic hyphema. Four cases were secondary to repeated vitreous hemorrhage from Eales's disease. Congenital or infantile glaucoma was present in two infants, one male of four months and one female of six months. Absolute glaucoma was present in 13 patients. In the patients who were operated on, the postoperative complications included uveitis, delayed formation of anterior chamber, choroidal detachment with flat anterior chamber, infection, and cataract formation. These postoperative complications are the subject of another paper in preparation. Alcohol injections, despite several repititions, failed to provide permanent relief in any of the patients.

DISCUSSION

The population of glaucoma patients in our clinic was 10% of all the eye patients seen during the period of this study. The incidence of glaucoma patients in

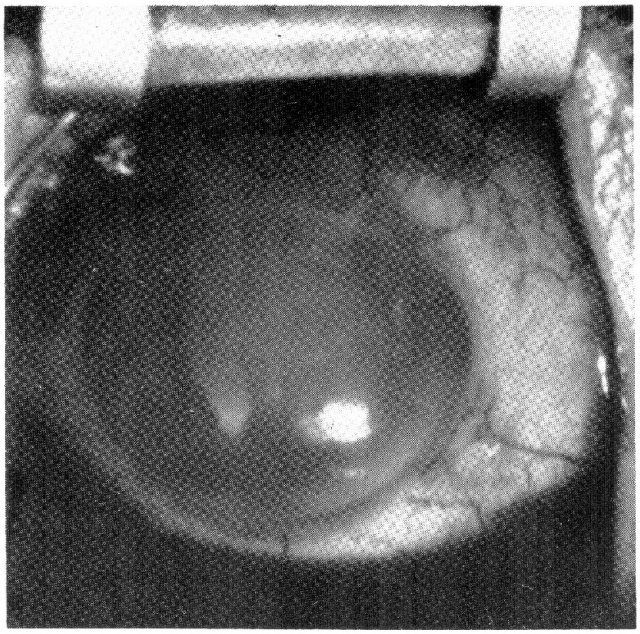

Figure 5. (Mahju): Acute phacolytic glaucoma from a hypermature cataract. Note the milky fluid filling the anterior chamber. The patient had lost sight due to cataract many years ago, but did not want surgery for it. (Courtesy of Khalid J. Awan, M.D.)

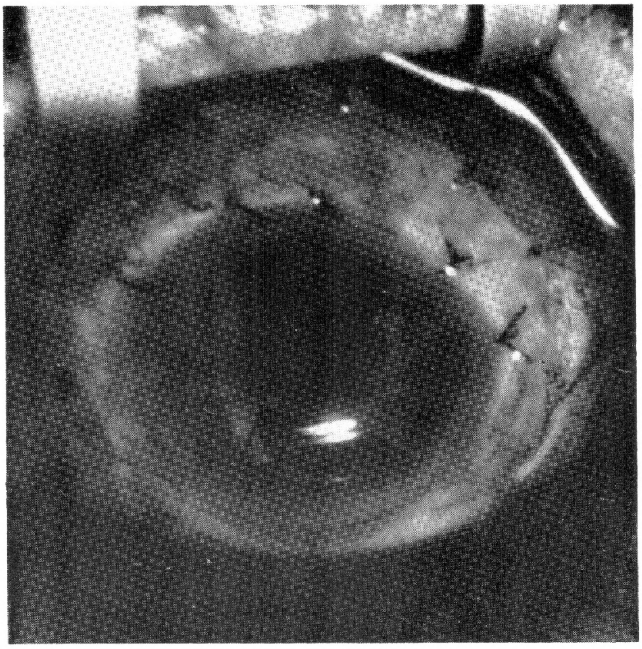

Figure 6. (Mahju): The eye shown in Figure 5 immediately postoperatively. The patient had intracapsular cataract extraction with Arruga capsule forceps. The postoperative visual acuity turned out to be 20/40 (6/12). (Courtesy of Khalid J. Awan, M.D.)

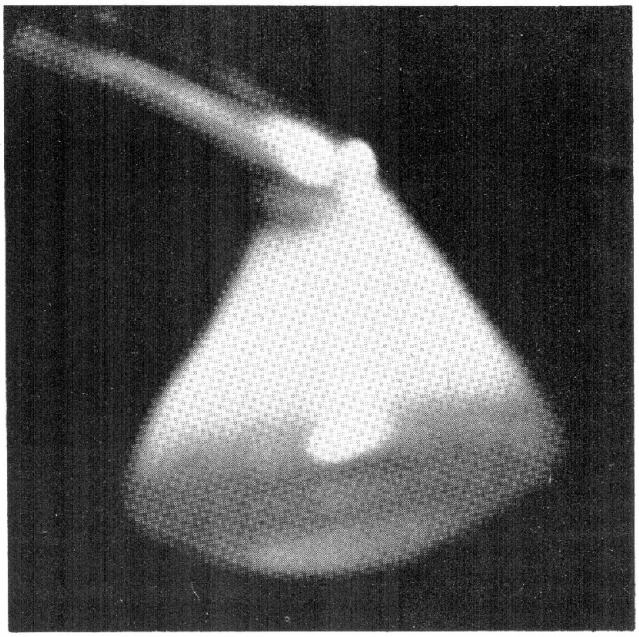

Figure 7. (Mahju): The lens removed from the eye in Figures 5 and 6. Note the fluid cortex in the capsular pouch with hard nucleus settled at the bottom. Surgeon must be very careful during intracapsular surgery on these cataracts because of fragility of the capsule. (Courtesy of Khalid J. Awan, M.D.)

the general population in Western countries is 1% to 2%.[1] To this author's knowledge no scientific study to determine it in Pakistan has been conducted. It has been, however, reported in lay press that 8%-10% of blindness in Pakistan is due to glaucoma. Chronic simple glaucoma has been reported to be most common after the sixth decade.[6] The highest incidence in Pakistan appears to be in people a few years younger. Sex distribution in our series showed no significant sex-difference, similar to other reported series.[6,7] It is interesting that in one series females were affected twice as much as males.[8] In our patients with chronic simple glaucoma, 4% pilocarpine solution at four hourly intervals proved to be effective in bringing the intraocular pressure below 20 mm Hg in a majority of the patients. The most difficult problem we faced was failure of continued compliance by the patients in using drops. This was because of the unavailability of medicine, financial inability of patient to purchase it, or the carelessness of the patient. It may not be a bad idea to perform surgery in patients that come from remote areas, are older, and have significant nerve damage rather than to risk blindness in them by giving a fruitless trial of medical therapy. We found that iridencleisis is a simpler procedure and has proven efficient in our hands in selected cases. Cassady[9] found iridencleisis as successful an operation in the Negro as in the White. Scheie[10] found it to be successful in 84% of the eyes with narrow angle glaucoma and in 86% in the eyes with chronic simple glaucoma. A healthy and intact pigment epithelium on the iris pillars is important for the successful fistula formation after iridencleisis. Where pigment epithelium of iris showed degenerative changes we did not do iridencleisis. In his own hands, Scheie[10] found his operation successful in 86% of cases of open angle glaucoma and in 90% of the cases of narrow angle glaucoma with compromised angles. The results in our series were almost comparable. Although cauterization of the limbal wound to achieve filteration was first introduced by Preziosi,[12] in 1924, it was Scheie[5] who made the idea workable and popular. In recent years, it has been claimed that trabeculectomy is the surgical method of choice and is virtually free of major postoperative complications when executed

properly.[13] We did not perform trabeculectomy in any of the patients in this series. Spaeth[14] compared the results of trebeculectomy and Scheie operation three years following the surgery and concluded that both of these procedures were equally effective. At the time of this study we were not equipped for laser treatment of glaucoma, but current literature shows that laser iridotomy and trabeculoplasty are successful modern alternatives for treating glaucoma.[15,16] It has also been found that Neodymium-YAG laser is a very effective therapy for aphakic malignant (ciliovitreal block) glaucoma by performing hyaloidotomy.[17] However, argon laser trabeculoplasty is not reliably effective in younger patients with primary open angle glaucoma.[18]

Primary narrow angle glaucoma is four to five times less common than primary open angle glaucoma.[1] In a study in three Welsh villages, Hollows and Graham[19] found that the ratio of narrow angle glaucoma to open angle glaucoma was 1:5.1. In our study this ratio was significantly higher at 1:1.34. It is interesting that although narrow angle glaucoma occurs with much higher comparative frequency in Punjab than the reported studies, its sex distribution is not much different from these studies. In our patients it occurred more frequently in women with a male to female ratio of almost 2:3. Another fact that came to light was that it was most common in the ages between 40-50, about 10 years earlier than in the West.[1] Of the 79 patients with narrow angle glaucoma, 70 came in with an acute attack. The others gave very strong histories of intermittent blurriness of vision and colored haloes. The selection of type of surgery for these patietns was based on the findings of gonioscopy. Anterior synechia extending over more than one third of the angle were considered an indication for the filteration procedure. A peripheral iridectomy was perfomed in the fellow eye when the patient agreed to have it. We came across several patients in our clinic who had undergone iridectomy without sufficient preoperative evaluation. This observation convinced us that it is imperative that physicians taking care of eye patients be properly trained.

A large number of cases in our series were associated with or were due to lens disturbances. Secondary glaucoma due to a hypermature cataract was present in 100 cases. The man to woman ratio was 2:3 in these 100 patients, showing a significantly higher incidence in women. As outlined by Awan,[20] acute glaucoma may be either due to intumescent cataract (Figure 4) or due to escape of lens material from the abnormally permeable or spontaneously perforated capsule causing phacoltyic glaucoma (Figures 5, 6, and 7). In a majority of cases lens extraction took care of the glaucoma. In 15 patients, however, a combined procedure for cataract and glaucoma had to be performed because of extensive peripheral synechia. In these cases, the patients had not sought medical attention in time. They were under the impression that eye surgery could be performed only at a certain time called "season." At one time, the socioeconomic and weather conditions made it necessary to do surgery on the eyes when the weather was mild and convenient for both the patients and the surgeons. This obsolete idea of surgical "season" still lingers in most of the population of Pakistan, entirely due to a lack of proper public education about eye diseases. In some cases typical Morgagnian cataract (Figure 8) was present. We had fairly satisfactory results in patients requiring the combined operation. A modified technique of Maumenee and Wilkinson[21] was used. In those patients where this failed, cyclodialysis was performed as a repeat procedure.

Traumatic dislocation of the lens was the cause of glaucoma in 14 patients. In four patients the lens was completely dislocated in the anterior chamber. All of these were young males who suffered blunt trauma to the involved eye. Unfortunately, the results of surgery on these cases were disappointing. The remaining ten patients had posterior dislocation of the

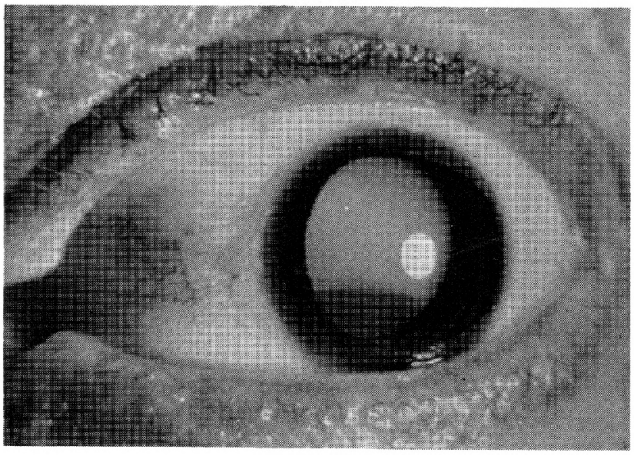

Figure 8. (Mahju): Typical Morgagnian cataract. Note the hard nucleus settled at the bottom. If not extracted, these cataracts may threaten the eye by causing phacolytic glaucoma (Courtesy of Khalid J. Awan, M.D.)

cataractous lens from couching that had been performed by quacks still roaming the remote areas of the country.

Couching is one of the most ancient surgical procedures. It was begun in India by the great Indian healer of his time Susrata.[22] It served splendidly in the past when the surgical procedures now possible and antibiotics were not available. Nowadays couching not only has no place in the treatment of cataract, it is sheer malpractice. In India one out of every 260 cataract patients still becomes a prey of couchers.[23] In couching, the procedure is intended to dislocate the lens completely into the vitreous but it has been estimated that in about 20% only subluxation results, and in another 8% the lens luxates anteriorly into the anterior chamber.[23] The complications of couching may appear in a few days or as long as 15 years later. The most significant complication of couching is secondary glaucoma, but chronic iridocyclitis, bullous keratopathy, and phthisis bulbi are also met with frequently. It is interesting that the infection rate is less than what one would expect considering the non-sterile techniques used in couching. Public awareness and education are the only way to prevent the

considerable amount of blindness caused by the quacks doing couching. We also need to inform the public that the old concept of "season" for eye surgery is a thing of the past, and encourage patients to seek medical advice the year round. This would definitely lead to a timely detection of glaucoma in a considerable number of patients who mistake their loss of sight to be due to cataract and keep waiting for the "season" to arrive.

Aphakic glaucoma was present in 13 patients due to

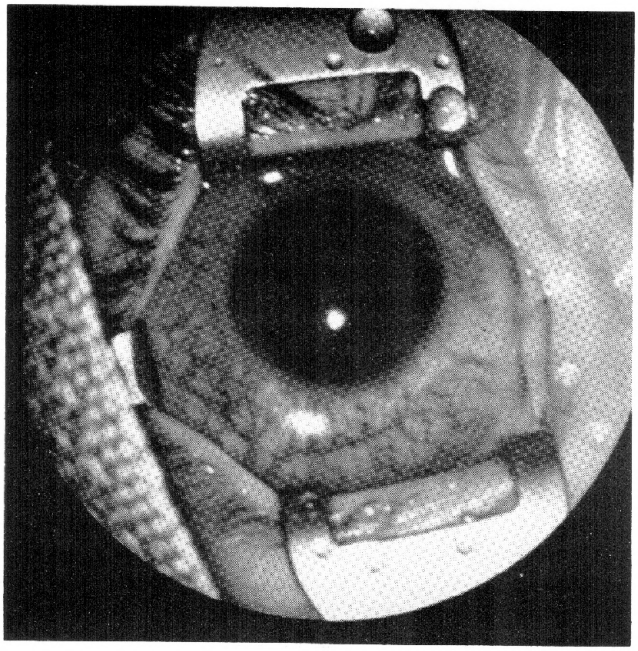

Figure 9. (Mahju): Retinoblastoma presenting as hyphema with acute glaucoma in a 2 year old male child. Aspirate from the anterior chamber confirmed the diagnosis when examined by an ophthalmic pathologist. Several general pathologists failed to make the correct diagnosis on examining this aspirate. (Courtesy of Khalid J. Awan, M.D.)

pupillary block or extensive peripheral synechia. The incidence of glaucoma following cataract extraction has been estimated at between 2% to 7%[1] Considering the large number of cataract operations done in Punjab, this number of aphakic patients in our series seems unusually low. It is possible that many of the patients who develop glaucoma following cataract operation do not return for evaluation, or fall in the hands of non-ophthalmologists who fail to make a correct diagnosis.

Of the 15 patients with glaucoma secondary to uveitis, eight had a confirmed diagnosis of tuberculosis, a disease still very common in Pakistan. In the remaining seven patients in this group no cause of uveitis could be determined. In 10 of these patients the intraocular tension was lowered to high teens or low twenties by cyclodialysis. Five patients did not respond to this procedure or any other mode of therapy. In patients with tuberculosis, the patients received antitubercular therapy as well. In all the cases in whom cyclodialysis was done, the inflammation was controlled as well as possible under the circumstances before the surgery. Short term results of this procedure appeared to be encouraging. The patients failed to return for followup, making it impossible to learn the long term effectiveness of this approach for treating this type of glaucoma.

Seven patients with glaucoma secondary to adherent leukoma, five men and two women, were treated with synechialysis and filteration surgery. As most of them also had lens changes, the control of intraocular pressure did not improve their visual capability. It might be a good idea to control the intraocular pressure in these cases medically until the patient consents to a combined procedure.

Traumatic hyphema with glaucoma was present in four children. These were the cases in whom secondary bleeding occurred and blood did not absorb following usual conservative therapy. We performed paracentesis to wash the anterior chamber. Unfortunately, when the anterior chamber cleared, lens opacities were found in all of them. From our experience, we have learned that all hyphemas should be treated conservatively, even if one has to resort to the systemic use of drugs to lower the intraocular pressure. If the pressure remains in the upper forties for several days despite full medical therapy, surgical intervention is justified. It is to be noted that the complication rate in hyphema is 44%, and that it increases directly with the size of initial hyphema.[24] To prevent rebleeding in hyphema several agents have been employed with controversial results. The use of systemic steroids has been advocated for it.[25] Recently, it was reported that oral administration of 100 mg/kg of body weight of aminocaproic acid every four hours orally, for five days, reduced the rate of rebleeding to 3% from the nontreated rate of 33%.[26]

Four patients who developed glaucoma from repeated massive vitreous hemorrhages were all diagnosed to have Eales's disease[27] (1882). The glaucoma that follows massive vitreous hemorrhage is usually caused by the obstruction of the trabecular meshwork by macrophages filled with the pigment and the extracellular debris of hemolyzed red cells.[28] or by ghost cells,[29] degenerated red blood cells with poor pliability. This type of glaucoma is called hemolytic glaucoma or ghost cell glaucoma. Another type of glaucoma is called hemosideric glaucoma, it is due to sclerosis of trabecular meshwork and hemosiderosis. It develops as a chronic process and needs to be differentiated from the hemolytic type. The hemosideric glaucoma is accompanied by other degenerative changes of hemosiderosis and develops after recurrent hemorrhages. In some instances, however, both of these may be present in the same eye.[28] There are three steps in the management of hemolytic glaucoma: 1) the standard medical treatment of open angle glaucoma; 2) if this fails, repeated anterior chamber washouts should be tried; and if this is not helpful 3) vitrectomy by one of the currently available techniques is the last resort.[28] We tried medical therapy initially, and this was followed by surgical intervention. We did not perform any vitrectomy as such techniques were not at our disposal.

The last group of patients with secondary glaucoma

consisted of 6 children, 3 male and 3 female, with retinoblastoma that had invaded the anterior chamber. It is not uncommon to see children with retinoblastoma in advanced stages in Pakistan, another aspect of lack of public education and awareness. On rare occasions, retinoblastoma may present as spontaneous hyphema with elevated intraocular pressure.[30] (Figure 6) In such cases differentation from juvenile xanthogranuloma may be difficult on clinical examination alone. We performed enucleation on all six patients.

Infantile glaucoma was found in two infants. Scheie[31] divided congenital glaucoma into infantile, in children under 3, and juvenile, in children and young adults under 30. As the glaucoma in young adults behaves no differently than chronic simple glaucoma of adults, this classification by Scheie appears confusing. However, one advantage to the use of term juvenile glaucoma is that it keeps the ophthalmologist alert to a possibility of young adults having glaucoma. Goniotomy is the procedure of choice in congenital glaucoma and gives 80% successful results in the hands of an experienced surgeon.[29] As soon as one is sure of diagnosis, prompt surgery is indicated for congenital glaucoma, particularly before the age of 3 when there is a high risk of enlargement of the globe. Because of a lack of facilities for and experience with goniotomy, we performed iridencleisis on our two patients. It controlled the intraocular pressure in the ward, but we are not aware of the ultimate outcome because the patients were never brought back for followup. Recently, very impressive results were reported in the use of drainage implants in the management of severe and complex cases of advanced juvenile glaucoma.[32]

Absolute glaucoma was found in 13 patients. Although the treatment of choice for this end stage of any type of glaucoma is enucleation, none of our patients were ready to have it. In four cases cyclodiathermy was done to lower the intraocular pressure to comfortable levels. The others were treated with repeated 90% alcohol retrobulbar injection with only temporary relief. Interestingly, 10% of the eyes with absolute glaucoma may have unsuspected malignant melanoma of the uveal tract,[33] making enucleation a more desirable approach in the management of this entity.

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Original Research

Massive Subfoveal Giant Cell Reaction: secondary to subretinal bleeding in retinoblastoma.*

J. Reimer Wolter, M.D.

ABSTRACT: Massive accumulation of epithelioid and giant cells of macrophage origin secondary to subretinal hemorrhage is demonstrated in the subfoveal space of a child's eye with an extensively necrotic retinoblastoma. This unusual observation is used as a basis for the discussion of the principles involved in the activation and settling of macrophages in avascular spaces of the inner eye. (Pak. J. Ophthalmol. 1: 60-63, 1985).

The central retina under pathological conditions frequently is involved with extended vascular leakage resulting in star-shaped intraretinal accumulation of so-called deep hard exudates exhibiting lipid-filled macrophages (microglia) within the spaces of Henle's fiber layer.[1-3] This may be associated with intraretinal hemorrhage. Under these circumstances the macrophages do not usually change into sessile epithelioid or giant cells. The potential space between central retina and pigment epithelium under pathological conditions also has a tendency for the accumulation of different types of exudates causing detachment of the central rods and cones. In this location the exudate usually exhibits only few macrophages and these do not typically contain much lipid – even after prolonged existence. Such subfoveal exudates without or with only few macrophages commonly are part of the histopathological picture of more or less fully developed cystoid macular edema (CME).[4-9] In addition to exudation, hemorrhage into the potential subfoveal space also is well known as a typical complication of ocular as well as general diseases. Hemorrhage into this plane typically attracts monocytes from retinal blood vessels. These

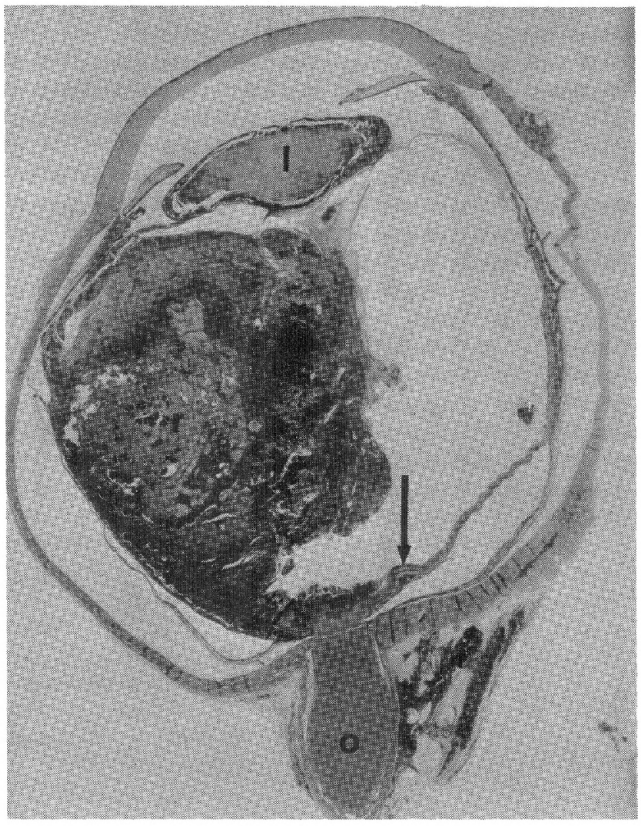

Figure 1. (Wolter): Cross section of the eye in horizontal plane exhibiting lens (l), extensively necrotic retinoblastoma (r), optic nerve (o), and locally detached foveal retina with sub-foveal exudate (arrow). - Paraffin section, H and E stain, photomicrograph X6.

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*Supported by the Research to Prevent Blindness, Inc., New York, N.Y. Offprint requests to: J. Reimer Wolter, M.D., Department of Ophthalmology, University of Michigan Hospitals, Ann Arbor, Mich. 48109.

change into free macrophages, penetrate without much difficulty the outer limiting membrane, and invade the subretinal blood with the obvious purpose to phagocytize and remove its remnants.

To observe and describe the pattern of the settling of macrophages and their change into epithelioid and giant cells in the very unusual association of a subfoveal hemorrhage with an advanced and extensively necrotic retinoblastoma is a rare opportunity. This is of practical importance, because the interactions between macrophages and blood in the avascular subfoveal space of this case allow for a new perspective in the consideration of the nature and purpose of a basically similar reactive situation with cells of macrophage origin seen on the surface of intraocular lens implants.

Case Report

This three year-old white female had a history of squint observed by her parents for a few weeks, before the pupil of the squinting right eye turned white. This caused the parents to take the child to an ophthalmologist. Vision decrease to barely light perception in the right eye associated with a suspected intraocular mass was found. The left eye was normal. A C-T scan revealed a mass filling most of the nasal half of the vitreous space of this right eye. On the scan the mass exhibited evidence of spotty calcification. The eye was enucleated on 8-26-80 for suspected retinoblastoma and it was fixed in 10% formalin immediately after removal.

Gross examination of the eye revealed this to measure 22 x 21 x 21 mm. The optic nerve was cut 17 mm in back of the posterior sclera and appeared grossly normal. Transillumination showed the diffuse shadow of a large mass filling the nasal half of the globe. The eye was opened in the horizontal plane. The nasal half of the vitreous space was filled by a bloody mass with non-distinct borders and this did not grossly resemble a typical retinoblastoma. However, the gritty sensation of spotty calcification was recognized, when the mass was cut. The mass covered the optic disk and extended for a short distance onto the inner surface of the temporal half of the retina next to the disk. The nasal half of the retina was absent, but the temporal half of the retina was preserved and in place. The foveal region of the retina appeared swollen and exhibited a small

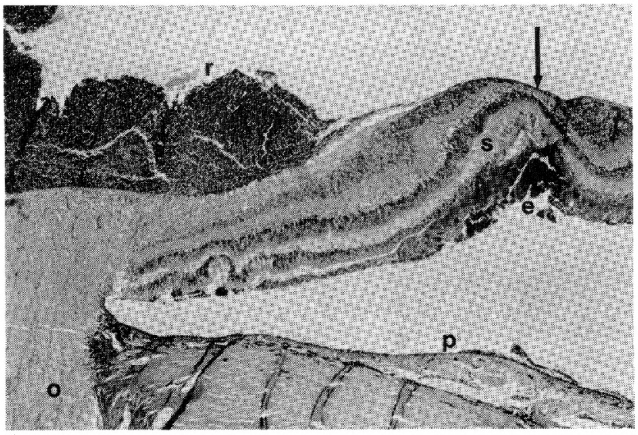

Figure 2. (Wolter): Foveola (arrow) exhibiting swelling of Henle's fiber layer (s) and partly hemorrhagic exudate (e) in subretinal space. Separation from pigment epitelium (p) is artificial. Optic nerve (o) with active retinoblastoma (r) in front of it on the left. - Paraffin section, H and E stain, photomicrograph X 140.

zone of selective detachment centered on the foveola and associated with a shallow vertical retinal fold running through its center and extending from this for a few millimeters in an upward and downward direction. The subfoveal space in the region of this fold contained a fixed exudate of yellowish color.

Microscopic study of paraffin sections cut in a horizontal plane and

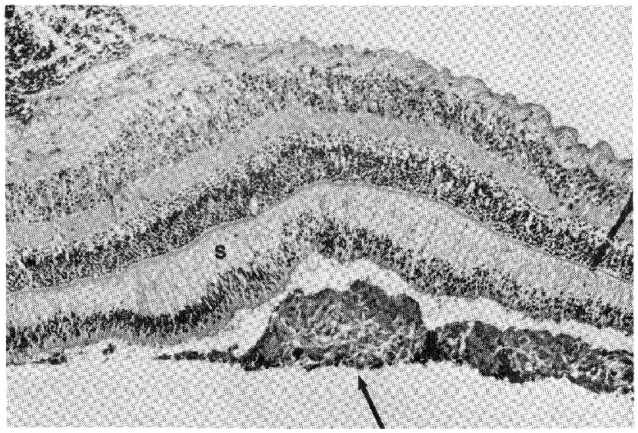

Figure 3. (Wolter): Section in parafoveal horizontal plane superior to foveola revealing swelling with microcystoid changes in Henle's fiber layer(s) and extension of subretinal exudate containing macrophages, epithelioid cells, and giant cells (arrow). Preretinal retinoblastoma extension on upper left. - Paraffin section, H and E stain, photomicrograph X 250.

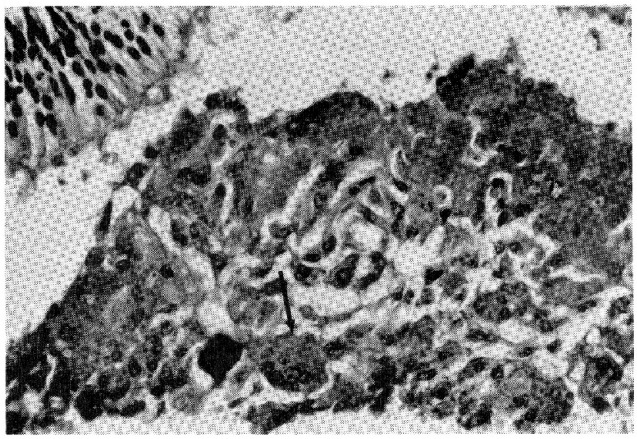

Figure 4. (Wolter): Macrophages, epithelioid cells, and multinucleated giant cells (arrow) in subfoveal exudate also exhibiting partly degenerated erythrocytes and irregular lipid spaces. Outer retina with partly degenerated rods and cones seen on upper left. - Paraffin section, H and E stain, photomicrograph X 550.

stained with H and E showed the cornea to be normal. Some fibrinous exudate was found in the anterior chamber. Iris and ciliary body exhibited some diffuse infiltration with mononuclear inflammatory cells. The lens was involved with early posterior subcapsular cataractous changes (Figure 1). The mass in the nasal half of the vitreous was composed of necrotic tissue with partly preserved new-formed blood vessels and with spotty calcifications in its anterior portion. Islands of a neuroepithelial neoplasm containing occasional pseudorosettes arranged around new formed blood vessels were found. Typical retinoblastoma without rosettes was preserved in its continuous growth pattern in the region in front of the optic disk. The nasal half of the retina was absent and replaced by the neoplasm. Irregular hemorrhage was seen all through the mass and there also was some blood on its outer aspect next to the preserved nasal pigment epithelium.

The temporal half of the retina was well preserved and mostly in place (Figure 1). Seeding of the same neoplasm seen in the nasal half of the vitreous was found on the surface of the temporal half of the peripheral retina. The foveal retina was locally detached and an exudate containing densely arranged macrophages, epithelioid cells, numerous large giant cells, preserved erythrocytes, and remnants of degenerated erythrocytes filled the subfoveal space (Figures 1-4). One dense hemorrhage was seen in the very center of the subfoveal detachment (Figure 2). The erythrocytes in this hemorrhage were densely compacted and partly degenerated. Most of the macrophages, epithelioid cells, and giant cells contained

pigment granules in their protoplasm (Figure 4 and 5). Special stains showed these granules to be mostly hemosiderin in nature, but some melanin granules were also found. The presence of lipids was recognized by the existence of empty appearing lipid spaces and vacuoles in the protoplasm of macrophages as well as in epithelioid and giant cells (Figures 3 and 5). Lipid spaces were also seen outside of cells in the piled-up remnants of degenerated erythrocytes. The foveal detachment had fold-like extensions in the superior and inferior directions. In the horizontal plane the accumulation of blood and sessile macrophages extended almost up to the temporal disk margin (Figures 1, 2 and 4). The central retina was slightly swollen and exhibited fluid accumulation in Henle's fiber layer with some wrinkling of the outer retinal layers directly temporal to the disk (Figure 2). The pigment epithelium in the foveal region was very well preserved (Figure 2). Choroid and sclera had a surprisingly normal appearance (Figures 1 and 2).

DISCUSSION

Active macrophages released from blood vessels into tissues and fluid spaces have a life of their own — responding to chemotactic gradients without innervation or central direction. Like pre-programmed unicellular organisms they set out to clean up, digest, or separate "foreign" substance. When they run into difficulties with the clean-up process they can change from free-moving cells to sessile elements. In this process they not only enlarge their protoplasm to form so-called epithelioid cells, but they also develop multiple nuclei either by nuclear division or by cellular fusion to form giant cells.[10]

The occurrance of active macrophages in their free-moving state within subfoveal exudates of long-standing CME is not unusual.[4-9] The subfoveal accumulation of sessile epitheliod and giant cells of macrophage origin in a multilayered formation, in contrast, is not a common occurrance. The pigment epithelium underlying this accumulation of reactive cells was remarkably uninvolved in the present case. The cell bodies of the detached central rods and cones were preserved, but the outer segments were mostly distorted and degenerated due to their separation from the pigment epithelium. The accumulation of blood remnants, epithelioid cells, and large multinucleated giant cells in the subfoveal space cannot be called a granuloma, since it does not contain the mesodermal components: blood vessels, fibroblasts, and chronic mononuclear inflammatory cells — typically seen in the outer layers of such formations. It, thus, represents a special kind of reactive formation that is also seen on lens remnants in the fluid spaces of the eye[11] as well as on intraocular lens implants,[12-14] on other foreign substances in avascular spaces of the eye,[15] or on the back surface of keratoprostheses,[16] for example. The fact that all these situations tend to be associated with recurrent bleeding is of special importance.

The lack of a more widespread inflammation, neovascularization, and fibrosis in association with the massive epithelioid and giant cell reaction to blood remnants seen in the subfoveal space of the present case is best explained by the overwhelming impact of the massive necrosis in the large retinoblastoma. This may well be responsible for the incomplete granulomatous response in the present case. The free-moving macrophages had fulfilled their assignment in seeking out and starting to phagocytose blood remnants in the subfoveal space. Under the impact of the massive necrosis in the retinoblastoma, however, the mesodermal and vascular components in the reactive chain of the removal of blood remnants from the subretinal space failed — and the macrophages reacted to this fact by containing and storing in their protoplasm as much of the blood remnants as possible. In this process they changed into epithelioid and giant cells. The presence of the retinoblastoma, thus, may have effected these cells somewhat like the suppressive action of a steroid medication — or of simple ischemia due to insufficient regional blood supply.

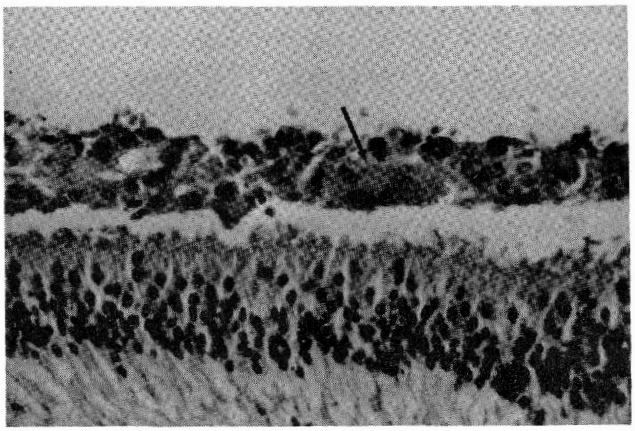

Figure 5. (Wolter): Shallow extension of subfoveal exudate towards disk with densely accumulated macrophages, epithelioid cells, and multinucleated giant cells (arrow). Outer retina with rods and cones exhibiting partly degenerated segments seen above. - Paraffin section, H and E stain, photomicrograph X 550.

Chronic accumulation of macrophages, epithelioid cells, and giant cells without much secondary vascular and mesodermal response is a new and practically very important reactive reality frequently seen under postoperative conditions after different types of implantation and tissue engineering in avascular regions of the inner eye. It is at this time not possible to say, whether or not this should be classified as granulomatous inflammation. However, the situation is not stable and it requires continuing inflow of new macrophages from adjacent blood vessels. Warren[17] has shown convincingly that "giant cells are exceedingly short-lived and will disappear rapidly if a supply of new macrophages is cut off." This can be observed after whole body radiation, for example. The subfoveal giant cell reaction in the present case, without doubt, was a highly active process that was limited in its full development by the influence of the massively necrotic retinoblastoma. Used like an experiment, the insights gained by this observation are useful for the evaluation of other processes with accumulation of sessile macrophages.

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Scholarship Schedules

Pakistan Academy of Medical Sciences
1985 Board of Trustees Meeting
November 9, 1985
Baltimore, Maryland, U.S.A.

Ophthalmological Society of Pakistan
1986 Annual Meeting, Quetta
March-April - (Dates to be announced)
Contact: Professor Sardar Ali Sheikh, President OSP
Nishtar Medical College, Multan, Pakistan

Association of Pakistani Physicians
7th Annual Meeting, July 18-21, 1985
Anaheim, California
Contact: APP 1300 Fairmount Avenue
Fort Worth, Texas 76104

Islamic Medical Association of North America 17th Annual Convention
Niagara Falls, New York - July 4-7, 1985
Contact: Bashir A. Chaudhary, M.D.
Department of Pulmonary Diseases
Medical College of Georgia
Augusta, GA 30912

American Academy of Ophthalmology
1985 Annual Meeting - September 29-October 3
San Francisco, California
Contact: Paul R. Lichter, M.D.,
Program Secretary AAO 1010 Wall Street
Ann Arbor, Michigan 48105

18th Panhellenic Ophthalmological Congress
May 23-25, 1985, Athens, Greece
Contact: Secretariat, 18th Panhellenic Ophthalmological Congress, 10, Loukianou St., GR-106 75 Athens, Greece.

The International Oculoplastic Society
Oculoplastic Surgical Fellowships 1985 & 1986 Applications
Training in pre- and post-op care; surgical assistance, staff appointment. Includes liability insurance, limited funding and locum tenens. Mini-fellowships can also be arranged with multispecialty surgeons.

The Far East Facial Plastic Surgery Congress
June 21-July 4, 1985
Taipei, Singapore, Bali & Hong Kong

17th Annual Oculoplastic & Facial Surgery Course
September 18-22, 1985
New York, New York.
Contact: Ms. A Burnett: c/o Dr. Pierre Guibor, The International Oculoplastic Society, 630 Park Avenue, New York, NY 10021 or telephone: (212) 734-1010, outside NY (800) 223-4500.

Current Concepts in Ophthalmology Courses
Johns Hopkins University
March 3-7, 1986, and Feb. 23-27, 1987
Dorado Beach, Puerto Rico
March 17-21, 1986, and March 16-20, 1987
Vail, Colorado
Contact: Office of Continuing Education, Johns Hopkins University School of Medicine, Turner 22, 720 Rutland Ave., Baltimore, MD 21205.

International Symposium on Vitreoretinal Surgery,
University Eye Clinic, Vienna
May 13-15, 1985, in Vienna, Austria
Contact: Susanne Binder, c/o Wiener Medizinische Akademie, Alser Strasse 4, A-1090 Vienna, Austria.

Practical Course in Radial Keratotomy
May 23 and 24, 1985, Philadelphia, Pennsylvania
June 28 and 29, 1985, in Houston, Texas
Contact: Frederick B. Kremer, M.D., course director. Barbara Cusumano, Pepper Pavilion, 19th and Lombard St., Suite 607, Philadelphia, PA 19146.

Camera Clinicals

Edited by: Khalid J. Awan, M.D.

In this section of the Journal, photographic documentations of interesting and challenging observatiosn will be presented to the readers. They should make their diagnoses from the given information and compare these with the ***expositions*** *given on page 97 – Editor.*

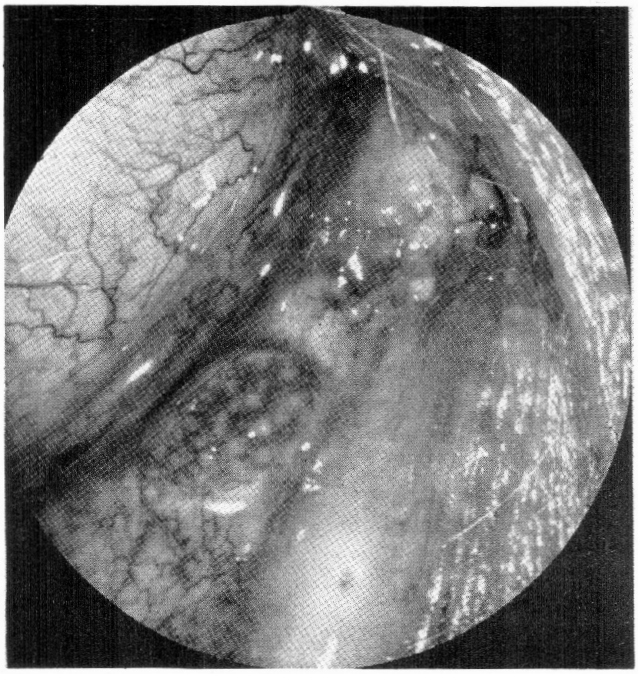


Figure 1

Figure 1: A 30-year-old woman complained of irritation in her right eye. A week before it was treated with antibiotic drops by her family physician. Eye examination showed the visual acuity to be 20/20 (6/6) in each eye. The left eye was normal. The right eye showed a fleshy, nodular, 11x8 mm tumor just behind the punctal area of the lower eyelid (Figure 1). Although the tumor was vascular, no congestion was noted in the surrounding conjunctiva. At least three lanugo hairs could be seen on the surface of the tumor slit lamp microscope. The tumor was excised and sent for histopathological studies. The recovery was uneventful with only a minor localized scarring of the conjunctiva.

Figure 2: A 19-year-old man came with history of blunt injury to his left eye. Eye examination showed the visual acuity to be 20/20 (6/6) in the right eye and 20/30 (6/9) in the left eye. The right eye was normal in all respects. Externally, the left eye had mild redness. A minimal cellular reaction in the anterior chamber was noted on biomicroscopy. Although the lens was clear, a circular deposit of brownish pigment granules was visible on its anterior surface when the pupil was dilated (Figure 2). This pigment ring was about 3mm in diameter and was centrally located. Ophthalmoscopic examination was normal in each eye. The intraocular pressure was 18 mm Hg in the right eye and 14 mm Hg in the left eye by applanation tonometry. The youth was treated with steroid-antibiotic combination drops and he regained 20/20 (6/6) vision in five days. After four weeks no trace of pigment ring on the anterior surface of the lens could be seen.

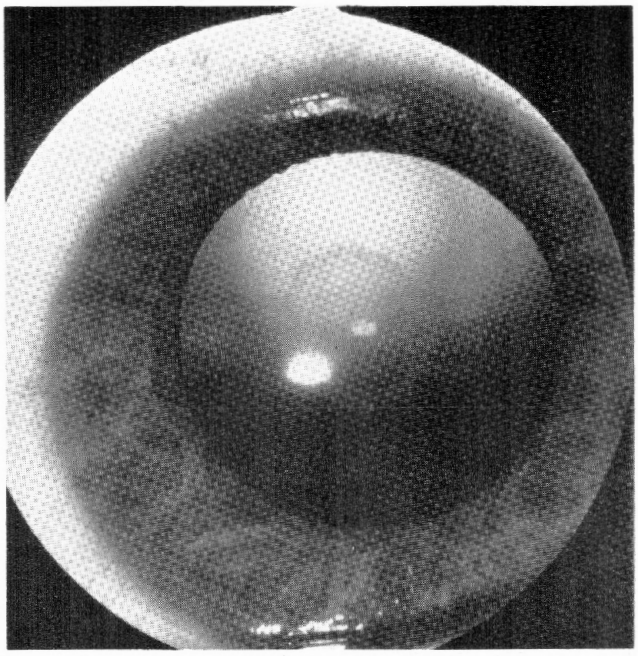


Figure 2

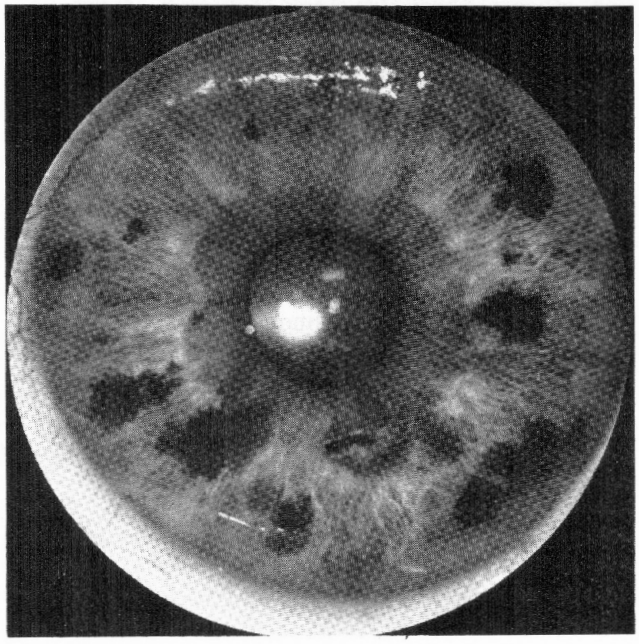
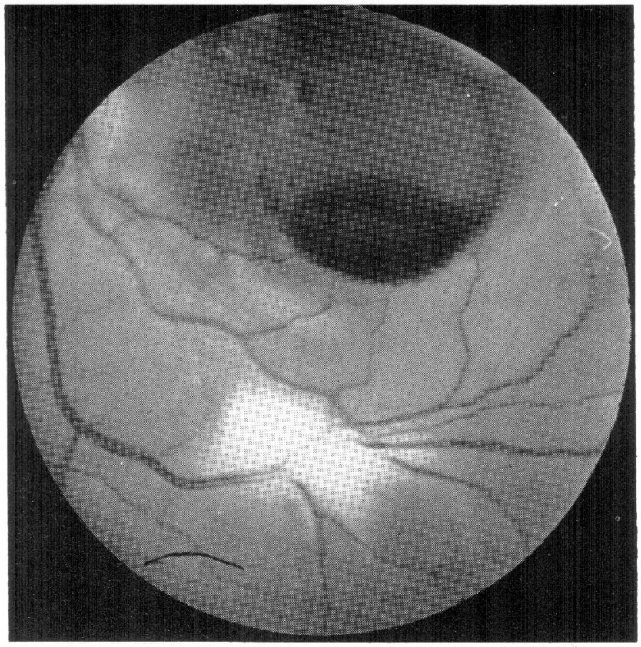


Figure 3

Figure 3: A 62-year-old man came for an eye checkup because of difficulty in reading. His visual acuity was 20/20 (6/6) at distance in each eye. External examination was normal in both eyes except for multiple darkly pigmented areas on both irises (Figure 3). In the right iris, a pigmented lesion at 5:30 o'clock had brownish discoloration and was elevated. There was no distortion of the pupils and they were normal in reaction. The intraocular pressure was 19 mm Hg in



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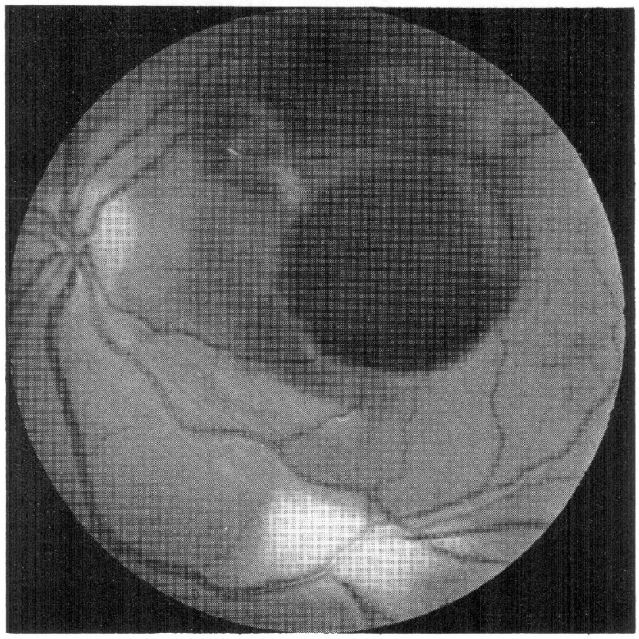

Figure 5

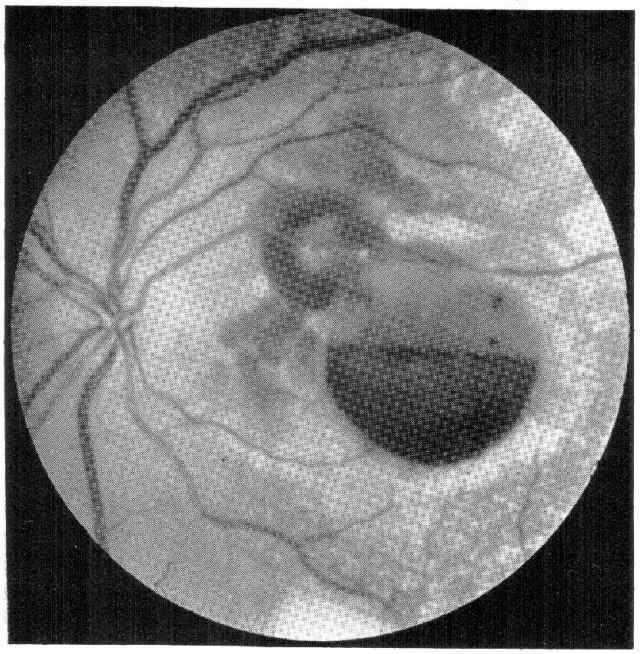

Figure 6

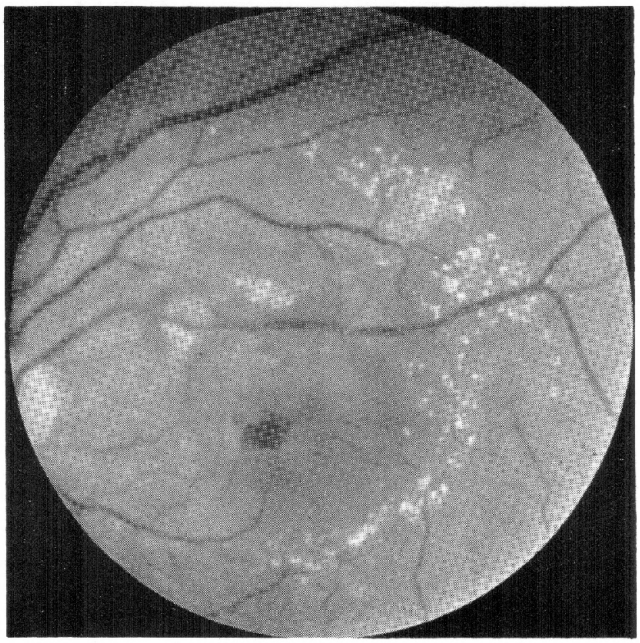

Figure 7

the right eye and 17 mm Hg in the left eye. No abnormality was noted in either eye on ophthalmoscopic examination. The patient said the elevated lesion had been there for at least 12 years and had not bothered his eye or his sight. The patient refused an excisional biopsy.

Figures 4, 5, 6 and 7: A 66-year-old man who previously had normal vision in both eyes complained of sudden painless blurriness in his left eye. His general medical history included longstanding hypertension and recent kidney trouble. His visual acuity was 20/20 (6/6) in the right eye and 20/100 (6/30) in the left eye with correction.

The eye examination excepting ophthalmoscopy in OS was normal. In the left fundus there was a central boat-shaped preretinal hemorrhage with deeper retinal hemorrhages around it (Figure 4). A white lesion was located just below the area involved in the hemorrhage (Figure 4). The hemorrhage appeared to ensue from the superior temporal vessels about 1½ disc diameter away from the disc border. The blood was squirting into the boat-shaped hemorrhage with each beat of the heart (arrow in Figure 4) and soon the hemorrhage increased to a disciform shape (Figure 5). After one month the bleeding stopped spontaneously (Figure 6). However, a central hemorrage with surrounding white exudates was left behind. In four months, the hemorrhage and the thick exudates cleared up to a great extent (Figure 7), but the vision went down to counting fingers.

Ophthalmic History

Ophthalmological Society of Pakistan: A History

Raja Mumtaz Quli Khan*

ABSTRACT: Author gives the history of the inception, the progress, and the present activities of the Ophthalmological Society of Pakistan. The Society came into existence in 1957 and has since actively strived for the Progress of Ophthalmology in Pakistan. The Society hosted the meetings of the Asia-Pacific Academy of Ophthalmology in 1979 and the Afro-Asian Congress of Ophthalmology in 1984. In 1979, the President of Pakistan, General Muhammad Zia-ul-Haq became the Patron-in-Chief of the Society and instituted the Ramzan Ali Syed Gold Medal to be annually awarded to an ophthalmologist who has distinguished himself by outstandingly serving ophthalmology in Pakistan. In 1982, the Society celebrated its silver jubilee. In 1984, the Society launched the Pakistan Journal of Ophthalmology, the first such Pakistani publication, under the patronage of the author and the editorship of Khalid J. Awan, M.D. (Pak J Ophthalmol 1:66-70, 1985).

At 2 p.m., Thursday, December 19, 1957, 32 physicians gathered in Lecture Theater No. 2 of the Patiala Block of King Edward Medical College, Lahore to launch Pakistan's first national association of ophthalmologists.

After introductory discussion of the needs and objectives for such an organization, the first team of officers was elected for the Ophthalmological Society of Pakistan. They were Lt. Gen Burki, President; Professor Ramzan Ali Syed, Vice President from Lahore, West Pakistan; Professor M. Riffat Ullah, Vice President from Dacca, East Pakistan; Dr. M.S. Faridi, Treasurer; and I, Secretary.

There had previously been one local ophthalmic organization, the Ophthalmological Society of Lahore, founded by Professor Ramzan Ali Syed in 1954, and interested individuals had formed the habit of gathering informally to share ideas and opinions when there was time to do so.

But seeds for a national organization had already been planted. They were a byproduct of an idea of William John Holms, M.D., of Hawaii, to found the Asia-Pacific Academy of Ophthalmology, an organization intended to promote ophthalmology and related sciences in Asia and the countries bordering the Pacific Ocean, and to cultivate social and fraternal relationships among ophthalmologists of this vast region through formal conferences, seminars, round table discussions, teaching clinics, work shops, and films. To inform the ophthalmologists in Pakistan about the planned Asia-Pacific Academy of Ophthalmology and to encourage their participation in it, Dr. Holms wrote us a letter on July 10, 1957. He stated "that following the International Congress of Ophthalmology in Brussels in 1958, a group of ophthalmologists from the West will visit various countries in Asia including Pakistan and hold joint meetings with part or all of your members. Conceivably the visitors could present the same papers in Pakistan that they will read at the International Congress. Your members in turn might want to reciprocate by holding wet or dry clinics or conferences on the subjects of local significance. With your interest and encouragement, the post International Congress meeting to be held in Pakistan in 1958 might become one of the outstanding ophthalmic conferences of the year."

Unfortunately, the proposed Asia-Pacific Academy of Ophthalmology did not materialize at that time, but its concept did prove catalytic in the formation of our national ophthalmic society.

The news of the establishment of the Ophthalmological Society of Pakistan was

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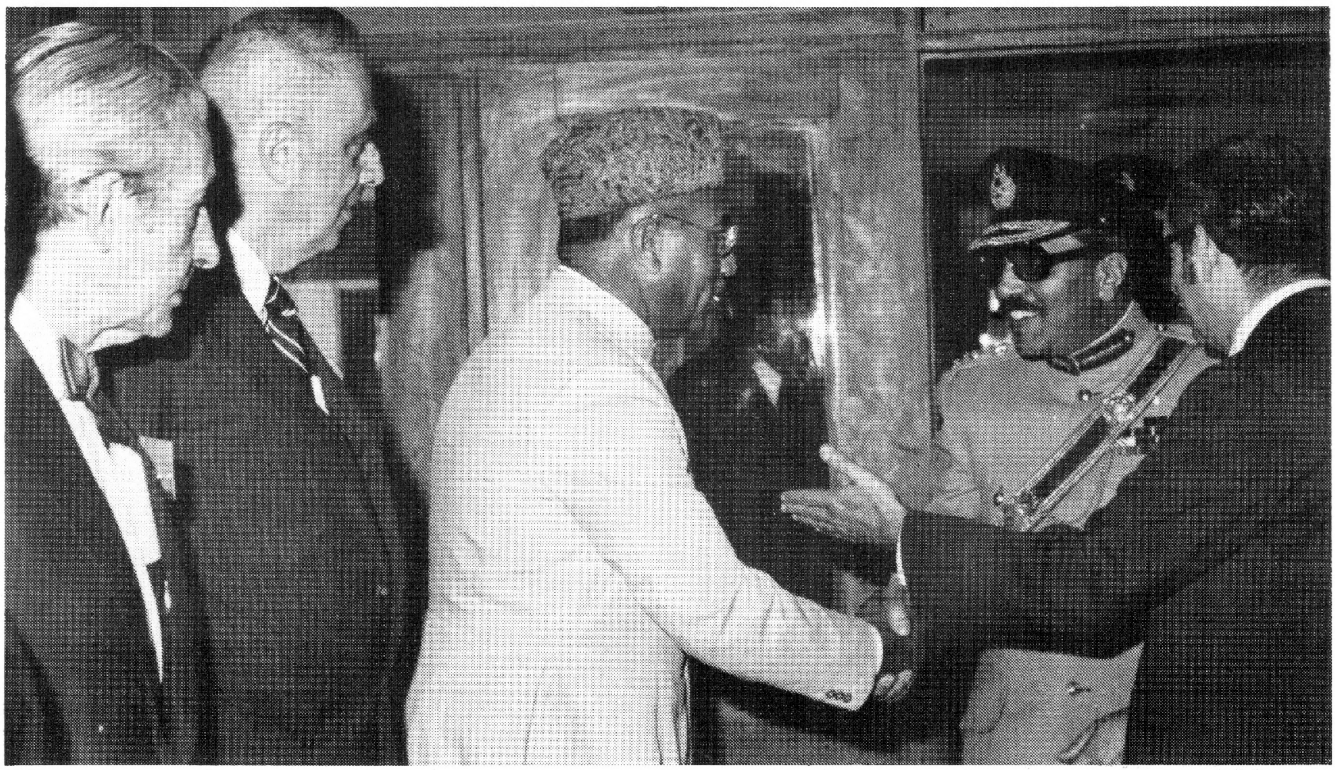

Figure 1. At the 1979 APAO Congress Dr. J. Wania (Regional APAO Sec.) is introducing Professor Rajor Mumtaz (Congress President) to the Chief Guest, Gen. Muhammad Zia-ul-Haq, President of Pakistan. Standing next to Professor Mumtaz are (left to right) the late (Baron) Prof. Jules Francois, President of International Council of Ophthalmology, and Dr. W.J. Holmes, President of APAO.

communicated to Mr. Hartman on March 28, 1958 for its enlistment with the International Council of Ophthalmology. Dr. Cooper was also contacted to request entry into the Index Ophthalmological. Our first foreign member was Dr. Roland I. Pritikin who joined the Society in August of 1958.

In February, 1959, Lt. Gen. Burki was given additional responsibilities by the Government of Pakistan and indicated he would be unable to continue as the President of the Society. In 1960, new officers were elected. They were: Professor R.A. Syed, President; Professor M.H. Alvi, Vice President; Dr. M. Ishaque, Joint Secretary; Dr. A.A. Sheikh, Treasurer; and I, Secretary.

The next year the Ophthalmological Society of Pakistan was happy to affiliate with the Ophthalmological Society of Pakistan, East Wing. A letter to this effect was sent to Prof. M.A. Jalil of Dacca on June 18, 1961. Now the Society held clinical meetings monthly. In 1963, I left to undertake a year-long Nuffield Foundation Scholarship abroad. Our thanks are due to Dr. M. Yaqin who looked after the affairs of the Society in my absence.

In 1965, very sadly, India's aggression in Kashmir and her invasion of West Pakistan led to an all out war between the two countries. The Society's activities came to a halt during this unhappy time. But in February, 1966, a meeting was held and new officers elected. The new officers included: Lt. Gen. S.M.A. Farooqi, President; Professor M.H. Alvi, Vice-President; Dr. Sajjad Ahmed, Joint Secretary; and Dr. S.M. Faridi, Treasurer; and I, Secretary.

The Society has played a very prominent role in the uplift of ophthalmology in Pakistan. On August 23, 1966, the International Federation of Ophthalmological Societies recognized the Ophthalmological Society of Pakistan. The activities of the Society gained momentum and on January 26 and 27, 1968, the first Annual Conference of OSP was held. A total of 68 member ophthalmologists participated in the business meeting and the following officers were elected: Professor Ramzan Ali Syed, President; Professor Muhammad Shafi, President-Elect; Professor Raja Mumtaz, Secretary; Dr. M. Latif Chaudhry, Joint Secretary; and Dr. M. Manzoor, Treasurer.

By this time, a branch of the Society had been formed in the Southern Region, and Dr. T.H. Kirmani participated in this Annual Meeting as the General Secretary of this new branch. To combine all the existing branches of the Society (North and South zone branches in West Pakistan and one branch in East Pakistan), into one all-Pakistan organization, a meeting was held on November 16, 1968, in Dacca. The meeting was successful, and the following officers were elected for the joint all pakistan society: Professor M.A. Jaleel, President; Dr. A. Alim Chaudhry, Secretary; and Dr. T.H. Kirmani, Treasurer. At this meeting the branches were reorganized into regional branches of the Center; hence, the OSP, Lahore, was renamed Ophthalmological Society of Pakistan-North Zone and the OSP, Karachi, was renamed Ophthalmological Society of Pakistan-South Zone.

In 1969, on November 7-9, an all Pakistan Annual Clinical Conference was held in Karachi under the Center. The Central Health Minister, Dr. A.M. Malik, an ophthalmologist himself, participated in the proceedings. The Government of Pakistan also supported the Society by allowing travel and daily expense allowances to Government employees who read papers at this Conference (Letter No. SO (ME-I)-178-2/63, dated April, 1969). The Second Annual Conference was held April 5, 1970, at the Hotel Intercontinental in Lahore under the chairmanship of Lt. Gen. S.M.A. Farooki. I was fortunate to have kept records of these events.

What happened to our country in 1971 during the conflict between the leaders of West Pakistan and the leaders of East Pakistan is a very dark chapter in history for all Pakistanis. The personal ambition of a few tore the whole nation apart. It hurts me to repeat it. The creation of Bangladesh from East Pakistan disrupted the momentum and the continuity of Society activities as it disrupted other institutions. We lost, of course, our East Wing Branch. A Biennial Conference was held on December 11 and 12, 1971 at Karachi. However, the next meeting could not be held until 1974, when we met on March 24 at King Edward Medical College, Lahore. Two more years went by before the next meeting was held in the Hotel Intercontinental at Lahore on March 5 and 6, 1976. Mr. P.D. Trever-Roper, the renowned British ophthalmologist was the guest of honor at this meeting. Dr. Abdul Khaliq, the Finance Minister of Punjab, presided. This proved to be one of the best attended conferences of the Society. The following officers were elected at this meeting: Professor M. Nawaz, President; Professor M. Latif Chaudhry, President-Elect; Dr. M. Khalil Rana, Secretary; and Dr. M. Hayat Akhtar, Treasurer.

Now the Society was firmly established and ready for bigger things. We had the honor of hosting the 7th Congress of the Asia-Pacific Academy of Ophthalmology. It was a fine experience and a delight to collaborate with APAO for its meeting in Karachi on March 4-9, 1979. (Figure 1 to 3) The late Professor Jules Francois, then President of International Council of Ophthalmology, was one of many internationally prominent men in ophthalmology who honored us with their presence. He presented a paper on **The Congenital Glaucoma and its Inheritance.** Other major topics at the Karachi Congress included glaucoma, the intraocular lens implantation surgery, diabetic retinopathy, and keratoplasty. The first Microsurgical Teaching Workshop organized by APAO was started at Karachi as a part of the APAO's Continuing Education Program. Professor Saiichi Mishima of Japan, Dr. Arthur S.M. Lim of Singapore, Professor Ian Constable of Australia, and Dr. Ian Robertson of Australia conducted this workshop. I am pleased to say that this occasion provided all of our members a view of what was being thought and done in ophthalmic circles around the world.

Our joint meeting with APAO was of great significance in another aspect. General Muhammad Zia-ul-Haq, the President of Islamic Republic of Pakistan very graciously presented the Inaugural Address. He said in his speech:

> "I consider the profession, of medicine a very noble profession like many other professions, but amongst the noblest of professions, the specialty of ophthalmology is the finest."

He also announced far reaching decisions for the promotion of Pakistan Ophthalmology in the Notification of April 24, 1979. This was actually the "Dastar Bundi" (convocation) of our Society which had attained the age of majority, 21. The prestige of the Ophthalmological Society of Pakistan reached a new

Figure 2. President Zia-ul-Haq viewing the First Scientific Exhibition held in Pakistan. Looking on are Drs. Mumtaz, Wania, Mukhtar, and Anklesaria (left to right).

Figure 3. A delightful cultural show of Pakistani dances.

height when President General Muhammad Zia-ul-Haq became Patron-in-Chief of the Society. During the 1979 APAO Congress, President Zia announced that the Government of Pakistan would begin allowing the import of ophthalmic instruments not being manufactured in Pakistan free of any customs duty charges. This naturally enhanced the quality of eye care and the expertise of ophthalmologists in Pakistan. In Pakistan, ophthalmology has always been considered as a sub-branch of general surgery. I was very pleased when President Zia acceded to my request that ophthalmology should have recognition as a separate specialty, independent of other branches of surgery. This will prove, Insha Allah, a milestone in the history of Pakistan Ophthalmology.

Professor Ramzan Ali Syed, one of the greatest ophthalmologists of Pakistan, was awarded the coveted "Jose-Rizal Medal" of the Asia Pacific Academy of Ophthalmology. (Figure 4). He was the first Pakistani to receive the honor. I am sure all Pakistanis felt proud when President General Zia-ul-Haq presented the medal to Professor Syed on behalf of the Asia-Pacific Academy of Ophthalmology.

President Zia also honored the Pakistani ophthalmologists by instituting the Ramzan Ali Syed Gold Medal to be annually awarded to a distinguished Pakistani Ophthalmologist. At the conclusion of the APAO Congress, the Society held its business meeting at which the following officers were elected: I, President; Dr. Jamshed H. Wania, President-Elect; and Dr. M. Khalil Rana, General Secretary.

Figure 4A. Professor Ramzan Ali Syed, the recipient of APAO's 1979 Jose Rizal Medal.

Figure 4B. Professor Mahmud A. Shah, the recipient of 1983 Ramzan Ali Syed Gold Medal.

The next Annual Meeting was held in Peshawar in February, 1980. This was the first meeting attended by a significant number of foreign delegates. The traditional hospitality of Pathans was one of the most memorable aspects of the event. In December of the same year, the Society held another conference in Lahore and called it "Ophthalmo 80." The conference was a great success with ophthalmologists, because in addition to high quality presentations, many instructional workshops were arranged at Fatima Jinnah Medical College for Women. A resolution was passed requesting the President of Pakistan to establish an independent Institute of Ophthalmology in Lahore. To the great delight of the Society Membership, it was announced in the next Annual Meeting in Karachi, in November 1981, that the President of Pakistan had granted this request and had issued directives to establish an Institute of Ophthalmology at King Edward Medical College, Lahore. In the Karachi meeting members also voted to allow any region of Pakistan with at least 10 ophthalmologists to establish a new branch and affiliate with the Center or the nearest zonal section of the Society.

Now the Society was approaching its "Silver Jubilee" and the Department of Ophthalmology of King Edward Medical College, Lahore was requested to arrange the 1982 meeting. It was held in Lahore, and one of the highlights of this occasion was the participation of the "Orbis" team from the United States of America. I feel that the celebration of the Silver Jubilee of the Ophthalmological Society of Pakistan was a genuine tribute to the professionalism, unity, and patriotism of all the members of this worthy organization.

As Muslims and Pakistanis, we have always loved to be hosts and profoundly enjoy opportunities to do so. A great opportunity came our way once again to host an international organization, the Afro-Asian Congress of Ophthalmology. The AACO's meeting was scheduled in March of 1984, so we decided to delay our Society's 1983 Annual Meeting and hold it at the same time as the AACO's meeting. The 8th Afro-Asian Congress of Ophthalmology and the Annual Meeting of the Ophthalmological Society of Pakistan were jointly held on March 5-9, at the Hotel Hilton in Lahore (Figures 5-7). The Congress was attended by foreign delegates from Asia, Africa, Europe, and America. The Congress program was divided into 14 Scientific Sessions spread over five days. Dr. Khalid J. Awan presented his data on two heretofore unrecognized entities. The first was **Essential Limbitis,** a nonspecific localized irritation of the limbus of unknown etiology that is accompanied by sharp pain and profuse tearing with very little injection of the eye. A focal edema with microscopic ruptures of the limbal conjunctive that stain with fluorescein when examined under slit lamp is seen in most cases of this disease with a benign course. Lively discussion followed Dr. Awan's presentation and several members of the audience said they had seen the entity but not recognized it before. Dr. Awan's feeling was that the etiology of essential limbitis may be allergy but is probably viral. His second interesting presentation was on a new syndrome of **Hypertelorism and Angle Closure Glaucoma.** He presented three patients who had this syndrome. All of them were in their sixth decade of life. Dr. Awan suggested that all patients with hypertelorism should have gonioscopic examination, particularly when they reach their sixties, to rule out narrow angles and avoid acute attack due to increasing thickness of the crystalline lens.

Dr. Muhammad Humayun and Dr. Khalid J. Awan presented their observations on **Persistent Hyperplastic Primary Vitreous in Adults.** Dr. Daljit Singh from India, Dr. T.H. Kirmani from Karcahi, Dr. Akira Momose from Japan, Dr. Khalid Eyada from Egypt, and Dr. Akhtar Jamal Khan of Karachi presented their individual experiences with intraocular lens implantation and cataract surgery. It appears that the interest in IOL surgery is steadily increasing in Asian and African countries, and by the number of papers read at the Congress one can see that IOL implantation will become the standard method of cataract surgery in these countries in the not very far future. Dr. B. Lavingia presented the interesting method of **donor corneal preservation in Honey/Dextron medium** at 79° after perfusion with Ringer's solution for 20 minutes. The corneas were kept viable up to 6 days, he said. Drs. N. Verma, J. Singh, and Y. Dayal presented **ultrasonographic studies in orbital and optic nerve lesions.** Drs. Wasif Kadri and A. Kamal gave an introductory talk on **fluorescein angiography in Pakistan.** Dr. S.R.K. Malik presented a paper on **hazards of timolol.** Dr. Maurice

Figure 5. (.Mumtaz): Afro-Asian Congress of Ophthalmology at Lahore, 1983. A group of participants: (L to R) Dr. Mustafiz-ur-Rahman, Dr. Jamshed Wania, (President OSP), Mrs. Mustafiz-ur-Rahman, Dr. M. Humayun, the author, and our Editor.

Rabb of the United States presented the papers on **The Spectrum of Sickle Cell Eye Disease** and on **Serous Detachment of the Macula** with color slides of highest clarity and beauty. Some of the papers with highest attendance were those presented by Professor Gholam A. Peyman of Chicago. His paper, on **Clinical Management of Endopthalmitis** included a discussion on intravitreal antibiotics and vitrectomy in the treatment of endophthalmitis. It was received with much enthusiasm, and his other paper, **Ten Year Experience With Eyewall Resection for Uveal Malignant Melanomas** included his results of this technique on 34 eyes (of which 11 had to be enucleated) and was quite interesting. Laser applications are becoming routine in ophthalmic practice the world over. Dr. Peyman gave a very informative talk on **Ocular Effects of Various Laser Wavelengths.** The **Management of Glaucoma** was expertly presented by Professor Akira Nakajima. Drs. R.K. Goulatia and N.K. Misra presented papers on the best use of **plain x-rays and computerized tomography in neurophthalmology.** Dr. Dil Muhammad Mirza from Lahore stimulated many among the audience by presenting his simpler, low cost techniques of ocular surgery. He also demonstrated various devices he had himself made from basic parts. Many other papers, workshops, technical exhibits, and auxillary activities were part of the activities. Dr. Jamshed Wania was the Chairman of the Congress and Dr. Muhammad Yaqin was the Organizing Secretary. We owe them our thanks for their superb efforts in making the 8th Afro-Asian Congress of Ophthalmology a great event.

In addition to hosting the 8th Afro-Asian Congress, the Ophthalmological Society of Pakistan was involved in several other significant events. General Muhammad Zia-ul-Haq, the President of Islamic Republic of Pakistan graced the occasion as Chief Guest and inaugrated the Congress. He also presented the Ramzan Ali Syed Gold Medal to Professor Mahmud Ali Shah (Figure 5) for his distinguished professional and academic achievements in ophthalmology. President Zia showed his generosity and regard for ophthalmology and for Professor Shah by conferring on him the rank of Honorary Rear Admiral. Professor Shah is an upright, discerning, learned, and revered member of the Society.

During the Society business meeting, it was favorably voted that all Ophthalmologists of Pakistani background living abroad may become full and active members of the Ophthalmological Society of Pakistan by fulfilling the standard requirements of membership.

Another historic milestone was achieved. The Society membership unanimously decided to launch the Pakistan Journal of Ophthalmology. I was given the honor of looking after this project and instructed to choose whomever I though best suited for the task of editing the journal. I decided to enlist Dr. Khalid J. Awan as Editor and Chaiman of the Editorial Board. I requested him to search out an internationally regarded Editorial and Advisory Board and work on the publication and printing of the Journal from his United States office. I felt that this was the best way to create a Journal with international standards. Dr. Awan has a solid background in writing and has published more original papers in international peer review ophthalmic journals than any other Pakistani ophthalmologist I know of. He is also a Life Member of the Ophthalmological Society of Pakistan. I thought it would be unwise not to utilize his expertise and energy for such an important project. It was entirely through his individual untiring labor and tremendous personal sacrifices that the First Issue of the Pakistan Journal of Ophthalmology came out in Muharram, 1405 (October, 1984). The comments of the leaders of world ophthalmology about the first issue appear on the pages of this issue. After reading these evaluatory remarks, I doubt that any truly patriotic Pakistani will not feel indebted for ever to Dr. Awan for bringing such international recognition to Pakistan and ophthalmology. Dr. Awan's accomplishment has made him the pioneer in this venture and Professor Shah's comments (on page 102 of this issue) reflect the feelings of all of us.

I have tried to state the facts according to the best of my knowledge. However, if there are any omissions in my account, they are totally unintentional, and I sincerely apologize to those I may have inadvertently failed to mention. The Ophthalmological Society of Pakistan is very dear and sacred to me, and to promote its interest, I shall do everything within my reach. May Allah give us unity, strengthen our faith, and discipline us to be one. Amin.

Original Article

Diabetic Retinopathy: Current Concepts and Two Signs for Early Diagnosis, Splinter Hemorrhages and Iridescent Dots*

Khalid J. Awan, M.D.**
and
Muhammad Humayun, F.R.C.S. (C)**

ABSTRACT: The authors discuss the current advances in the understanding of clinical, pathogenetic, and therapeutic aspects of diabetic retinopathy by giving clinical illustrations from their personal experience. The management of proliferative diabetic retinopathy and of macular edema in non-proliferative (background) retinopathy is detailed. They additionally report on the significance of isolated flame-shaped hemorrhages on or about the optic disc and a new finding, named "iridescent dots" of the retina by the authors, in the early diagnosis of diabetes and its effects on the retina. The paper also includes illustrative case reports-one each of isolated flame-shaped hemorrhage near the optic disc in a 32-year-old woman, venous "iridescent dot" in a 32-year-old man, lipemia retinalis in a 50-year-old woman, lipemic diabetic retinopathy in a 67-year-old woman, and bilateral acute optic disc swelling in a 21-year-old man. (Pak. J. Ophthalmol. 1: 71-90, 1985)

Recent advances have improved our understanding of diabetic retinopathy, but they have also brought with them some complex methods of examination and complicated criteria for patient evaluation. Thus, stereo fundus photography and fluorescein angiography, facilities not available in every ophthalmic office, are now regarded as important for the evaluation of diabetic retinopathy,[1] and one is confronted with the task of classifying the patients one sees with this condition into one of as many as thirteen categories, according to the types of retinal lesions and the severity of retinopathy.[2,3] The situation has been further aggravated by the use of seemingly endless numbers of abbreviations in the writings of 1980's on diabetic retinopathy (Table 1). Despite great strides in the understanding of the pathogenesis of diabetic retinopathy in recent years, the fundamental change leading to retinal microangiopathy has eluded the researchers. Until this basic change is clearly understood, diabetic retinopathy will remain a leading

TABLE 1

Abbreviations in Current Writings on Diabetic Retinopathy

DR	IDD	IDDM
NIDDM	NVE	SE
NVD	IOP	DRS
ETDRS	CWP	BDR
PDR	IRMA	FAZ
PPDR	CBMT	HE
VB	NPDR	H/Ma
DCCT	AODM	MODY
IODE	JDM	JODM
MODY	LDR	HbAc
E/IMP	NIA	IRNV
NCR	MDD	etc.

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*Study was funded by the Pakistan Academy of Medical Sciences. Also the section on pathogenesis of diabetic retinopathy includes some portions of the thesis by Dr. Humayun for the Fellowship of the Pakistan Academy of Medical Sciences.

**Both Dr. Awan and Dr. Humayun are alumni of Nishtar Medical College, Multan, Pakistan.

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cause of blindness in the world. Fortunately, this blindness may be temporarily cured, or warded off, in many diabetics by the application of modern modes of treatment at the disposal of ophthalmologists, such as laser photocoagulation and vitrectomy. An earlier diagnosis of diabetic retinopathy is important for more efficient management and better prognosis. The intended purpose of this communication is to present in a simpler form the present status of knowledge about clinical fundus changes, the current concepts about the pathogenesis, and the management of diabetic retinopathy, and to describe two new signs, "iridescent dots" and isolated flame-shaped disc hemorrhages, that we believe may be helpful in the early diagnosis of diabetes and its effects on the retina.

INCIDENCE

"Diabetic retinitis is very rare." This statement was made by Juler[4] exactly one hundred years ago in 1884. Today, it is considered the commonest type of retinopathy and one of the leading causes of blindness in the world.[5-7] However, it is still uncommon in children and adults under 30 who have had diabetes for 10 years or fewer.[8] Diabetic retinopathy occurs in about 40 percent of all diabetics.[5]

During the last decade several classifications of diabetic retinopathy based on the type of lesions or on the severity of retinal changes were proposed, but their complexity renders them almost impractical for routine use in the management of the patients. Now the trend is toward simpler classification of diabetic retinopathy into nonproliferative, or "background", retinopathy; preproliferative retinopathy; and proliferative retinopathy. The earliest changes in the retina are not ophthalmoscopically visible, but may be detected by fluorescein angiography or vitreous fluorophotometry.[9]

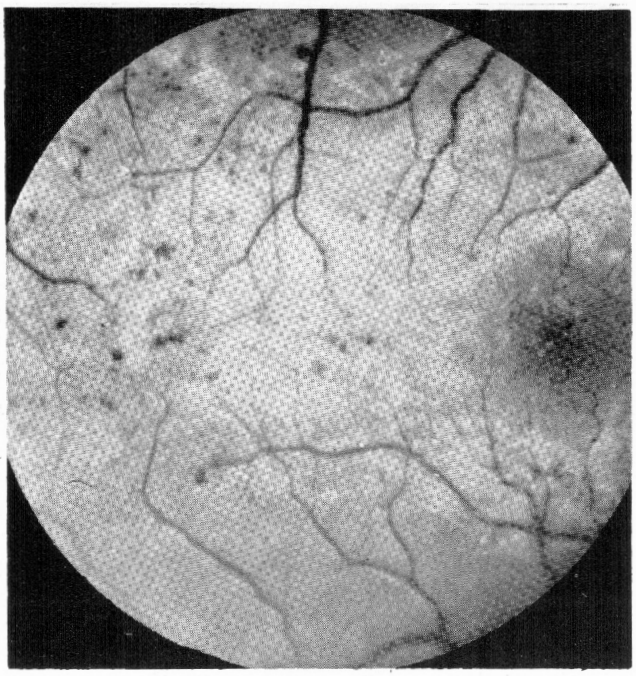

Figure 1. (Awan & Humayun): Right eye. Diabetic retinopathy. Microaneurysms temporal to the macula in early stages of nonproliferative (background) retinopathy.

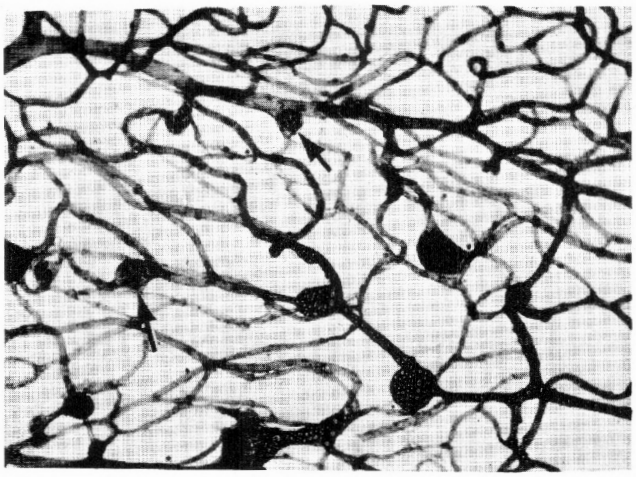

Figure 2. (Awan & Humayun): Retina trypsin-digested preparation. Microaneurysms clustered around acellular capillaries in diabetic retinopathy. (From Yanoff, M: Ocular pathology of diabetes mellitus. Amer. J. Ophthalmol. 67:21-38, 1969. With permission from the Ophthalmic Publishing Company and Dr. Yanoff) Periodic acid-Schiff and hematoxylin X45.

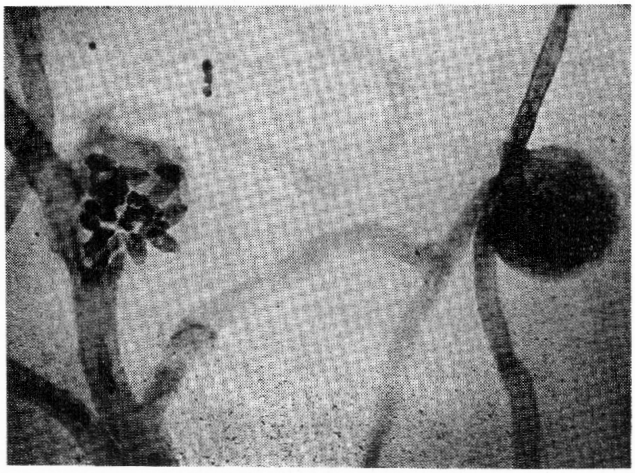

Figure 3. (Awan & Humayun): Trypsin-digested retina preparation. Diabetic microaneurysms appear as outpouchings from the capillaries. Microaneurysm on left is probably in early stages and has thin wall with proliferated endothelial stages. Microaneurysm on the right is in late stages and has its lumen obliterated by basement membrane material. (From Yanoff, M: Ocular pathology of diabetes mellitus. Amer. J. Ophthalmol. 67:21-38, 1969. With permission from the Ophthalmic Publishing Company and Dr. Yanoff.) Periodic acid-Schiff and hematoxylin X220.

NONPROLIFERATIVE (BACKGROUND) RETINOPATHY:

Nonproliferative, or background, retinopathy is characterized by intraretinal changes that are visible by direct ophthalmoscopy. It is currently believed that the earliest ophthalmoscopically demonstrable sign of diabetic retinopathy is venous dilation.[6,10,11] The more familiar finding of retinal microaneurysms is also an early manifestation. Usually they lie on the venous side of circulation in the posterior pole of the fundus and appear in groups (Figures 1 and 2). Their number usually depends on the duration of diabetes, increasing with longer duration. Their size is

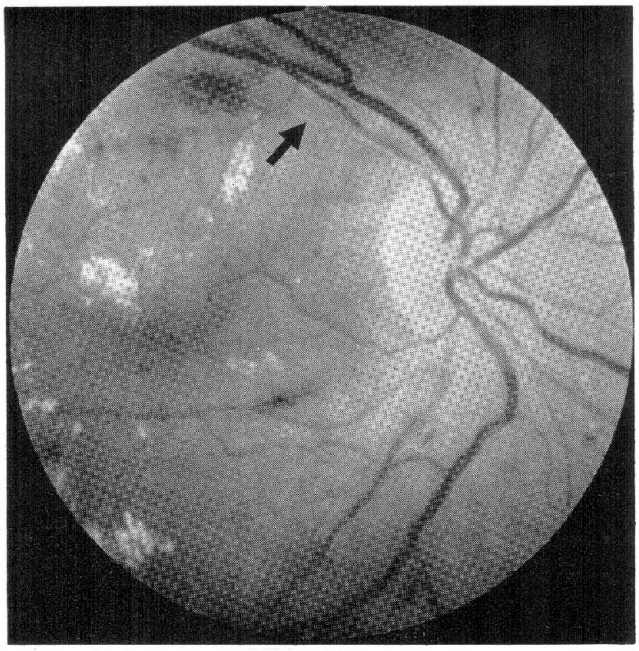

Figure 4. (Awan & Humayun): Right eye. Diabetic retinopathy. Exudates and "dot" or "blot" hemorrhages. Also present are a few splinter hemorrhages and microaneurysms. The area pointed out by the arrow shows no microaneurysms by ophthalmoscopic examination but had them by fluorescein angiography. Compare with Figure 5.

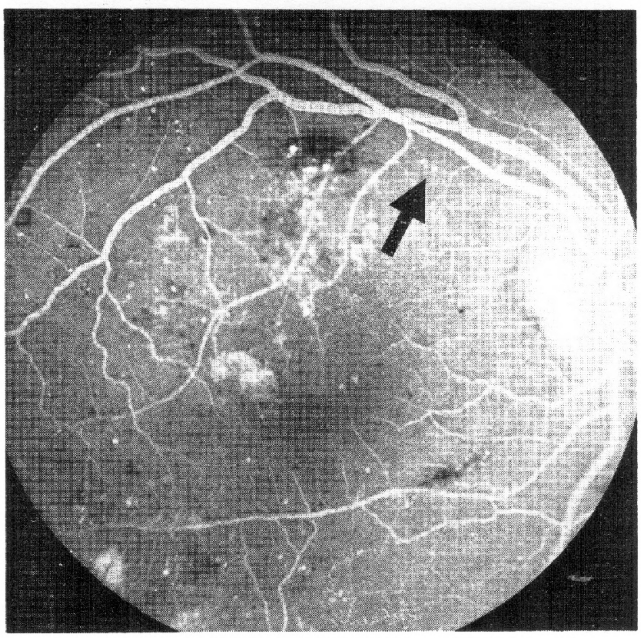

Figure 5. (Awan & Humayun): Fluorescein angiogram of the eye shown in Figure 4. Note the microaneurysms that were not visible with plain ophthalmoscopy (arrow). Also many more microaneurysms are visible here than in Figure 4.

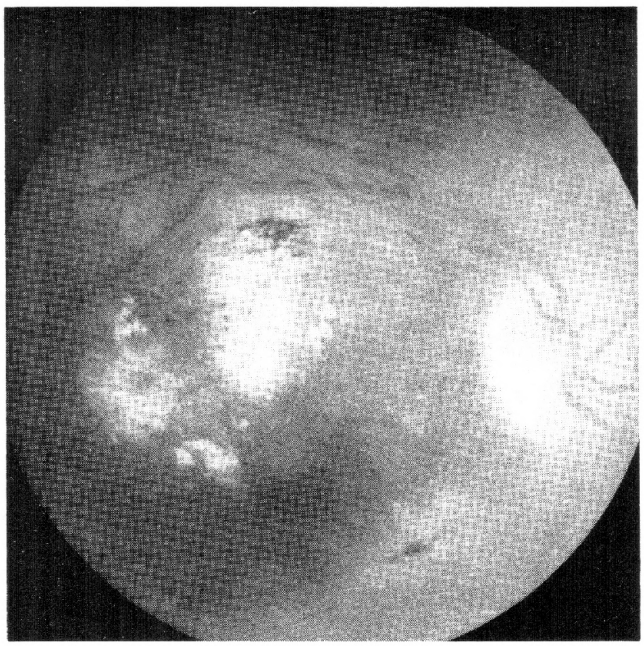

Figure 6. (Awan & Humayun): Late phase of fluorescein angiography of the eye in Figures 4 and 5. Note the leakage in the areas of the largest number of microaneurysms. Further extension of this to macula could seriously affect the sight. This type of lesions are ideal for focal photocoagulation with very good results.

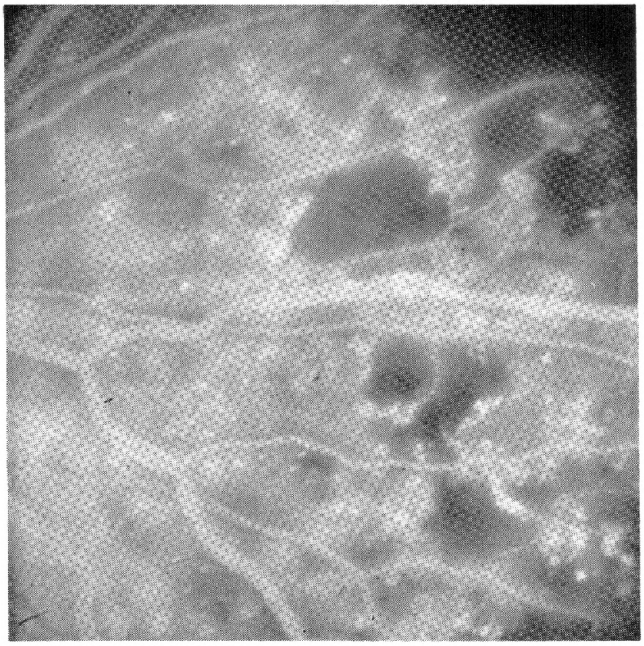

Figure 7. (Awan & Humayun): The darker geographic spots are areas of non-perfusion on fluorescein angiography.

somewhere between 25 and 100 microns, and they may be saccular or fusiform in shape. Usually, areas of capillary nonperfusion are present next to the groups of microaneurysms. The capillaries in the neighborhood of these nonperfusion areas dilate because their walls have become weakened due to the loss of pericytes.[12] The ratio of endothelial cells to pericytes changes to a mean of 4.2 from the normal mean of 1.1.[13] The focal dilation of a capillary, or an outpouching of the weakened wall, leads to formation of a microaneurysm. The endothelial cells multiply to support the weak walls of microaneurysm, and their unrestricted proliferation eventually may lead to the total obliteration of the lumen (Figure 3). The process of hyalinization may make the microaneurysms ophthalmoscopically obscure, [6,7] one of the reasons for

some observers' mistaken impression of improvement in diabetic retinopathy. All microaneurysms are not observable by an ophthalmoscope, and fluorescein angiography is the best available way to demonstrate them, and areas of nonperfusion, in the greatest number (Figures 4, 5, 6, and 7). On fluorescein angiography, the microaneurysms appear as white dots that show leakage of dye in the late stages (Figure 8). Sooner or later the microaneurysms are accompanied by exudates and hemorrhages.

Hemorrhages of diabetic retinopathy are called "dot" or "blot" because of their circular appearance imparted by their location in the deeper retinal layers: the inner plexiform, inner nuclear, and outer plexiform layers (Figures 9 - 13). Flame-shaped hemorrhages, usually seen in hypertensive retinopathy, may be seen in established nonhypertensive diabetic retinopathy.

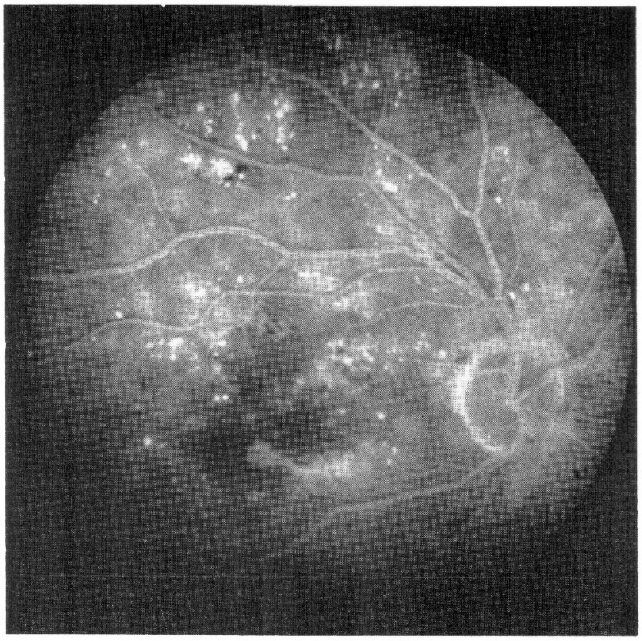

Figure 8. (Awan & Humayun): Fluorescein angiogram in venous phase showing a large number of microaneurysms.

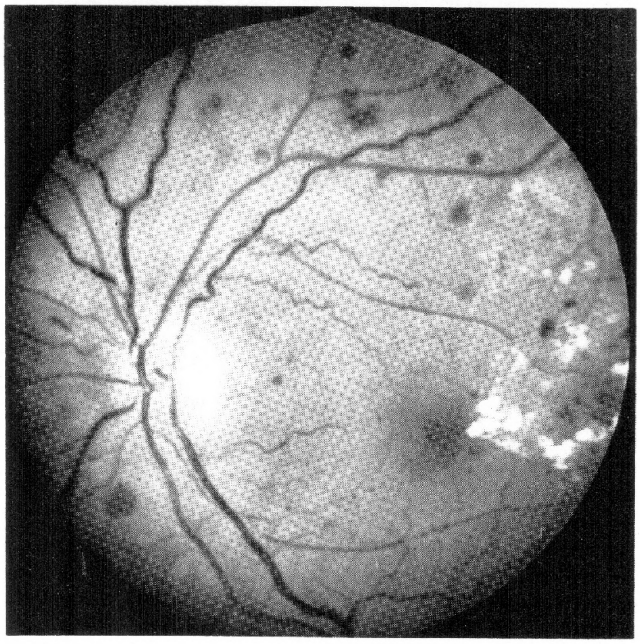

Figure 9. (Awan & Humayun): Ophthalmoscopic view of fundus. Hard exudates and "dot" or "blot" hemorrhages in diabetic retinopathy.

ISOLATED FLAME-SHAPED HEMORRHAGES ON DISC

Recently it was reported that small hemorrhages in the peripheral retina, next to the ora serrata, may appear in diabetics before any other sign of retinopathy is seen in the posterior pole.[15] Awan[15] is reported on flame-shaped isolated hemorrhages on or about the optic disc as one of the early indicators of diabetes.

Case 1.

A 39-year-old woman came with a complaint of difficulty in reading and intermittent blurriness of vision. A physical examination by her family physician two years prior to her visit was normal. She was a moderately build healthy appearing woman. Eye examination (by KJA) showed her vision to be 20/20 (6/6) without glasses in each eye. External examination, slit lamp examination, extraocular muscle functions, intraocular pressure (15 mm Hg-OD, 16 mm Hg-OS), convergence, and pupillary reactions were normal. Cycloplegic retinoscopy showed +225 +25 X90 in the right eye and +200 +50X90 in the left eye. Ophthalmoscopy revealed a flame-shaped hemorrhage at 10 o'clock near the right optic disc (Figures 14 and 15). The retinal vessels and fundi were otherwise unremarkable. The patient was referred to an internist for full systemic evaluation. Only positive finding was oral glucose tolerance test that showed a fasting glucose of 106 mg/dl; 230 mg/dl after one hour; and 215 mg/dl after two hours.

Comments: Of nine patients who had this finding without the presence of the factors usually associated with flame-shaped retinal hemorrhages in otherwise healthy fundi, six had positive oral glucose tolerance tests.

The leakage of plasma from defective vessel walls into the retina gives rise to yellowish, hard, waxy exudates that are so characteristic of diabetic retinopathy (Figures 13, 14 and 15). These exudates usually form clusters, but may lie in a circular pattern around the site of leakage, called "circinate retinopathy" (Figure 16), or they may lie in the macular area as a "macular star". Histochemically, these hard exudates are lipoprotein deposits, in the outer plexiform layer. Very large and thick exudates may become permanent, but smaller exudates are gradually absorbed by macrophages over a period of months or years.

"IRIDESCENT DOTS" - A NEW FINDING

While doing a fundus examination on a young woman with a detached retina, one of us (KJA) noticed a sparkling dot, resembling Hollenhorst cholesterol emboli, only much smaller and non-arterial, at the beginning of the smallest ophthalmoscopically visible vein in the central retina (Figure 17) that was not involved in the retinal detachment.[15] When the patient was thoroughly evaluated for any systemic abnormality, the only abnormal findings were a positive glucose tolerance test and slight elevation of serum triglycerides. The

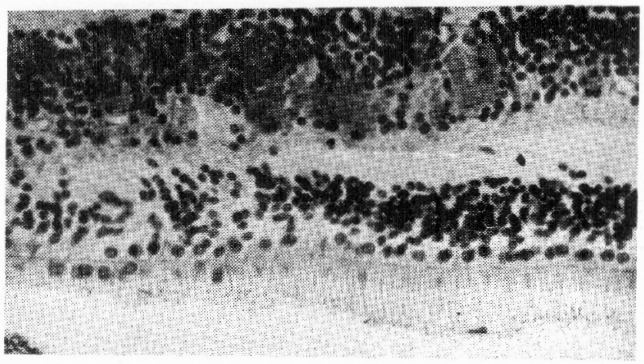

Figure 10. (Awan & Humayun): Histopathologically the "dot" or "blot" hemorrhage shown in Figure 9 are located in the inner nuclear and outer plexiform layers of the retina. (From Yanoff, M: Ocular pathology of diabetes mellitus. Amer. J. Ophthalmol. 67:21-38, 1969. With permission from the Ophthalmic Publishing Company and Dr. Yanoff.) (Hematoxylin-eosin X260).

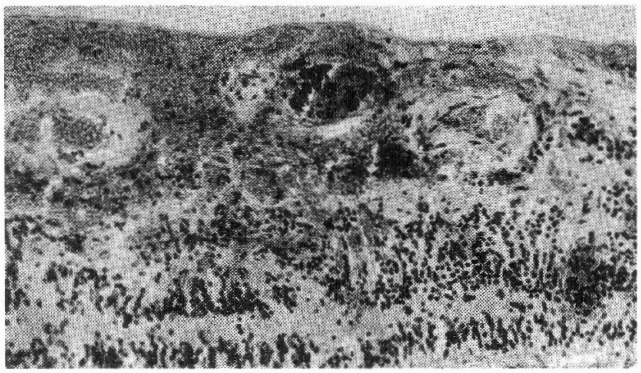

Figure 11. (Awan & Humayun): A large hemorrage involving all the layers of retina. (From Yanoff, M: Ocular pathology of diabetes mellitus. Amer. J. Ophthalmol. 67:21-38, 1969. With permission from the Ophthalmic Publishing Company and Dr. Yanoff.) (Hematoxylin-eosin 130).

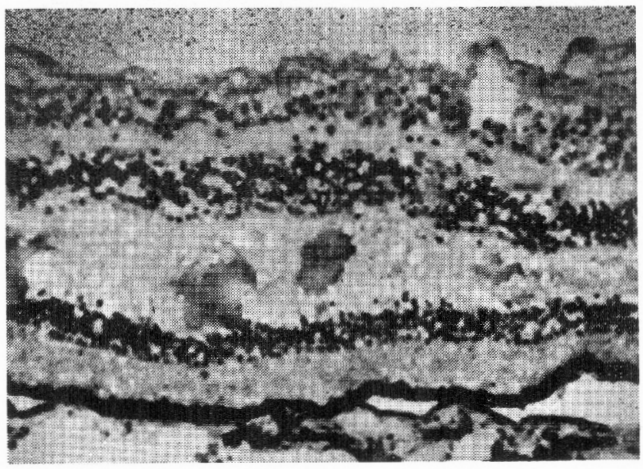

Figure 12. (Awan & Humayun): Histopathology of hard exudates. Note the homogenenous, eosinophilic deposits in outer plexiform layer of retina. (From Yanoff, M: Ocular pathology of diabetes mellitus. Amer. J. Ophthalmol. 67:21-38, 1969. With permission from the Ophthalmic Publishing Company and Dr. Yanoff.) (Hematoxylin-eosin X160).

patient's fundi had no abnormality other than the retinal detachment in the right eye.

Case 2.

A 34-year-old man came in for an eye examination for burning of his eyes and eyes feeling tired at the end of day. He appeared

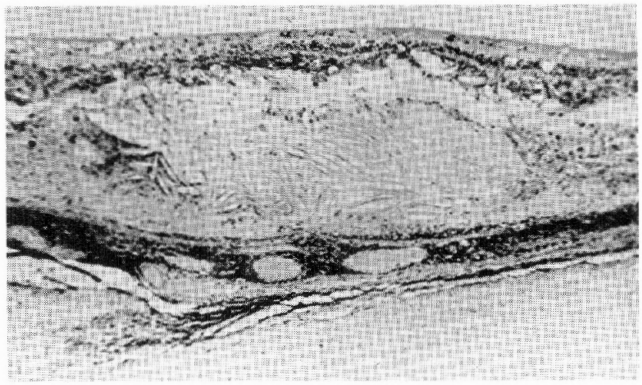

Figure 13. (Awan & Humayun): Absorbing hard wexy exudate (fatty) with cholesterol clefts. (Courtesy of Michael S. Ramsey, M.D.) (Hematoxylin-eosin X44).

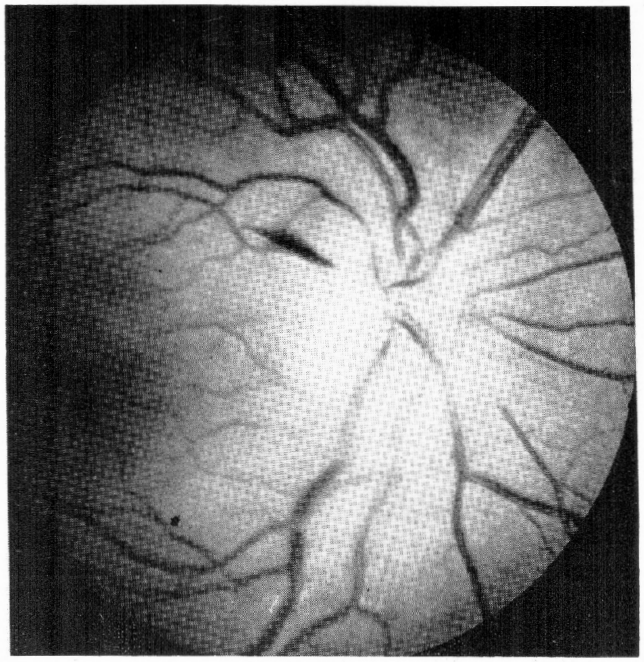

Figure 14. (Awan & Humayun): Case 1. Right eye. An isolated flame-shaped hemorrhage in an apparently healthy woman of 39. Glucose tolerance test was positive. (From Trans. Asia-Pacif. Acad. Ophthalmol., 1985)

healthy and had no serious systemic problem according to his family physician who had examined the man a year before. Eye examination (by KJA) showed his vision to be 20/20 (6/6) in each eye without any correction. External eye examination, slit lamp examination, intraocular pressure (16 mm Hg each eye), extraocular muscle functions, and pupillary reactions were normal. On ophthalmoscopic examination both fundi were completely normal except for an iridescent and refractile dot at the beginning of a nasal macular vein in the left eye (Figure 18). Its reflectivity varied with the tilting of the light beam of ophthalmoscope. He was carefully evaluated by an internist who found the oral glucose tolerance test to be positive. No changes were found in the serum lipid levels.

Comments: A careful ophthalmoscopic survey of eye patients led to a discovery of these isolated "iridescent dots" at or near the tips of the smallest veins of the posterior pole of 14 otherwise healthy patients between the ages of 19 to 42. Of these, 10 were found to have positive oral glucose tolerance tests. Although a rare and easily overlooked finding, these venous "iridescent dots" may be an early sign of retinal changes in diabetes warranting an evaluation of the

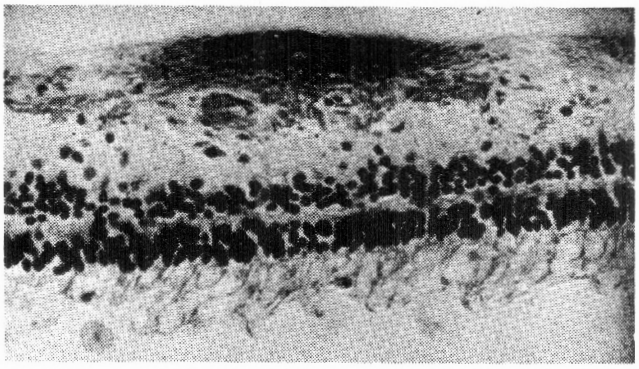

Figure 15. (Awan & Humayun): A flame-shaped hemorrhage in the nerve fiber layer similar to the one in Figure 14. (From Yanoff, M: Ocular pathology of diabetes mellitus. Amer. J. Ophthalmol. 67:21-38, 1969. With permission from the Ophthalmic Publishing Company and Dr. Yanoff.) (Hematoxylin-eosin X260).

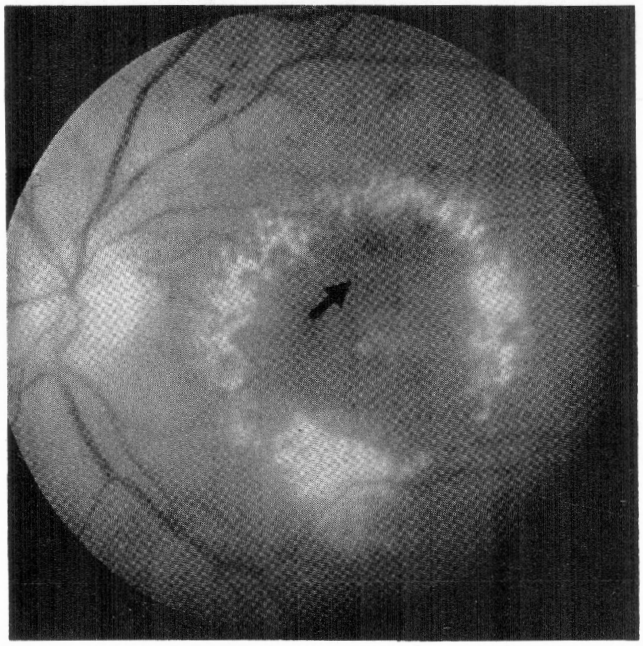

Figure 16. (Awan & Humayun): Diabetic circinate retinopathy involving the central retina. Patient responded favorably to focal photocoagulation of group of microaneurysms within the ring of exudates (arrow) and triple row circle of laser treatment temporal to the macula and nasal to the optic disc.

patient for this disease. The exact cause of these shiny dots is difficult to determine. They might be a result of altered blood components precipitated on the venous side by a decreased perfusion pressure, increased blood viscosity, increased platelet aggregation,[10] abnormalities of fibrinogen, etc.[16] Histopathologic studies have shown that lipid thrombi containing luminous crystals may develop in the retinal capillaries of diabetics.[17] It has been histopathologically demonstrated that lipid may accumulate in and around the retinal blood vessel in idiopathic hyperlipidemia, [18,19] and in diabetes.[20] Hyperlipemia is relatively common in patients with diabetes mellitus.[21,22,23] It appears that such lipid thrombi may explain the iridescent dots just described. It has been suggested, however, that in addition to accumulation of fatty components, the hard exudates are made up of debris of neuronal degeneration engulfed by the retinal microglia.[20] In

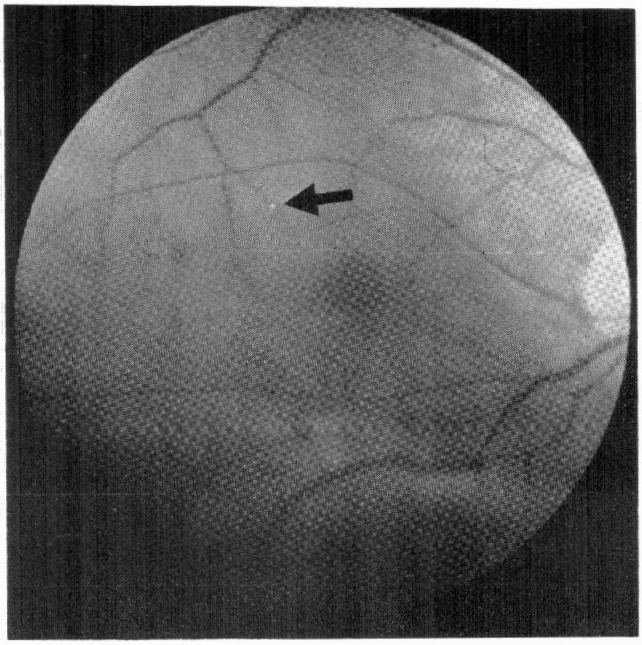

Figure 17. (Awan & Humayun): Right eye of a young woman with venous "iridescent dot" (arrow) of the retina. Glucose tolerance test was positive. (From Trans. Asia Pacif. Acad. Ophthalmol., 1985).

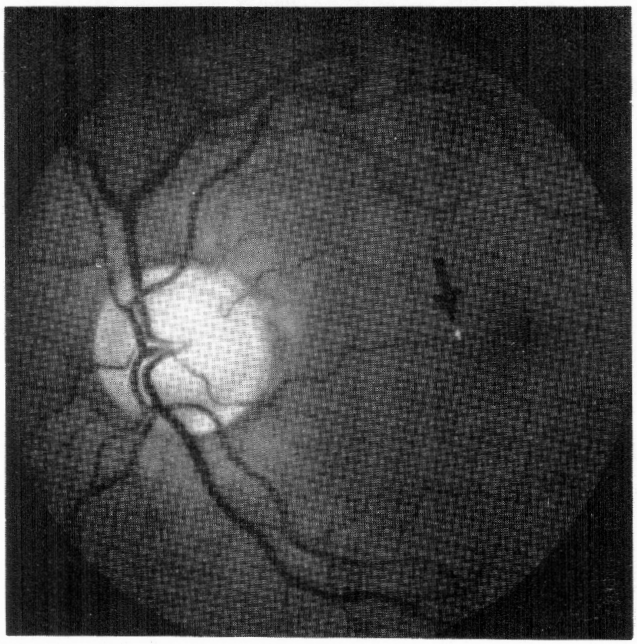

Figure 18. (Awan & Humayun): Case 2. Left eye. Venous "iridescent dot" (arrow) of the retina. The eye was entirely normal otherwise. The photograph is taken at an angle of maximum reflectivity of the shiny dot, making it appear larger than its actually size.

two patients with these iridescent dots and diabetes, fluorescein angiography did not reveal any abnormalities. Retinal edema, particularly when it involves the macula, is the most serious consequence of leakage from defective retinal capillaries and microaneurysms. The sight is seriously affected by cystoid macular edema caused by fluid that leaks into the foveal area, (Figures 19, 20, and 21), and it may be irreparably affected if the occlusive phenomenon of diabetic retinopathy involves the arterial side of the

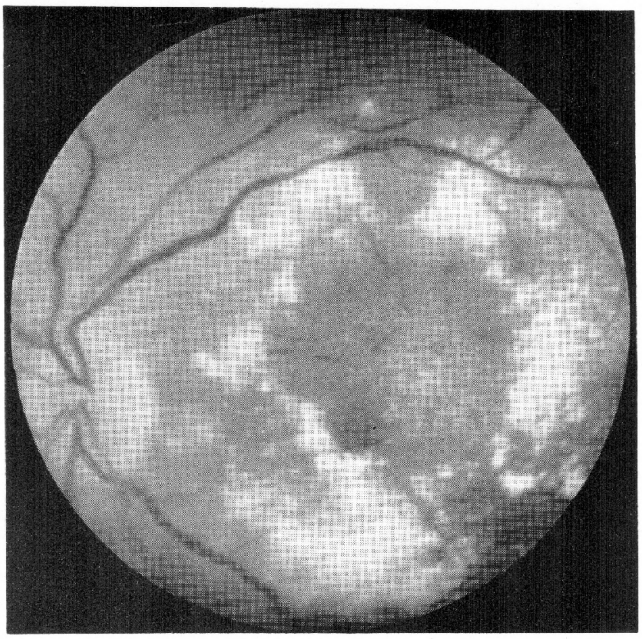

Figure 19. (Awan & Humayun): Left eye. Ophthalmoscopic view. Extensive hard exudates with significant edema of macula and the retina around it Ophthalmoscopic view.

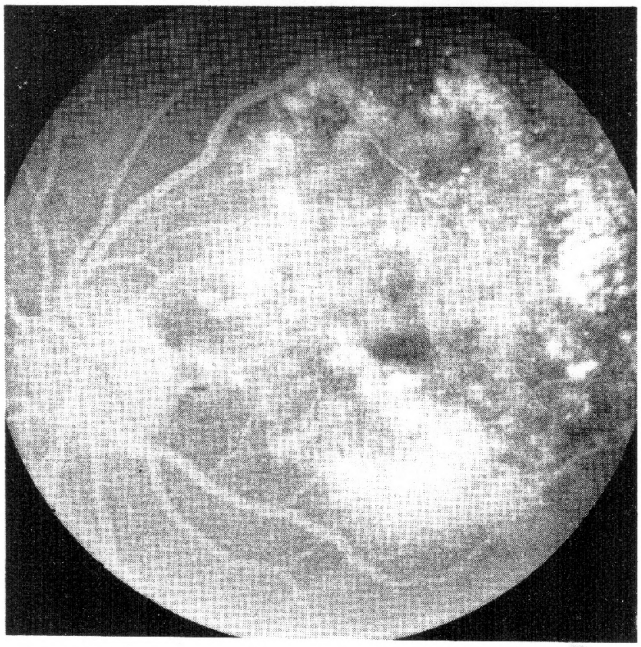

Figure 20. (Awan & Humayun): Fluorescein angiogram of the eye in Figure 19. Note the marked edema of the central retina characterized by extensive leakage.

circulation in the macular area. Similarly, hard exudates that accumulate in the macular area affect the vision seriously. If there is no other accompanying condition that affects the retinal arteries directly, such as hypertension, some authors feel the large retinal arteries remain unaltered in diabetic retinopathy.[11] Fluorescein angiography is the best way to evaluate the macular edema of diabetic retinopathy. This technique also clearly demonstrates the nonperfusion of perifoveal capillaries. Hemorrhages and traction phenomenon may directly

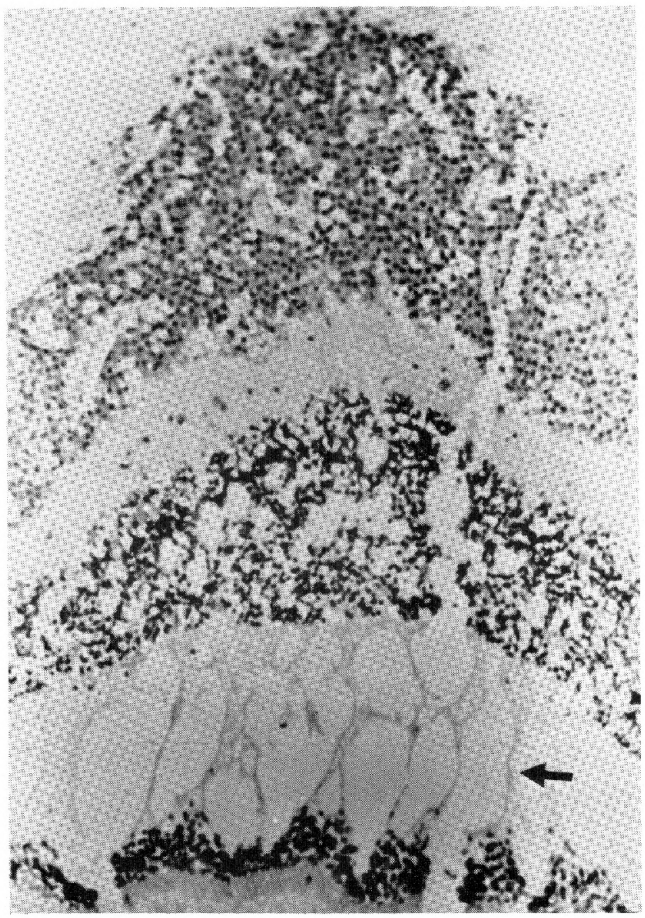

Figure 21. (Awan & Humayun): Cystoid edema of the macula (arrow) involving the outer plexiform and inner nuclear layers. (Courtesy of Michael S. Ramsey. M.D.) Hemotoxylin-eosin X120.

involve the macula. Another way of evaluating diabetic maculopathy is by noting the thickness of the retina by slit-lamp examination. It has been suggested that in addition to the leakage from the abnormal retinal vasculature, abnormalities of the retinal pigment epithelium may allow the fluid from choriocapillaries to pass into the sensory retina.[24] Ferris and Patz [25] suggest that, although spontaneous recovery in diabetic macular edema is not uncommon, over half of diabetics with macular edema will lose two or more lines of visual acuity within two years of diagnosis. They found photocoagulation the most promising therapy of diabetic maculopathy, but also recommend that attempts be made to normalize elevated blood sugar, decrease elevated blood pressure and improve cardiac and renal status of the patients.

PREPROLIFERATIVE RETINOPATHY

Preproliferative retinopathy is an advanced stage of background retinopathy and is an indication of increasing ischemia and, in some patients, the imminent onset of proliferative retinopathy. Three changes characterize the preproliferative stage: nerve fiber layer ischemia and infraction (soft, or cotton wool, exudates), beaded veins, and intraretinal vascular changes (intraretinal microvascular

abnormalities, recanalizations, and intraretinal neovascularizations) (Figures 22 and 23). Although soft exudates indicating nerve fiber layer infarcts are a hallmark of hypertensive retinopathy, they may occur in diabetes without hypertension.[26] An increased number of soft exudates is found in patients with severe kidney involvement (Figure 24). Because the retinopathy progresses to the proliferative stage, the soft exudates become less prevalent. As there is nonperfusion of capillaries in the areas of these nerve fiber layer infarcts, the infarcts block the underlying fluorescence during fluorescein angiography. "Beaded veins" are veins that appear engorged and show fusiform dilated areas with a sausage-like appearance along their course (Figure 25). It has been histologically shown that traction from vitreous strands leads to pulling of abnormal veins through the internal limiting membrane in the shape of loops. These loops have been called "tractional venous loops," and their walls show staining with flourscein.[27] "Intraretinal microvascular abnormalities" is a clinical term that is applied to dilated capillaries with increased number of endothelial cells or hypercellular arteriovenous communications without an increased rate of flow. These intraretinal microvascular abnormalities are found in the areas of capillary closure (Figures 22 and 23).[28] Unlike normal capillaries, they may be seen by direct ophthalmoscopy and may be easily demonstrated by fluorescein angiography. Intraretinal microvascular abnormalities appear with increasing ischemia, but disappear as neovascularization develops.[29] Intraretinal microvascular abnormalities leak fluorescein,[29] a feature that distinguishes them from recanalization in the previously nonperfused areas and from intraretinal neovascularization.[30,31] The intraretinal neovascularizations also differ from intraretinal microvascular abnormalities in that they have no arterial connection; they develop from and drain into the venules. The intraretinal neovascularization is differentiated from epiretinal and retinovitreal neovascularization by the profuse leakage of flourescein from the latter.[31] It is estimated that eyes with advanced preproliferative changes have a 50% chance of developing neovascularization in one year.[29] In some patients the preproliferative retinoapthy remains stationary or shows progression only after a long time. This makes the decision to treat or not to treat the patients with preproliferative retinopathy very difficult. The best approach might be to follow them closely and treat at the first sign of proliferative changes. Another finding that may suggest preproliferative ischemia in background retinopathy is the appearance of large, dark blot hemorrhages, representing hemorrhagic infarcts of the retina (Figures 26 and 27). Although diabetic retinopathy is believed to affect the posterior pole most severely, a recent study has shown that the midperipheral retina is far more prone to capillary nonperfusion than the posterior retina.[32]

PROLIFERATIVE RETINOPATHY

Of the patients with diabetic retinopathy, 90% have background retinopathy, and the remaining 10% have proliferative retinopathy.[29] Whereas background retinopathy is slowly progressive without catastrophic loss of sight, proliferative retinopathy often leads to total blindness. (However, the vision may be seriously reduced in background retinopathy if the macula is involved and it may remain excellent in proliferative retinopathy despite severe changes if a clear window remains in front of the macula till the last stages of the disease.) Proliferative retinopathy differs from background retinopathy in that it has neovascularization growing intraretinally, in the

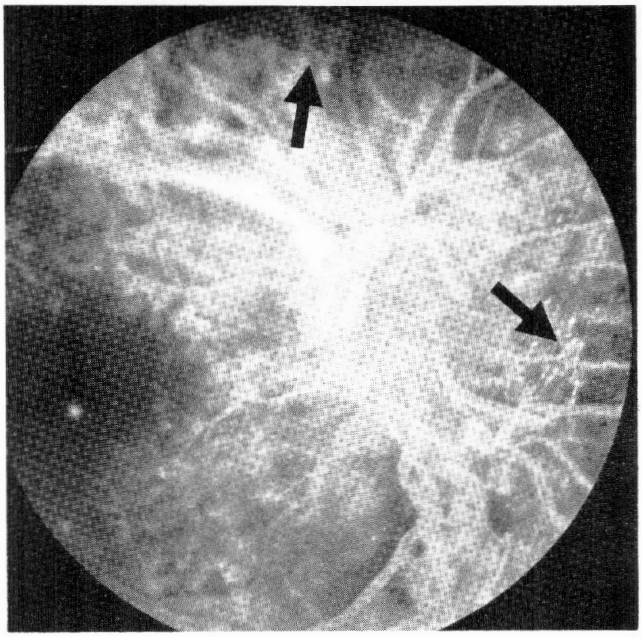

Figure 22. (Awan & Humayun): Fluorescein angiogram of eye with proliferative diabetic retinopathy, showing intraretinal microvascular abnormality (upper arrow) and intraretinal neovascularization (lower arrow).

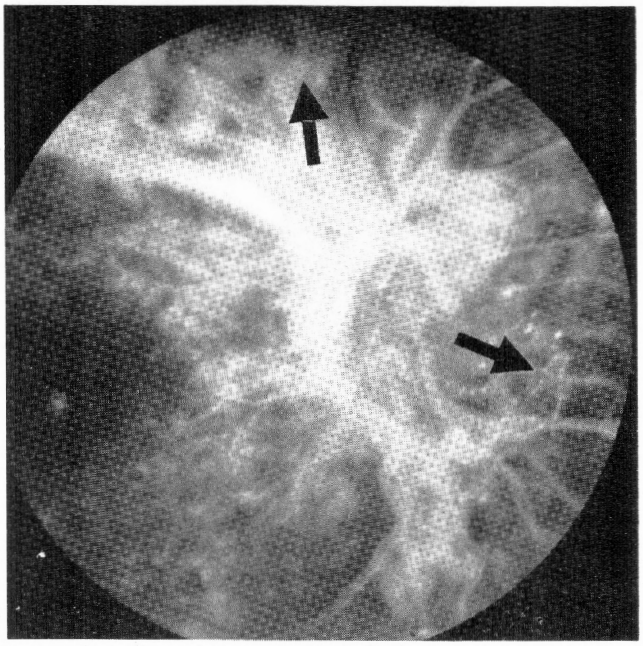

Figure 23. (Awan & Humayun): Same eye as in Figure 22 in the later phase. Note the leakage from the intraretinal microvasular abnormality (upper arrow) but no leakage from the intraretinal neovascularization (lower arrow).

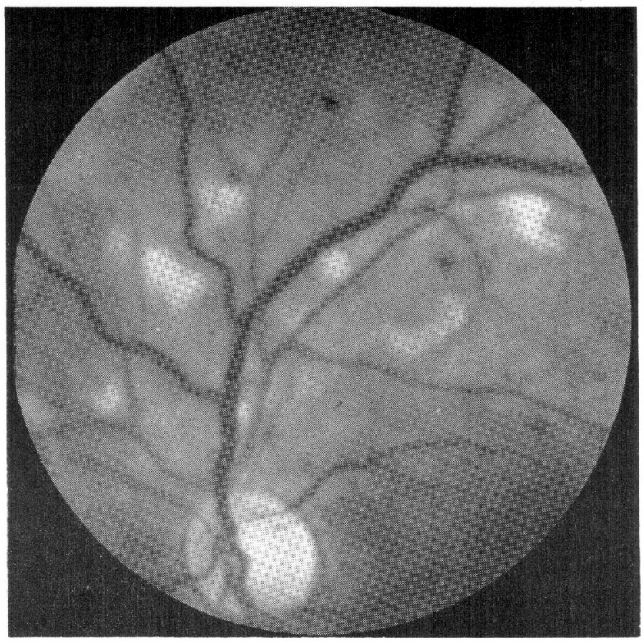

Figure 24. (Awan & Humayun): Preproliferative diabetic retinopathy. Left eye of a 28-year-old diabetic woman with severe kidney involvement. The retinopathy is dominated by large number of cotton wool spots. The patient died soon after this photograph was taken.

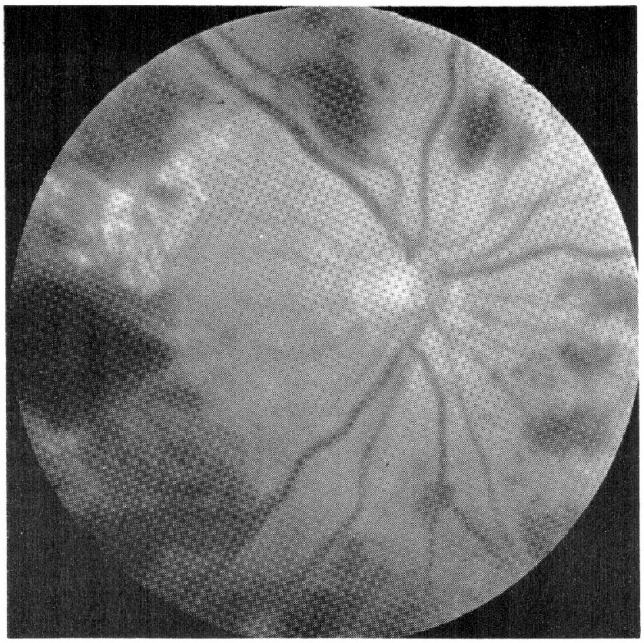

Figure 26. (Awan & Humayun): Preproliferative retinopathy. Dark, large hemorrhages in the deeper layers of retina in a 30-year-old woman. Soon after this photograph was taken, the patient developed severe proliferative retinopathy with severe vitreous changes.

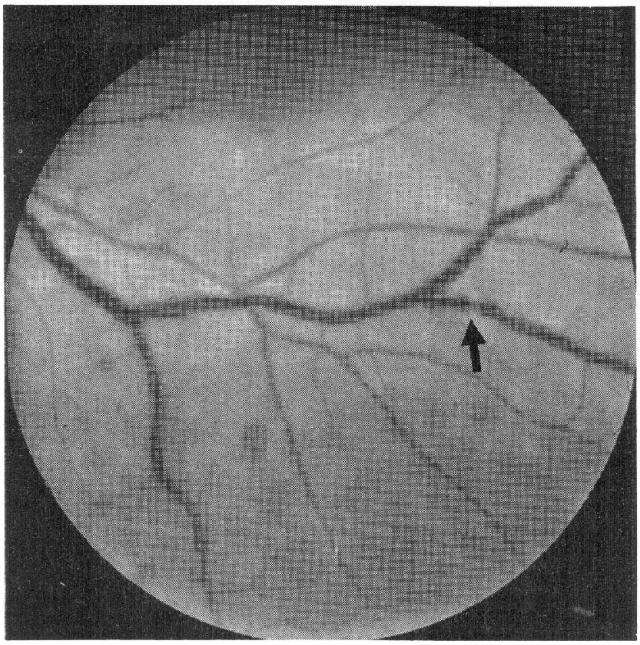

Figure 25. (Awan & Humayun): Beaded, dilated retinal veins (arrow) in preproliferative diabetic retinopathy.

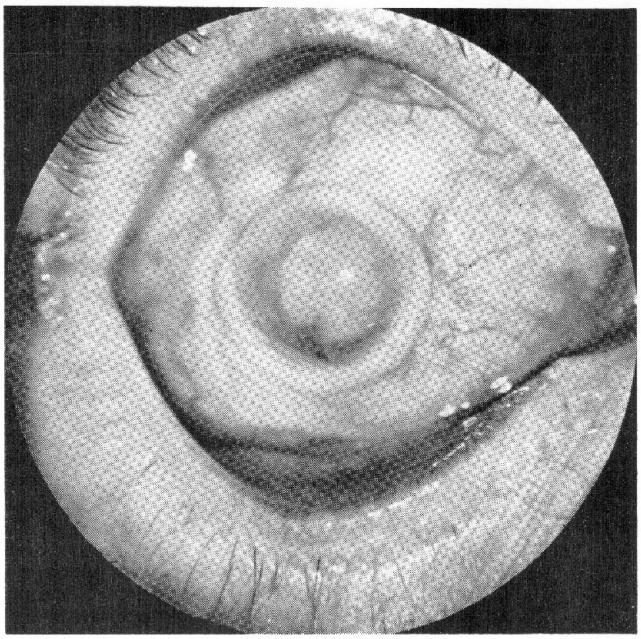

Figure 27. (Awan & Humayun): Same eye as in Figure 26. Vitrectomy failed, and the eye became phthisical immediately postoperatively.

nerve fiber and inner plexiform layers, and into the vitreous cavity from the surface of the retina and from the peripapillary area. (Figures 28, 29, 30 and 31). It is presumed that a hypoxic retina produces a vasoproliferative factor to stimulate the growth of new vessels.[33] The new vessels most commonly grow on or near the optic disc[34] because the presumed retinal vasoproliferative factor is more concentrated over the optic disc; because there is no internal limiting membrane of the retina over the disc; because the fluid vitreous carrying vasoproliferative substance flows posteriorly; and because the radial peripapillary capillary plexus is present. In the extrapapillary areas, neovascularization grows through the breaks in the internal limiting membrane. The new vessels are located anterior to the normal retinal blood vessels and have multiple and irregular branching (in contrast to the normal vessels' normal dichotomous branching). The neovascular tissue is more permeable to fluorescein on angiography. The neovascularization

usually occurs at the posterior edges of areas of nonperfusion and respond involutionally to panretinal photocoagulation. At the earliest stages these new vessels grow as naked channels. Subsequently, fibrosis and gliosis develop around these vessels with resultant scarring and shrinking. The growth of new vessels followed by fibrosis and involution of vessels may continue for years, with eventual disappearance of vessels and formation of large white sheets of fibrous tissue in front of the optic disc and retina known in the past as "retinitis proliferans." (Figure 32). In later stages, these fibrous strands contract and cause extensive traction or rhegmatogenous detachment of the retina by pulling it toward the center of the vitreous cavity (Figure 33).

Spontaneously or from the rupture of fragile new vessels by a tug of vitreous stand, massive vitreous hemorrhages may develop, causing sudden severe loss of sight (Figure 34). These hemorrhages clear up with

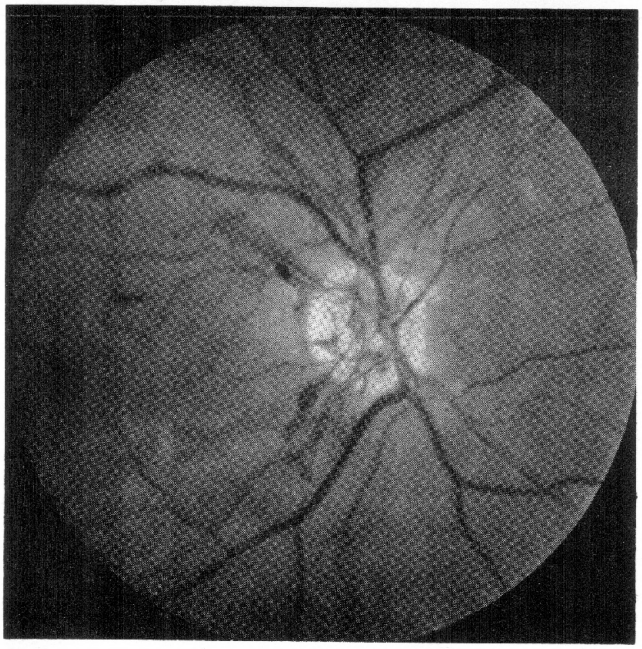

Figure 28. (Awan & Humayun): Proliferative diabetic retinopathy with neovascularization at the disc.

time but their repeated occurrence and further fibrosis lead to blindness in untreated or nonresponsive cases. Although patients with unilateral proliferative retinopathy with bilateral background retinopathy have been described,[35] the disease unfortunately is bilateral and symmetrical in 90% of the patients.[36] In one report, cells with many bundles of contractile protein action were found in the vitreous of a patient with proliferative retinopathy.[37] This may open a new direction for the investigation of the contraction phenomenon. As we just mentioned, it has also been shown that vitreous traction bands may pull on retinal veins, causing the formation of venous loops and ruptures in the internal limiting membrane (Figures 35 and 36).[27] These venous loops may be a source of vitreous hemorrhages. The high risk factors for blindness in eyes with proliferative retinopathy include 1) neovascularization on the optic disc equal to or greater than ¼ of the disc's diameter, 2) any neovascularization on the optic disc with associated hemorrhage, and 3) neovascularization elsewhere equal to or greater than one disc diameter when associated with hemorrhage.[33]

Although 90% of diabetics who have had the disease for 30 years, are expected to develop diabetic retinopathy[7,38] some have no or very little evidence of diabetic retinopathy other than dilation of veins after such duration of disease (Figure 37). (Cogan[7] called this the "venous type" of retinopathy.) We have several such patients. This group has been thoroughly studied to discover their reason for being refractory to retinal disease but without success. In one recent study it was felt that low mean blood glucose concentrations were the explanation.[39]

ACUTE OPTIC DISC EDEMA

An idiopathic swelling of the optic disc has been

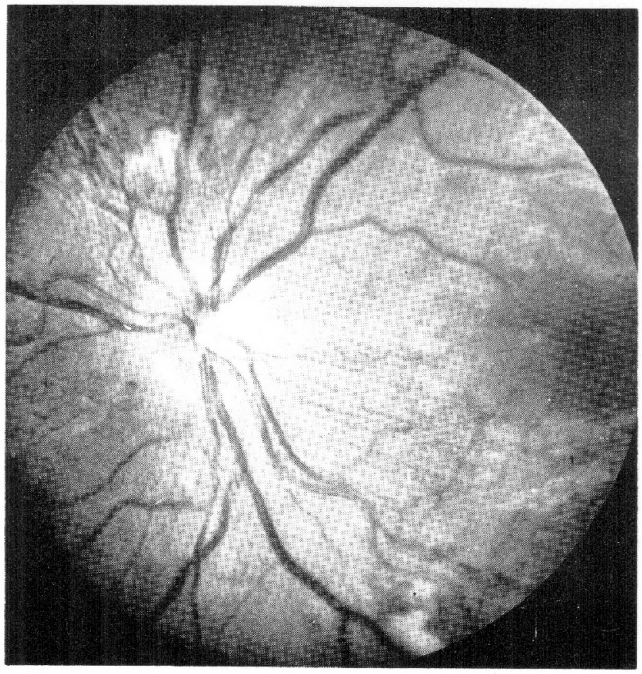

Figure 29. (Awan & Humayun): Proliferative diabetic retinopathy with extensive neovascularization spreading across most of the posterior pole. Such eyes respond poorly to photocoagulation and may need pituitary ablation.

reported as a rare involvement of the optic disc in diabetic retinopathy.[40 - 43] Called acute disc swelling, diabetic papillopathy, papillitis, pseudopapilledema, or anterior ischemic optic neuropathy, its exact cause is not known. The onset is acute and the prognosis, fortunately, is good in a majority of cases. However, it may be an indication of an impending proliferative retinopathy if one is not already present. The symptoms may be blurriness of vision or peripheral visual field defects. The recovery may be complete or followed by some degree of optic atrophy. Resolution may take two months to two years.[43] It seems to be more common in patients with juvenile-onset diabetes mellitus.

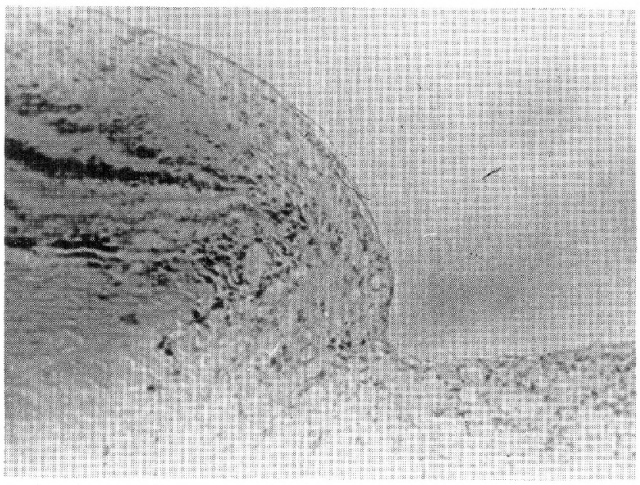

Figure 30. (Awan & Humayun): Histopathologic section of the eye showing fibrovascular growth from the optic disc. (Courtesy of Michael S. Ramsey, M.D.) Hematoxylin-eosin X 40.

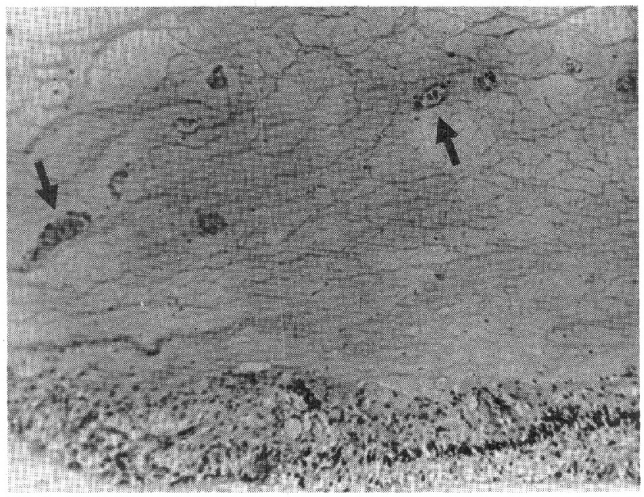

Figure 31. (Awan & Humayun): Neovascular membrane in the vitreous in proliferative diabetic retinopathy. Arrows point to blood vessels in the vitreous. (Courtesy of Michael S. Ramsey, M.D.) Hematoxylin-eosin X180.

Case 3

A 21-year-old man who had had diabetes for seven years suddenly developed "haziness" in his peripheral field of vision of both eyes. An eye examination (by KJA) 3 years before had shown normal fundi. His visual acuity was 20/25 in each eye and the only field defect was enlargement of his blind spots. The typical picture of papilledema was seen in both eyes (Figures 38, 39, and 40). His blood pressure and CT scan were normal. No treatment was given for the papilledema, but he was advised to gain good control of his diabetes. Three months after the first visit, the swelling of the discs had significantly improved without any evidence of optic atrophy.

Comments: It is important that this rare condition be carefully differentiated from actual diabetic neovascularization of the disc and true papilledema.

LIPEMIA RETINALIS AND LIPEMIC DIABETIC RETINOPATHY

In 1888, Heyl[44] first described a condition in which the fundus and its vessels lose their normal color and appear creamy. More commonly the color of vessels is pale creamy pink rather than white. The condition, which does not affect the function of the retina, is called lipemia retinalis. It usually is seen in patients under the age of 40.

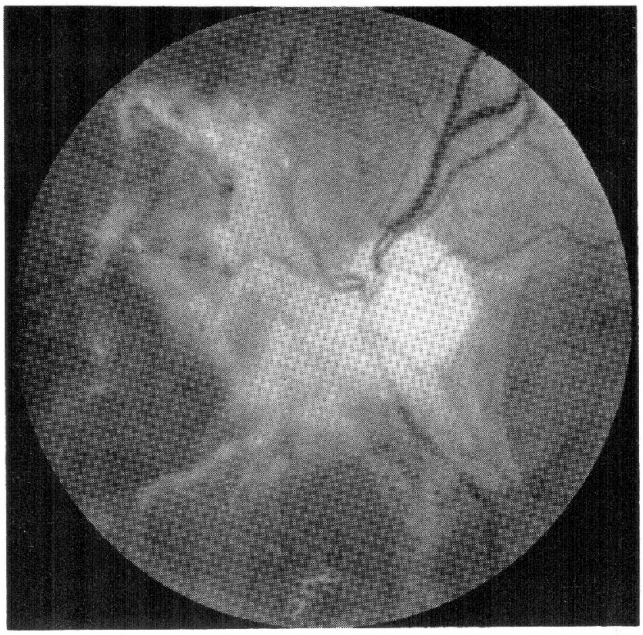

Figure 32. (Awan & Humayun): Retinitis proliferans.

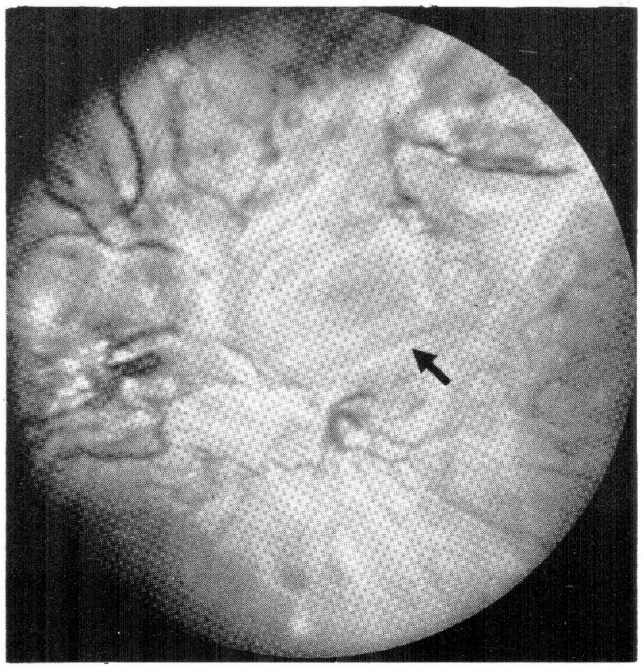

Figure 33. (Awan & Humayun): Same eye as in Figure 32 after repeated vitreous hemorrhages and severe traction of retina. Only a central opening (arrow) is without fibrous tissue in it but the retina behind it was also covered with greyish sheet of glial tissue.

Case 4

A 50-year-old woman complained of bilateral irritation of the eyes. She had been a diabetic for seven years and her blood sugar was normal two months before her visit to us (KJA). Eye examination showed visual acuity of 20/20 (6/6) in each eye with hypermetropic correction of +175s in each eye. Externally and biomicroscopically the eyes were normal. Intraocular pressure was 15 mm Hg in each eye. On ophthalmoscopic examination the fundi appeared pale with creamy pinkish vessels (Figure 41). Her fasting

triglyceride level was 12,600 MG/DL. Other laboratory findings were: cholesterol, 1315; glucose, 286; hemoglobin, 12; hematocrit, 34; WBC, 6,100; plasma, milky; chylomicrons, present; beta, prebeta, and alpha proteins, all increased; and phospholipids, 1,896%. Lipemia retinalis disappeared when triglyceride level was controlled.

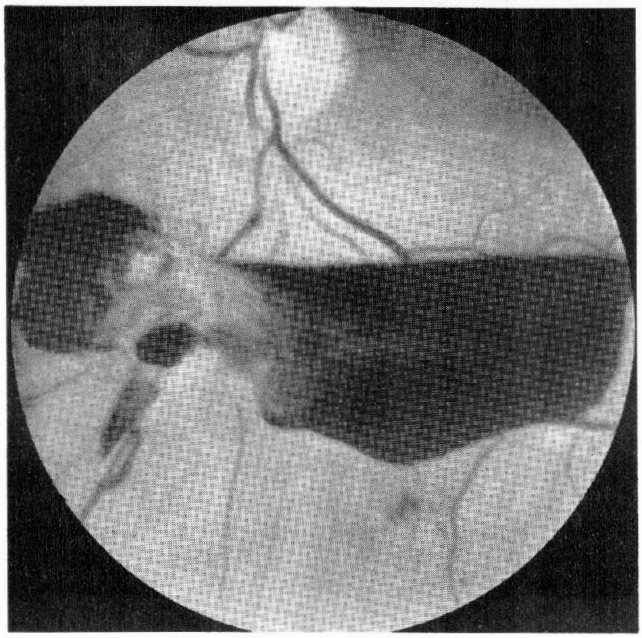

Figure 34. (Awan & Humayun): Vitreous hemorrhage in an eye with proliferative retinopathy.

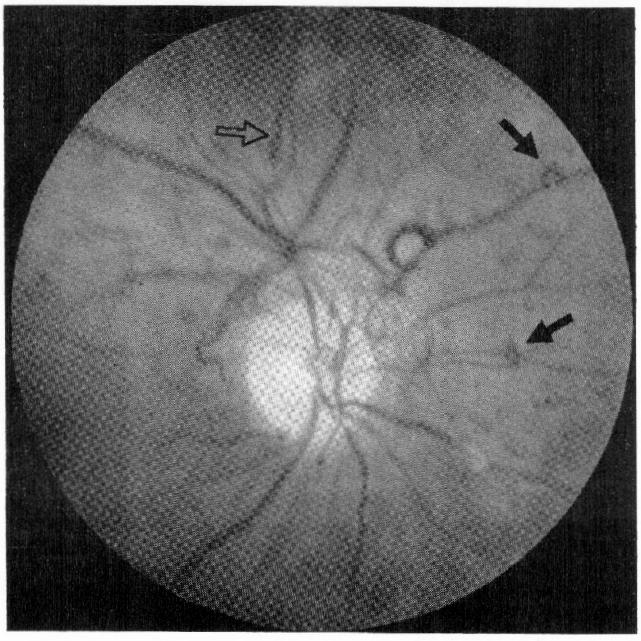

Figure 35. (Awan & Humayun): Tractional loops of the veins (solid arrows), and proliferative retinopathy with area of retinitis proliferans (open arrow).

COMMENTS

Lipemia retinalis is related to plasma triglyceride levels and usually becomes manifest when they are greater than 2,500 mg%.[45] The changes are much more pronounced in the peripheral vessels. It is important to examine the fundus at the same time as the time of drawing of blood specimen. The blood specimen must be taken after a 12- to 16-hour fast to avoid erroneous elevations in the triglyceride levels after meals. In very anemic patients the triglyceride levels below the level of 2,500 mg% may show lipemia of retinal vessels because of decreased number of red blood corpuscles that cannot obscure the creamy plasma. Out of 78 cases reported in the literature, 65 were associated with diabetes mellitus.[46] Lipemia retinalis is a rare and fleeting ophthalmoscopic manifestation. Only 78 cases were reported in 75 years following the first report of Heyl. Today, a better understanding and improved management of diabetes has made it even rarer.

Recently, it was reported that some diabetics with hyperlipemia develop a particular kind of retinopathy with extensive accumulation of hard exudates in the posterior pole with resultant severe loss of acuity in about half of the patients affected with this lipemic diabetic retinopathy.[21]

Lipemia retinalis must not be confused with lipemic diabetic retinopathy. Lipemia retinalis is a benign manifestation for sight and the serum lipids do not escape in pathologic quantities from the vessels; whereas, lipemic diabetic retinopathy is seriously sight threatening and heavy exudation of serum contents leads to extensive hard exudates in the retina.

In a study of recently defined lipemic diabetic retinopathy, no patient had lipemia retinalis.[21] Also the triglyceride levels are much lower in lipemic diabetic retinopathy.

Case 5

A 67-year-old woman came with a seven year history of diabetes and poor sight for two years. Eye examination showed the vision to be counting fingers in the right eye and perception of movements of hand in the left eye. Her vision could not be improved with correction of refractive error of +150 +100X5 in OD and + 100 + 100X170 in OS. A distinct Marcus Gunn pupillary reaction was present in the left eye, but the pupils were otherwise normal. Slit lamp examination, extraocular muscle functions, and intraocular pressure (14 mm Hg in each eye) were normal. Ophthalmoscopic examination after the dilation of pupil showed clear vitreous, but extensive exudative deposits in the posterior poles and nasal to the optic discs (Figure 42). Several areas also showed large intraretinal hemorrhages with shiny hard exudates bordering them. Some of the hard exudates were refractile like the cholesterol crystalline deposits. Whitish sheathing of the blood vessels was also present in many areas some of which appeared like white cords. The optic discs were normal. The fasting triglyceride level was 268 (normal, 180). The diagnosis was lipemic diabetic retinopathy.

Lipemia retinalis must not be confused with lipemic diabetic retinopathy. Lipemia retinalis is a benign manifestation for sight and the serum lipids do not escape in pathologic quantities from the vessels; whereas, lipemic diabetic retinopathy is seriously sight threatening and heavy exudations of serum contents leads to extensive hard exudates in the retina. In a study of recently defined lipemic diabetic retinopathy, no patient had lipemia retinalis.[21] Also the triglyceride levels are much lower in lipemic diabetic retinopathy.

PATHOGENESIS

Tissue hypoxia in diabetes has long been suspected a major factor in the development of diabetic retinopathy. Current concepts of the pathogenesis of diabetic retinopathy suggest that its development and severity depend on the duration of abnormal carbohydrate metabolism and poor metabolic control

of diabetes.[47] Nevertheless, clinical and laboratory findings suggest that the development of diabetic retinopathy is a complex process, thus making it difficult to come to a common understanding about its pathogenesis The imbalance of carbohydrate metabolism characterized by hyperglycemia and low levels of insulin along with the associated imbalance of lipid and protein formation is believed to be responsible for causing structural damage to microvasculature.

The earliest clinical evidence of diabetic retinopathy is venous dilation. Leakage of blood products at this stage cannot be observed with simple ophthalmoscopic examination or even by fluorescein angiography, but newer methods of investigation like vitreous flurophotometry have shown leaking of small molecule fluorescein.[48] Increased retinal venous blood flow and decreased pulsatility has also been demonstrated by another sophisticated technique called laser doppler velocimetry.[49] The chronic increased blood flow with prolonged dilation could well lead to the fatigue of the normal autoregulatory mechanism of the retina. At which stage structural damage within the vessel walls starts becoming apparent. Chronic hyperglycemia appears to be the primary pathogenic agent in diabetic microangiopathy. It has also been shown in vitro that insulin stimulates and hyperglycemia inhibits the proliferation of retinal pericytes,[50] the cells whose selective loss is a hallmark of angiopathy in diabetic retinopathy. It has been also postulated that primary biochemical lesion of diabetic retinopathy may lie in the neuronal or glial cells of the retina, with the retinal vessels only secondarily involved.[50]

The earliest histologic lesion known at present is loss of intramural cells or pericytes of the retinal capillaries.[12, 13, 16] Several factors and mechanisms have been shown to play a role in the pathogenesis of diabetic retinopathy.

SORBITOL OVERLOAD: Normally, the enzyme aldose reductase has a low affinity for glucose, which at normal blood concentrations is metabolized through the pathways of Embden-Meyerhof, the pentose shunt, and Krebs (tricarboxylic acid) cycle. Under the conditions of hyperglycemia, aldose reductase becomes active to convert the excess glucose to sorbitol, a sugar alcohol that is metabolically relatively inert and cannot permeate through the cell membranes with resultant high intracellular accumulation.[50] Intracellular sorbitol increases intracellular osmotic pressure which ultimately leads to the cellular edema. This intracellular edema in turn may impair oxygen diffusion causing further tissue hypoxia. These changes may account for the death of the pericytes and loss of endothelial cell function. Some controversy surrounds the role of sorbitol in diabetic retinopathy. Some authors have found no aldose reductase in the retinal microvessels.[51,52] Whereas others hold different opinion.[50,53] Other questions raised are absence of loss of pericytes or microaneurysms in the cerebral vessels,[54] and why only pericytes are lost when equal sorbitol accumulation also occurs in the endothelial cells.[53] Recently, immunoreactive aldose reductase was

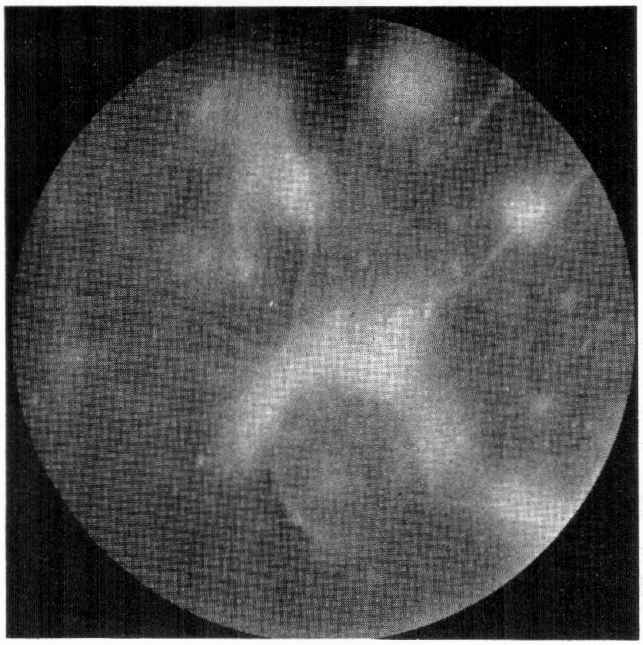

Figure 36. (Awan & Humayun): Same eye as in Figure 35. Intense staining of the vessel wall of the loops and leakage during fluorescein angiography.

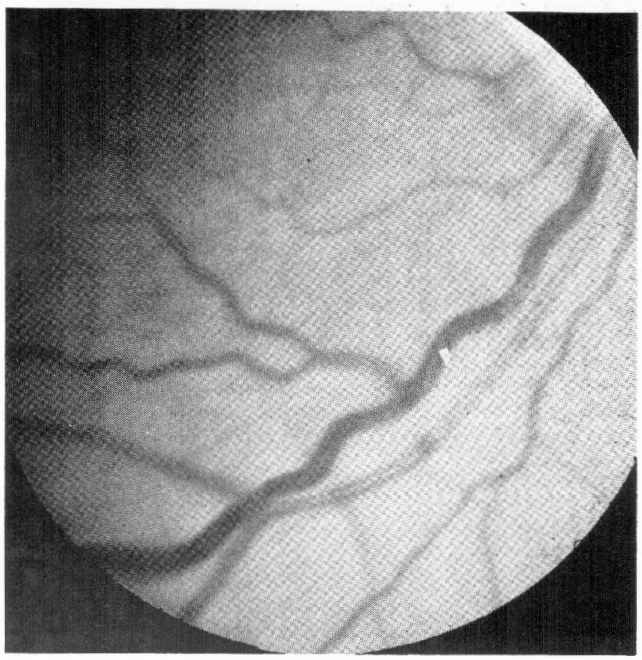

Figure 37. (Awan & Humayun): Left eye of 61-year-old woman with diabetes mellitus of 32-year duration. Other than the engorgement of large veins and a few blot hemorrhages, the eyes and the vision were normal.

demonstrated only in the pericytes, and not in the endothelial cells, of human retinal capillaries.[55] The function of retinal pericytes is unknown. However, it is speculated that they are capable of contractile properties which is similar to the characteristics of pericytes grown in the tissue culture of the blood vessels of the aorta.[56] It seems pericytes support the capillaries and their degeneration may lead to microaneurysm formation.[57]

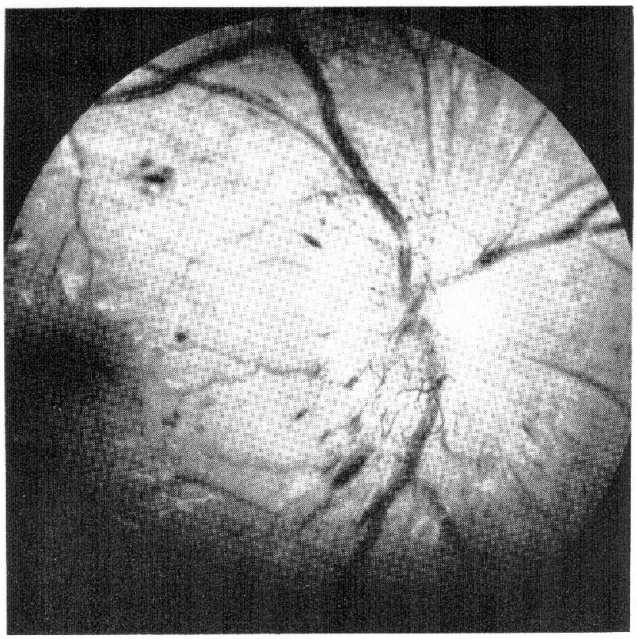

Figure 38. (Awan & Humayun): Case 3. Right eye of a 23-year-old man with diabetes of 7-year duration. Acute swelling of the optic discs.

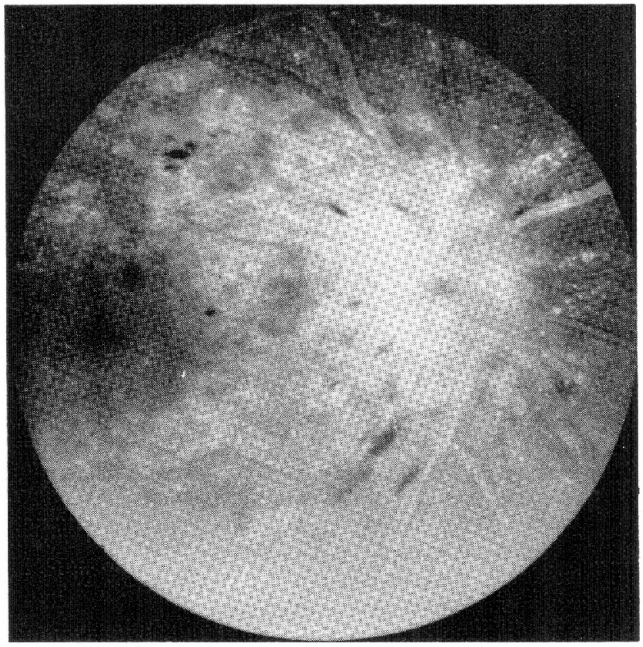

Figure 39. (Awan & Humayun): Case 3. The same eye as n Figure 38. Late venous phase of fluorescein angiography showing filling of dilated capillaries on the optic disc. There is also leakage in the area of disc.

THICKENING OF BASEMENT MEMBRANE: In diabetes, thickening of basement membranes, (Figure 43) vascular and non-vascular, is a generalized phenomenon that has been recorded extensively.[58, 59, 60] Siperstein, Unger, and Madison[61] claimed it to be the basic defect in diabetic microangiopathy. As the exact function of basement membrane is unknown, it is not possible to determine the significance of its thickening in diabetes. Thickening of vascular basement membrane appears to be correlated to the

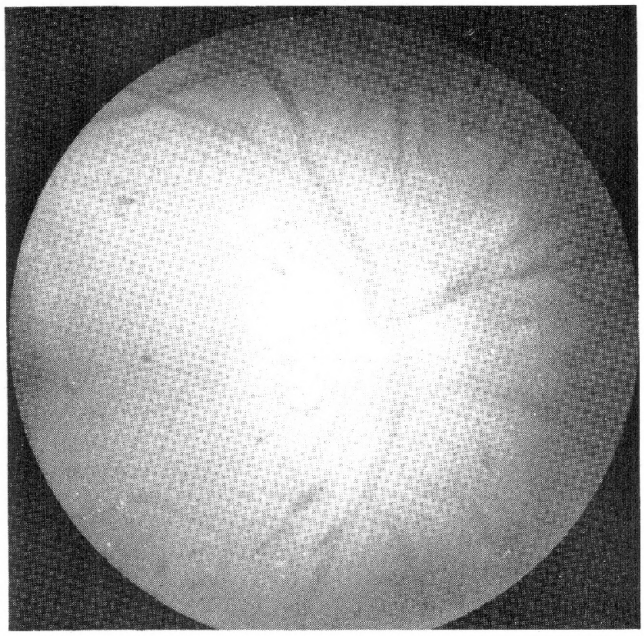

Figure 40. (Awan & Humayun): Case 3. Late phase of fluorescein angiography in eye shown in Figures 39 and 40. Note the retinal vessels with no dye against the stained disc and perpapillary area.

degree of hyperglycemia and the duration of disease.[62] The source of this extracellular material and the mechanism of its formation are not known. However, it has been postulated that it is derived from increased production of basement membrane material from regenerating cells,[63] organization of leaking blood products, and vascular cell debris, etc.[64] Retinal pericytes do not produce extracellular material whereas overgrown endothelial cells have a capacity to form basement membrane material. Thickening of the arterial and precapillary retinal vessels leads to narrowing and gradually occluding the lumin of the vessels. This ultimately leads to slow blood flow further complicating the already hypoxic situation. The finding that an aldose reductase inhibitor prevented thickening of the basement membrane in the retinal vessels of galactosemic rats supports the theory that retinal vessel cell dysfunction can result from elevated levels of glucose and galactose.[65] It is also possible that this abnormal basement membrane prevents proper diffusion and transport of important nutrients from the blood leading to relative tissue ischemia.

HEMATOLOGIC FACTORS: Platelets with an increased tendency to clump and red blood cells with decreased pliability are thought to be characteristics of diabetes.[66] The control of platelet aggregation is influenced by two conflicting prostaglandins: thromboxin A2 (which is synthesized and released by platelets and promotes aggregation) and prostacyclin which is produced by vascular endothelium and is antiaggregatory. Disturbed metabolism of either of these has been suggested as a cause of increased platelet aggregation in diabetics. Linoleic acid may indirectly affect the balance of platelet aggregation and studies have shown the reduced platelet

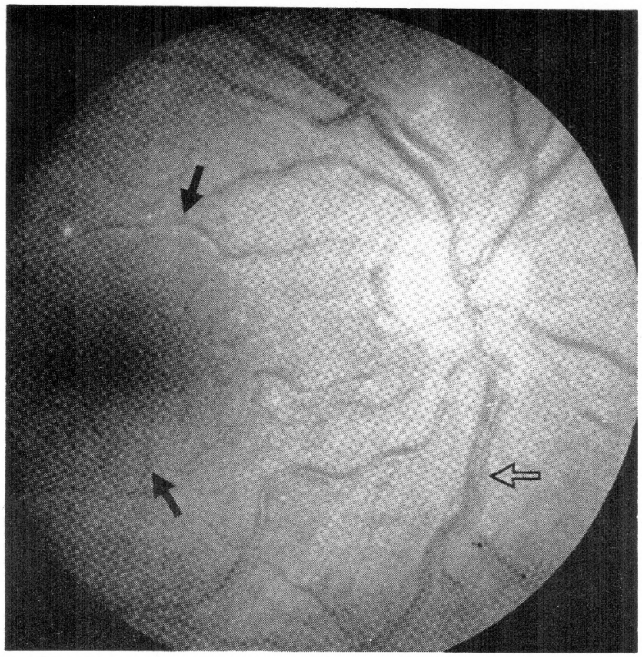

Figure 41. (Awan & Humayun): Case 4. Right eye. Lipemia retinalis. Note the creamy color of smaller vessels around the macula (solid arrows). The larger vessels (open arrow) had lost there normal color and appeared salmon against the faded background of lipemic fundus.

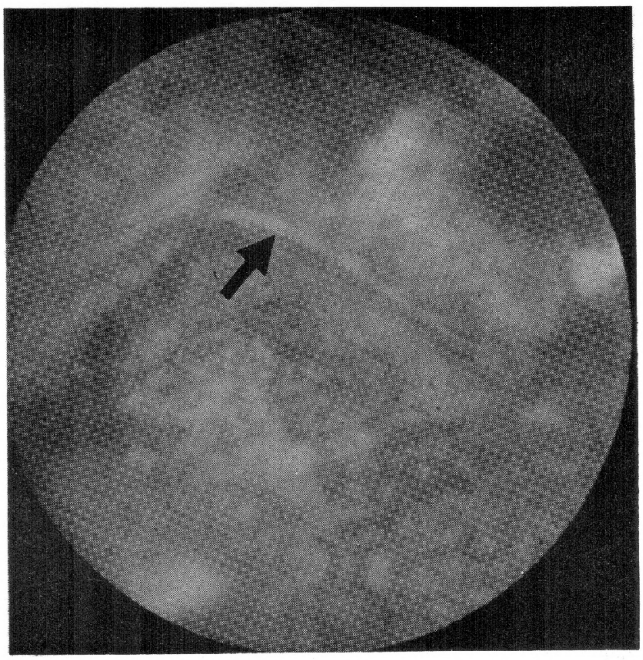

Figure 42. (Awan & Humayun): Case 5. Lipemic diabetic retinopathy. Note the white walled vessel, which was patent on fluorescein angiography. Also note massive and widespread lipoid exudation.

aggregation in the subjects fed linoleic acid supplements and increased platelet aggregation in subjects fed arachadonic acid.[67]

The von Willebrand factor is a plasma protein which has been found necessary for platelet adhesion. This factory is a protein normally having Antihemophilic Factor A activity which can be detected in the blood by hematological techniques. It has been shown that the von Willebrand factor is increased in diabetics particularly those with retinopathy. The high level of this factor may play a role in the increased platelet adhesion causing development of retinopathy. Consistent with this hypothesis is the observation that the von Willebrand factor is selectively confined to the endothelium of the retinal vessels where it can readily interact with blood platelets.[68]

It has been noted that diabetics have a higher concentration of hemoglobin A1c.[69] The normal concentration of this hemoglobin is between 3 to 6 percent of the total hemoglobin level but in diabetics this concentration increases to 10 to 20 percent of the total hemoglobin level. This leads to the greater than normal oxygen binding power in diabetics which impairs oxygen release and diffusion into retinal tissues ultimately leading to tissue hypoxia. In addition, the level of 2-3 diphosphoglycerate, the substance that facilitates the release of oxygen from normal hemoglobin, is decreased so that oxygen release is impaired further complicating the hypoxic situation. At an oxygen pressure (venous) of 40 mm of mercury, blood from a diabetic will give up approximately 30 percent less oxygen than that from a normal person. This is all made much worse by the fact that the diabetic erythrocyte is extremely rigid and so incapable of the easy pliability normally needed for the 7 micron red cell to squeeze through capillaries

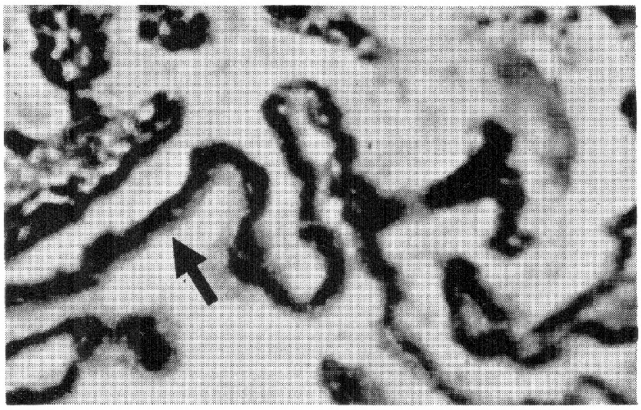

Figure 43. (Awan & Humayun): Basement membrane of the ciliary body is diffusely and markedly thickened (arrow). (Courtesy of Michael S. Ramsey, M.D.) Periodic acid-Schiff and hematoxylin X 160.

which are sometimes 4 to 5 micron in diameter.

HORMONAL FACTORS: The relationship between pituitary gland and diabetes has been well known. In some patients pituitary ablation causes regression of hemorrhagic diabetic retinopathy, reversal of new vessel formation and resorption of retinal edema.[70] These effects have been attributed to the absence of growth hormone. Recently elevated levels of an insulin-like growth factor (somatomedin C) which could mediate the effect of growth hormone was found in Type 1 diabetics who had retinopathy.[71] The increased level of growth hormone in diabetics is proportionate to the degree of hyperglycemia. Synthesis of various proteins like fibrinogen and alpha 2 macroglobulin is enhanced by the liver which in turn is stimulated by the elevated growth hormone levels in the blood. These proteins may decrease the

repulsive forces between red cells and make more likely aggregation of red cells that may not be able to pass through small capillaries. This causes focal occlusions believed to be responsible for nerve fiber infarcts known as soft exudates or cytoid bodies.

Sex hormones appear to play a role as well. Hence, some studies have shown that retinopathy does not develop till about the time of puberty.[72,73] It has also been stated that severe retinopathy develops with greater frequency in younger males,[74,75] and that blindness is more common in males than in females before the age of 45, approximately the time of female menopause.[76] This suggests that female hormones may exert a beneficial influence on diabetic retinopathy. What is the exact role of sex hormones and other hormones in the pathogenesis of diabetic retinopathy is yet unclear.

ANGIOGENIC FACTOR: Under hypoxic conditions the retina produces a vasogenic factor which stimulates microaneurysm formation, shunt development, and neovascular growth. The growth of new vessels near the nonprofused retinal area strongly suggests that the vasogenic factor is derived from the ischemic tissue.[77] The occurrence of iris neovascularization following vitrecotmy supports the concept that the vasogenic factor is diffusable in nature. It is also interesting to note the rarity of retinal neovascularization following occlusion of central retinal artery or the vein.[78] The precise mechanism of retinal neovascularization remains elusive despite extensive research. Panretinal photocoagulation increases oxygen diffusion to the inner retina from the choroid by destroying the high oxygen consuming tissues like photoreceptor cells and retinal pigment epithelium. Increased oxygen concentration within the inner retina may decrease levels of angiogenic factor production by the hypoxic tissue. Laser doppler velocimetry has established that the panretinal photocoagulation reverses hemodynamic changes in advancing diabetic retinopathy by decreasing blood flow and improving pulsatility. Recently, an angiogenic factor was isolated from the omental fat. This lipid factor created dramatic angiogenesis in the rabbit cornea after only a single injection; whereas, no such response was noted in control corneas.[79] Isolation of such substances may open the door to the understanding of the process of angiogenesis. It seems that such exciting developments are not very far in the future.

RELATIONSHIP OF RETINOPATHY AND SYSTEMIC CONTROL OF DIABETES

That a good control of hyperglycemia favorably influences the development of or the course of established diabetic retinopathy remains controversial. Evidence that a high degree of control may delay or slow the progression of diabetic retinopathy is accumulating,[32,39,80,81] and some authors go so far as to say that strict metabolic control may prevent the development of retinal microvascular changes [80] Although reversal of clinically established retinal changes has not been proven, some authors think that the earliest changes of capillary permeability are reversible by a rigid control of carbohydrate metabolism.[32] It has also been stated that the early results of studies of the effects of tight control of blood glucose on the diabetic macular edema are promising.[25] On the other hand, some recent studies have shown that prolonged restoration of near-normal glucose metabolism does not reverse established changes in the retina[3] Whether it slows the progression, or prevents the development, of diabetic retinopathy, remains to be proven.[82] From the information at hand, it appears that good control of diabetes is a preferable course to adopt.

FACTORS INFLUENCING DIABETIC RETINOPATHY

It has long been recognized that the duration of systemic disease directly influences the appearance and the degree of retinal changes. In one study 60% of the patients with insulin-dependent diabetes of 5-10 years had some degree of retinopathy.[2] On the other hand, in another series published during the same year only 12% of patients in a rural diabetic population showed diabetic retinopathy during the first ten years.[8] Age seems to confer a certain degree of immunity against retinopathy; hence, it is rare in children under 10 and in adults under age 30 with juvenile-onset diabetes.[8, 83] One study concluded that the frequency and severity of retinopathy is considerably influenced by genetic factors in non-insulin-dependent diabetics, but this is not sure in insulin-dependent diabetes.[84] The type of diabetes may also determine the type of retinopathy; hence, proliferative retinopathy is more often associated with insulin-dependent diabetes, and macular edema with non-insulin-dependent diabetes. Clinical and experimental studies have given rise to hypothesis of immunogenic vasculitis as a factor in the retinal microangiopathy.[8,85] Immune reactions from the appearance of high titers of antibodies against insulin may occur in diabetics receiving insulin therapy, causing focal vasculitis in the retina. This hypothesis may explain why the incidence of diabetic retinopathy is higher in persons receiving insulin than in those receiving oral hypoglycemic agents.[8] The severity of retinopathy has also been linked to high levels of glycosylated hemoglobin, to proteinuria, to higher diastolic blood pressure, and to being male.[83] However, no relationship between hypertension and diabetic retinopathy was seen in diabetes of less than 10-year duration.[83] It has been said that systemic hypertension or ocular hypotension will have an adverse effect whereas systemic hypotension or ocular hypertension will have a good effect on diabetic retinopathy.[7] However, a recent study concluded that elevated intraocular pressure did not prevent the development of retinopathy.[35]

MANAGEMENT

"The prognosis is very unfavorable. The treatment must be entirely directed to the diabetes." Wrote Juler[4] regarding the treatment of diabetic retinopathy a century ago, and denounced the usual methods of treatment of his time that included local "bloodletting, by leeches or other means, blisters, scarification, etc."

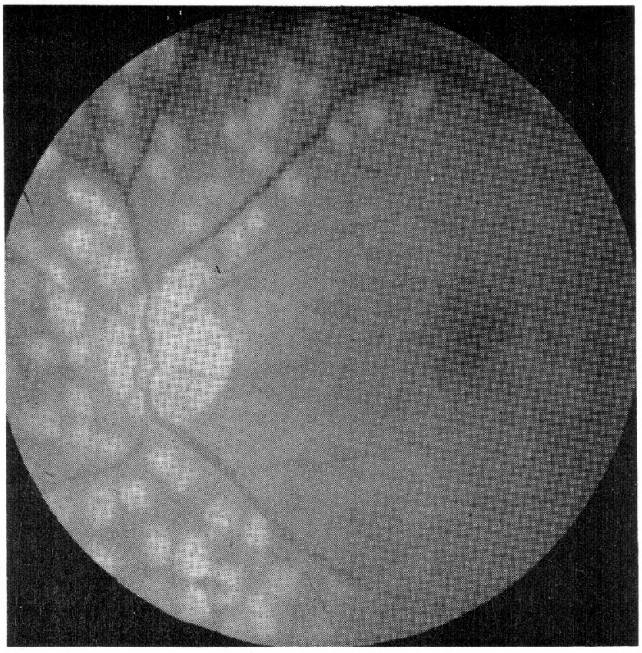

Figure 44. (Awan & Humayun): Panretinal photocoagulation.

In modern times, his advice that treatment should be directed, but not "entirely," to systemic control is gaining popularity, and evidence in favor of its usefulness is accumulating. As far the "blisters;" the modern technology has made it possible for today's oculist to create them in the eye rather than away from it, and the last few years have witnessed the effectiveness of panretinal photocoagulation in the prevention of blindness from proliferative diabetic retinopathy. The overall prognosis of diabetic retinopathy, however, still leaves much to be desired, if it is not "very unfavorable." The effects of controlling diabetes with insulin or hypoglycemic agents has already been discussed. It has been observed that diabetics who also have arthritis and are taking aspirin have less diabetic retinopathy.[89] It is believed that aspirin helps restore to normal the abnormal aggregation of platelets found in diabetics.[90] This effect of aspirin may be achieved by small doses of 5 gr daily. How effectively aldose reductase inhibitors decrease the transformation of glucose into sorbitol, which damages the endothelial cells of vessels, is being studied. Reduction of serum lipids and lipoproteins have been tried without any convincing usefulness in the treatment of diabetic macular edema. However, control of hypertension and improved cardiac or renal status appear to favorably affect macular edema.[25] Capillary permeability decreasing agents, such as calcium debosilate, have proved ineffective in controlled studies.[91]

PHOTOCOAGULATION: The observation that eyes with chorioretinal scars, secondary high myopia, and previous vascular occlusion causing retinal and optic atrophy often developed no or very little diabetic retinopathy led to the idea of artificially destroying portions of the peripheral retina as a treatment of diabetic retinopathy. Photocoagulation (Figure 44) and cryotherapy are employed to achieve this aim. It is speculated that by reducing the number of viable retinal cells, production of vasoproliferative factor in amounts sufficient to stimulate neovascularization is diminished.[33] Although photocoagulation was first employed in the treatment of diabetic retinopathy about a quarter of a century ago by Meye micron Schwikerath,[92] Spalter[93] was the first to show its success in treating circinate retinopathy. In 1976, Diabetic Retinopathy Study conclusively showed that photocoagulation is beneficial in the treatment of proliferative diabetic retinopathy.[87] Now its usefulness in treating the macular edema of background retinopathy has also been proved.[25] The success of photocoagulation in the treatment of diabetic retinopathy is thought to be due to its ability to destroy neovascular complexes, leaking vessels around the macula, and areas of microinfarctions and capillary closure. It is also possible that chorioretinal scars created by laser treatment help retina resist the deleterious effects of vitreoretinal traction.[94] These scars also bring retinal elements in closer aposition to the choriocapillaris, thereby allowing a greater perfusion of oxygen from choroid to retina.

There are several contraindications to the use of panretinal photocoagulation in the treatment of proliferative diabetic retinopathy. They include extensive glial proliferation in the posterior pole, large vitreoretinal tractions, non-perfusion of 60% or more of paramacular area, extensive surface neovascularization on most of the posterior pole, grossly edematous retina, additional advanced renal retinopathy, and severe combined diabetic and hypertensive retinal changes.[94]

Panretinal photocoagulation seriously affects the peripheral and night vision. Other complications include: loss of one to four lines in acuity, vitreous hemorrhages, tractional retinal detachment, retinal striae, preretinal membrne formation, decreased foveal perfusion in eyes with preoperative severe ischemia of the posterior pole, and persistent post-laser macular edema causing loss of vision.[95]

Photocoagulation Technique: There are several approaches to panretinal photocoagulation. In one technique,[91] the full scatter treatment involves the application of 1,200 to 1,600 lesions of 500 micron at 0.1 second exposure, and an intensity barely sufficient to cause moderately intense white burns and one-half burn diameter apart. In mild-scatter treatment the number of spots is reduced to 400 to 600 spots that are at least one burn diameter apart. The treatment is applied outside of a 2-disc-diameter circle centered at the fovea. Neovascularization, excepting that on the optic disc, are focally treated to obliterate the vessels. Study in the usefulness of photocoagulation for treating diabetic macular edema is still going on.[25] However, three treatable lesions have been identified: focal leaks more than 300 micron from the center of the macula, avascular zones, and areas of diffuse leakage. Light intensity sufficient to create grey lesions of 100 micron, to 200 micron, is applied. If no improvement occurs, the leaks may be closed with 50 micron to 100 micron burns using a 0.1 setting.

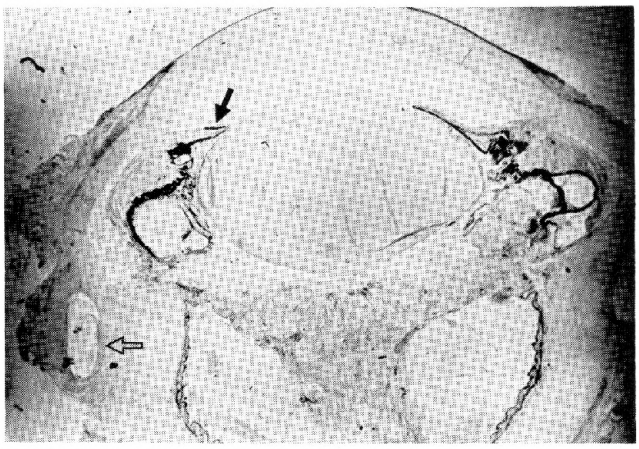

Figure 45. (Awan & Humayun): Histopathologic section of an eye with end stage proliferative diabetic retinopathy and hemorrhagic glaucoma. Note the ectropion uveae (solid arrow). Eye had undergone retinal detachment surgery and the encircling element is visible on both sides of the globe (open arrows). (Courtesy of Michael S. Ramsey, M.D.) Hematoxylin-eosin X6.

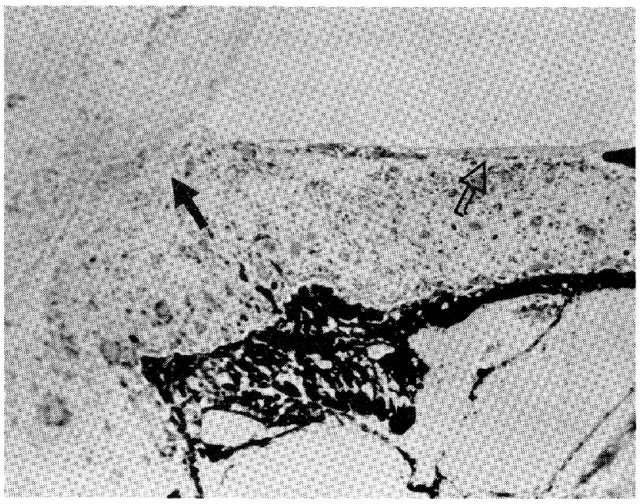

Figure 46. (Awan & Humayun): Hemorrhagic glaucoma. Note the vascular membrane on the anterior surface of iris (open arrow) and peripheral synechia closing the angle (solid arrow). (Courtesy of Michael S. Ramsey, M.D.) Hematoxylin-eosin X75.

Avascular zones and diffuse macular edema are treated with a grid pattern of 100 micron to 200 micron spots separated by one burn diameter. Treatment is started at 500 micron away from the center of the macula, followed by treatment up to 300 micron away from the center of the macula when the visual acuity is less and 20/40 (6/12). The evaluation, and treatment if indicated, of macular edema patients is carried out twice or thrice a year. Currently, argon lasers producing blue (488 nm) light and green (514 nm) light are considered most effective. This light can be absorbed by the melanin of the pigment epithelium and choroid, by retinal xanthophyll, and by hemoglobin. Krypton red laser (647 nm) light and ruby laser (694 nm) light are absorbed by melanin but not by hemoglobin and retinal xanthophyll. They produce deeper burns and, hence, may be of slight advantage in treating the central areas. the Xenon laser has a white light and has been found to have more side effects. Photocoagulation is usually applied in two sessions two weeks apart, or in three or more sessions four days apart. In certain circumstances, the whole treatment may be completed in one sitting, but doing this increases the risk of morbidity.

VITRECTOMY: Removal of vitreous bands causing traction retinal detachment and the vitreous hemorrhage that fails to absorb has been justified by the visual success rate in selected patients.[96 - 98] This is in spite of the high incidence of complications of vitrectomy. The indications for vitrectomy include (1) vitreous hemorrhage that fails to absorb in 6 months; (2) traction detachment with a recent involvement of macula; (3) rhegmatogenous retinal detachment in which holes cannot be closed by buckling alone; (4) fibrovascular membranes that affect the vision by covering the posterior retina; (5) a need to clear media for photocoagulation in eyes with early rubeosis iridis; and (6) a reasonable chance of recovering the vision.[99] The usual complications are (1) iatrogenic retinal holes, (2) repeated hemorrhages in the vitreous, (3) no improvement of vision in about one third of the patients, and (4) development of rubeosis with subsequent hemorrhagic or thrombotic glaucoma (Figure 45 and 46), and (5) persistent corneal edema. Rubeosis iridis is the commonest complication that leads to failure of a technically successful vitrectomy. Michels,[99] Aaberg,[88] and Peyman, Humamonte, Goldberg, et al.[100] report a 10% incidence of hemorrhagic glaucoma in eyes that develop rubeosis iridis after vitrectomy, a complication seen in 35- to 45% of operated cases. The risk of hemorrhagic glaucoma increases when lens is removed, or when the patient has rhegmatogenous retinal detachment.[88] Retinal tears develop anywhere between 2.5% and 22% of the cases, depending on the type of technique, selection of patients, and approach of surgeon.[99, 100] Eyes with posterior vitreous detachment have better results from vitrectomy. In some instances the vitrectomy has to be combined with removal of the lens (lensectomy) either due to prior opacification or due to injury during vitrectomy. Vitrectomy not only removes the opaque media also eliminates the external traction on the retinal vessels which contributes to the vessel wall instability and endothelial cell proliferation.[101] Studies are underway to determine the optimal time for vitrectomy and to further improve the techniques. Hypophysectomy has been almost abandoned because of the serious complications that accompany it. However, it may be the only means available to prevent blindness in patients with florid surface neovascularization covering most of the posterior pole.[94]

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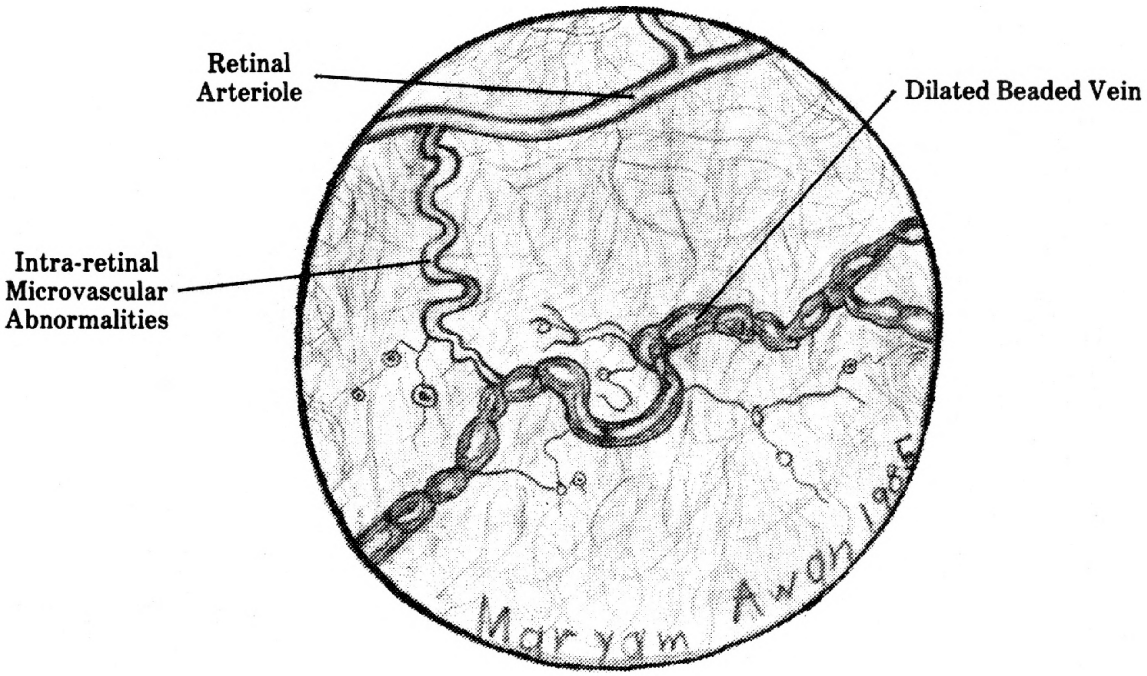

Intraretinal Microvascular Abnormalities in Diabetes

(A pencil sketch by Maryam T. Awan, age 9 years.)

Original Article

Secondary Intraocular Lens Implantation

I. An Analysis of 25 Cases

Abdul Khaliq, M.D.*

ABSTRACT: A retrospective study of 25 cases of secondary intraocular lens implantation showed that over 90% of patients achieved a postoperative visual acuity equal to or better than the preoperative level. One patient had a hemorrhage that extended into the vitreous from a lens haptic eroding into the iridociliary structures. The followup period ranged from 2 months to 5 years. Secondary pseudophakia is a reasonable alternative for patients who are unhappy with aphakic spectacles and cannot wear contact lenses. (Pak. J. Ophthalmol 1: 91-92, 1985).

Secondary intraocular lens implantation has remained embroiled in controvesy in the United States and some maintain the incidence of cystoid macular edema is higher following this procedure than after primary pseudophakia.[1] In Pakistan, most cataract surgery consists of intracapsular extraction without intraocular lens implantation. Obviously, the popularity enjoyed by pseudophakia in the western hemisphere is bound to reach Pakistan, where many of the patients will need secondary intraocular lens implantation. The purpose of this paper is to present our experience with this procedure.

MATERIAL AND METHODS

Medical records of 25 consecutive patients who had secondary intraocular implantation from 1979 to 1984 were reviewed. All of these patients, ranging in age from 30 to 74, had been unhappy with aphakic vision in the operated eyes, and could not wear contact lenses. In some of the patients the acuity was satisfactory with contact lens for a few years but eventually contact lens intolerance developed. In several others, contact lens care and protein deposits became a difficult problem. Out of these 25 patients, 10 had one of the pupillary lens implants and the remaining 15 had anterior chamber implants. The surgical technique included a 2 mm conjunctival flap followed by a 7 mm limbal incision to enter the anterior chamber. A small amount of air or Healon was injected to replace the aqueous that was lost at the time of entry into the anterior chamber. Healon or air was injected to prevent the escape of vitreous and to keep its face pushed back. In two patients vitrectomy had to be performed to clear the anterior chamber of vitreous. After the satisfactory insertion of the pseudophakos, done in 3 to 9 o'clock axis, the incision was closed with interrupted 10-0 nylon sutures. The Healon was aspirated from the anterior chamber and then it was formed with saline. The postoperative care included topical steroids-antibiotic combination drops and, in some instances, weaker cycloplegics. The patients were checked postoperatively at one week, six weeks, six months, and one year intervals.

RESULTS

Interestingly, no patient had cystoid macular edema that could appreciably affect the vision. A few patients who showed an excessive postoperative iritis responded well to local treatment. This reaction was slightly more in patients who had pupillary lenses. Up to the time of this report, no signs of retinal detachment have appeared in any of the patients. One patient had a bipolar tuck of the iris with horizontal elongation of the pupil, but this did not affect the visual outcome. In one patient a hemorrhage from the ciliary body due to an eroding lens haptic settled in the vitreous resulting in significant loss of sight.

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COMMENTS

Many of our patients were successfully wearing extended wear soft contact lenses just prior to undergoing secondary intraocular lens implantation. After several years, these patients developed discomfort, intolerance, and poor vision despite repeated changes in lenses. Hence, the extended wear contact lenses are not an all encompassing answer to problems of aphakia. A surgeon and a patient who are going to undertake secondary intraocular lens implant must weigh several aspects of this procedure. Lindstrom and Harris[2] reported 6% incidence of retinal detachment after this procedure. Athough none of our patients had this complication, the risk is real and higher than with the primary procedures. Similarly, in a patient who had good vision prior to secondary surgery the loss of vision due to cystoid macular edema cannot be ignored.

One interesting observation we made was that one patient who had vision of 20/30 and exotropia with a contact lens preoperatively became straight after secondary lens implantation. We are inclined to believe that this improvement in his exotropia was due to better quality of vision and reduced prismatic changes from intraocular lens implant. Another patient complained of multiple images before the surgery. His complaint disappeared after a secondary intraocular lens implantation. We speculate that this was also due to improved quality of image. An overwhelming majority of our patients was very happy with secondary lens implantation even if the visual acuity was no better than the preoperative vision. We think this is because of enhancement of visual perception that the patient can feel but the surgeon cannot measure.

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Ophthalmic "Past-Pourri"

CREDIT TO CREDE'

INTRODUCTION: Crede "recommends putting the children into the bath immediately after ligating the umbilical cord, carefully cleansing the eyes with a linen rag, or, better still, prepared cotton, and then instilling a two-percent solution of nitrate of silver with glass rod."

An 1884 abstract of THE PREVENTION OF BLENNORRHOES NEONATORUM, THE MOST FREQUENT AND MOST IMPORTANT CAUSE OF BLINDNESS by Crede
(1-14-337)

APPLICATION: "9-22.4 percent of all the new-born were affected with blennorrhoea at the Greifswald gynaecological clinic from 1870-1880; since the introduction of Crede's method in 1881-1882, only 1.7-9 percent."

ON BLENNORREHOEA NEONATORUM
By Beumer and Peiper
Arch f. Gyneakol. 23:479, 1884
(1-14-337)

PERFORATING EYE INJURY: MANAGEMENT A CENTURY AGO

"Col. J.H.H., aet. fifty, married, was brought to my office by my friend, Dr. John S. Kirkendall, of Ithaca, for consultation in regard to his eyes. (In 1878)... One dark night he reached up to a closet shelf for a goblet and knocked another down. It broke and a piece of the glass flew and struck his *left* eye. It cut through the upper lid and wounded the globe at the sclero-corneal margin. He was treated at an institution on Elmira where the eye was bandaged and he was kept in a dark room for *eleven weeks.* *He grew gray in those eleven weeks."

David Webster, M.D.
New York
June 29, 1892
*Italics in this sentence by the Editor

Secondary Intraocular Lens Implantation

II. A Discussion of Merits and Demerits of Use in Pakistan

Khalid J. Awan, M.D.*

ABSTRACT: Author discusses pros and cons of Secondary Intraocular lens inplantation with reference to its use in Pakistan. He illustrates his points of view with examples from personal experience. (Pak. J. Ophthalmol. 1: 92-95, 1985)

The impressive results of secondary pseudophakia in many reports from the United States, including Khaliq's report, were achieved by performing surgery under highly aseptic conditions, in fully equipped operating rooms, and by highly skilled and experienced intraocular lens implantation surgeons.[1-6] This aspect of the favorable reports on secondary intraocular lens implantation must be fully emphasized to any Pakistani surgeon who wishes to embark upon this very critical procedure, both for the surgeon and for the patient. A poorly executed secondary operation under less than optimum conditions could rob the patient of even what vision he had before the surgery. Good results like those mentioned above did not occur at the outset. In fact, in a survey of secondary lens implantation in the United States in 1977, some implant surgeons strongly opposed this procedure on the basis of their own statistics.[7] Pakistani surgeons must approach this procedure with greatest of caution, keeping in mind that performing this type of surgery in less well equipped facilities might prove even more discouraging than the earlier experiences of surgeons in the United States.

Other conclusions of this survey were: 1. If a patient is doing satisfactorily with contact lenses, do not do a secondary implant. 2. Many implant surgeons would not do secondary implant surgery in an eye with vitreous in the anterior chamber. 3. It is safer to do a secondary implant in an eye that has intact posterior capsule than in an eye that has undergone intracapsular surgery. 4. Anterior chamber lenses are most suitable for secondary implantation. 5. There is an extremely wide difference of opinion among implant surgeons concerning indications, techniques, type of lenses, and the kind of complications. Although these conclusions have become less relevant today for American surgeons,

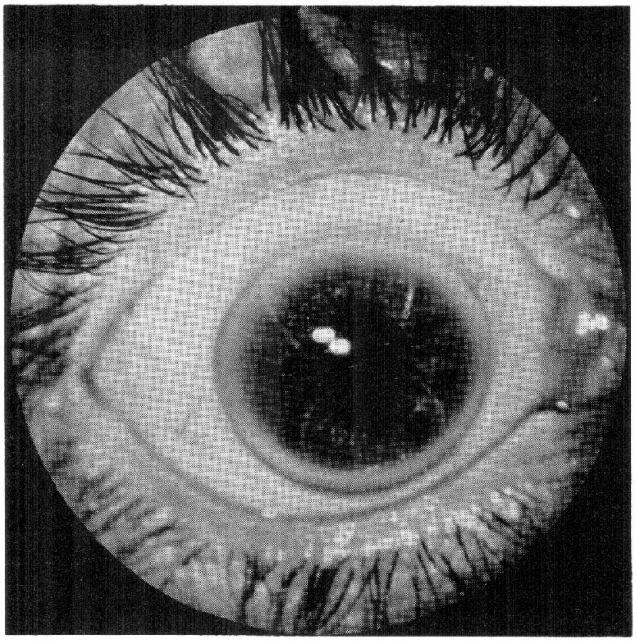

Figure 1. (Awan) Secondary anterior chamber intraocular lens implant with flexible loops. Visual acuity is 20/20 after 2 years.

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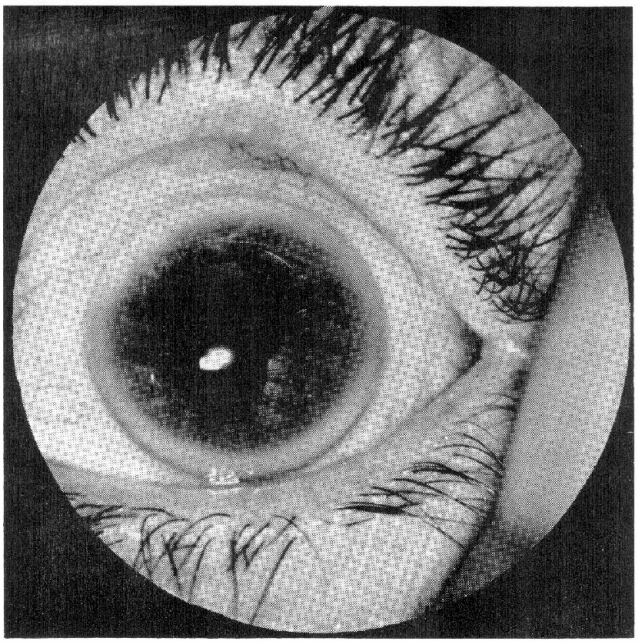

Figure 2. (Awan): Secondary intraocular lens implant in the other eye of patient in Figure 1. Vitrectomy was done at the time of operation and rigid anterior chamber lens inserted. One and a half years later the visual acuity is 20/20.

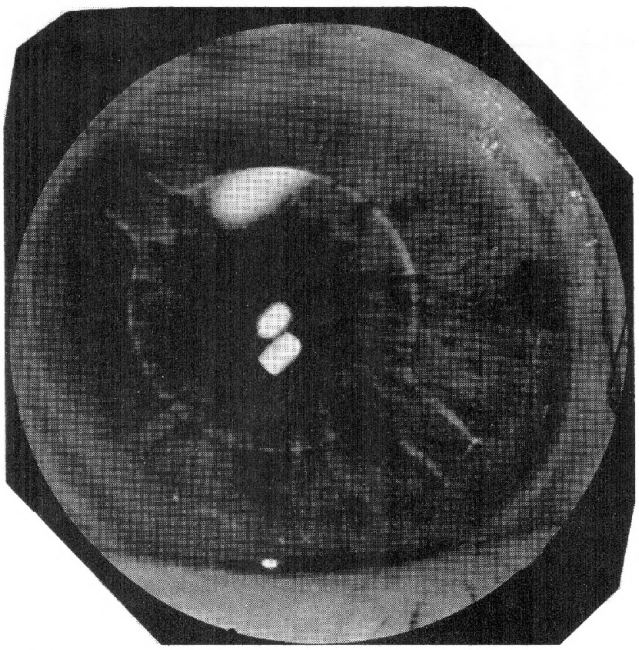

Figure 3. (Awan): The upper anchorlike haptic of anterior chamber lens has slipped thru the peripheral iridectomy and is rubbing on the ciliary body causing a recurrent microhemorrhage in the anterior vitreous with episodes of blurriness of vision. (It is possible that the haptic was intentionally pushed thru the iridectomy by the surgeon himself to fixate the smaller size implant.)

they might be a good place to start for a surgeon in Pakistan.

Secondary intraocular implant surgery is less complicated and less time consuming than the primary procedure, and the results are excellent in the hands of skillful surgeons in the carefully selected patients. Choyce[3] considers his Mark VIII anterior chamber implant a very suitable pseudophakos for secondary implant surgery. Khaliq used pupillary supported lenses in 40% of his cases. I personally am not in favor of pupillary lenses for secondary implantation except in rare instances. This type of lens has been almost entirely abandoned in favor of posterior chamber or anterior chamber lenses.[4] The pupillary lenses are associated with too high an incidence of cystoid macular edema and corneal decompensation to make them suitable for secondary implantation.[4,8] Some authors strongly recommend that when secondary intraocular lens implantation is considered, a rigid anterior chamber lens should be given preference.[4] I have found that flexible lenses give equally good results. I studied this aspect of lenses by implanting a rigid anterior chamber lens in one eye and a flexible lens in the other eye in several patients undergoing secondary implantation (Figures 1 and 2). No difference was noted in vision or in comfort between the two eyes by these patients.

It is felt that disturbance of vitreous, such as by vitrectomy or vitreous loss, leads to higher incidence of cystoid macular edema and retinal detachment.[1] In one report with an incidence of 6% retinal detachment, vitrectomy had been performed during operation in 15% of cases.[2] On the other hand no retinal detachment was seen postoperatively in another series.[4] However, it was not mentioned in this report whether vitrectomy was performed or not in

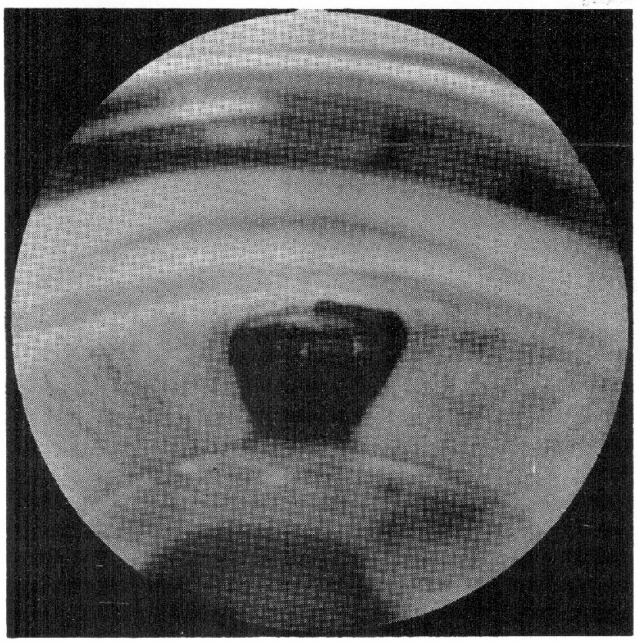

Figure 4. (Awan): Gonioscopic view of Figure 3.

patients during the operation. In another series of 190 cases, 3 developed retinal detachment. This was not considered a direct result of lens implantation.[6] Khaliq and I have not seen any retinal detachment in our secondary implantations, but our number of patients is not large enough to give statistically significant results. Retinal detachment is, nonetheless, a risk to be carefully considered in patients before secondary implantation. Some authors advise a preoperative full indirect ophthalmoscopic examination with scleral

indentation before this procedure.[2]

Cystoid macular edema following secondary intraocular lens implant deserves very serious consideration. Its higher incidence after secondary implantation has made the primary procedure more popular with some surgeons.[1] In patients with preoperative visual acuity of less than 20/40 more than six months following the initial surgery, fluorescein angiography should be performed to detect any persistent cystoid macular edema.[1,2,5] Usually, if vision remains satisfactory one year after the initial surgery and there is no operative complication, serious cystoid macular edema does not develop. However, there is no certainty that cystoid macular edema will not be a major postoperative problem after secondary implantation. Performance of secondary implant within 6 months after the first procedure increases the incidence of macular edema.[5] The cystoid macular edema may occur in 3% to 25% of the cases after secondary lens implant surgery.[1,2,5] Some surgeons advocate the use of steroids, antiprostaglandins, and indomethacin preoperatively and aspirin postoperatively to deminish the incidence or the degree of cystoid macular edema.[2,4] It is interesting that out of two patients of Khaliq and four patients of mine who had vitrectomy during the secondary implant procedure, none developed persistent cystoid macular edema. One of my patients showed cystoid swelling of his macula that disappeared in three months.

Corneal edema is fortunately not as common as cystoid macular edema. It occurred in 4% of patients in one report.[1] In one patient it was successfully treated with hypertonic sodium chloride q.i.d. It developed in one of my patients and it was treated with penetrating keratoplasty. Corneal edema is much more common in pupillary lenses but does occur with anterior chamber lenses, particularly with the flexible kind.[1,4,5,8]

The complications of glaucoma,[1] hyphema,[1,2] subchoroidal hemorrhage,[1] pupil capture,[2] vitreous hemorrhage,[1,5] posterior synechia,[4] and ischemic optic neuropathy,[6] have been reported. The footplate of the anterior chamber implant in some cases, primary lens implants in particular, may dig into the corneoscleral wound necessitating reoperation. I have seen a patient who consulted me after having been operated on somewhere else. He was having episodes of blurriness. On examination, the anterior chamber lens appeared tilted with one anchor type foot having slid behind the iris thru one of the two iridectomies (Figures 3&4). The other foot was in the anterior chamber but was tilted up toward the cornea (Figure 5). The foot behind the iris was rubbing against the ciliary body, resulting in a severe reaction in the anterior chamber and streaklike hemorrhages in the anterior vitreous (Figure 6).

Postoperative astigmatism must also be kept in mind. Induced prism and aniseikonia may lead to functionally bad results even in a perfectly executed and noncomplicated secondary implant procedure. The differences in the spectacle prescription of two eyes may make the reading very difficult for the patient.[9] It is calculated that an error of 0.1 mm in the axial length of an eye can result in a change of 0.33 diopter in postoperative refraction.[10] It is to be remembered that a corrected tissue velocity (1532 m/sec) is required for ultrasonography in the aphakic eyes.[11] In one of my patients with primary pseudophakia in one eye, diplopia resulted from a tilt in the pupillary supported lens implant. It required repositioning of the implant to treat this diplopia.

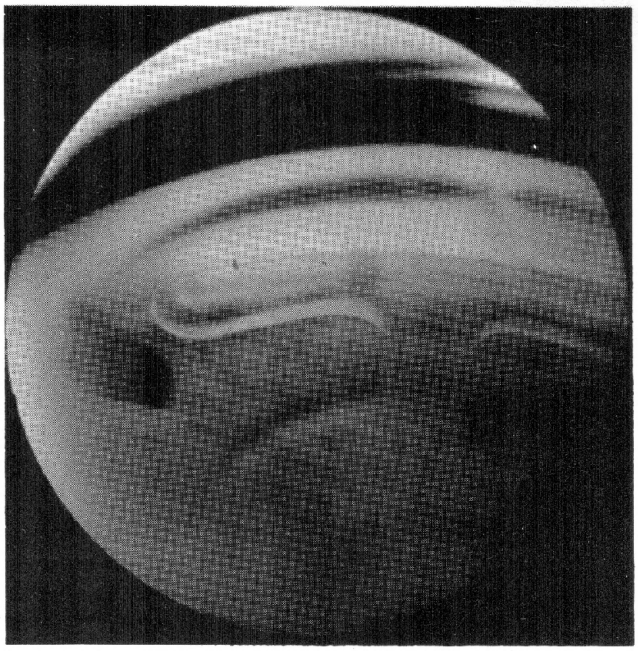

Figure 5. (Awan): The other haptic of the anterior chamber lens implant shown in Figures 3 and 4 is tilted anteriorly and is rubbing on the endothelial surface of the cornea.

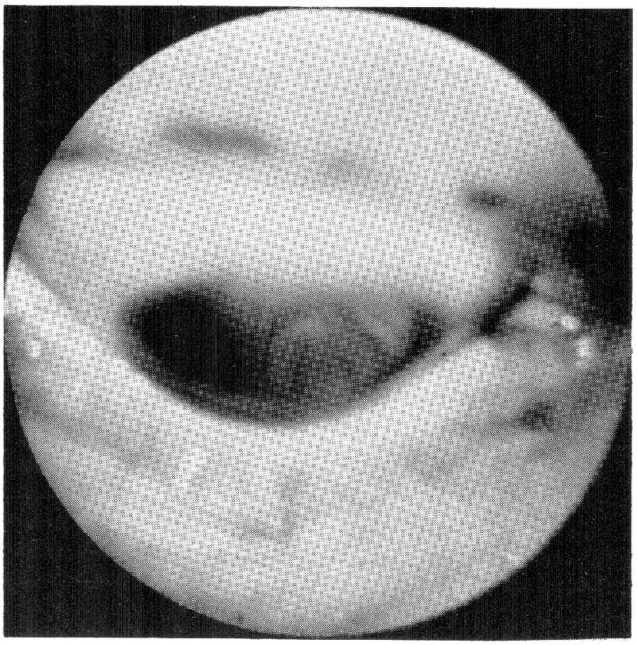

Figure 6. (Awan): Streaks of blood in the anterior vitreous from the recurrent microhemorrhages from irritation of ciliary body by implant haptic.

Hence, the power of the lens implant must be calculated very carefully.

Finally, the secondary implant is an excellent alternative to aphakic vision with glasses or contact lenses, and only in 0.5% of the patients can the visual decrease be directly attributed to the secondary implant procedure.[6] This is only when the surgeon is meticulously careful in the evaluation of the patient and when the surgery is executed skillfully under optimum conditions.

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Ophthalmic "Past-Pourri"

WHAT YOU WOULD WISH YOU HAD NEVER KNOWN ABOUT SEX

"A GROUP OF FOUR CASES OF OPTIC ATROPHY DUE TO SEXUAL EXCESS: Four young men became affected with optic-atrophy. No cause could be found but sexual excess."

Dr. J.A. Spalding, of Portland, ME
Presented at the 33rd Annual Meeting of the
American Ophthalmological Society in 1897
(1-26-433)

Good Tidings

PAKISTAN JOURNAL OF OPHTHALMOLOGY has now been included in the list of journals being indexed and abstracted by:

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– Editors

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Figure 1:

Supernumerary or Accessory Caruncle

ABSTRACT: A 30-year-old woman had a rare supernumerary caruncle inside the right lower eyelid. The lesion was mostly symptom free. The diagnosis was confirmed by histopathologic studies. (Pak. J. Ophthalmol. 1: 64, 97, 1985)

Histopathologic studies of the specimen showed the tumor to be covered with stratified epithelium. It contained sebaceous glands, hair follicles, and fatty and connective tissue elements, all consistent with the diagnosis of supernumerary caruncle.

In 1896, Stephenson[1] reported the first case of *supernumerary caruncula lacrymalis.* The condition must be extremely rare because in the half century that followed only six more cases were documented.[2,3] The condition is most commonly found on the lower eyelid, as the normal caruncle arises from the nasal part of it,[4] but a case involving the upper eyelid has been reported.[5] The supernumerary caruncle may be confused with a healing chalazion, a granuloma, or a neoplastic lesion. It really is in no way troublesome to the eye, but may be hazardous to one's amorous ambitions since in one case the fiancee of the patient refused to marry him until the unsightly thing "had been removed."[5]

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Figure 2:

Vossius (or Vossius's) Ring

ABSTRACT: A 19-year-old man developed the Vossius ring following blunt trauma to his left eye. The author discusses the pathogenesis and the clinical significance of this rare phenomenon. (Pak. J. Ophthalmol. 1: 64, 97-98, 1985).

It is a ring of brownish or reddish-brown amorphous granules deposited on the pupillary area of the anterior surface of the lens following a blunt injury of the eye. A. Keller first described the phenomenon in six patients in his dissertation. After reading Keller's dissertation, Vossius[1]1published the first report on it. (Keller's ring or Keller-Vossius ring would be a more justified eponym.) He thought the ring was due to either "a cast of the pupil on the anterior surface of the lens" or the "transient degenerative changes in the epithelium of the capsule" from "the pressure of the iris against the capsule." In the latter case the ring is non-pigmented and "undergoes complete regeneration and becomes invisible." Vossius felt that the contact of the iris with the lens was brought about by the inward dimpling of the central cornea by the force of injury.[2] This theory was discredited by observations that the depression sufficient to cause this contact would lead to the rupture of this structure in normal eyes. This led to the explanation that the iris was forced against the lens by sudden elevation of the intraocular pressure. Some authors hold the opinion that the ring is produced by the deposition of blood on the anterior capsule in eyes with hyphema from any cause.[2,3] It must be remembered, however, that it has never been reported in a case of non-traumatic hyphema.[3]

The ring is about 1 mm in width and nearly 3 mm in diameter, and appears only in the young, because the iris must possess considerable elasticity to deposit the imprint.[4] This is confirmed by my own observation that at times deposits of pigment are seen on the anterior lens capsule following an iridectomy in children and young adults; whereas, I have never observed it in patients over 50 even when they are diabetics. Gundersen[3] reported on 19 cases of this ring, and none of his patients were over 30. He was also of the opinion that an intraocular hemorrhage would be a constant finding. The patient described here had no intraocular hemorrhage, but the observation of Gundersen warrants a careful examination of eyes with this ring. That the Vossius ring is a rarely seen phenomenon even in eyes with blunt trauma shows that many aspects of its pathogenesis remain unexplained.

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Figure 3:

Old Iron Foreign Body In Iris Mimicking a Tumor

ABSTRACT: A 62-year-old man had a 12 year old retained iron foreign body in the iris of his right eye without any adverse effects on the ocular structures or the sight. The foreign body was totally encapsulated and was initially mistaken for an iris tumor or malignant melanoma arising form one of the many dark iris freckles. (Pak. J. Ophthalmol. 1: 64-65, 97-98, 1985).

The patient had suffered an injury of the right eye while working with a hammer and chisel. A piece of steel had flown into the eye, but had caused no discomfort or loss of sight. The patient was under the impression that the foreign particle had only hit the eye and had fallen off. A small scar of corresponding perforation of the cornea was present. The eye has never been inflammed.

The eyes sometime show a remarkable tolerance to encapsulated foreign bodies which may remain without exciting inflammatory reaction for many years.[1-5] This observation led Lohlein[2] to suggest that under some situations, such as when the fellow eye is blind or has been lost, operation is indicated only when symptoms or signs of inflammation are present. This advice appears more logical if attempting to remove a foreign body in an inaccessible position may risk more damage than leaving it alone. There is a great risk of damage by leaving foreign bodies containing iron or copper in the eye. There are, however, cases on record in which the foreign bodies containing these elements remained in the eye for as long as half a century without exciting any reaction.[3-5] This case is an additional example of the eye's ability to tolerate without complication an iron foreign body. On initial observation, the encapsulated foreign body in this patient made me think of malignant melanoma of the iris that might have arisen from one of the many dark iris freckles present in his eyes.

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Figures 4, 5, 6 and 7:

Pulsatile Macroaneurysm of the Superior Temporal Artery of the Retina

ABSTRACT: A 66-year-old man had a pulsatile macroaneurysm of the left superior temporal artery of the first order of branching about 1½ disc diameter away from the disc border. A photographic documentation of the course of the disease from beginning to total regression is given. Although the aneurysm completely regressed spontaneously, the vision was lost due to involvement of the macula in recurrent hemorrhaging and exudation. (Pak. J. Ophthalmol. 1: 65, 97-98, 1985).

Although aneurysms of larger retinal arteries have been observed for almost a century,[1] it was in 1973 that Robertson[2] first established it as a definite entity. This has been confirmed in histopathologic studies by others.[3] The retinal arterial macroaneurysms are mostly a disease of persons in the sixth or seventh decade of life, an overwhelming percentage of whom are women.[4,5] It is interesting that pulsatile aneurysms of retinal arteries have always been seen in the age group over sixty. This may point to some additional factor involved in the pathogenesis of these aneurysms in the older age group.

The aneursyms may remain entirely asymptomatic throughout their course if the macular area escapes involvement in hemorrhage or exudation.[5] The usual clinical features are abnormality in the course of a retinal artery within the first three orders of arterial bifurcation, hemorrhage that surrounds the aneurysm and which may be deep retinal, peretinal or in rare instances, vitreal, and deposition of hard exudates in a complete or incomplete circle at the periphery of the hemorrhage. Fluorescein angiography shows staining of the wall of the aneurysm and dilated capillaries around the aneurysm that show leakage. Most of the patients have hypertension and arteriosclerosis. The isolated retinal arterial aneurysm appears to be a self-limited disease; a progressive fibrosis of the wall leads to eventual regression of the aneurysm and of the

Continued on Page 99

ملک میں قانونی پابندیاں لگانی چاہئیں تاکہ نیم حکیم قسم کے معالج کم علم اور بھولے بھالے مریضوں کو اپنے جال میں نہ پھنسا سکیں۔ باعثِ افسوس ہے کہ دنیا کی مصنوعی اندرونِ چشم عدسے implant کر رہی ہو اور پاکستان میں ابھی تک جعلی معالج couching کرتے پھر رہے ہوں۔ دواؤں کی خرید و فروخت پر بھی پابندی لازمی ہے۔ ہر فارمیسی کسی تجربہ کار اور تربیت یافتہ Pharmacist کے زیرِ نگرانی چلنی چاہیئے۔ اور ڈاکٹر کے نسخہ کے بغیر دوائی ہرگز نہ فروخت ہو۔ دواؤں جیسی اہم چیز کی فروخت اِس اصول پر چلانا مضحکہ خیز ہے کہ

مینیم جی پیسے لے لیں اور سودا دے دیں

حالیہ قریب میں آنکھوں کے علاج میں بہت سے نئے اضافے ہوئے ہیں۔ مثلاً لیزر Trabeculoplasty جیسی TIMOLOL نئی دواؤں کی ایجاد، اور Automatic Perimetry وغیرہ ایسے ذرائع ہیں جن سے کالے موتیے کی تشخیص اور علاج میں انقلاب برپا ہو گیا ہے۔ لیکن یہ ذرائع بہت زیادہ قیمتی ہیں۔ اور ان کا فائدہ ہمارے ملک کے اقتصادی حالات کے تحت بہت محدود ہے۔ عین ممکن ہے کہ ہم مستقبل میں TIMOLOL جیسی ادویات اپنے ملک میں بنانے لگیں۔ لیکن اگر ہم محنت اور جانفشانی سے کام کریں تو یہ حقیقت بھی ہم پر فاش ہو جائے گی کہ تنظیم اور جدوجہد سے ہم وسائل کی کمیابی کے باوجود منزلِ مقصود تک پہنچ سکتے ہیں۔

یہ بھی بہت اہم ہے کہ ماہرینِ امراضِ چشم پاکستان کی آئندہ سالانہ سوسائٹی میں تندہی سے حصہ لیں اور مجلہ طب العیون پاکستان کی ارتقاء میں پورہ کردار ادا کریں۔

خالد جاوید اعوان

منتفرقاتِ ماضی

اِس شمارے کے مختلف صفحات پر پرانے زمانے کے ماہرینِ امراضِ چشم کے نظریات نذرِ قارئین ہیں۔ ہمارے خیال میں ماضی کا مطالعہ حال کو تجسس افزاء اور مستقبل کو پُرامید بنانے میں بہت دخل رکھتا ہے۔ ماضی کا جائزہ لیتے وقت غیر شعوری طور پر ہمارے ذہن میں یہ خیال پیدا ہوتا ہے کہ آنے والی نسلیں ہمارے متعلق کیا رائے قائم کریں گی۔ "Ophthalmic Photo-Pourri" ایک مستقل عنوان کی حیثیت سے اِس مجلّے میں شامل کیا جا رہا ہے۔ اِس کے تحت امراضِ چشم کے متعلق پرانی تصنیفات میں سے اختصارات کا ئے بہ گاہے پیش کئے جائیں گے۔ جیسا کہ موجودہ شمارے میں شامل شدہ حقالے سے اندازہ لگایا جا سکتا ہے۔ یہ حصہ جن ایسے اقتباسات پر مشتمل ہو گا جن میں کچھ نہ کچھ سائنسی، تاریخی، یا لحاظ لطفِ طبع کا پہلو ہو گا۔

اگر قارئین اِس عنوان کے تحت اشاعت کے لئے نگارشات ارسال فرمائیں تو ادارہ بخوشی اِن پر سنجیدگی سے غور کرے گا۔ مضامین بھیجتے وقت ایک چیز کا خیال رکھنا ضروری ہے کہ مواد کے ساتھ حبلہ اور ماخذات کی فہرست شامل ہونی چاہیئے تاکہ قبل از اشاعت اِن کی توثیق میں آسانی رہے۔ آپ اپنے مضمون کا عنوان تجویز کر سکتے ہیں لیکن ادارے کو اِس میں تبدیلی کا حق ہو گا۔ خ ج ا

Camera Clinical-Exposition

Continued from Page 98

bleeding from it, as in the case of this patient.[3] The aneurysms in the peripheral area that do not involve macula are asymptomatic and even may go unnoticed. Those affecting the macula may require photocoagulation of the area surrounding the aneurysm to obliterate the abnormal capillaries there. A direct photocoagulation of the aneurysm or the artery is not advised. The retinal aneurysms may be associated, in rare instances, with central nervous system aneurysm, branch retinal vessel occlusion, or serous detachment of the retina. Unfortunately, when the macula becomes involved, the vision is almost invariably lost. In some instances if one aneurysm undergoes regression another might appear somewhere else, or in the other eye, fortunately a rare phenomenon.

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اداریہ

پاکستان میں کالا موتیا اور اس کا تدارک

اس شمارے کے اوّلین عنوان کے تحت پاکستان میں کالے موتیے کے مسئلے پر توجہ مبذول کی گئی ہے۔ پروفیسر ماہجو صاحب کے مقالے میں مترشح ہے کہ یہ مسئلہ ہمارے ملک میں فوری طور پر توجہ طلب ہے۔ اس معاملے میں ہمارا نقطۂ نظر اور طرزِ عمل متمول قوموں سے مختلف ہونا ضروری ہے۔ فنی طور پر ترقی یافتہ اور مالی آسودگی سے بہرہ ور قوموں کے طریقۂ کار پر عمل کرنے کی بجائے ہمیں پاکستان کے اقتصادی، سماجی، اور تکنیکی حالات کے پیشِ نظر اس میں ترمیم کرنا ہوگی۔

اس سلسلے میں بہت سے امور جواب طلب ہیں۔ مثلاً" ترمیم کہاں تک ممکن ہے؟ کونسا طریقِ علاج ہے جو عملی طور پر کارآمد بھی ہو اور اعلیٰ پیمانے پر عوام الناس کو میسر بھی آسکے؟ ترمیم شدہ طریقۂ علاج سے کون کونسے مسائل اور مشکلات پیدا ہوسکتی ہیں؟ ہماری یہ کوششیں اقتصادی طور پر کس حد تک قوم کے لئے قابلِ برداشت اور صحت کے لحاظ سے فائدہ مند ثابت ہو سکتی ہیں؟ ان سوالات کے جوابات دینے کیلئے پاکستانی ماہرینِ امراضِ چشم کو انفرادی تجربات کے بعد اپنے مشاہدات شائع کرنے چاہئیں۔ اس طرح سے ایک مجموعی خاکہ اُبھر آئے گا جس سے ایک مسلمہ طرزِ علاج مرتب کیا جاسکتا ہے۔ زیرِ نظر مجلہ پاکستانی ماہرینِ امراضِ چشم کی حوصلہ افزائی کرنا چاہتا ہے تاکہ وہ اپنے مشاہدات و تجربات بغرضِ اشاعت ارسال کریں۔

یہاں تک اس بیماری کے تدارک کا تعلق ہے اسکی حفاظتی تدابیر اوّلین اہمیت کی حامل ہیں۔ اس راستے میں بہت سی مشکلات حائل ہیں۔ مثلاً" تربیت یافتہ ڈاکٹروں کی کمی، مالی تنگدستی، آلات و ادویات کی کم یابی، اور سب سے بڑھکر عوام کی حوصلہ شکن کم علمی۔ الغرض

ع بہ راہِ مئے کدہ حائل ہزار دیوار است

پاکستانی عوام امراضِ چشم کے متعلق کم آگہی کا شکار ہیں۔ اکثر لوگ ابھی تک اس خیال میں مبتلا ہیں کہ جب تک سفید موتیا پوری طرح پکتا نہیں اس کا آپریشن نہیں ہوسکتا، اور آنکھوں کے آپریشن سال میں ایک خاص "موسم" میں ہی کئے جاسکتے ہیں۔ بدقسمتی سے یہ لوگ سفید موتیے اور کالے موتیے میں امتیاز نہیں کر پاتے۔ لہٰذا جب کوئی شخص کالے موتیے کا شکار ہوتا ہے تو اس کو سفید موتیا خیال کرکے اس کے پکنے کا انتظار کرتا رہتا ہے اور ہمیشہ کیلئے اپنی بینائی کھو بیٹھتا ہے۔ یہ اور اس قسم کی بے شمار غلط فہمیاں صرف اس صورت میں مٹائی جاسکتی ہیں جب کہ ہم خبر رسانی اور اطلاعات کے تمام ذرائع بلا استثناء پوری شدت کے ساتھ اس مقصد کے لئے استعمال کریں۔ میرے خیال میں میڈیکل کالجوں کے اعلیٰ کلاسوں کے طلباء کے نصاب میں یہ شامل کر لیا جائے کہ ہر طالب علم سال میں چند روز دور کے دیہات میں جاکر عوام کو ان بنیادی مسائل سے آگاہ کریں۔

پروفیسر ماہجو صاحب لکھتے ہیں کہ کئی مریض ایسے بھی ہیں جن کے کالے موتیے کا آپریشن کرنے سے پہلے سرجنوں نے پوری طرح تحقیق و تشخیص نہیں کی۔ اس سے صاف ظاہر ہوتا ہے کہ ہمیں ایسے سرجن درکار ہیں جنہوں نے بالخصوص آنکھوں کی بیماریوں کا علاج کرنے کی تربیت لی ہو۔ اس چیز کی شدت سے ضرورت ہے کہ پاکستان میں ایک بورڈ حضرر و متعین کیا جائے جس کی ذمہ داری اور اختیار ماہرینِ امراضِ چشم کی تعلیم اور تربیت کی جانچ پڑتال کرنا اور ان کو لائسنس دینے پر مشتمل ہو۔ دوسری طرف اپنے ملک کے اندر ایسے ادارے خود ہونے چاہئیں جو امراضِ چشم کی مناسب تربیت کے مواقع مہیا کریں۔

ماہرینِ امراضِ چشم کے علاوہ باقی ڈاکٹروں کو بھی ایسے حقائق نظر انداز نہیں کرنا چاہئیں جن سے مریض کی بینائی کو خطرہ لاحق ہو۔ میں نے خود پاکستان میں ایسے نوعمر مریض دیکھے ہیں جن کو موسمی سوزشِ چشم کے علاج کے لئے مسلسل CORTICOSTEROIDS دئے گئے اور وہ کالے موتیے کا شکار ہو کر بینائی کھو بیٹھے۔ بہت سے مریض ایسے ہوتے ہیں جن کو کالا موتیا ہوتا ہے اور ڈاکٹر ان کو ANTIBIOTICS دیتے جاتے ہیں حتیٰ کہ بیماری لاعلاج حد تک ترقی کر جاتی ہے۔ کالے موتیے کے ان المیاتی پہلوؤں کے تدارک کا واحد طریقہ مریض اور ڈاکٹر دونوں کی مناسب تربیت ہے۔ ڈاکٹروں پر لازم ہے کہ وہ آنکھوں کے مریضوں کو مناسب تربیت یافتہ سرجنوں کے پاس بھیجیں۔ یہ صرف اخلاقی فریضہ ہی نہیں بلکہ مرض کے مناسب علاج کا واحد طریقہ ہے۔ مزید برآں ہر ماہر ہر بیماری کا علاج نہیں کرسکتا۔ مثال کے طور پر پیدائشی کالے موتیے کو لیجئے۔ بہت کم ماہرینِ امراضِ چشم ایسے ہیں جنہوں اس مرض کے کافی مریض دیکھے اور ان کے علاج کا تجربہ حاصل کیا ہے۔ ایسے شاذونادر امراض کے علاج کے لئے ملک میں خصوصی مراکز ہونے چاہئیں یہاں صرف ان ہی بیمار بچوں کے مریض بھیجے جائیں اور وہاں کے سرجن ان کے علاج کا خوب تجربہ رکھتے ہوں۔

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

Glaucoma and Its Management in Pakistan

The lead article in this issue focuses attention on the formidable problem of glaucoma in Pakistan. The paper by Professor Mahju demonstrates that the problem of glaucoma is in urgent need of re-evaluation and must be viewed differently in Pakistan than it is in the rest of the modern world. The professional, social, and financial circumstances in Pakistan are such that the methods and philosophies of therapeusis employed by more prosperous and scientifically more advanced nations in the management of glaucoma cannot be effectively utilized by us without significant modifications.

What are the possible modifications? What therapy might be practical and useful? What could be done on a mass scale? What new problems would a modified approach create? How productive and cost effective would these efforts be? These are questions that can only be answered by ophthalmologists interested in suggesting such modifications and showing their effectiveness and practicality by controlled studies. The individual efforts by ophthalmologists of Pakistan must be recorded and published to bring about a collective solution to the problem of glaucoma in Pakistan. The Pakistan Journal of Ophthalmology encourages the ophthalmologists of Pakistan to begin making written records of their ideas and experiences and sending them to the Journal for possible publication.

Concerning glaucoma in Pakistan, the preventive and prophylactic aspects need to be addressed first of all. The lack of fully qualified physicians, the paucity of resources, a limited availability of therapeutic modalities, and most of all dismally poor public awareness inhibit an effective control of glaucoma in Pakistan. The people of Pakistan must be educated about eye diseases, glaucoma in particular. Many of them still believe that surgery for cataract cannot be done unless it is "ripe" and many confuse "Chitta Motia"* with "Kala Motia"**. A large number with kala motia go blind waiting for their sight to completely go out as a sign of ripeness of what they mistakenly believe is chitta motia. I have personally seen a large number of patients with absolute glaucoma who let their disease progress to this hopeless stage because of this erroneous concept. It is possible that the common term "Motia" is also a source of confusion between "Kala Motia" and "Chitta Motia." These and numerous other wrong concepts about eye diseases can only be removed by educating people through all media available. Public education meetings in remote villages may be made a part of the curriculum for the senior medical students.

Professor Mahju notes that some patients with glaucoma had been operated on without proper evaluation by surgeons. This clearly shows a need for proper training of the physicians who take care of eye patients. A regulatory board of qualified ophthalmologists to scrutinize the training, skill, and specialty education of these physicians is urgently needed. At the same time, the physicians interested in ophthalmology must have available to them regular, well organized, and well staffed education opportunities inside Pakistan. Physicians in other fields also need to know some important facts about blinding diseases of the eye. I have seen healthy and intelligent teens with the end-stage steroids induced glaucoma because of prolonged use of topical steroids in treatment of allergic conjunctivitis under the direction of family practioners. A large number of patients with easily detectable acute attacks of glaucoma are treated with antibiotics until the eyes develop irreversible damage. All these and other tragic aspects of glaucoma in Pakistan can be eliminated by an aggressive approach to public and physician education. Physicians in Pakistan also need to be made conscious of the importance of referring patients to properly trained specialists. This is not only their moral and professional duty but the best way to provide optimum care to the patient as well. Some disorders cannot be treated by every specialists, no matter how knowledgeable and skillful. One example is congenital glaucoma. It is very rare and few ophthalmologists see enough cases to become proficient in its proper management. Hence, it is logical to refer such patients to a particular ophthalmologist or institution in the region and establish a center where physicians can become more familiar with the disease and more capable in providing care for it.

Legal controls are necessary to stop couching and other unscientific methods practiced by quacks upon unsuspecting and trusting patients. Some sort of restrictions on the dispensing of drugs without prescriptions from qualified physicians must be implemented. All pharmacies and drug stores must be required either to employ or work under the supervision of a qualified pharmacist. Drugs and medicines are too important to be left in the care of

*cataract
**glaucoma

store cashiers alone.

The advent of laser therapy, popularization of techniques like trabeculoplasty, and the introduction of newer drugs, such as timolol, automated perimetry, and better methods of evaluating optic disc changes in glaucoma have revolutionized the management of glaucoma. But these are too costly and technical to be very useful in Pakistan in our fight against glaucoma. I know that timolol, for example is either too expensive or altogether unavailable for many patients in Pakistan. Pakistan might produce these items locally but Pakistanis may also find that organization and hard work accomplish much even when material is limited.

It is also important that Pakistani ophthalmologists more actively participate in the activities of the Ophthalmological Society of Pakistan and enthusiastically contribute to the Pakistan Journal of Ophthalmology.

Khalid J. Awan

Appreciation, Encouragement

Dear Khalid, I congratulate you, most sincerely, on bringing out the first issue of P.J.O. Judged from any angle it is an excellent journal. It shows your total dedication to the task entrusted to you and brings honor to Pakistan and to yourself. I can well imagine how much physical, mental, and emotional effort is required to bring out a scientific publication consistent with current international standards.

The list of Editorial and Advisory Board is staggering. These internationally famous men have become legends in their own lifetimes and you are certainly to be commended for pursuading them to give their precious, hardpressed time to this Board. If you had done nothing else for the P.J.O., and only assembled this Board, you would have laid a claim to the gratitude of your countrymen. With such an Editorial and Advisory Board, the papers selected attain international status. You also succeed in paying attention to the minutest details of format, arrangement of papers, reproduction of photographs, and error free printing on first class paper. All this would ordinarily have required the concerted efforts of a number of people working in specialized areas. It is amazing that one man could do this alone.

Sir Alexander Fleming the discoverer of penicillin visited us in the early fifties. He had occasion to comment that worth while things in life have always been done by individuals. As far as P.J.O. is concerned, you are that single individual. To do something for the first time for one's country is a privilege given only to the very lucky and to the very few. You are such a lucky one.

The time and effort you have expended is apparent from the result. I should really be most surprised if anyone failed to see this. God Almighty above is watching and history is there to record your efforts. Coming generations will salute you for your efforts to enhance the prestige of your country and its ophthalmologists in the eyes of the world, even as I salute you now.

M.A. Shah, M.D.
Ex Dean and
Chairman of Department
of Ophthalmology
Dow Medical College, Karachi

and

President,
Ophthalmological Society of
Pakistan, Karachi
January 20, 1985

Ophthalmic "Past-Pourri"

Scattered in the pages of this issue the reader will find interesting excerpts from the ophthalmic literature of days gone by. A knowledge of how the things were in the past is perhaps the strongest stimulus and the greatest encouragement for a worker in the present, particularly the one in a scientific field. Furthermore, our peek into the past is the best learning about how the future shall view us.

The Journal is introducing a new section of OPHTHALMIC "PAST-POURRI" in which we intend to periodically publish brief quotes from older writings related to the eye. The items that are of historic value, have true scientific significance, or are simply amusing will be included in this section. Entries illustrative of each of these are part of this issue's OPHTHALMIC "PAST-POURRI."

The readers are invited to submit their contributions that fulfill these requirements. Editors request that each contribution be accompanied with information necessary for verification of the sources or references. The contributors may also suggest appropriate titles for the items they submit. Such titles shall be subject to editing.

Khalid J. Awan

We wish our readers a very happy and successful 1985.

—Editors

First Issue Feedback

"This is to offer you my very best wishes and congratulations on having achieved this milestone. I note that The American Journal of Ophthalmology this year celebrated its 100th anniversary and I hope that in October 2084, the Pakistan Journal of Ophthalmology will celebrate a similar anniversary. I thought the issue was well prepared. Your article on *angioid streaks* looked extremely good. I was particularly impressed with the skillful reproduction of the illustrations. I thought that Figure 4 showing the peau d'orange was likely the best example of this I had ever seen."

Frank W. Newell, M.D.
Publisher and Editor
American Journal of Ophthalmology

"There is no doubt that this is an historic event in publishing and the Ophthalmological Society of Pakistan and those involved deserve congratulations for this accomplishment. I was quite impressed with the format of the journal and the ease with which it could be read. The illustrations are very clear and the article selections are quite attractive, as well as informative. You have selected an excellent Editorial and Advisory Board."

Irving H. Leopold, M.D., D.Sc.
Department of Ophthalmology
University of California

"Congratulations. You've done a marvelous job."

George L. Spaeth, M.D.
Director, Glaucoma Service
Editor, Ophthalmic Surgery

"Having seen it, I now regret to have missed the chance of rubbing shoulders with other contributors in this issue. I send my most sincere congratulations to you and to Professor Mumtaz for bringing out this magnificent Journal, which undoubtedly puts Pakistan on the ophthalmic map. The ophthalmologists in Pakistan must feel proud of this achievement and I feel sure their good wishes and consistent patronage will guarantee its survival."

A.H.S. Rahi, M.D., PhD, FRC Path.
Reader in Immunopathology
Institute of Ophthalmology
University of London

"Congratulations to the splendid first issue of your Pakistan Journal of Ophthalmology. I especially admired your *lead article and memorial lecture.* You have done a *splendid job* and I know from personal experience how much work is involved. Your *obituary of Professor Francois* added a warm personal touch."

Frederick C. Blodi, M.D.
Director, Medical Education
King Khaled Eye Specialists Hospital, Fiyadh
Formerly the Editor,
Archives of Ophthalmology

"Thank you very much for...the copy of the Pakistan Journal of Ophthalmology. I have looked at the articles contained therein and am very impressed at the quality both of the presentations and of the reproductions. You are certainly to be congratulated."

Robert L. Stamper, M.D.

"It was a pleasure to receive the first copy of the Pakistan Journal of Ophthalmology. It illustrates much preparation and thought and you are to be commended on a wonderful publication. I foresee many years of successful publication for this new journal. It was particularly interesting that you included the *Camera Clinicals* and the *abstracts* from other journals. This should certainly broaden the appeal of your journals. Congratulations on an outstanding journal."

Alice McPherson, M.D.

"The issue is of excellent quality and I commend you for initiating this fine mechanism for scientific communication."

Bradley R. Straatsma, M.D. Director
Jules Stein Eye Institute
University of California, Los Angeles

"Congratulations on the very fine Volume 1, No. 1 of the Pakistan Journal of Ophthalmology. you have undertaken a monumental task. The early results indicate that you are up to the task."

Eugene M. Helveston, M.D.
Professor
School of Medicine
Department of Ophthalmology
Indiana University

"I must say that I am really impressed about the excellent makeup of this first edition. Congratulations! I can easily imagine how much work you have had since I, myself, am involved in working on a journal. I think the personal flavor that you put in is very appropriate.

Robert Machemer, M.D.
Editor, Graefe's Archives of
Clinical and Experimental Ophthalmology

"I wish to congratulate you on the successful launching of the first issue of the Pakistan Journal of Ophthalmology. I commend your efforts in publishing an exceptional journal."

Randall T. Bellows, M.D.
Associate Director
The American Society of
Contemporary Ophthalmology
Chicago

"I did get your first publication (of the Pakistan Journal of Ophthalmology.) I thought it was very well done. Congratulations on the splendid job you did on this.

A. Edward Maumenee, M.D.

"I am quite impressed by both the Editorial and Advisory Board that you have assembled for the Journal and by the quality of material that has been a part of the first issue. By reading through the issue, it is obvious... that this Journal represents, almost completely, your great efforts in the project. Sometimes, it takes this type of commitment to get a Journal started and your efforts are commendable. I am impressed both by the quality of material in the Journal and by the professional layout and organization."

Henry S. Metz, M.D.
Editor
Journal of Pediatric
Ophthalmology & Strabismus

"I want to congratulate you for personally lauching a major eye journal, The Pakistan Journal of Ophthalmology. I read quickly through each of the articles, examined the illustrations, and paid attention to the references as well. It is a remarkable accomplishment and one of which you should be justifiably proud."

Stuart L. Fine, M.D.
Professor of Ophthalmology
The Wilmer Institute
The Johns Hopkins Medical Institutions

"Congratulations on the appearance of your new journal. It shows evidence of considerable care in its preparation."

Morton F. Goldberg, M.D., Editor
Archives of Ophthalmology

"The first issue of the Pakistan Journal of Ophthalmology is something of which you can be extraordinarily proud. Congratulations on a splendid product. I know that your colleagues will be most appreciative."

George O. Waring, M.D., F.A.C.S.
Professor of Ophthalmology

"Your journal comes at a most coincidental time. Just last week I operated on a gentleman from Pakistan, so I had the opportunity to tell him about your journal and asked him regarding some of the expressions used in the journal. Such as the introductory sentence, Bismillahir-Rahmanir-Raheem, which he tells me means, with the Grace of God. I noticed that this was the introductory sentence in many of the comments and letters in this most interesting journal. I congratulate you on an excellent journal, and an outstanding group of initial subjects."

Jack Hartstein, M.D.

"My dear Dr. Awan, I enjoyed the articles thoroughly. I found the reproduction exceptionally fine. It must give you great pleasure to be the founder of a national ophthalmic journal for your country."

Harvey Lincoff, M.D.
Professor of Clinical Ophthalmology
Cornell University Medical College

"I am very much impressed by the quality of the journal, and you ought to be congratulated for all your efforts. Please accept my congratulations for your efforts in this excellent new journal in an attempt to improve the academic aspects of ophthalmology in Pakistan."

Muhammad A. Khan, M.D.
Associate Professor of Medicine
Case Western Reserve University
Cleveland, Ohio

"Indeed the quality of the papers and your beautiful photographs highlighted this journal remarkably. The overall lay out and context is extremely well done. I am extremely interested in the *carcinoma of the lacrimal sac* paper by A.J. Khan, M.D. and I would like to congratulate you on your beautiful paper on angioid streaks which is the lead article. I think that you have an excellent choice on your editorial and advisory board.

Pierre Guibor, M.D.
President,
The International Oculoplastic Society

"It is certainly very elegant."

Claes H. Dohlman, M.D.
Professor and Chairman
Harvard Medical School
Department of Ophthalmology

"Lead article on *angioid streaks* of the ocular fundus was particularly fascinating and should be very useful for all ophthalmologists. The other articles also proved quite interesting and I am sure the readers will find this a good first issue."

Paul Henkind, M.D., Ph.D.
Professor and Chairman
Department of Ophthalmology
Albert Einstein College of Medicine
Editor, Ophthalmology

"Thank you for sending me the first issue of the Pakistan Journal of Ophthalmology. It is certianly attractively done and I enjoyed reading the articles, particularly the one by you on *Drug Induced Myopia and Angle Closure Glaucoma in Drug Addiction.* Perhaps in a future issue you might explain the significance of the logo on the cover. I, of course, recognize the eye and the lens but an explanation of the arabic writing and the dangling star would be of interest as well as the tower below this."

David Shoch, M.D.
Professor of Ophthalmology
Northwestern University

Note: All italics by the Editor.

A Logo Lesson

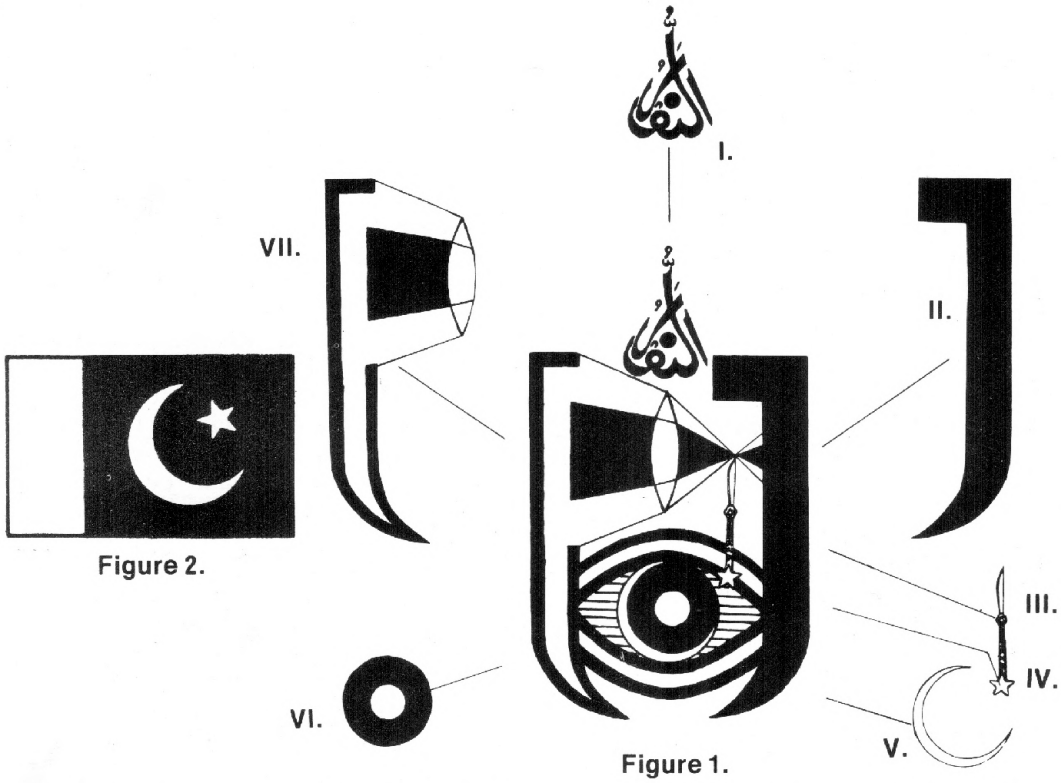

Figure 1.

Figure 2.

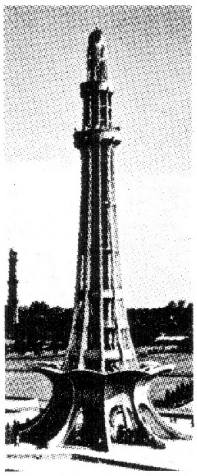

Figure 3.

It is hard to say why a logo catches the eye of an observer. It may appeal to an appreciation of creativity, arouse curiosity or even generate some confusion. Whatever the source of the attraction, however, it is gratifying to know the logo of the Pakistan Journal of Ophthalmology has it. I have been asked on many occasions, in chats and in chits, what those interesting drawings in our logo mean. In fact, on a few occasions the whole front cover of the first issue became a subject of discussion. I am obligated, since I designed and drew it, to unravel the elements of the logo and explain their significance to our readers. To make my description easier to follow, I have spread the components of the logo in Figure 1. The calligraphic writing I at the top of the logo is the Urdu-Arabic word "Annoor," meaning the "light". Annoor is also an attribute of God Almighty and is many times used to represent sight. The letter J shown at II stands, of course, for the word Journal. The rays focusing on it symbolize the hope that the Journal will act as a storehouse of ophthalmic knowledge from and for Pakistan. The knife at III is there to indicate that the Journal is an instrument of ophthalmology and not of optometry. The star at IV is part of the national symbol of "Crescent and Star" and its location at the handle end also symbolizes the hand of the surgeon that puts the light back into the blind eye. The crescent at V is the other half of the Pakistan's national symbol of "Crescent and Star." The arrangement of the crescent and the star follows the exact pattern of the symbol on the national flag (Figure 2). In the logo, the crescent is embracing the left side of the iris and pupil of the eye, that represent the letter 0 at VI for Ophthalmology in the title of the Journal. The letter P at VII represents Pakistan in the title. It is made up of a biconvex lens focusing the rays of light from the column of P, symbolizing what has been already explained in the description of letter J.

I had intended to interweave the representations of our faith, country, and profession, in the logo of the journal without losing the significance of one at the expense of the other; and yet to come up with a design that was tasteful and appealing. If readers think that I have met my objective and approve of the logo, I shall feel thoroughly compensated for the two months of labor I employed in its creation.

While I am at it, let me explain about the tower on the front cover. This is mainly for the benefit of our non-Pakistani readers. It is a line sketch of the Minar-e-Pakistan (Pakistan Tower) in Lahore (Figure 3). This tower stands in Iqbal Park at the site where our founding fathers passed the Pakistan Resolution. Though not a skyscraper in physical height, it stands tall indeed in the hearts of all patriots. The credit for placing it on the cover of the journal goes to Professor Raja Mumtaz. He was also responsible for the screen of the geographical map of Pakistan which provides such an attractive background for the logo on the Editorial Board page.

-Khalid J. Awan

Book Review

THE SURGEONS GUIDE TO INTRAOCULAR LENS IMPLANTATION. By Henry M. Clayman, M.D., Slack Inc., Thorofare, N.J., 1984, hardcover, 169 pages, $49.50.

This handsome hardcover book with a smoothly written and superbly organized text also offers very informative supportive illustrations and it serves very well to convey the presently dominant views on lens implants and lens implantation techniques at a practical level. These views are presented very carefully on the basis of the history of intraocular lens implantation. Very reasonable examples and rules for the selection of surgical procedures and implant types are given - not an easy task in view of the great differences of opinion between proponents of various surgical techniques and implant types. Limits of only six printed lines on "IOL Precipitates and Membranes" and of only fifty lines on "Cystoid Macular Edema" in the chapter on complications indicate that the book does not dwell on difficulties and side effects sometimes seen with lens implantation.

This well-made book offers pleasant and informative reading for an afternoon and it will serve very well as an introduction and a guide for physicians in presently popular thought on its subject matter. It is highly recommended for that purpose — *J. Reimer Wolter, M.D.*

COMPUTERIZED VISUAL FIELDS: WHAT THEY ARE AND HOW TO USE THEM. Edited by William R. Whalen, M.D. and George L. Spaeth, M.D., Slack Inc., Thorofare, N.J. 1984, hardcover, 414 pages, $75.00.

No doubts about it: the time for computerized perimetry has arrived - in its convincing practical value and usefulness ahead of most other automation in our field. Automated threshold perimetry is progress; it is different from and offers more than manual perimetry.

This well-made hardcover book is very well written by clinically and scientifically established experts and teachers. It covers all theoretical, technological, and clinical aspects. In sixteen chapters the practical technology is carefully discussed and the clinical application is considered by its purposes as well as by its normal and abnormal values. Special chapters on use in glaucoma, neuro-ophthalmology, and retinal diseases are complemented by chapters on the operation of computerized perimetry, statistical analysis, and a comparison with manual techniques. Numerous good illustrations and references on the end of each chapter are offered. More than fifty pages of bibliography in the end add to the permanent scientific value of the book as a basis and reference for the future.

This book belongs in every departmental and serious private ophthalmological library. It is most highly recommended. — *J. Reimer Wolter, M.D.*

Scanning Electron Microscopy of Medically Important Arthropods. By Vicar Zaman. Singapore, Maruzen Asia PTE. LTD., 1983. 176 pages, no index, illustrated. Price not shown.

The author, Professor V. Zaman, is internationally famous in the field of entomology, and Scanning Electron Microscopy of Medically Important Arthropods shows that the reputation is well deserved. The contents of this book were also submitted by the author as his thesis for the Fellowship of the Pakistan Academy of Medical Sciences (PAMS). Zaman photographed all the illustrations in the book and they are a collection that will be very profitable for research and teaching. As mentioned in the preface, the book is essentially a pictorial presentation and should be studied in conjunction with a standard text. However, it contains sufficient brief description with each illustration that it can serve as a quick review of the subject.

The illustrations are in black and white and exquisitely beautiful. They are reproduced crisply and with an artistic touch. Because of my interest in the subject of phthiriasis palpebrarum,[1] I particularly enjoyed pages 118-127 (Figures 1-6). Going over the section on mites brought to mind the interesting motion picture of *Demodex Folliculorum* Dr. Jacobson [2] presented at the 75th Annual Meeting of American Academy of Ophthalmology and Otolaryngology in Las Vegas, in 1970. (Mites infest the eyelashes of over 90% of the people, but are probably not directly responsible for the so-called blepharitis acarica.) All the material in the book may not be of direct relevance to ophthalmology, but the volume is a scholarly treat for the eyes. I recommend this book enthusiastically.
Reviewed by Khalid J. Awan

References

1. *Awan KJ: Cryotherapy in phthiriasis palpebrarum. Amer. J. Ophthalmol 83:906, 1977.*
2. *Jacobson, JH: Demodex Folliculorum,* Infestation of eyelids. Trans. Amer. Acad. Ophthalmo. Otolaryngol. 75:1242, 1971

Books Received

Computer Essentials for Ophthalmologists. Edited by Donald R. Sanders, M.D., Ph. D., and Gerald E. Meltzer, M.D., Slack Inc. Thorofare, N.J. 1984, spiralbound.

Real Time Ophthalmic Ultrasonography and Biometery. By Richard S. Koplin, Martin Gerstin, and Barton Hodes, M.D., Slack Inc., Thorofare, N.J., 1984, spiral-bound.

Ocular Syndromes & Systemic Diseases. By F. Hampton Roy. Grune & Stratton, Inc., New York, 1985.

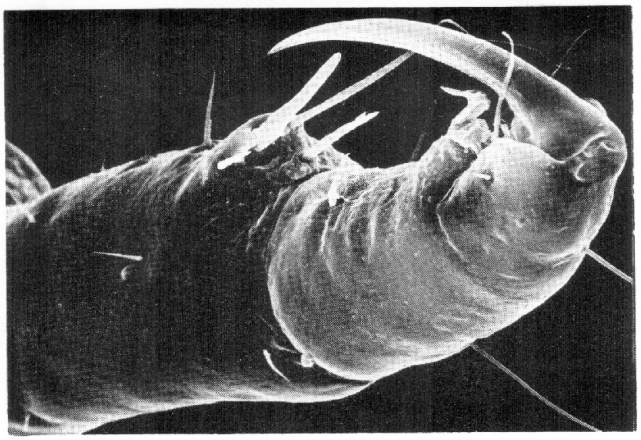

Figure 1. (Zaman) *Pediculus humanus* showing the typical large curved claw and smaller thumb like spine on the inner side. This arrangement is ideal for gripping the hair of the host. X1000.

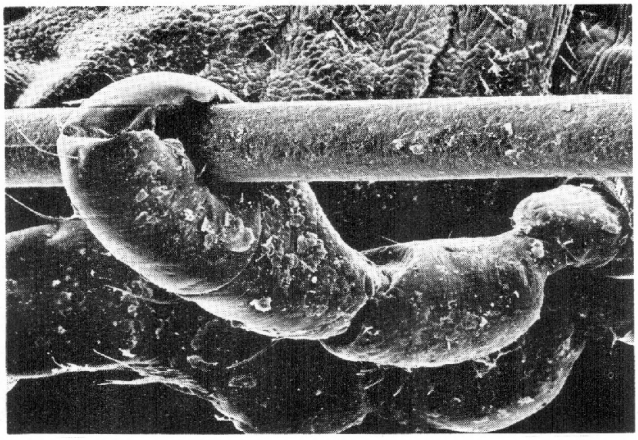

Figure 2. (Zaman) *Pediculus humanus* showing the claw gripping rod like structure, which is a hair. X1000.

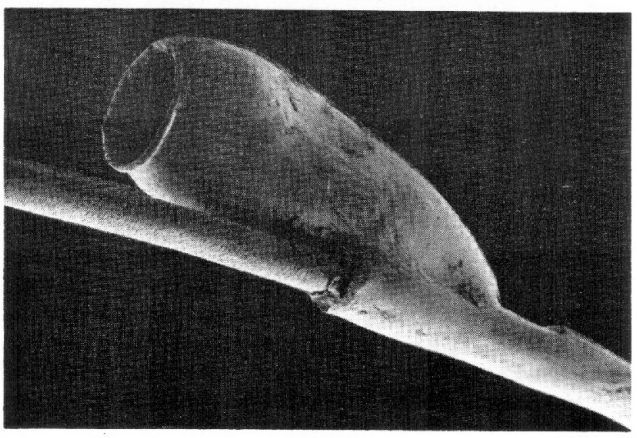

Figure 3. (Zaman) *Pediculus humanus* egg. This is commonly called a nit, is oval in shape and is glued to a hair. In this case the operculum is lost as the larva has hatched. X500.

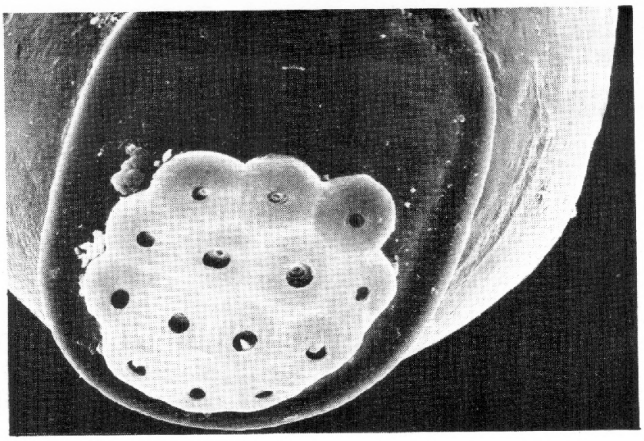

Figure 4. (Zaman) *Pediculus humanus* egg showing the operculum which has small perforations sitting on a circular disc like structures. X2000.

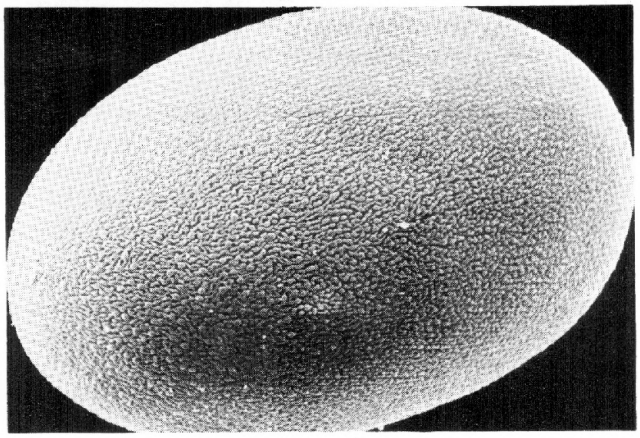

Figure 5. (Zaman) *Mite Egg* (Family-Acaridae). It is oval in outline and has a tuberculated surface. X2000.

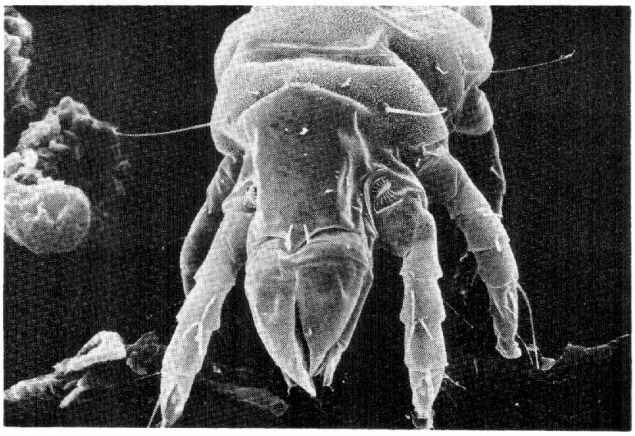

Figure 6. (Zaman) *Mite* (Family-Acaridae), probably belonging to a *Tyrophagus* sp., which is very common in tropics. Note that mite is sitting on dust particles and the dorsum is finely tuberculated. Between the neck and the first leg is the supracoxal seta. X1000.

Abstracts from Elsewhere

AMA Archives of Ophthalmology

SURGICAL TECHNIQUE FOR ADVANCED JUVENILE GLAUCOMA. A.C.B. Molteno, FRCS; E. Ancker, MD; G. Van Biljon, MD. The main causes of drainage operation failure are early hypotonia, blockage of the surgical opening, and excessive bleb scarring. By using draining implants, inserted in two stages, together with the temporary administration of medication to control bleb fibrosis, the surgeon can circumvent these causes of failure and drain the most severe and complex cases of glaucoma with good immediate and long-term results. This surgical technique failed in only four of the 83 eyes with advanced juvenile glaucoma. (Arch Ophthalmol 102:51-57; 1984) *Author's Abstract.*

A GRAPHIC THREE-STEP TEST. R.L. Vazquez, MD. A graphic technique is used to analyze cyclovertical muscle palsies. This technique is similar to that presented in the *Ophthalmology Basic and Clinical Science Course*, but has the following advantages: only one diagram is required to apply the method, and knowledge of the actions of the cyclovertical muscles, when analyzing the Bielschowsky head tilt test, is not required. (Arch Ophthalmol 102:98-99; 1984). *Author's Abstract.*

OPHTHALMOLOGIC FINDINGS IN ACQUIRED IMMUNE DEFICIENCY SYNDROME (AIDS). M. Khadem, MD; S.B. Kalish, MD; J. Goldsmith, MD; C. Fetkenhour, MD; R.B. O'Grady, MD; J.P. Phair, MD; M. Chrobak. Forty-one homosexually active men had ophthalmologic and immunologic evaluations. Four of eight with acquired immune deficiency syndrome (AIDS) had abnormal ocular findings that included cotton-wool spots, retinal hemorrhages, cytomegalovirus, retinitis and conjunctivitis due to cytomegalovirus, and keratoconjunctivitis sicca. The other four patients with AIDS and 33 homosexual male controls had normal ocular examinations. Patients with AIDS and abnormal eye findings had a notably lower total leukocyte count, absolute lymphocyte count, percentage T-helper lymphocytes, helper-suppressor lymphocyte ratio, hematocrit level, and platelet count than patients with AIDS and normal results on eye examination or controls. All patients with AIDS and abnormal eye examination results died; the four other patients with AIDS and normal eye findings remain alive. These observations suggest that ophthalmologic abnormalities are common in patients with AIDS, are associated with severe immunoregulatory abnormalities, and carry a poor prognosis. (Arch Ophthalmol 102:201-206; 1984) *Author's Abstract.*

BACTERIAL ENDOPHTHALMITIS AFTER CLOSED VITRECTOMY. P.C. Ho, MD; F.I. Tolentino, MD. Four cases of bacterial endophthalmitis occurred after more than 2,800 closed vitrectomies. Despite vigorous antibiotic therapy, all four eyes were lost. The poor outcome seems to result from difficulties in diagnosing this condition in its early stages. Important clinical indications, such as orbital pain, corneal edema and infiltrate, excessive intraocular inflammatory reaction, hypopyon, and diminished fundus reflex, are often masked by the usual postoperative course. According to our study, the incidence of endophthalmitis after closed vitreous surgery is 0.14%. Three of the four patients with endophthalmitis were diabetic. Our clinical findings are compared with those in the four other cases reported in the literature. (Arch Ophthalmol 102:207-210; 1984) *Author's Abstract.*

BIRDSHOT RETINOCHOROIDOPATHY. D.J. Fuerst, MD; H.H. Tessler, MD; G.A. Fishman, MD; M.M. Yokoyama, MD, PhD; G.J. Wyhinny, MD; C.M. Vygantas, MD. Nine patients had birdshot retinochoroidopathy. Associated ophthalmologic findings included rhegmatogenous retinal detachment, rubeosis iridis, glaucoma, and a high incidence of disciform macular degeneration. Four patterns of birdshot spot distribution were noted on fundus examination. Immunologic studies showed a significantly elevated percentage of EA rosettes and an elevated C4 complement level. Electroretinograms showed b-wave amplitude reduction, with a disproportionate implicit time prolongation. The etiology of this syndrome remains unknown. (Arch Ophthalmol 102:214-219; 1984) *Author's Abstract.*

TREATMENT OF ESSENTIAL BLEPHAROSPASM. II. A MODIFICATION OF EXPOSURE FOR THE MUSCLE STRIPPING TECHNIQUE. C.D. McCord, Jr., MD; J. Shore, MD; J. R. Putnam, MD. The refinement of a muscle excision technique for the correction of essential blepharospasm has been a major contribution to treatment. In this procedure, incisions are made directly above the eyebrow to excise the brow muscles, often resulting in adherent scars and poor brow position. The bicoronal scalp flap has been used by many other surgical subspecialities for exposure of the frontal bone area, eg, in neurosurgical procedures, sinus surgery, and cosmetic forehead lifts. The use of the coronal flap exposure for excision of the corrugator and procerus muscles has allowed them to be more easily excised and has avoided the problems associated with the direct brow

incisions. The Anderson muscle stripping technique, combined with the coronal flap exposure for the brow muscles, provides the best correction for the spasms in patients who have essential blepharospasm. The frequency of complications is low, and patient acceptance is high. (Arch Ophthalmol 102:269-273; 1984) *Author's Abstract.*

TRABECULECTOMY V THERMOSCLEROSTOMY A FIVE-YEAR FOLLOW-UP. R.A. Lewis, MD; C.D. Phelps, MD. Thirty-seven eyes with open-angle glaucoma were treated by trabeculectomy and 34 eyes were treated by thermosclerostomy. Thermosclerostomy lowered intraocular pressure to a slightly greater extent than trabeculectomy; however, the average difference was not statistically significant except for the second year. After five years, IOPs were less than 22 mm Hg without additional medications or surgery in 24 (65%) of eyes undergoing thermosclerostomy. When eyes were included that were treated with medication or additional glaucoma or cataract surgery, over 90 % of eyes in each group had an IOP less than 22 mm Hg. Visual acuity declined frequently in both groups, and progression of cataract was the most common cause. Loss of visual acuity occurred much more frequently in patients who were older than 60 years than in younger patients. (Arch Ophthalmol 102:533-536; 1984) *Author's Abstract.*

POSTTRAUMATIC ENDOPHTHALMITIS. G.S. Brinton, MD; T.M. Topping, MD; R.A. Hyndiuk, MD; T.M. Aaberg, MD; F.H. Reeser, MD; G.W. Abrams, MD. Nineteen consecutive cases of culture-proved posttraumatic endophthalmitis occurred. Over an eight-year period, 19 (7.4%) of 257 patients with penetrating trauma had endophthalmitis develop, and 19 (31.1%) of 61 cases of endophthalmitis were due to trauma. Eleven (10.7%) of 103 patients with intraocular foreign bodies had endophthalmitis develop. Final visual acuity was 20/200 or better in eight (42.1%) of 19 and 20/30 or better in five (26.3%) of 19 cases of posttraumatic endophthalmitis. Organisms cultured were similar to those in the other types of endophthalmitis, except that Bacillus species were seen only in posttraumatic endophthalmitis (five [26.3%] of 19.) Virulent organisms or retinal breaks or detachments seen at the time of primary repair indicated poor prognoses. (Arch Ophthalmol 102:547-550; 1984) *Author's Abstract.*

ARGON LASER TRABECULOPLASTY AS INITIAL THERAPY FOR GLAUCOMA. J.V. Thomas, MD; A. El-Mofty, MD; E.E. Hamdy, MD; R.J. Simmons, MD. Argon laser trabeculoplasty (ALT) was used as the initial treatment in 30 eyes of 20 patients with uncontrolled open-angle glaucoma in Egypt. None of the patients had received previous medical or surgical therapy. The average reduction of intraocular pressure with ALT alone was 10.1 mm Hg. Medical and surgical glaucoma therapy was avoided in 83% (25 eyes) of the treated eyes during an average follow-up period of 7½ months. The data indicate that ALT can be used as a safe and effective initial treatment for glaucoma in developing countries where socioeconomic factors make the medical treatment of glaucoma impractical. (Arch Ophthalmol 102:702-703; 1984) *Author's Abstract.*

***ACANTHAMOEBA* KERATITIS POSSIBLY ACQUIRED FROM A HOT TUB. J.R. Samples, MD; P.S. Binder, MD; F.J. Luibel, MD; R.L. Font, MD; G.S. Visvesvara, PhD; C.R. Peter, PhD.** An irritated left eye followed by a geographic epithelial corneal defect developed in a 42-year-old man. Disciform edema developed in the cornea, and the lesion progressed to a ring-shaped abscess. The lesion failed to respond to medical therapy. After two penetrating keratoplasties histopathologic examination and electron microscopic studies established the diagnosis of *Acanthamoeba* keratitis. Subsequent cultures and immunofluorescent studies identified the organism as *Acanthamoeba castellani.* Following treatment with antibiotics and corneal cryotherapy, there has been no evidence of recurrence. Morphologically and immunologically identical amebae were also cultured from the patient's hot tub and surrounding garden. (Arch Ophthalmol 102:707-710; 1984) *Author's Abstract.*

PROLIFERATIVE LUPUS RETINOPATHY. A.K. Vine, MD; C. C. Barr, MD. Preretinal neovascularization in response to retinal vascular occlusions developed in two patients receiving treatment for systemic lupus erythematosus. In both patients, the preretinal neovascularization was asymptomatic and was discovered on routine ocular examination. One patient required bilateral peripheral ablative argon-laser therapy for florid preretinal neovascularization. This patient had severe occlusive retinal vasculitis despite serologic evidence of remission of the underlying disease process. (Arch Ophthalmol 102:852-854; 1984) *Author's Abstract.*

FORMATION OF PERIPHERAL ANTERIOR SYNECHIAE FOLLOWING ARGON LASER TRABECULOPLASTY. A PROSPECTIVE STUDY TO DETERMINE RELATIONSHIP TO POSITION OF LASER BURNS. C.E. Traverso, MD; K.C. Greenidge, MD; G.L. Spaeth, MD. One hundred eighteen eyes with primary open-angle glaucoma underwent argon laser trabeculoplasty. Laser burns were placed in the anterior trabecular meshwork (ATM) in 58 eyes and directly over Schlemm's canal in the posterior trabecular meshwork (PTM) in 60 eyes. Twenty-nine eyes received 50 burns distributed equally over 360°. Peripheral anterior synechiae (PAS) developed in seven (12%) of the eyes having anterior trabeculoplasty and in 26 (43%) of those having posterior trabeculoplasty. There was no correlation between the development of PAS and sex, width of the anterior chamber angle, or the number of laser burns. Eyes having anterior trabeculoplasty had a final decrease in intraocular pressure equal to those

having posterior trabeculoplasties. Our results strongly suggest that trabeculoplasty is a safer but no less effective procedure when the laser burns are placed on the ATM rather than the PTM. (Arch Ophthalmol 120:861-863; 1984) *Author's Abstract.*

CORNEAL ULCERS ASSOCIATED WITH CONTACT LENS WEAR. P.G. Galentine, MD; E.J. Cohen, MD; P.R. Laibson, MD; C.P. Adams, MD; R. Michaud, MD; J.J. Arentsen, MD. We reviewed the experience with ulcerative keratitis associated with contact lens wear at Wills Eye Hospital, Philadelphia, from Jan. 1, 1978 through July 1, 1983. Of the 322 cases of ulcerative keratitis, 56 cases (17%) were associated with the use of contact lenses. Twenty-nine (52%) of the 56 cases of contact lens-associated ulcers were culture positive. *Pseudomonas* was the most common isolate, occurring in 13 (23%) of the 56 cases. *Staphylococcus* species were the second most common, occurring in 11 (20%) of the 56 cases. In this series, contact lens-associated ulcers were seen frequently in those wearing soft lenses (48/56 cases or 86%) and in those wearing aphakic lenses (32/56 cases or 57%). Contact lens use is an increasingly important risk factor for the development of corneal ulcers. Prompt, appropriate, and intensive treatment is necessary to prevent visual loss. (Arch Ophthalmol 102:891-894; 1984) *Author's Abstract.*

AN INTERNATIONAL CLASSIFICATION OR RETINOPATHY OF PREMATURITY. The Committee for the Classification of Retinopathy of Prematurity. Because of modern life-support systems capable of keeping tiny premature infants alive, retinopathy of prematurity has recurred. No classification system currently available adequately describes the observations of the disease being made today. A new classification system, the work of 23 ophthalmologists from 11 countries, is presented in an attempt to meet this need. It emphasizes the location and the extent of the disease in the retina as well as its stages. The term "plus" is employed with the stage to denote progressive vascular incompetence. A computer-compatible diagram for recording the results of the examination employing the new classification system is furnished. (Arch Ophthalmol 102:1130-1134; 1984) *Author's Abstract.*

SURGICAL TREATMENTS OF PROLIFERATIVE VITREORETINOPATHY. A.E. Jalkh, MD; M.P. Avila, MD; C.L. Schepens, MD; C. Azzolini, MD; J.E. Duncan, MA; C.L. Trempe, MD. Four hundred ten eyes with retinal detachment and proliferative vitreoretinopathy underwent closed vitrectomy with membrane peeling, scleral buckling, and intraocular air injection. The retina was reattached in 243 eyes (59.3%). Useful vision was attained in 223 eyes. The preoperative proliferative vitreoretinopathy was clinically graded in six groups of increasing severity (C-1, C-2, C-3, D-1, D-2, D-3). From grades C-2 to D-3, a significant trend related a decreased rate of retinal reattachment with increased severity. However, grade C-1 showed a significantly lower success rate than did grade C-2. Scleral buckling alone may be the treatment of choice in grade C-1 eyes. Grade D-3 eyes had the worst anatomic and functional results. Preoperatively, there was a significant relationship between increasing severity of proliferative vitreoretinopathy and frequency of aphakia, and aphakic eyes showed a significantly lower rate of retinal reattachment than did phakic eyes. (Arch Ophthalmol 102:1135-1139; 1984) *Author's Abstract.*

NEURORETINITIS. C.G. Maitland, MD; N.R. Miller, MD. Twelve patients had neuroretinitis characterized by optic disc swelling with marked peripapillary and macular exudates. Individual cases demonstrated bilateral involvement, associated chorioretinitis, and symptoms and signs indicating extraocular CNS involvement. Detailed diagnostic investigation, performed in half of the cases, failed to demonstrate a cause. A temporal relationship to viral disease was present in five of 12 cases, although clinical findings in some cases indicated the condition does not simply represent a monophasic response to viral illness. Regardless of the degree of initial visual impairment or the severity of disc swelling and retinal involvement, ultimate visual outcome was generally excellent, but visual impairment persisted in some patients. (Arch Ophthalmol 102:1146-1150; 1984) *Author's Abstract.*

INCOMITANT VERTICAL STRABISMUS. TREATMENT WITH POSTERIOR FIXATION OF THE INFERIOR RECTUS MUSCLE. R.A. Saunders, MD. Three patients with evidence of inferior rectus muscle paresis were surgically treated for diplopia in downgaze following blowout fracture of the orbit or operative trauma. In each case, surgery involved the placement of two posterior fixation sutures 13 or 14 mm behind the physiologic insertion of the inferior rectus muscle in the contralateral eye. In one case, posterior fixation was combined with a small inferior rectus muscle recession. All three patients experienced relief of their diplopia and improvement in their binocular field of vision. There were no untoward sequelae and no surgically induced changes in primary position alignment. (Arch Ophthalmol 102:1174-1177; 1984) *Author's Abstract.*

DIAGNOSIS OF ALLERGIC CONJUNCTIVITIS. M.H. Friedlaender, MD; M. Okumoto, MA; J. Kelley, MS. Itching was cited as a major symptom of their ocular disease by 49 (80%) of the 61 patients with allergic conjunctivitis. Conjunctival scrapings from 51 patients (84%) demonstrated intact eosinophils or eosinophil granules. Eosinophil granules were a useful and recognizable cytologic feature of allergic conjunctivitis even in the absence of intact eosinophils. We believe that a history of itching and the presence of eosinophils or eosinophil granules in conjunctival scrapings are helpful in diagnosing allergic conjunctivitis. (Arch Ophthalmol 102:1198-1199; 1984) *Author's Abstract.*

COMPLICATIONS OF CONTINUOUS-WEAR SOFT CONTACT LENSES IN A NONREFERRAL POPULATION. T.C. Spoor, MD; W.C. Hartel, MD; P. Wynn, MD; D.K. Spoor, CO/COT. Serious corneal complications occurred in an otherwise successful experience with continuous-wear soft contact lenses (SCLs) for aphakic correction. One hundred twenty eyes were fit, and 92% attained visual acuity of 20/40 or better. Severe corneal complications were observed in 13 eyes, including bacterial ulcers (six), apical erosions (three), and severe superficial vascularization (four). Corneal ulcers occurred in nondiabetic as well as diabetic subjects. Continuous wear SCLs are not innocuous; as for any other drug or device, continuous, long-term medical supervision is necessary to minimize potentially severe complications and visual loss. (Arch Ophthalmol 102:1312-1313: 1984) *Author's Abstract.*

RETROBULBAR ANESTHESIA IN STRABISMUS SURGERY. S.M. Szmyd, MD; L.B. Nelson, MD; J.H. Calhoun, MD; R.D. Harley, MD. Forty-nine patients, ranging in age from 12 to 77 years, underwent strabismus surgery under local anesthesia. With standard preoperative medication and a retrobulbar injection of 2% mepivacaine hydrochloride (hyaluronidase added in 12 patients), effective anesthesia was obtained. Twenty-four patients were observed during the immediate postoperative period, and return of extraocular muscle function and visual acuity was found to be complete an average of 3.8 hours after the injection. The addition of hyaluronidase significantly altered the duration of the anesthetic. Local anesthesia may be a preferable technique in terms of decreased morbidity, especially in the older patient. The short duration of anesthesia and lack of side effects also enable the surgeon to use adjustable sutures and make the final adjustment relatively early in the postoperative period. (Arch Ophthalmol 102:1325-1327; 1984) *Author's Abstract.*

ISOLATED SIXTH-NERVE PALSIES IN YOUNGER ADULTS. M.L. Moster, MD; P.J. Savino, MD; R.C. Sergott, MD; T.M. Bosley, MD; N.J. Schatz, MD. Acquired sixth-nerve palsies are relatively rare in younger adults. We re-examined 49 patients, aged from 15 to 50 years, with isolated sixth-nerve palsies who were seen between 1972 and 1982 at the Wills Eye Hospital in Philadelphia. In this group, the following etiologies were encountered: vasculopathy (14 patients (29%), tumors (eight patients (16%), multiple sclerosis (six patients (12%), presumed inflammation (four patients (8%), trauma (three patients (6%), postlumbar puncture (two patients (4%), and orbital amyloidosis (one patient (2%). Eleven patients (22%) had no determined cause of their sixth-nerve palsy. The implications for the clinical management of isolated sixth-nerve palsies in younger adults are discussed. (Arch Ophthalmol 102:1328-1330; 1984) *Author's Abstract.*

CLASSIFICATION AND INCIDENCE OF SPACE-OCCUPYING LESIONS OF THE ORBIT. A Survey of 645 Biopsies. J.A. Shields, MD; B. Bakewell, MD; J.J. Augsburger, MD; J.C. Flanagan, MD. Six hundred forty-five consecutive biopsies of orbital lesions performed at a major ophthalmic hospital during a 20-year period were used to develop a comprehensive classification of orbital tumors and pseudotumors, excluding thyroid orbitopathy. Although this series has certain bias, it probably closely parallels the incidence of orbital lesions that would prompt a biopsy in an ophthalmic practice. It is hoped that this review, combined with a familiarity of the signs and symptoms of various orbital lesions, will aid the clinician in the diagnostic evaluation of the patient with an orbital mass. (Arch Ophthalmol 102:1606-1611; 1984) *Author's Abstract.*

TREATMENT OF BLEPHAROSPASM WITH BOTULINUM TOXIN. A PRELIMINARY REPORT. B.R. Frueh, MD; D.P. Felt, MD; T.H. Wojno, MD; D.C. Musch, PhD. The effects of botulinum A toxin injections for the treatment of facial spasm were analyzed for 22 patients. Sixteen patients had unoperated on essential blepharospasm, three had essential blepharospasm with residual spasm following previous surgical treatment, and three had unoperated on hemifacial spasm. Treatment was effective for most patients, but transient, with the mean interval of relief of spasm after the first injections being ten weeks. The injection of botulinum toxin reduced the maximum lid force by about 10%. While side effects were common, they were generally mild and well tolerated. No cumulative effect of botulinum toxin was evident in those receiving three series of injections. There is no significant difference in either the interval free of spasm or the rate of lid-force recovery following any of three sequential injections of increasing doses of botulinum toxin. (Arch Ophthalmol 102:1464-1468; 1984) *Author's Abstract.*

CLINICAL CLASSIFICATION OF GRAVES' OPHTHALMOPATHY. IDENTIFICATION OF RISK FACTORS FOR OPTIC NEUROPATHY. S.E. Feldon, MD; S. Muramatsu, MS; J. M. Weiner, DPH. Clinical signs of Graves' ophthalmopathy were correlated with extraocular muscle volumes in 50 patients. Significant correlations were obtained for horizontal, vertical, and total extraocular muscle limitation, as well as periorbital swelling. Proptosis correlated to a lesser extent. Optic nerve involvement was found to be correlated with both total extraocular muscle volume and limitation of ocular motility. From this information, no risk, future risk, and immediate risk categories of disease were defined quantitatively. The results of the study imply that optic nerve involvement is more likely to occur in association with noncompliant, fibrotic muscle than with more supple muscle of the same total volume. (Arch Ophthalmol 102:1469-1472; 1984) *Author's Abstract.*

IN THE NAME OF ALLAH, THE BENEFICENT, THE MERCIFUL

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