DOS TIMES

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for
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NOTICE

ANNUAL GENERAL BODY MEETING

The Annual General Body Meeting of Delhi Ophthalmological Society will be held on **Saturday the 28th August 2004 at 01.00 P.M.** at Sir Ganga Ram Hospital, Rajender Nagar, New Delhi.

All members are kindly requested to make it convenient to attend.

Dr. Jeewan S. Titiyal
Secretary, DOS

EDITORIAL

Dear Colleagues,

I welcome you all to the new academic year of DOS Times. Last year saw a lot of effort being taken to change the format and to improve the style & substance of the contents of DOS Times.

We also thank all of you for your invaluable suggestions and feedback which we honestly tried to incorporate in the subsequent issues. This year too, we invite from all of you, not only suggestions to improve DOS Times but also invite readers to contribute in the form of articles, practical tips, modifications of techniques, appliances and letters to editor which I am sure would be of immense help to all our readers. There are many senior members in our DOS fraternity who have vast experiences and I appeal to them the most, to share their views so as to benefit our young readers.

The major bulk of an ophthalmologist is cataract related and today one cannot imagine of performing a cataract surgery without implanting an IOL. Every launch of a new IOL is treated with interest in the ophthalmic fraternity. We all marvel at the long and interesting journey of this implant with its twists and turns. So DOS Times feels it is a high time to stop for a while and salute the person who invented and first implanted this lens in a human eye – Sir Harold Ridley.

We dedicate this first issue to Ridley and to his contributions to ophthalmology.

Thanks

Dr. Jeewan S. Titiyal
Secretary, DOS

Programme for DOS Monthly Clinical Meeting for July 2004

Venue: Army Hospital (Research & Referral), Near Dhaula Kuan, (on NH-8), Delhi Cantt-110010
Date & Time : 1st August, 2004 (Sunday) at 10.00 A.M.

Case Presentation

1. A case of nodular scleritis .....Lt. Col. Dr. R. Maggon (10 min)
2. A case of sterile corneal melting .....Lt. Col. Dr. (Mrs.) M. Bhadauria (10 min)

Clinical Talk

- Clinical profile of retinal vasculitis in serving soldiers ..... Col. Dr. Ajay Banerjee (20 min)

Mini Symposium: Infectious Keratitis

Chairman : Brig. Dr. Suresh Chandra
Co-Chairman : Col. Dr. D.P. Vats, S.M., VSM

1. Management of Bacterial Keratitis
2. Management of Fungal and Acanthamoeba Keratitis
3. Management of Viral Keratitis
4. Surgical Management of Infectious Keratitis

Panel Discussions : **20 min.**

Letter to Editor

Dear Dr. Titiyal,

My greetings to you. I wish to inform that my article on "Simplified Pterygium surgery with no Recurrence" has been published in Ocular Surgery News in Vol. 22., No. 8 April, 15, 2004. I have been doing this technique, which is my own innovation since 3 decades with no recurrence of both primary and recurrent pterygium. I wish to share this technique with the DOS Times Readers.

Technique

After surface anesthesia and placing of the speculum, 0.2 cc of xylocaine is injected under the body of the pterygium to facilitate dissection. The apex of the pterygium is dissected out carefully and is lifted upward. The assistant holds its tip, and the surgeon dissects the subepithelial tissue of the pterygium towards the base.

All attachments of this tissue are freed from all sides. Then it is slightly pulled to the base of this tissue. It is cut off just above the artery forceps.

The cut end is held by the forceps is cauterized and released. This tissue, because of its elasticity, recedes back deep into the orbit, from which there is no chance of its growing forward. The apex of the pterygium is cut, and the upper and lower edges of the conjunctiva are closed by placing two sutures, leaving a small bare area of sclera.

The idea is that by the time the denuded cornea is covered by epithelium, the conjunctiva will grow to the limbus. Mitomycin-C is available in India as a 2 mg vial of powder. We add 5 cc of water to this to make an eye drop. One drop is placed daily 3 times a day starting 48 hours postoperatively. On the operating table, antibiotic ointment is applied and the eye is bandaged. After 24 hours the eye is left open, and any steroid-antibiotic combination eye drop is prescribed three times a day for 1 week.

In the case of a recurrent pterygium, recurrent pterygium, the same procedure is performed, but the subconjunctival tissue does not have as much elasticity as in a primary surgery. Hence the tissue is pushed backwards as much as possible.

In the past 32 years I have done a large number of primary pterygium surgeries using this technique with no recurrence. I have also done the technique in 21 cases of recurrent pterygium, and none of them recurred.

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New Delhi – 110048

Where is my copy of DOS Times ?

Dear DOS members, anyone who could not receive
DOS Times from the month of July, 2004 onwards.

Please Contact:

President DOS : **Dr. GURBAX SINGH**
Email : dospresident@yahoo.com

or

Secretary DOS : **Dr. JEEWAN S. TITIYAL**
Email : dosonlin@vsnl.net

!! Attention DOS Members !!

Applications are invited for DOS Fellowship for Partial Financial Assistance to Attend Conference(s). The last date for receiving application is ***31st July, 2004***.

For details please see page no. 20

Sir Nicholas Harold Ridley : The Inventor of the Implant and a Pioneer in the Quest to Eradicate World Blindness

Suresh K. Pandey, MD[1,2]
David J. Apple, MD[3]

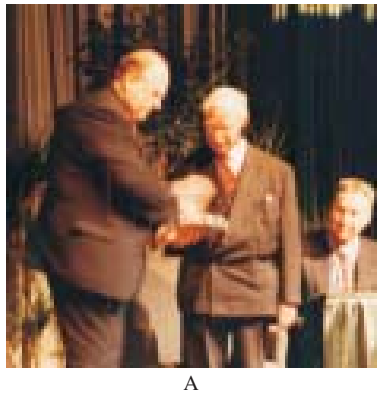
Sir Harold Ridley (Fig. 1)

Sir Harold Ridley, the inventor of IOL, passed away at the age of 94, on 25 May 2001, and ophthalmology lost one of its greatest and most influential practitioners (Fig. 1). We are happy that he lived to enjoy the fruits of his labour to see the amazing improvements and the expansive growth that evolved in the cataract-IOL technique, from early and unsatisfactory operations in previous decades, to the superb results attainable today. This article presents a brief biographical sketch of Sir Harold Ridley and lists his major inventions and contributions to ophthalmology.

Sir Harold Ridley: Education & Academic Recognition

Nicholas Harold Lloyd Ridley, MA, MD, Cantab. (Cambridge); FRCS, England; D.H.L. Medical University of South Carolina, Charleston; D.S. City University of London; Fellow of the Royal Society (FRS), was born at Kibworth, Leicestershire, July 10, 1906. After completing studies at Cambridge in 1927, he proceeded with medical training at St. Thomas' Hospital, London, and in 1930, he completed his basic medical education. In 1938, he was appointed full surgeon and permanent consultant at Moorfields Eye Hospital. Ridley married Elisabeth Wetherill in Surrey on May 10, 1941, and soon thereafter he entered the Royal Army Medical Corps.

Sir Harold Ridley was elected a Fellow of the Royal Society of London in 1986. His first academic honor was an honorary doctorate degree, Doctor of Humane Letters (DHL), conferred in 1989 by the Medical University of South Carolina, Charleston. In 1992, he received the Gullstrand Medal (conferred by the Swedish Society of Medicine), and in 1994, he received the Gonin Medal (conferred by the Club Jules Gonin, Lusanne). The matter came full circle July of 1997, when Sir Harold Ridley was honored by the delegates of the Oxford Ophthalmological Congress—the venue of his first presentation of the IOL. In April 1999, at the annual meeting of the American Society of Cataract and Refractive Surgery in Seattle, Washington, Sir Ridley was honored in a special anniversary session as one of the most outstanding and influential ophthalmologists of the 20th century (Fig. 2). At that meeting, he also received a medal from Rayner, Ltd., acknowledging their collaboration with Sir Ridley on his original lens, a most outstanding advance in the field of cataract surgery (Figure 3). He received similar accolades at the 1999 meetings of the European Society of Ophthalmology (Stockholm, July 1999) and the annual meeting of the European Society of Cataract and Refractive Surgery (Vienna, September 1999). In



A

B

C

Fig. 2: Sir Harold Ridley was awarded for being one of the most influential ophthalmologists of 20th century. This event commemorated the 50 th Anniversary of the IOL.

A. Sir Harold Ridley receiving award during the American Society of Cataract and Refractive Surgery in Seattle, Washington, April 1999.

B. Sir Harold Ridley with authors of his biography during the American Society of Cataract and Refractive Surgery in Seattle, Washington, April 1999. (Left to right- Drs. Jim Sims, MD, Sir Harold Ridley, David Apple, & Mrs. Elisabeth Ridley).

C. Sir Harold Ridley with some young ophthalmologists during the American Society of Cataract and Refractive Surgery in Seattle, Washington, April 1999. (Left to Right- Drs. Qun Peng, Elisabeth Ridley, Suresh K. Pandey, Sir Harold Ridley, Marcella Escobar Gomez, Nithi Visessook, Liliana Werner).

[1]John A. Moran Eye Center, Department of Ophthalmology and Visual Sciences, University of Utah, 50 North Medical Drive Salt Lake City, Utah-84132, USA; [2]Intraocular implant Unit, Sydney Hospital and Sydney Eye Hospital, Macquarie Street, Sydney, Australia; [3]Save Sight Institute, The University of Sydney, Sydney, NSW, Australia.

February 2000, knighthood was conferred on him by Queen Elizabeth II (Figure 4). These honours finally helped erase what had indeed been a very difficult memories for him, for most of his professional life. Prior to these honours and recognitions, Ridley was indeed a classic example of a "prophet -without-honour" in his own country.

At 1999 American Society of Cataract and Refractive Surgery meeting, Sir Harold Ridley also received a medal from Rayner, Ltd., acknowledging their collaboration with Ridley on his original lens, a most outstanding advance in the field of cataract surgery.

Fig. 4: Sir Harold Ridley was conferred the knighthood in his homeland by Queen Elizabeth II, in February 2000.

Sir Harold Ridley: Invention, Reaction and Recognition of the Implant

The story of Sir Ridley and his ground-breaking invention of the intraocular lens is well known and became one of the high points of our specialty in the 20th century. During the new millennium, we in ophthalmology and the visual sciences have recently celebrated the 50th anniversary of one the 20th century's most important innovations in eye care—the invention of the intraocular lens (IOL) by Sir Harold Ridley. The 50th anniversary of this invention literally straddled the turn of the century. The first operation, done at St. Thomas' Hospital, London on November 29, 1949. The pseudophakos was manufactured by Rayner, Ltd., United Kingdom.

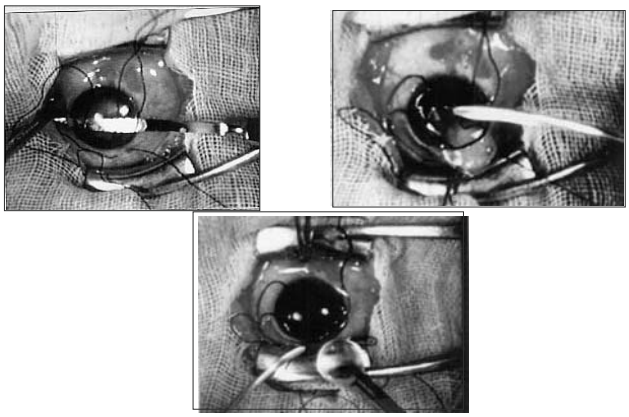


Fig. 5: Photographs of Sir Harold Ridley's eighth implant operation, May 10, 1951. These cuts were taken from the original film.

A. von Graefe incision.

B. Crystalline lens removal.

C. Intraocular lens insertion.

Sir Ridley's first cataract extraction and IOL operation marked the beginning of a major change in the practice of ophthalmology. The idea of an artificial lens had been mentioned earlier (including mostly apocryphal sources), but credit for the invention and implantation clearly goes to Sir Ridley. His collaboration with John Pike at Rayner, Ltd., the manufacturer of the first lenses, began a new era. He filmed some of his early cases, including case number 8, segments of which are shown here (Figure 5).

Figure 7.

A B C

Sir Ridley's first presentation of the new procedure was given at the Oxford Ophthalmological Congress on July 9, 1951. His presentation of two patients with successful implants evoked much interest, but also marked resistance. Although Sir Ridley had a handful of early supporters throughout the world, including David Peter Choyce (a participant in the early operations), he had numerous detractors. Some authorities in the 1950s and 60s had a conservative attitude toward this procedure, as there was little available experimental or animal data and little analysis of material in those early years. Some surgeons referred to the IOL as a "time bomb." Governmental funding sources showed little interest in the device. Even as the success of the IOL became assured, doubts and questions still lingered—which, in retrospect, were probably healthy, as they helped stimulate more basic research into the procedure.

Young residents and practitioners today implant IOLs routinely and with ease, not realizing the extraordinary struggle that Ridley and many other contemporary surgeons, who implated Ridley IOL had to undergo for many years to develop this

A.

B.

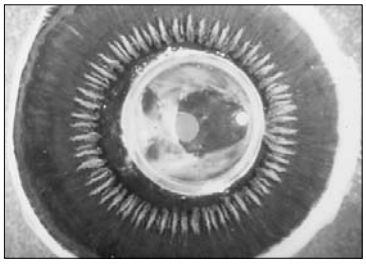


Fig. 6: Miyake-Apple view of a Ridley intraocular lens implant taken 30 years after surgery.

A. IOL decentration.

B. Capsular bag opacification.

technique. A prime example is the issue of lens fixation, which was one of Sir Ridley's biggest problems and has taken over 40 years to solve (Figure 6A). A solution was not achieved until the mid-late 1980s, when surgeons began to understand the advantages of in-the-bag (capsular) fixation of posterior chamber IOLs. Posterior capsule opacification (PCO, secondary cataract), another complication seen in Ridley's earliest procedures, is now being controlled, and its incidence has decreased to single digit percentages (Figure 6B).

Sir Harold Ridley: Other Inventions:

Sir Harold Ridley was sent to Ghana in the Gold Coast of West Africa, a challenging assignment that turned out to be a blessing in disguise. It was during this period that he performed his original work in the field of tropical eye disease, especially onchocerciasis. His monograph, *Ocular Onchocerciasis*, with his classic fundus painting, sometimes termed the *Ridley fundus,* was published in 1945. This work constitutes one of Ridley's major contributions.

In addition to his contributions to tropical medicine and enhancing eye care worldwide, and prior to his invention of the intraocular lens, Sir Ridley showed creativity and innovation in several other areas. He was the first to televise eye operations, and he devised a system of examining the inner eye by electronic methods. It has only recently been appreciated that Ridley developed many of the basic principles of what has evolved into modern confocal microscopy and scanning laser ophthalmoscopy (SLO).

Sir Harold Ridley & IOL Implantation in Developing World:

Forty million people in developing countries are functionally blind over half of them from cataract. Cataract removal and IOL implantation is by far the most common and most successful of all operations in medicine. We are pleased that Sir Harold Ridley lived to see the amazing improvements and the expansive growth that has evolved in the cataract-IOL technique, from early and unsatisfactory operations in previous decades, to the superb results attainable today. Our challenge is to *"cure aphakia"* on a global basis; and we are pleased that concerted efforts now are being made toward achieving widespread "pseudophakia" in the developing world. The advantages of an IOL implant over aphakic spectacles have also been confirmed in recent studies done in leading ophthalmic centers in developing countries.

Without exaggeration and there are very few doubters today Sir Ridley's IOL invention has both directly and indirectly led to visual restoration and cure for multi-millions of visually handicapped people worldwide, a huge step towards the eradication of cataract blindness. Because of Sir Harold's early work, implantation of IOLs on a broad scale in the rural areas of these countries is now a definite possibility. It would be a spectacular legacy to Sir Harold Ridley - *whose heart and intellect was strongly devoted to the problems of tropical medicine and blindness in the developing world* - to accelerate and expand efforts to provide the benefits of IOLs to needy individuals throughout the world.

In summary, as we say good-bye to Sir Harold Ridley, we hope that all of us who have shared the experiences of relatives or friends (even ourselves!) regarding IOL implants after cataract surgery, will reflect back in history on his *tiny piece of plastic* and be grateful for his sight-saving invention. The year 1949 began a new era, the formative years of the IOL. The intermediate years represented the period of growth of the cataract-IOL procedure. His death came at a time of near perfection of his invention, indeed during a period of maturation of not only IOLs, but also of other hi-tech implantable biodevices, many of which he pioneered. The second-half of the 20th century has truly benefited from Sir Ridley's contributions, and we are likely to continue deriving benefits from his work.

Suggested Reading

1. Ridley NHL. Intraocular acrylic lenses. *Trans Ophthalmol Soc UK & Oxford Ophthalmol Congress*. 1951;LXXI:617-21.
2. Ridley NHL. Intraocular acrylic lenses after cataract extraction. *Lancet*. 1952;1:118-19.
3. Apple DJ, Sims J. Harold Ridley and the invention of the intraocular lens. *Surv Ophthalmol*. 1996;40:279-92.

Dry Eye Disease

Gurbax Singh Bhinder, M.D.; M.S.
Hanspal Singh Bhinder, M.B.B.S.

Dry eye disease is a chronic inflammatory condition of the eye in which the precorneal film gets altered in function due to dysfunction of tear volume or tear quality alone or both leading to a complex symptomatology. The incidence varies from 8% to 30.5% in different countries.

Classification: There are many types of classifications:

(a) National Eye Institute of America: They classify it in two basic categories

a. Decreased aqueous production
   i. Sjogren's syndrome
   ii. Non-Sjogren's syndrome
b. Increased evaporation of tears
   i. Inflammatory (meibomian gland disease)
   ii. Atrophic meibomian gland disease

(b) Dohlmann classification (1972): It depends upon the deficiency of the precorneal film layers

i. Fluid deficiency: There is decrease in production of tears by the lacrimal gland.

Causes:

(a) Sjogren's syndrome

(b) Collagen diseased (S.L.E., Riley and Day syndrome)

There is usually increased lymphocytic infiltration of the lacrimal gland tissue leading to decrease in tear production.

ii. Mucus deficiency

- Steven's Johnson syndrome
- Ocular pemphigus
- Avitaminosis A.

iii. Altered corneal surface: It is seen in trachoma, herpes simplex, corneal dystrophies. The elevated spots become vulnerable to infection or ulceration.

iv. Insufficient spread of tears:

- Neuroparalytic keratitis
- Dallen

(c) Clinically based on severity of symptoms

a. Mild 1 – 2 symptoms

b. Moderate 3 – 5 symptoms

c. Severe >5 symptoms

(d) Murube & Rivas classification (2003)

a. Grade-0 = Normal

b. Grade-1 = Occasional symptoms no signs

c. Grade-2 = More often symptoms no signs

d. Grade-3 = Symptoms present in daily life and signs also present

e. Grade-4 = Symptoms present always. Signs present

f. Grade-5 = Symptoms and signs in form of scarring, vascularization of cornea

(e) Tsubota et al classifications

a. Non-immune (no antibody in serum)

b. Immune antibodies in serum detected (Sjogren's syndrome)

(f) Based on Sicca score

a. Grade-0 = Normal

b. Grade-1 = Symptoms present sometimes

c. Grade-2 = Symptoms present half of the time

d. Grade-3 = Symptoms present mostly

e. Grade-4 = Symptoms present always

	Epithelial cells	Nuclear to cytopl ratio	Goblet cells	Cytoplasmic Cells
Grade-0	Small round	1:2	Abnormal	Eosinophilic
Grade-1	Larger	1:3	-do-	-do-
Grade-2	Larger	1:4 (Multi nuclear)	?	-do-
Grade-3	Larger polygonal	1:6 or 7	Absent	Basophilic

Guru Gobind Singh International Eye Research and Cure Centre
31, Defence Enclave, Vikas Marg, New Delhi - 110 092 India

(g) Based on conjunctival cytology (Nelson)

Aetiology of Dry eyes

Exact causes are not known? Certain factors are blamed: -

Idiopathic: Oestrogen deficiency as in post menopausal; Rheumatoid arthritis; Ectodermal dysplasia & Androgen deficiency.

Metabolic: Diabetes mellitus; Hypothyroidism; Gout; Anaemia & Hypercholesterolemia.

Drugs:

- **General:** Imprepramine; Promazine; Benzylhexol & Dicyclomine
- **Local:** Beta blockers & Preservatives in drugs

Collagen disease: Sjogren's syndrome; Rheumatoid; Pemphigus & Steven's Johnson syndrome.

Others: Chemical burns; Rosacea blepharo-conjunctivitis; Dacryoadenitis; Post cataract surgery & Post Lasik

Local factors: Contact lens wearers; Lasik surgery; Local medication & Preservatives of local medicines

Environmental Factors: Low Humidity; Pollution; Immune influence; Allergy & Stress

Clinical Picture

It depends upon the stage of dry eye. It varies from no symptoms to groups of severe symptoms which are counted 13 in number: - gritty sensation or irritation[1], itching[2], redness[3], pain[4], photophobia[5], tearing[6], frequent blinking[7], stickiness[8], photophobia[9], dryness of eyes[10], fluctuating vision[11], tiredness[12] and discharge[13].

Clinical signs:

(a) Chronic papillary conjunctivitis

(b) Chronic meibomitis

(c) Blepharitis

(d) Debris in the tear film as seen on slit lamp as oily particles, mucus blobs, sheet or strands.

(e) Presence of LIPCOF (Lid Parallel Conjunctival Fold): This is visible as a raised bulbar conjunctiva along with the posterior lower lid margin (Fig-1 & 2). It is seen first on the outer ¼ of lid length clinically it is divided into four grades (Grade-1=Outer ¼ of lid length; Grade-2 = Half of the lid length; Grade-3= ¾ of lid length and 4/4 of the entire lid length). It becomes prominent with Lissamine green stain and disappears with fluorescein stain.

(f) Tear marginal meniscus: The height and breadth of the meniscus gets decreased in dry eye disease.

(g) Meibomian gland health

a. Gland orifice metaplasia: If a white shaft is protruding from any meibomian gland, it shows abnormal growth and keratinisation of the ductal epithelium. It is seen in atrophic meibomian or inflammatory condition of lid.

b. Meibomian gland expression test: Hold two lids close to each other drawn in front of the eye ball and try to rub together in an area of five glands. The inference derived:

i. Grade-0	Expression of five glands
ii. Grade-1	Four glands expressed
iii. Grade-2	Three glands expressed
iv. Grade-3	Two glands expressed
v. Grade-4	No glands expressed

c. Trans illumination of inferior tarsus: If there is high percentage (67% - 100%) of acinar dropout, it shows the patient has meibomian gland disease.

Diagnostic tests

Schirmer-1 test: This is carried out with Whartmann-41 paper 5mm x 35mm whose end is bent to adjust on the lid. It is placed between inner 2/3rd and outer 1/3rd lid for 5 minutes. The room should have controlled humidity and temperature. In original test, the eye was kept open. However it can be kept closed. The inferences drawn from wetting of filter paper are:

- If wetting < 3mm = V. Severe dry eyes
- If wetting 3 – 5mm = Severe dry eyes
- If wetting 5 – 10mm = Moderate dry eyes
- If wetting is 10mm = Mild dry eyes
- If wetting is >10mm = Normal

Note: This test is crude, unreliable and pseudo positive, type of paper and environmental factors affect the results.

Schirmer-2 test: If the above test reveals <10mm wetting, irritate the nasal mucosa with a cotton bud and note the additional wetting. If no wetting or <1mm then it is Sjogren's syndrome. If wetting increases by 1 mm it is non-Sjogren's syndrome.

Schirmer-3 test: To note wetting of the filter paper after local anaesthesia of conjunctival sac. This is carried out to note reading of basal tear function.

FTBUT (Fluorescein Stain test): Touch the temporal conjunctiva with a wet fluorescein dipped filter paper and asks the patient to blink few time and see under slit lamp using a cobalt blue filter with 10x magnification

Normal = >10 seconds
Grade-1 = =10 seconds
Grade-2 = 5 – 10 seconds
Grade-3 = 3 – 5 seconds
Grade-4 = < 3 seconds

Note: It is good test but abnormal values have been noted in normal eyes.

Fluorescein stain test: Examine the conjunctiva and cornea for staining under blue filter.

- No staining = Grade-0
- 1/3 = Grade-1
- 2/3 = Grade-2
- 3/3 = Grade-3

Lissamine green stain or Rose Bengal test: It stains the cells which are not covered by albumin or tears. It does not stain dead cells as already reported–. A wet strip of filter paper dipped in Lissamine green stain is touched to the lower tarsal conjunctiva. The patients are asked to blink few times. The slit lamp examination with 10x magnification using a yellow filter is carried out between 30 seconds to 2 minutes of the installation of dye and observation noted. It is graded on Oxford score. Each quadrant is graded from 0 – 4 and the total score is averaged (0= No stain; 1= Mild staining dots; 2=Staining dots multiple; 3=Confined staining area; 4=Very big patches of staining). If cornea is stained, it is further graded into 0 – 4 scores as in conjunctiva.

Note: Even staining is present in normal people ranging in 48% - 90% of the cases hence we cannot attach much value to this dye test.

Phenol Red Impregnated thread (Yokoi et al): This is a type of cotton thread Schirmer test. The thread end is put in the lower fornix and readings are taken after two minutes (120 seconds). The wetting of thread is noted:

Normal = >15mm wetting (15 – 24mm)

Abnormal = <15mm wetting – Aqueous deficiency

Slit lamp fluorophotometry: Three micro litre of 0.5% fluorescein solution was applied to cornea in untouched fashion. The ocular surface is washed after 10 minutes. After 20 minutes, corneal fluorescein is measured and converted into fluorescein conc. or matched with standard fluorescein solution. Interpretation: -

Grade-0 = No superficial punctum corneal stain

Grade-1 = No severe SPK at center of cornea

Grade-2 = Mild SPK at center of cornea

Grade-3 = Severe SPK at center of cornea

Fluorescein uptake: -

Normal = 22.4±16.9ng/ml

Grade-1 = 96.4±51.2ng/ml

Grade-2 = 318.6±146.0ng/ml

Grade-3 = 1479.1±671.9ng/ml

Meibometry: It is for noting the mucus gland dysfunction. It is of two types:

(1) Direct meibometry: Lipid impressions are taken on a plastic paper and its lipid contents are read on a meibometer.

(2) Integrated meibometry: It is done by taking scans of impregnated lower meniscus and measuring densometry on computer.

Direct tests:

- Normal = 268.5±6.3
- Incomplete dysfunction = 248.6±13.2
- Aqueous deficiency = 306.4±9.2
- Mucus gland dysfunction = 127.24±24.4

Tear Meniscus Measurement: Put a small drop of fluorescein in inferior tear meniscus. Examination on slit lamp with 12x magnification and then scanning the developed images into a computer programmer.

	TM Curvature	TM Height	TM Width
Normal	0.54±0.25mm	0.461±0.173mm	0.0176±0.013mm
Dry eyes	0.314±0.16mm	0.244±0.089mm	0.0082±0.0048mm

Significance: It may keep false positive in hypersecretion stage of dry eyes.

Ocular protection index: It is TBUT time in second divided by interblink interval in seconds.

TBUT (Time in seconds)

Blink interval in seconds

Inference:

OPI <1 = Patient at risk

OPI>1 = Not at risk

Lacunae: The inter blink time can only be accurately measured with a camera interfaced with a computer.

The subjective facial expression rating scales: It was used by Berry 1990. It consists of a facial schematic photographs from 1 (happy face) to 9 (unhappy). It is analysed in five grades from pictures.

Tear Osmolarity: It is measured by taking 0.24 micro litres of tears and measuring the freezing point of dispersion.

Normal = 302Mos±6.3/litre

Dry eye = >350 Mos/litre

Closed chamber infrared thermometry: It is very useful for quick diagnosis for dry eyes–.

Method: An infrared thermometry and a closed chamber is devised to measure temperature on a fixed point and fixed distance from the eye. The temperature is recorded with eye closed and then after opening the eye after five seconds.

Normal: The temperature increased by 0.1^0C after opening the eye.

Dry eye: No increase in temperature after opening the eyes.

Closed Chamber humidity of the eye: Closed chamber is applied to the eye. The humidity is measured with eye closed and then 5 seconds after opening of the eye. The difference in humidity in:

Normal = <1RH%

Dry eye = >1RH% (1RH% to 4RH%)

It is most reliable test, non-invasive, quick test for early diagnosis of dry eyes even when other tests declare normal values.

Tearoscope: This is used to see the tear film as such or an attachment to the slit lamp.

Confocal microscopy: This is used to visualise the tear film, status of epithelium in detail.

Lab Diagnosis

(1) Impression cytology: It has a great confirmatory value.

(2) CA 19-9 Elisa test: It expression as KU (kilo unit)

Normal=39.4 Ku ±22.21/igm (>17.17Ku)

Dry eyes= ≤25.8 Ku ±17.3/igm (<17.17Ku)

(3) Estimating of pro-inflammatory form of interleukin 1.4m in tear fluid and conjunctiva of dry eye patients.

- IL_1 alpha ? in tears
- Mature - IL_1 Beta ? in tears
- IL_1 – Beta -? in MGD

(4) Ocular ferning test: Install a drop of propazacaine hydrochloride into conjunctival sac. The palpebral conjunctiva of lower nasal is scrapped with a platinum spatula. The specimen is put on the clear glass slide and allowed to dry. After drying the slide is evaluated with the microscope (63x).

- **No Ferning:**
  - o Pemphigus
  - o Stevenson Johnson syndrome
- **Non-dry eye:** Ferning is present in 91%.

Dry eye Diagnosis at G.G.S.I. Eye Research & Cure Centre

(1) History

(2) Thermometry (Closed chamber): If there is no difference in temperature in closed and open eye position, it is dry eye. These cases are investigated further.

(3) Humidity (Closed chamber)

(4) Schirmer-1 test

(5) Slit lamp examination

a. LIPCOF

b. Meibomian gland

(6) FTBUT

(7) Fluorescein stain test

(8) Lissamine Green test

Management of dry eye

(A) Try to find out trigger factor and remove it.

a. Avoid work in dry and hot places

b. Contact lens wearers should use wetting solutions as a mandatory requirement.

c. Correct systemic disease likes diabetes, hypothyroidism.

d. Use eye drops mims without any preservative

(i) Profession

(A) Detail of place where working especially stress on humidity and temperature at that place

(1) Some workers were working under low tin sheets without any cooling device and all of them were developing dry eye a different stages.

(2) Computer operation.

(3) Watching TV for long periods.

(4) Less sleep at night or day as in students.

(5) Playing long hours or working long hours in sun light.

(6) Contractor working in dust environments.

(7) Electronic engineers used to different waves

(B) <u>Ocular disease treatment</u>

(i) Glaucoma B-blockers gives dry eye

(ii) Local anaesthetic eye produce dry eye

(iii) Long use of cortico steroids causes dry eyes.

(iv) Preservative in local eye drop produces dry eyes (Banzalkonium chloride).

(v) Lid abnormalities – Ectropion coloboma or lid.

(vi) Local scarring producing diseases: Trachoma; Pemphigus; Steven Sjogren syndrome & Acid alkali burns.

(C) Systemic drugs: They produce dry eye

(1) Anti-Rheumatoid drugs

(2) Anti-hypertension drugs

(3) A diuretics

(4) Anti inflammatory drugs non-steroids

(5) Rheumatoid artheritis

(6) Diabetes mellitus

TREATMENT

Removal or correction of the triggering factor and the main aim of the therapy is to correct the basic disease, which is producing dry eye, and at the same time provides artificial supplements and measures to reduce evaporation of tears. I will outline the management as follow: -

(A) Medical

(B) Surgical

(A) General to improve the lacrimal gland secretions if they are affected: How will you know that lacrimal gland is producing enough tears or not. It is easy to find out by Schirmer-1 test and two. If there is <1mm of wetting in Schirmer-2, it is Sjogren syndrome or secretion deficiency. If wetting difference in Schirmer 1 & 2 is >1mm, it rules out the lacrimal gland (Sjogren disease).

(B) Surgical

a. Amniotic membrane transplantation

b. Auto conjunctiva

c. Rectal mucosa

d. Lips mucosa

e. Frontal sinus drainage

f. Parotid duct transplantation (stenson duct transplantation)

(C) (Foetal tissue or placental tissue or amniotic fluid or lacrimal gland area transplantation.

It is preferable to classify it into following headings

(A) Medical

1. To increase and correct the production of tears at lacrimal gland level

a. Oral cyclophosphide: - It is given in a dose of 75mg/ daily. It corrects the autoimmune response in the cellar level of the lacrimal gland. It has been used by with good result.

b. Zidovudine: - Dose 250mg/twice a day. A significant improvement seen with its use.

c. Pilocarpine hydrochloride tab (Salagen or Pilomax): - It has its action on the lacrimal gland. It is given as 5mg Tab four times a day for 12 weeks and more. In 60% of the case good response is seen on first dose. Side effect of drug is (diaphoresis) excessive sweating. Vivino et al; Papas et al and Takya et al have used it.

d. Oral antioxidants: - They have been tried by (Blades et al 2001) who noted change in number and morphology of goblet cells by impression cytology and improvement in tear quality.

e. Effect of acupuncture: - It is effective only in functional disorders which include toxic, allergic, drug induced and ocular surface inflammation. However in Sjogren syndrome it is ineffective: -

Method: - 10 session per week of ½ hour each.

(Nepp et al 199 used this in 102 patients and noted good reality in younger patients)

f. Laser and short wave diathermy stimulation of lacrimal gland. (Kecik et al 1994)

g. Androgens (They too improve the dry eye disease):

i. Systemic

2. Local immunosuppressive agents

(A) Cyclosporin: - It is used in a conc of 0.05% to 0.1% two times a day and has been seen efficacious in correcting the dry eye syndrome. (Sall K et al 2000; Stevenson et al 2000) However it is used 0.05%, 0.1%, 0.2% or 0.4% solution twice daily for 12 weeks followed by four weeks of post observation period. They noted that 0.05% or 0.1% solution produced the most consistent improvement in objective and subjective points (Rober et al 2001) noted good results. Cyclosporin allows local immuno-regulation without systemic side effects. Gao et al 1998 noted that the cyclosporin

a. Facilitate epithelial cell apoptosis

b. Suppression of lymphocytic apoptosis in the lacrimal gland and ocular surface tissues

c. Kunner et al 2000 too observed reduction in number of activated lymphocytes within the conjunctiva.

d. It modulates the goblet cell function in secondary Sjogren.

e. It decreases level of cytokine 2 in the conjunctival epithelium.

(B) Autologous Serum (Tsubota et al; BJO 1999; 83; 390-5)

Mechanism: -

(i) Autologous serum provides essential components to maintain ocular surface (Wilson et al 1986; Pales et al 1986; Van Setten et al 1989; Van et al 1992).

(ii) EGD (Epidermal growth factor) Vit. A and various cytokines, fibronectin, TGFB (Transforming growth factor beta) preserved in serum up to 1 month at 4^0C and 3 months at 20^0C.

Method of preparation: -

(a) Take 40ml blood

(b) Centrifuge at 1500rmp for 5 minutes

(c) Serum so obtained was diluted by saline to 20%.

(d) Placed in 5ml bottle

(e) Store in freezer until required

The serum was instilled in the eye 6-10 times a day along with hyaluronic acid and artificial drops 4 times a day.

Mechanism: -

(1) EGI is effective for acceleration of corneal epithelial profilteration (0.7-8.1mg/ml)

(2) Increased mucin expression

(3) Retinol in human tears as 0.4 – 10.6mg/ml

(4) TGFB

i. In tears 10mg/ml

ii. In serum 50mg/ml

1. Anti-proliferative

2. Suppress wound

(5) Serum is self-sterilizing as it has antibacterial agents such 1gg, lysozine and complement

(6) It improves cytologic changes in severe dry eyes (Tananuvat et al 2001)

Note: Auto serum is really useful in our study.

(C) Long term treatment with sodium hyaluronate containing artificial tears used by Aragona et al (BJO 2002);

DOSE: 0.14% - 0.1%

MECHANISM:

(1) Reduction in cell degeneration (Cordon et al, BJO 1999)

(2) Protects the corneal epithelium (Wysnback et al, Invest Ophth. 1988)

(3) It does not change degree of cell metaplasia (Nelson 1988)

(4) It promotes cell migration

(5) It stabilizes ocular surface epithelial barrier (Inque et al 1993 and Nishida et al 1991).

(6) Activation the CD_{44} receptors which are present in conjunctiva and cornea (Lerner et al 1998; Baudouin et al 2001).

(7) Binding of hyaluronate to CD_{44} may stimulate cell proliferation through a mechanism involving a kinase cascade (Rosales et al 1995).

(8) Hyaluronate may play a part in controlling a localised inflammation (Stern et al 1998).

The use of sodium hyaluronate has been recommended by Shimmura et al 1993; Hamano et al 1996; Hamano et al 1996; Papas et al 2001; Nepp et al 2001; Solomon & Merin 1998; Argona et al 2002; Debbasch et al 2002; Cordon et al 1999; Yokoi et al 1997.

Note: - We too noted it very useful in our hands

(D) Trehalose:

DOSE: 50, 100 200mm trehalose solution

MECHANISM: It is a disaccharide, a key element for anhydrobiosis.

(1) It prevents corneal epithelial death from desiccation.

(2) It stabilizes lipids and proteins of the cell membrane in the condition of desiccation (Crowe et al 1984; Crowe et al 1998 & Allison et al 1999).

(E) Castor oil eye drops for non-inflammatory obstructive gland dysfunction

DOSE: 2% Castro oil and 5% polyoxyethylene emulsifier

SOURCE: Seeds of ricinus communis LINN

MECHANISM:

(1) Significant improvement in TBUT

(2) Lipid spreading

(3) Ease of Meibomian gland expression.

(4) Prevention of evaporation

(5) Lubricating effects

(6) Enhance secretion of glands by detergent action

(F) Carbomer gel: 0.3%

(a) It is efficacious to control symptoms of dry eyes.

(b) It is safe.

(G) Dexapanthenol: Containing artificial tears marketed as Sicca

protect (Gobbel and Gross 1996)

(a) Favourable and more effective

(b) It decreases the permeability of corneal epithelial cells

Supracutaneous administration of calcium ointment 10% (Tsubota et al BJO 1999).

The ointment 10% is applied to lower lid twice a day containing calcium carbonate which is bland powder.

MECHANISM:

(a) It improves symptoms.

(b) Improve tear dynamics.

(c) Improve ocular surface staining.

(d) It improves electrolytes in tears.

Basis: McKeen et al; Gilbard & Rossi; Bernal and Ubels; they emphasized that the ION content of tears play an important role in the maintenance of epithelium integrity. Since calcium ION control various gene expression as well as formation of cell – cell adhesions (Schwaz et al 1996; Buexton & Magee 1992) and this ION may be one of the most vital in tears involved in various physiological activities of the ocular surface. Recently MacKeen et al reported that by the application of ointment to skin of lower lid, the ointment moves up slowly with the ocular surface and this movement has been named as supracutaneous (McKeen & Roth 1995).

(H) Retinoic Acid 0.01% of All Tansretinoic acid (Tretinoin)

(a) Driot & Bonne 1992; Murphy et al 1996; Soong et al 1988; Selek et al 2000 reported that although there is an improvement in Schirmer test, FTBUT & Rose Bengal staining but symptoms of dry eye did not relieve.

(b) Kobayashi et al 1997; Tseng Si 1986 however reported good results and improvement in metaplasia in dry eye by impression cytology.

(c) There is increase in goblet cells (Schilling et al 1989).

Inference: It is not used now for dry eyes.

(I) Tetracycline

- Decrease in bacteria that break down lipid into the irritating force acids.
- Act as anti-inflammatory

(J) Androgens in dry eye

The level in female is 10 to 20 times lower than men. Mostly it exists in bound form with albumin except 1 – 2% in free form. Post menopausal women a deficient in androgens (Sullivan et al 2002) and hence dry eye occurs on them.

MECHANISM:

(1) Increase the activity of sebaceous glands and meibomian glands.

(2) They promote retention of water and electrolytes.

(3) The meibomian gland contains androgen receptor MRNA with acinar epithelial cell nuclear and hype 1, 2, 5 alpha reductive (Rocha et al BJO 2000).

(4) Anti-androgen therapy in men leads to dry eye symptoms (Sullivan et al)

(5) Immuno modularity or anti-inflammatory effect.

(6) Positively influence on epithelial cells by enhancing the expressing of certain genes, protein synchronization and processes.

(7) Alter the composition of meibomian gland secretion, stimulating production and release of meibomian gland fluid.

(K) Secretogogues: There are agents which increase the secretion from the glands when applied locally:

(a) GERANYL- GERANYLACETONE (Gefarnate)

Cytprotective PGE_2 (Prostaglandin E_2):

(1) Increases the secretion of mucus from goblet cells and increases the blood flow to mucosa.

(2) Promotes regeneration of cells (DNA content)

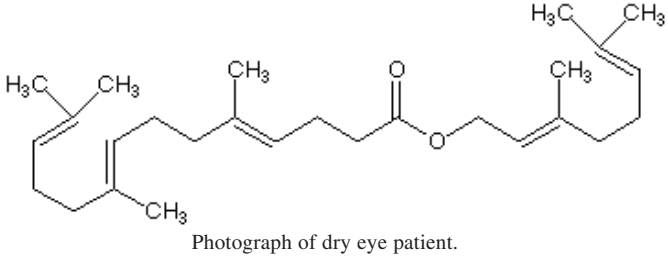
(3) Enhancement of tight functions of cell membrane architecture.

(4) Increases Mucosal and mucus circulation.

(5) Increases PAS positive cell density.

CHEMICAL:

Gefarnate (3, 7 – dimethyl – 2, 6-Octadienyl-5, 9, 13 trimethyl-4, 8,1, 2-tetradec atirienoate.



Photograph of dry eye patient.
LIPCOF highlightened with lissamine green.

DOSE: Eye Drops 1% Solution given 6 times a day.

Nakamura et al 1997 – 1998 BJO did exclusive study on rabbit with 1% Gefarnate eye drops and noted increases +ve cell on impression cytology. Vehicle: 0.5% polysorbate 80 in saline

Toshida et al 2002: used Gefarnate in squirrel monkey 1% - 6 times a day for 5 days a week, for 4 weeks and noted increase in repopulation of the goblet cells and increase its mucin production.

(b) 15-S HETE (Hydroxyeicosate traenoic acid)

It is produced from Arachidonic acid by enzyme 15-Lipoxygenase (15-Lo) and is formed in human beings in respiratory epithelium, leukocytes and reticulocytes.

MECHANISM: -

(1) It is anti-inflammatory

(2) It lowers leukotrine B4(LTB4) concentration

(3) It may regulate T lymphocyles by inhibiting 5 and 12 Los.

(4) It is vasoconstrictor

(5) Inhibits Apoptosis

(6) It increases mucus secretion of goblet cells.

The glycocalyx is formed by apical cells of the conjunctiva and cornea. The specific mucus1, Muc2, Muc4 is formed from the glycocalyx. Muc5AC forms the major structural mucus of the tear film.

Surgical

(1) Subcutaneous abdominal artificial tear pump-Reservoir for severe dry eyes

(Murube J; Murube E; Chenzhou L & Rivas L)

60ml reservoir operated through a gas pump through silicon tube via chest, neck, lateral part of head and entering conjunctivital sac over the lateral canthal ligament. It pumps tears 1.5ml/daily and the pump has to be filled with subcutaneous injection every 40 days.

(2) Preservation of tears by occluding with punctal plugs

(1) Silicone punctal plugs (Removable Gio-vagnoli & Graham 1992)

a. Increases the contact lens wear

b. Reduces dependency on tear drops

Disadvantages:

1. Difficult to tolerate
2. Side effects:

a. Migration down and producing dacryocystitis and canaliculitis (Rumelt et al 1997)

b. Spontaneous plug loss (Balaram et al 2001)

c. Colonization of bacteria around plugs (Sugita et al 2001)

d. Spontaneous Extrusion (Fayet et al 2001)

(2) Permanent intracanalicular silicone plug

a. Vicha and Lichaka 2000 & Riviere and Lerory 1998 reported good results.

(3) Autologous Limbal Transplantation (Stem cell transplantation)

It was first proposed by Kenyon and Tseng (1989).

METHOD: 150 µm depth adjusted knife could make circumferential incisions and two radial cuts at 2 – 3^{o} Clock hours and conjunctivial tissue were harvested. In the recipient it was set to 100 µm to allow for absent epithelium.

(4) Occlusion of the punctum or with laser or diathermy

a. It is effective to close the punctum in 60% of cases.

b. Useful in some cases

Note: However most of the dacryologists hate to use this procedure as it is non-physiological.

(5) Soluble Collagen Discs (Shaker et al 1989)

(a) Significant improvement in symptomatology

(b)Reduced necessity for artificial tears.

(6) Amniotic Membrane transplantation

Amniotic membrane consists of a single layer of ectodermally derived columnar cells firmly fixed to the underlying layer of mesenchyme which contain large amount of collagen (Pollard et al 1976; VanHerendail et al 1978). According to Shimazaki et al (1998), the epithelium of amniotic membrane survives for up to 70 days after preservation. Dua HS (1999), noted that at -70^{0}C the membrane survives for 6months to one year.

The apical surface of amniotic cells has many microvilli. At the base, cell processes or pedicels extend into the basement membrane in podocyte fashion. The basal cell processes have a hemidesmosome type of attachment to the basal membrane with tonofilament, and the subjacent basement membrane substance is partly amorphous and partly microfibrillar.

MECHANISM:

(1) Basement membrane facilitates migration of epithelial cells (Tseng et al 1997; Terranova & Lyall 1986).

(2) It reinforces the adhesion of basal epithelial cells (Khodadoust et al 1968)

(3) Promotes epithelial differentiation (Kurpakus et al 1992; Guo & Grimmell 1989)

(4) Prevents epithelial apoptosis (Boudreau et al 1995).

(5) It produces various growth factors such as basic fibroblast growth factor; hepatocyte growth factor and transforming growth factor which can stimulate epithelialization (Sato et al 1998; Shimazaki et al 998).

(6) Inhibits protease activity (Kin et al 1998; Sato et al 1998)

(7) Acts as a bandage contact lens, allowing epithelialization to occurs under the career (Azuara Blanco et al 1999).

(8) Acts as inhibitor of fibrosis (Tseng et al 1998; Li DW et al 1995).

Indication:

(1) Persistent epithelial defects unresponsive to medical treatment (Azuara et al 1999; Loe SH et al 1997).

Use:

(a) Single layer (Azuara Blanco et al 1999).

(b) Multilayer (Kruse et al 1999; Dekaris et al 2001; Prabhasawat et al 2001)

(2) Severe neurotrophic ulcers (Chen et al 2000): 2 – 3 layered amniotic membrane hold with tarsorrhaphy.

(3) Chemical and thermal burns

(4) Diffuse limbal cell deficiency

(5) Stevens-Johnson syndrome

(6) Ocular Pemphigoid

Preparation of Amniotic membrane

- From caesarean delivery
- Sero-negative donor
- Under a lamellar flow hood, the placenta is first washed free of blood clots with balanced salt solution containing antibiotics (Penicillin 50µgm/ml; Streptomycin 50µgm/ml; neomycin 100µgm/ml and amphotericin B 2.5µgm/ml.
- The inner amniotic membrane is separated from the rest of chosen by blunt dissection.
- The membrane is flattened onto a nitro-cellulose paper with epithelial side up and cut into 4 x 4 cm pieces and placed in sterile vial containing DULBECCO, modified eagle is medium and glycerol at a ratio of 1:1 (Vol / Val).
- The vials are frozen at -80^{0}C. The membrane is defrosted before use by warning the container at room temperature for 10 minutes (Tseng et al 1997).

(7) Transplantation of the autologous submandibular gland for most severe cases of KCS: The submandibular gland is moved from its natural site into the temporal fossa. The glands supplying vessels are connected to the temporal artery and vein, its secretory duct is implanted into the conjunctival fornix (Geerling et al 1998). The review of 1 year followup showed promising results. Mcleod AM & Robbin SP (1992) too noted good results in 8 patients out of 12 operations.

(8) Parotid Duct transplantation: It was carried out by Zhang et al 1998 in 40 dry eye cases with a followup of 3 weeks to 6 years. In 82.5% of cases tearing occurred while in 27.5% had no tearing vision however increase in 72.5% of cases.

Advantages:

(a) The tears are replaced with almost natural tears

Disadvantages:

(a) Profuse tearing during eating in 96% of the cases.

(b) Short parotid duct

(c) Twisting of duct and blocking

(d) Abscess formation

(e) No tearing.

(f) Failure in ocular pemphigoid

(9) Tarsorrhaphy in dry eye

Union of upper and lower lid margins.

Temporary: Using nylon suture

Permanent: To unite the raw surfaces of lid margin of upper and lower lid.

Indication:

(a) Persistent epithelial defect

(b) Exposure keratopathy

(c) Neurotrophic ulcers

(d) P.K.

(e) Ocular cicatrical pemphigoid

(f) Steven Johnson syndrome

(g) Entropion

Complication:

(1) Trichiasis

(2) Adhesion between upper and lower lids after tarsorrhaphy lysis.

(3) Premature opening of eye lid

Inference:

(1) Useful

(2) Simple

(3) Effective in 90.9% of the cases.

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!!Attention!!

Case Presentation in the Monthly Meetings by non institutional members

There will be one non Institutional case presentation/Clinical talk by one of the DOS member during the monthly meeting. The presentation will be done by a non Institutional member where monthly meetings are not being held. The presenter will be allowed to present a case or a clinical talk for same amount of time as it is given for other presentations in the monthly meeting. Interested members should contact secretary DOS at least two weeks before the monthly meeting with details of their presentation. If there are more than one request then they will be given opportunity in the next monthly meeting. The President and Secretary will review the presentation for its clinical and scientific contents. These non Institutional presentation will be graded for the best case presentation/Clincal talk as it is done for Institutional presentations and they will be eligible for best presentation award.

DOS Credit Rating System (DCRS)

DOS has always been in the forefront of efforts to ensure that its members remain abreast with the latest developments in Ophthalmology. Among the important objectives formulated by the founders of our constitution was the cultivation and promotion of the Science of Ophthalmology in Delhi.

The rapid strides in skills and knowledge have created a need for an extremely intensive Continuing Medical Education programme.

In a bid to strengthen our efforts in this direction DOS had DOS Credit Rating System (DCRS), the details of which are given below. Our Primary objective is to promote value-based knowledge and skills in Ophthalmology for our members and give recognition and credit for efforts made by individual members to achieve standards of academic excellence in Ophthalmic Practice.

DOS CREDIT RATING SYSTEM (DCRS)

		DCRS	*Max.*
1)	Attending Monthly Clinical Meeting*† (For full attendence)	10	90
2)	Making Case Presentation at Monthly Meeting**	15	—
3)	Delivering a Clinical Talk at Monthly Meeting**	15	—
4)	Free Paper Presentation at Annual Conference (To Presenter)**	15	30
5)	Speaker / Instructor** in : Monthly Symposium	15	30
	: Mid Term Symposium	15	30
	: Annual Conference	15	30
6)	Registered Delegate at Mid Term DOS Conference	20	—
7)	Registered Delegate at Annual DOS Conference	30	—
8)	Full Article publication in Delhi Journal of Ophthalmology / DOS Times	30	60
9)	Letter to editor in DOS Times	10	20
10)	Letter to editor in DJO	15	30

If any of the presentations is given an Award – Additional 20 bonus Credits.

Member who have earned 100 Credits, are entitled to:

a) Certificate of Academic Excellence in Ophthalmic Practice.

b) Eligible for DOS Travel fellowship for attending conference.

If any member earns 200 Credits, he / she shall, in addition to above, be awarded Certificate of Distinguished Resource-Teacher of the Society.

Institutional assessment for best performance will be based on the total score of members who attend divided by number of members who attended. Institutional assessment regarding decision to retain the institute for the next year will be based on total score by all delegates who attend the meeting divided by average attendence of all 8 meetings.

Please note that the Institutions' grading increases if the attendance at its meeting is higher (i.e. more than the average attendence of the eight monthly meetings).

* Based on Signature in DCAC

** Subject to Submission of Full Text to Secretary, DOS

† Credits will be reduced in case attendence is only for part of the meeting.

DCRS !! Attention !!

* Members are requted to sign on monthly meeting attendance register and put their membership number.

* The DCRS paper will be issued only after the valid signature of the member in the attendance register.

* Please submit your DCRS papers to the designated DOS Staff only.

* The collected DCRS papers will be countersigned by President and Secretary and sealed immediately ofter the meeting is over.

Attention D.O.S. Members

The Hi-tech DOS Library has started functioning on Ground Floor, Dr. R.P. Centre, Delhi Ophthalmic Sciences, AIIMS, New Delhi-110029 from 12.00 Noon to 9.00 P.M. on week days and 10.00 A.M. - 1.00 P.M. on Saturday, Sunday. The Library will remain closed on Gazetted Holidays. Members are Requested to utilise the Facilities Available i.e. Computer, Video Journals Viewing, Latest Books and Journals. We are planning to subscribe two journals member can give suggestion in this regard.

Dr. Lalit Verma
Library Officer, D.O.S.

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   Editors - Thomas C Spoor
2. Manual of Fundus Fluorescein Angiography
   Editors - Amresh Chopdar
3. Complications of Glaucoma Therapy
   Editors - Mark B. Sherwood. M.D. George L. Spaeth M.D.
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4. Survey of Ophthalmology
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5. Survey of Ophthalmology
   Vol.43 No.6 May-June-99
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3. Ocular Surgery For The New Millennium Part I (Dec 1999 12:4) Ophthalmology Clinics Of North America - Georgel Spath. Md)
4. Retinal Vascular Disorders (Dec 1998 11:4) (Ophthalmology Clinics Of North America (Dr. Pran N. Nagpal - Donated By Dr. B. Patnaik)
5. Survey Of Ophthalmology (Vol 44 No.4 Jan-Feb 2000)
6. Survey Of Ophthalmology (Vol 44 No.5 March-April 2000)
7. Survey Of Ophthalmology (Vol 44 No.6 May-Jul 2000)
8. Survey Of Ophthalmology (Vol 45 No.1 July-August 2000)
9. International Ophthalmology (Vol 23 No.1 Pp-1-60 1999)
10. Retina The Journal Of Retinal And Vitreous Diseases (Vol 20 No.1 2000)
11. Journal Of Cataract Refractive Surgery (Vol 26 No.8 August 2000)
12. BJO Journal Ophthalmology (Vol. 7 No. 1 Jan. 2004)

Methodology for Monthly Clinical Meeting: Criteria for Selection

Formula: Institution's Marks

$$\text{Average marks A (outside delegates)} \times 0.7 + \frac{\text{Attendance of institution (N)}}{\text{maximum attendance in any monthly meeting (Nx)}} \times 3$$

$$A = \frac{\text{Total marks by outside delegates (M)}}{\text{Total number of outside delegates (N-n)}}$$

N = Total Attendance of an instituton (Outside + internal delegates)

Nx = Highest attendance of all meetings

N = Total number of delegates

n = Total number of internal delegates

Delhi Ophthalmological Society Fellowship for Partial Financial Assistance to Attend Conferences

Conferences

International: two fellowships per year

- Maximum of Rs. 25,000 / -will be sanctioned

National: three fellowships per year (only for AIOS)

- Maximum of Rs. 5,000 will be sanctioned

Eligibility

- DOS Life Members (Delhi Members only)
- Accepted paper for presentation / poster / instruction course

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Preference will be given to member who has not attended conference in last three years. However if no applicant is found suitable the fellowship money will be passed on to next year. Members who has availed DOS fellowship once will not be eligible for next fellowship for a minimum period of three years.

Authorship

The fellowship will be given only to presenting author. Presenting author has to obtain certificate from all other co-authors that they are not attending the said conference or not applying for grant for the same conference. (Preference will be given to author where other authors are not attending the same conference). If there is repeatability of same author group in that case preference will be given to new author or new group of authors. Preference will also be given to presenter who is attending the conference for the first time.

Quality of paper:

The applicant has to submit abstract along with full text to the DOS Fellowship Committee. The committee will review the paper for its scientific and academic content. The paper should be certified by head of the department / institution. In case of individual practitioner he or she should mention the place of study.

Credit to DOS:

The presenter will acknowledge DOS partial financial assistance in the abstract book / proceedings.

The author will present his or her paper in the immediate next DOS conference and it will be published in DJO.

Points awarded:

		Points
1)	Age of the Applicant	
	a) ≤ 35 years	10
	b) 36 to 45 years	07
	c) 45 years plus	05
2)	Type of Presentation	
	a) Instructor / Co-instructor of Course	10
	b) Free Paper (Oral)	08
	c) Poster	05
3)	Institutional Affiliation	
	a) Academic Institution	15
	b) Private Practitioner	20
4)	DCRS Rating in the immediate previous year	
	a) > 100	10
	b) 50-100	05
	c) < 50	not eligible

Documents

- Proof for age. Date of Birth Certificate
- Letter of acceptance of paper for presentation / poster / instruction course
- Details of announcement of the conference
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- Copy of letter from other national or international agency committing to bear partial cost of conference if any.
- At least one original document should be provided, that is ticket, boarding pass or registration certificate along with attendance certificate of the conference.
- Fellowship Money will be reimbursed only after submission of all the required documents.

Dr. Gurbax Singh (President DOS), Dr. Noshir M. Shroff (Vice President DOS), Dr. Kamlesh (Editor) Dr. Lalit Verma (Library Officer), Dr. Sudipto Pakrasi (Member) Dr. J.C. Das and Dr. Jeewan S. Titiyal (Secretary DOS) will be the members of ***DOS Fellowship for Partial Financial Assistance to Attend Conferences Committee****.*

Application should be addressed to President, DOS. Application should reach secretary's office before **31st July** and **31st January** for international conference and before **30th September** for national conference. The committee will meet thrice in a year in the month of August, October and February with in 2 weeks of last date of receipt of applications. The committee will reply within four week of last date of submission in yes / no to the applicant. No fellowship will be given retrospectively, that means prior sanction of executive will be necessary.

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The Art of Probing in Management of Congenital Nasolacrimal Duct Obstruction (CNLDO)

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Introduction: *CNLDO is a well established entity in infants and children and its incidence varies from 5 to 6%.*

Diagnosis: Most of the cases present with tearing (epiphora); crusting of lids and a boggy swelling on inner canthal area which on pressure regurgitates discharge from the punctum

Other clinical presentations: Subacute, Acute dacryocystitis, lacrimal abscess and fistula & orbital cellulitis.

Management: Many modalities of treatment have been tried:

(a) Local antibiotics eye drops and pressure massage for a period of three months to three years with uncertain results.

Success of results: 29% - 93% (Score) Exponent of this procedure is Nelson; Nucci; Nucci et al; Noda et al & Paul TO. We do not recommend waiting of more than one week for the conservative treatment to cure CNLDO to increase risks of serious immediate and late complications.

(b) No treatment: Modality (Spontaneous cure) occurred in 3 months – 3 years in about 30 – 60% of cases. However epiphora persisted. The exponents of this modality are Kucher et al; Young et al and Levin et al. It is not followed however by other researchers.

(c) Syringing of nasolacrimal passages: Kim et al and Bellard used this procedure with antibiotic drops. However the results were inconsistent and unsatisfactory and is not recommended.

(d) Office probing:

a. Under Local Anaesthesia was recommended by Gold Blum et al and Stager et al in infants with good results.

b. Under GA exponents are Young et al; Robb and Cibis met with good results.

(e) Fracturing of inferior turbinate was recommended by Wesley; Havins & Wilkins; McEwen et al and Ingels et al in cases where orthograde probing failed and met with better results.

(f) Endoscopically controlled retrograde probing: It could correct the end of probe going submucosally by rerouting it (Choi et al; McEwen et al and Ingels et al) and recommended against waiting.

(g) Silicone intubation and balloon dilatation was recommended in failed cases by McEwen et al; Magliori and Putterman Lueder; Grin et al; Roy et al; Kaufmann et al and Wolyter. Results: 70% success and rest failure. Equivocal.

(h) Orthograde Probing: The modality was preferred by all at all ages.

Advantages:

i. Easy

ii. Simple procedure

iii. 87-95% success rate (Robb et al 92%; Elmonsoury et al 93.5%; Xiao et al 98.2%; Clark 92%).

Method of Probing:

(1) The punctum upper and lower is dilated with a fine punctum dilator.

(2) OO-Number Probe end is then introduced vertically 1-2mm and then moved horizontally towards the inner canthus till it meets bony resistance.

(3) The probe end is slightly withdrawn and probe is moved to vertical position and gently pushed downwards, backwards and laterally till it reaches the lower end of nasolacrimal duct.

How you are sure that the probing is correctly performed:

(1) The vertical outside end of probe does not move side ways.

(2) Do endoscopy to visualize the lower end of probe.

(3) Sounding of lower end of probe with the endoscopy.

(4) Removing of probe does not result in oozing of blood from punctum.

(5) Dye test which will show easy passage of dye into nose.

(6) On syringing, no regurgitation of fluid is seen

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Basis of one week of conservative treatment before probing: We recommend probing at any age if the conservative treatment for one week as local antibiotic eye drops and massage to sac region failed. Rationale of use of antibiotic drops in conservative treatment in one week protocol are: -

(A) Many cases of subacute, acute, chronic dacryocystitis with cellulitis and fistula formation is seen as complication of congenital nasolacrimal duct obstruction (Gurbax S & H Bhinder 2004).

(B) Demonstration of streptococcus, pneumonia and haemophilus influenza in culture of the sac discharge (Kucher et al 2000; Bareja & Ghose 1990; Huber-Spitz et al 1987).

(C) Demonstration of pure and mixed fungal infections by candida and aspergillus from sac discharge (Ghosh and Mahajan 1990).

Note: However McEwen et al 1994 found no difference in bacterial growth in nasal, conjunctival sac and sac discharge.

Inference: We do not agree with McEwen as (3.5%) case of acute infection in CNLDO is seen even in our series.

Timing of probing

However there were diverse views in the world literature:

(I) Some workers recommend immediate probing from 2nd day to 1 week of conservative treatment. (Nelson 1985; Levin et al 2003; Leuder 1995; Campolattario et al 1997; Paysee et al 2000; Boynton et al 1989; Leone 1989)

(II) Some recommend immediate probing after 3 – 4 months of conservative treatment. (Paul TO 1985; Noda et al 1991; Burns & Kipiote 2001; Shashy et al 2003; Paul TO & Shepard 1995; Katowitz & Welsh 1987)

(III) Some recommend immediate probing after 4 – 6 months of conservative treatment (De Pozz et al 1995; Baggio et al 2001).

(IV) Some recommend immediate probing before 9 months of conservative treatment (Noda et al; Ekineller et al 1994; Peterson & Robb 1978; Stager et al 1992).

(V) Some recommend immediate probing before 13 months of conservative treatment

(VI) Some recommend immediate probing before 24 months of conservative treatment (Sturrock et al 1994; Kashkounle et al 2002; Young & McEwen 1997).

(VII) Some recommend immediate probing before 36 months of conservative treatment (Peterson et al; Robb and many others).

(VIII) Some recommend immediate probing at all ages (Chiese et al 1999; Young et al 1996; Golblum et al 1996; Clark 2002; Robb 1986; Khasanov et al 1991).

Advantages of early probing:

(a) High success rate > 90%.

(b) Immediate cure results

(c) Patient has not to suffer long with repeated visits to the ophthalmologists

(d) Risks are avoided which are likely to occur e.g. lacrimal abscess, acute dacryocystitis, fistula, orbital cellulitis and its complication.

What to do if first probing fails:

We recommend repeated 2 – 3 times probing at an interval of one week which cured our failed cases. A similar view is expressed by Robb who also noted 90% success of 1st probing and 60% additionally in 2nd probing. Xiao et al performed probing in 1 – 3 times and noted 98.2% success rate. We achieved a success rate of probing (1st time = 98.10%; 2nd time = 98.64%; 3rd time = 100%).

Consensus of timing of probing is doing as early if 1 week of conservative treatment fails. The younger the age, the better the results.

Disadvantages of probing: If proper technique is adopted, there is no side affect seen in our series of 1800 cases over the period of 1965 – 2004. Some authors reported punctum occlusion (Lyon et al) and false passages (Cibis & Jazbi).

Point to stress: The diversity of views is to wait for 3 years with conservative treatment or not to wait. If your probing is not successful in 4-10% of cases due to immature technique or anatomical difficulty in nasolacrimal passages, yet you will give immediate cure to >90% of infants or children. Your failed cases may wait for spontaneous cure. We however find repeated probing 2 to 3 times as a set protocol for 100% cure in 5-10% of difficult cases.

What next to do of 3rd probing also fails?

(1) Wait and watch. Some people reported spontaneous cure.

(2) Do a DCR endonasal in routine

(3) Dacryoplasty with Endoscopically controlled cutter.

(4) Silicone tubing

(5) Ballooning of nasolacrimal duct obstruction.

Note: However this eventuality has not seen in our large series of 1800 cases.

Points to Ponder

(1) Learn probing procedure from an adept in this technique.

(2) Do dilatations with 0 diameter probe.

(3) Repeat probing 2 – 3 times with 0 diameter or 0.01 diameter probe. This is called sequential probing.

(4) Never use any force while pushing the probe.

(5) Do not forget to sound the probe end or see the end with an endoscope.

(6) Check the potency by syringing with antibiotic solution.

When do you declare a case as a successful?

In our series we noted that tearing and discharge usually disappears within a week and hence we declare it successful as success in maintained. Burns & Kipioti however noted a case to be cured if he remains symptom for 3 months and Yap & Yip maintained 1 month period. Honaver et al maintained 6 months observation. Others have noted case of dry test is negative if dye disappears in 2 weeks.

What will you do if a case presents you with Acute-dacryocystitis, abscess & fistula?

We recommend antibiotics systemically and local antibiotic drops for a week. As the acute stage is seen resolving, do probing under antibiotic cover. We noted that the lacrimal abscess or fistula closed by its own after a successful opening of nasolacrimal passages. A similar view is expressed by Levin et al 2003; Mansour et al 1991; Shashy et al 2003; Compolattaro 1997; Payasse et al 2000; Boynton et al 1989; Berson & Landou 1978; Elorza et al 1989; Kauskouli et al 2003).

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Clinical Assessment in Neuroophthalmic Trauma

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Abstract:

In cases of neural trauma, the ophthalmologist must be prepared to substitute inference for observation. Sometimes neural lesions involving the optic nerve, chiasma, visual pathway, trauma to internal carotid artery, ocular motor nerve injuries, raised ICT and intracranial haemorrhage are initially missed. The ophthalmologist must nevertheless be prepared to recognize, evaluate & manage retrobulbar lesions.

Introduction:

Ocular lesions are not the only cause of impaired visual function following trauma. The optic nerve, optic chiasma and posterior visual pathways are all vulnerable in patients with open or closed head injuries. The incidence of closed head injury has increased with rapid industrialization. Roadside accident trauma is also on the rise which has both blunt and perforating elements associated with it.

Since the care of road-injured patients with visual impairment is shared between ophthalmologists and non-ophthalmologists, it is essential that the latter be familiarized with the ocular signs associated with head injury.

Preliminary Assessment:

Injuries to the retrobulbar visual pathways may have a low priority in the trauma scheme, but whenever possible in alert, cooperative patients a quick preliminary bedside examination should be carried on. This includes:-

1) Visual acuity testing with each eye tested individually.
2) If subnormal acuity is detected, an attempt should be made to improve vision with a pinhole and refraction if patient is stable.
3) Colour vision testing to check for any injury to optic nerve.
4) Pupillary evaluation is a key element in the recognition and differential diagnosis of anterior visual pathway trauma. Check for pupillary size, direct and consensual light reflex along with a proper swinging flashlight light.
5) A positive swinging flashlight sign in a patient with a normal fundus implies the presence of a retrobulbar lesion or lesion of the anterior visual pathway. Anisocoria is never, the result of a sensory lesion. A patient with uncorrectable reduced acuity in one eye and a dilated pupil has both a sensory and a motor lesion.
6) Fundus examination is a must in all the patients to look for any optic nerve injury, avulsion, haemorrhage in and around the optic nerve, pallor of disc, commotio retinal, retinae breaks or tears, traumatic macular degeneration etc.
7) Doll's eye movements can be elicited, preservation of which informs that coma is probably not due to an upper brain stem lesion but defect lies in the cerebral diencephalic structures.
8) Nystagmus if present should be properly assessed.
9) Confrontation testing for visual fields is a must.

Never dilate pupils of an acutely injured patient with altered consciousness.

Differential Diagnosis:

There are many causes of visual loss after trauma, and neural causes are much less common than ocular causes. However even if a retinal lesion is identified, we should remain open to the possibility that there is also a retrobulbar lesion. Two other entities deserve consideration:-

1) Preexisting neuropathies
2) Factitious amblyopia

Given below are some of the ocular signs associated with head injury.

Optic Nerve:

The injuries are of two types:

1) Direct
2) Indirect

1. Direct :- Refers to injuries resulting from objects that penetrate the orbit and impinge on the nerve. There is partial or complete transection by the intruding object with haemorrhages within and around the nerve. In addition to local changes at the wound site, there is ptosis, mydriasis or ophthalmoplegia. In the acute stage the patient has a normal disc, optic atrophy supervening, only after weeks have passed.

2. Indirect injuries are further divided into an Anterior and Posterior variety.

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➢ Anterior variety is extremely rare, involving short (1mm) intraocular segment of optic nerve.

➢ In posterior, the intracanalicular segment is damaged with associated traumatic loss of vision which occurs without external or initial ophthalmoscopic evidence of injury to the eye or its nerve. It is usually associated with Frontotemporal blows which create an orbital deformation concentrated at the optic canal resulting in monocular blindness.[1]

In partial damage to optic nerve there is relative afferent pupillary defect (RAPD) and Marcus Gunn pupil.[1] In case of complete damage there is total afferent pupillary defect and amaurotic pupil with equal pupillary size in both eyes under diffuse illumination.[1] Visual deficits cover the entire spectrum from small Bjerrum scotomas unassociated with dyschromatopsia or reduced acuity to complete blindness. Optic nerve atrophy sets in within 4-5 weeks.

The immediate course of management is use of high dose of intra venous bolus of 500mg of methylprednisolone followed by 250mg IV every 6 hours for 24-36 hrs; patients who appear to improve are switched to a gradually tapering dose of Prednisolone. In case of relapses surgical decompression should be considered. Visual evoked response is used to monitor visual status in comatose patients.

OPTIC CHIASMA:

The optic chiasma is not immune to the effects of mechanical head trauma.[2] True splitting of chiasma leads to a bitemporal hemianopia that is inapparent during binocular viewing without reducing central acuity. The fundus is initially normal, but later on optic atrophy ensues.

Associated loss of consciousness and other non visual neurologic deficits like anosmia and transient diabetic insipidus is known to develop in some patients.

RETRO CHIASMAL VISUAL PATHWAY TRAUMA:

Since retrochiasmal injuries are usually associated with aphasia, confusion or coma neuroimaging is preferred over clinical examination in detecting, localizing and characterizing acute intracerebral damage to the visual pathways. Unilateral damage to tract, geniculate body, optic radiations or visual cortex does not alter snellen acuity,[3] which remains normal even with absolute macula splitting homonymous hemianepia, ***Snellens acuity is reduced only if there is a bilateral involvement of visual pathways.***

Optic tract lesion produces incongruous homonymous hemianopic as well as RAPD. Wernickes hemianopic pupil can be elicited with a very fine beam of light.[4,5] All the defects are on the contralateral side. Optic atrophy typically involves fibres temporal to fovea ispsilaterally and fibers nasal to fovea contralaterally.[5,7] Optic radiation may be involved in temporal or parietal lobes. Apart from quadrantanopias other neurological deficits like, dysphasia, agnosia uncinate fits and hemi paresis may be present. Optokinetic nystagmus should be tested in alert cooperative patients since parietal lobe hemianopias will be accompanied by asymmetric responses.

Table Enumerating Field Changes & Clinical Presentation Acc

S.No.	SITE OF INJURY	FIELD CHANGES	
1.	Optic Nerve	Mono ocular Blindness, Bjerrum Scotomas	RA di co Ne
2.	Optic Chiasma 1) Crossed Fibres → 2) Uncrossed Fibres →	Bitemporal hemianopia Binasal Hemianopia	As Ar
3.	Optic Tract	Incongruous homonymous hemianopia with macular splitting	RA sn bil
4.	Lateral Geniculate Body	Homonymous hemianopia	Sp
5.	Optic Radiations → 1) Parietal Lobe → 2) Temporal Lobe →	Homonymous hemianopia with macular sparing a. Inferior quadrantic hemianopia (pie on the floor) b. Superior quadrantic hemianopia (pie in the sky)	
6.	Occipital Cortex	Congruous homonymous hemianopia with macular sparing	

Bilateral damage to occipital cortex may cause bilateral homonymous hemianopia with macular sparing to complete

loss of vision.[4,5] Confrontation testing of visual fields is the best bedside tool for the above mentioned.

Transient Cortical Blindness after Blunt Trauma:

The patient gives a history of concussive blow to the occipital region. The patient is either blind on awakening or becomes blind within a few hours. Pupils respond to light and fundii are normal. Amnesia and confusion are commonly present. There is spontaneous recovery of vision within hours.[3]

Traumatic Aneurysm of the internal Carotid Artery:

This is a devastating lesion that may cause not only blindness but death. Most of these traumatic aneurysm are associated with basal skull fractures. Following severe head trauma, a false aneurysm may develop in the cavernous portion of the internal carotid artery[6,7]if the aneurysm points into the sphenoid sinus the patient will have epistaxis and loss of vision. The role of ophthalmologist is to alert neurosurgeons and otolaryngologists to possibility of aneurysm in patients who manifest the triad of severe head injury, posterior indirect optic nerve trauma and epistaxis.[3]

Raised ICT and Intra Cranial Haemorrage:

This is secondary to trauma because of obstruction of CSF flow and related subarachnoid haemorrhage. Typical presentation is headache, nausea, projectile vomiting and horizontal diplopia secondary to abducens palsy.

There is associated papilloedema which is usually bilateral, but may be asymmetric. Vision is impaired only with macular edema, haemorrhage or optic atrophy.[4]

Acute subdural haemorrhage becomes symptomatic within minutes to hours. It is characterized by unilateral headache and mild ipsilateral pupillary enlargement.

Pupil:

One sided unreactive and enlarged pupil or one that is poorly reactive signifies a compression or stretching of the third nerve from the effects of a mass above. Enlargement of pupil contralateral to a mass may occur but is infrequent. An oval and slightly eccentric pupil is a transitional sign that accompanies early mid brain or third nerve compression. Unilateral miosis in coma has been attributed to dysfunction of sympathetic efferents originating in the posterior hypothalamus and descending in the tegmentum of the brainstem to the cervical cord. Anisocoria is never the result of a sensory lesion. Trauma also accounts for about 4% of all Horner's syndrome cases[3].

Ocular Motor Nerve Injuries:

The most common ocular nerve to be affected by trauma is the sixth nerve:

1. Injury to oculomotor nerve is either a direct or indirect result of transtentorial herniation. The third nerve may suffer damage to orbital branches, contusion at the superior orbital fissure, or at its entry into the cavernous sinus, shearing forces in the midbrain[9] or avulsion at its rootlets. Clinically it presents with diplopia which is oblique and variable in different positions of gaze with ptosis of upper eyelid, pupil sparing in young patients but if affected, defective accommodation is present. Trauma to third nerve nucleus results in ipsilateral third nerve palsy with paresis of contralateral elevators.[4] Tentorial herniation secondary to extradural hematoma compresses third nerve and produces delayed total third nerve palsy preceded by fixed dilated pupil.[4] Involvement of superior division at anterior part of cavernous sinus or at superior orbital fissure results in levator paresis. Lower division involvement produces limitation of adduction and depression with pupillary dilatation.[4] Unapposed action of lateral rectus and superior oblique results in abduction and intortion in downgaze. Delayed responses include nystagmus, skew deviation, vertical gaze paresis, and internuclear ophthalmoplegia because of intrinsic brain stem injury.[10]

2. Trochlear nerve injury – Trauma causes one half to one third of all cases of isolated fourth nerve palsies.[8] It is bilateral and is characterized by elevation, excyclotorsion of ipsilateral eye. Diplopia may be there which is vertical and increase in downgaze.[4] This is due to avulsion of the nerve from the posterior aspect of the midbrain or contusion against the tentorium.

3. Abducent Nerve Injury – Crush injuries of the head are more likely to affect the sixth nerve at the petrous tip, especially if there is simultaneous involvement of the trigeminal nerve. It is characterized by horizontal diplopia that is worse at distance and in the direction of action of the weakened extraocular muscle. Combined ocular motor nerve injuries is much less frequent than isolated injury.

To conclude even with the recent advances in neuroimaging techniques, a good and thorough clinical checkup, strong index of suspicion and a good knowledge of the signs and symptomatology still remain a hallmark for the interpretation of any clinical problem.

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Vernal Keratoconjunctivitis - Recent Advances in its Management

Kamna Verma, MS, **Namrata Sharma,** MD, **Rajesh Sinha,** MD, FRCS, **Jeewan S. Titiyal,** MD

Vernal, is derived from Greek meaning "occurring in the spring" is an infrequent but serious form of allergic conjunctivitis. It is a bilateral, recurrent, interstitial inflammation of the conjunctiva, of periodic seasonal incidence, self-limited in nature, and of unknown etiology. It is characterized by flat-topped papules usually on the upper tarsal conjunctiva resembling cobblestones in appearance, a gelatinous hypertrophy of the limbal conjunctiva, either discrete or confluent, and a distinctive type of keratitis, associated with itching, redness of eyes, lacrimation and a mucinous or lardaceous discharge.

It is essentially a disease of youth, occurring most frequently between the ages of 6 and 20; with remissions by the late teens, it is rare over 40 years. It is more common in males as compared to females (2:1). No genetic predisposition of the disease is known. The most striking point in its presentation is the seasonal variation i.e. it is more common in the summers than in the winters.

Clinical features

Vernal keratoconjunctivitis (VKC) is a bilateral disease and manifests as intense itching, hyperaemia, tearing, photophobia and ropy mucus discharge. Astigmatism can also be seen in cases of severe palpebral from of VKC.

The conjunctival manifestations may be found in two distinct forms: palpebral and limbal although certain percentage of cases occur in combination.

Palpebral form: It is essentially seen in the upper tarsal conjunctiva. The earliest sign is generalized conjunctival hyperemia with minimal discharge and itching. Papillae on the tarsal conjunctiva are either discrete or clumped together. However in chronic cases, the tarsal plate is covered by a mosaic of flat papules *(fig.1)*. Occasionally, papillae may become very hypertrophic and develops into large cauliflower like excresences, sufficient to cause mechanical injury to the cornea. On the outside of the lids there is no disturbance, but a certain amount of ptosis is seen, giving the patient a sleepy appearance.

Limbal or bulbar form: There is a thickening, broadening and opacification of the limbus, especially at the superior margin of the cornea. Here, discrete limbal nodule appears, latter in crops becoming confluent. Nodules are most commonly present either nasally or temporally. It is not uncommon for them to run round the entire limbus making a ring of raised, gelatinous, hypertrophic tissue *(fig.2)*. In the raised mass chalk-white spots may occur known as Horner-Tranta's dots.

VKC has strong association with keratoconous, corneal opacification, hydrops and pseudogerentoxon.

Complications

Superficial punctate keratitis : These present as minute dull greyish points in the epithelium.

Vernal ulcerative keratitis (Shield ulcer) : It is rare and present in the upper part of the cornea, transversely oval, shallow, and not vascularized. There is whitened roughening of the epithelium and a greyish opacification of the exposed Bowman's membrane around its edges. It is extremely indolent in nature and develops into sodden plaque *(fig.3)*.

Dry eyes: It is frequently seen complication in chronic form of VKC.

Diagnosis

Diagnosis of VKC is based on history and typical clinical presentation it should be differentiated from trachoma, which has large papillae and scarring in upper tarsus. It can mimic Giant Papillae Conjunctivitis (GPC) where there is presence of triggering agent and genetic endowment,and atopic keratoconjunctivitis.

Pathogenesis

The pathogenesis of VKC is not fully understood. The hypothesis that VKC is a manifestation of an allergy has the most definite supporting evidence, but even that is not conclusive and the initiating event is unknown. More than one immune mechanism is responsible for its clinical presentation.

The presence of elevated tear histamine and IgE levels, and presence of degranulated mast cells on histopathological examination indicate it to be type-I hypersensitivity reaction mediated by Ig-E.However presence of helper T cells (CD4) on immunohistochemical studies indicate it to be of type-IV hypersensitivity reaction.

Histopathology

There is intense collagen proliferation in the cobblestones of VKC with ground substance and cellular accumulation. There is marked increase in the density of the mast cells in conjunctiva, of which 80% are degranulated as seen in electron microscopic sections. Eosinophils are also found to be increased in numbers. Horner-Trantas dots consist mainly of eosinophils and degenerated cellular debris with few polymorphonuclear cells and type- 1 hypersensitivity reaction mediated by Ig-E is supported by lymphocytes. The ropy discharge contains inflammatory cells, specially eosinophils and their Charcot-leyden granules, mucopolysaccharides and hyaluronic acid.

How to diagnose ?

- Seasonal variation
- Intense itching : vigorous knuckle rubbing
- Ropy discharge: thick strands of mucus
- Conjunctival congestion : pink in color
- Mild to moderate chemosis: seen only with slit lamp as pinkish fluid separating the conjunctiva from underlying episclera
- Large cobblestones of varying height and width in upper

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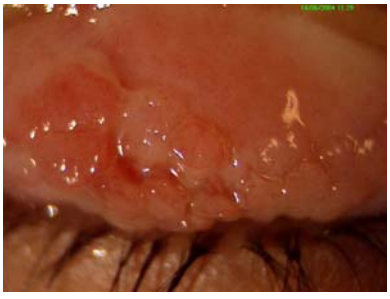

Fig. 1 : Cobblestones in upper tarsal conjunctiva

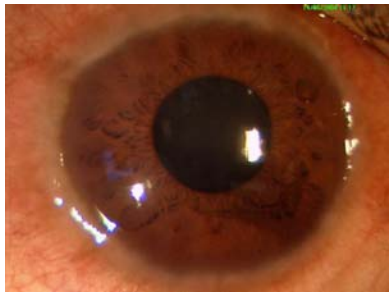

Fig. 2 : Ring of raised nodules in limbar form

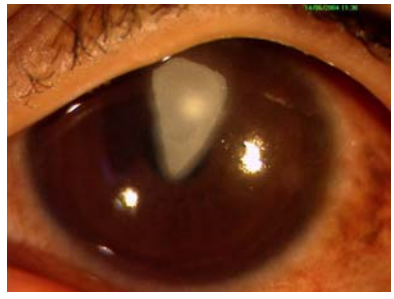

Fig. 3 : Shield ulcer in VKC

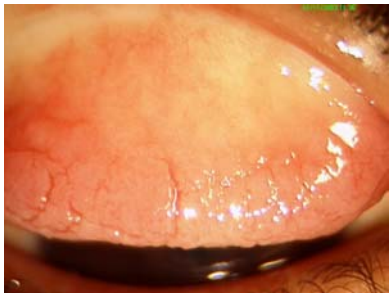

Fig. 4 : Six months post treatment with Topical Cyclosporin A 2%

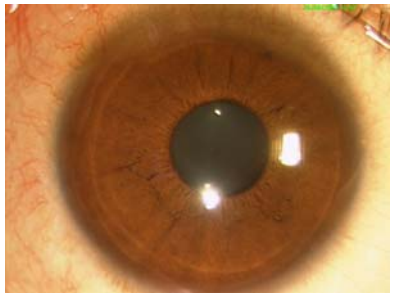

Fig. 5 : Recalcitrent limbal VKC treated with Topical Cyclosporin A 2%

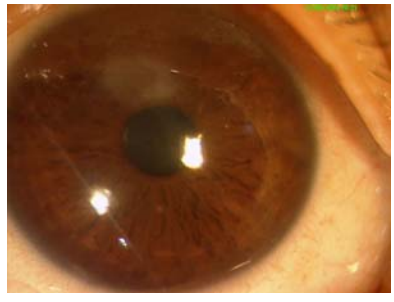

Fig. 6 : Shield ulcer in VKC treated with Topical Cyclosporin A 2%

tarsal plate

- No scarring
- Absence of rose bengal staining pattern
- Shield ulcer with white plaque
- Prompt response to topical steroids

Management

Management of VKC is primarily aimed at alleviating symptoms. The most effective but least practical is to prevent exposure to the allergen. Since this is not usually possible, instruct patients to frequently use cold compresses, artificial tears soothe to lubricate and wash away the allergens.

Definitive therapy of VKC includes the use of topical vasoconstrictors, antihistamines, inhibitors of mast cell degranulation, and corticosteroids alone or in combination according to severity.

Topical vasoconstrictors:

These drugs cause vasoconstriction, retarding the release of the chemical mediators into the tissues from the blood stream. This reduces hyperemia, chemosis and other symptoms.

(a) *Phenylepherine HCL* 0.12% solution 2 to 4 times/day.

(b) *Naphazoline hydrochloride* 0.012 to 0.1% ophthalmic solution 3-4 times/day.

Topical antihistamines:

Antihistamines decrease itching by virtue of their effect on H_1 activity. In combination with vasoconstrictors, they provide adequate control in mild conditions and delay the use of topical steroids.

(a) First generation topical antihistaminic:

Pheniramine maleate 0.3% solution in combination with 0.025% nephazoline HCL 3 to 4 times/day. *Pyrilamine maleate* 0.1% ophthalmic solution with 0.12 percent phenylephrine HCl 3-4 times/day.

(b) Second generation topical antihistaminic: *Levocarbastine hydrochloride* 0.05% ophhthalmic suspension 3 to 4 times/ day.

Topical mast cell stabilizers:

Cromolyn sodium is a synthetic compound, which dramatically decreases the disease activity and often eliminate the use of topical steroids. 2% or 4% ophthalmic solution 4-6 times/day.

Lodoxamine tromethamine is more potent than cromolyn sodium. 0.1% ophthalmic solution 4 times/day.

Nedocromil sodium is used as 1 to 2% solution 3 to 4 times/day.

Azelastine is a phthalazine derivative drug prevents release of contents of mast cells and also has potent H_1 antagonist activity. 0.025% soution 3 to 4 times a day

Olapatidine is new topical ocular dibenzoxepin derivative drug with promising role. It inhibits the release of preformed and newly synthesized inflammatory mediators from mast cell upon antigenic challenge and also exerts antihistaminic effects.[1] The dual action renders the drug superior in terms of clinical effectiveness, rapid onset and length of action. 0.1% solution 3 times/day.

Ketotifen fumarate antagonize calcium, blocking the rise in intracellular calcium, which is necessary for degranulation. It blocks histamine H_1 receptors and prevents eosinophil accumulation.[2] 0.5% solution 3 times / day.

Topical non-steroidal antiinflammatory drug:

Ketorolac inhibits cyclooxygenase responsible for production of production of prostaglandins. 0.5% solution 4 times / day

Topical corticosteroid:

There is dramatic response to topical corticosteroids in patient of VKC but it should be used with caution because of potential risk of superinfection. Also, steroid induced cataract,

Clinical Features of Vernal Keratoconjunctivitis

Symptoms	Intense itching, hyperaemia, watering,
Signs	ropy discharge, photophobia.
Discharge	Scanty, thick ropy, lardaceous
Palpebral form	Generalized conjunctival hyperaemia, papillae in upper tarsal border, cobble-stones.
Limbal / bulbar form	Thickening, broadening and opacification of limbus, solitary/ multiple/ring of gelatinous tissue, Horner-Tranta's dots.
Cornea	Superficial puntcate keratitis, shield ulcer

glaucoma and delayed wound healing are known complications with the topical use of corticosteroids. Morever the steroids do not immediately resolve the cobbles. *Topical pulse therapy used six to eight times per day, for up to 1 week, followed by rapid tapering to the lowest levels gives better control.*

Topical immunosuppressant:

Cyclosporine A is an immunomodulator that specifically inhibits CD4+ T lymphocyte production via inhibition of interleukin –2 receptor expression. Cyclosporine A also has inhibitory effects on eosinophil and mast cell activation and release, which is important in control of inflammation in VKC.[3]

Cyclosporine A 2 % in artificial tears or olive oil given 4 times per day for three months in combination with mast cell stabilizers in recalcitrant VKC improves the signs and symptoms.

Management of cobblestones :

Supratarsal injection of corticosteriods[5]

Supratarsal injection of dexamethasone sodium phosphate 2 mg, triamcinolone acetonide 10.5 mg, hydrocorticone sodium succinate 50 mg were found to be equally effective in temporary suppression of severe inflammation associated with VKC.

Cryotherapy

It should be avoided as this result in scarring of the conjunctiva that can lead to lid and tear film abnormalities.

Excision

Excision of giant papillae with intraoperative use of Mitomycin C can be tried in severe and refractory cases of VKC with corneal lesions.

Immunosuppression

Topical cyclosporine A 2 % four times /day is an effective treatment for cobblestones[5] *(fig.4)*

Management of shield ulcer

Patching with topical antibiotic-steroid combinations is highly effective, however keratectomy should be avoided. Topical cyclosporine A 2% four times /day is a better alternative. *(fig.6)* Seconadry infection should be treated as infective keratitis.

Management of mechanical ptosis

Tarsal plate resection can be done if severe ptosis results due to chronic VKC.

Supportive management

It is recommended to use tears substitute, oral antihistamines, goggles and limiting the outdoor activity of the patient.

Duration of treatment depends upon the type of therapy given, and response to the treatment regime.

Ideally in the *acute phase* topical diluted corticosteroids as pulse therapy along with topical mast cell stabilizer should be given to control the inflammation. Following which topical mast cell stabilizer with tears substitute to be continued for atleast three months.

For the treatment of the *chronic phase* of VKC, use of topical mast cell stabilizer along with topical immuno-suppression is advocated to control the inflammation till the patient is free of symptoms or for a maximum of six months along with tears substitute.

Always do the baseline and repeated intra-ocular pressure checkup and fundus evaluation in patients on topical corticosteroids therapy.

Mild VKC

- Topical vasoconstrictor (Naphazoline) + Topical antihistamines (Pheniramine maleate) in combination

or

- Topical antihistamines (Levocabastine)

Moderate VKC

- Topical antihistamine (Levocabastine) and NSAID (Ketorolac)

or

- Topical mast cell stabiliser (4% cromolyn sodium/ Lodoxamine tromethamine/Olopatidine/ Ketotofen fumarate Axelestive)

Severe VKC

- Topical corticosteroids as pulse therapy

or

- Topical mast cell stabiliser (Lodoxamine tromethamine, Olopatidine or Ketotofen fumarate)

Recalcitrant VKC

- Topical mast cell stabiliser with topical immunosuppressant. *(fig.5)*

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DOS QUIZ NO. 11

1. Drug used in treatment of Essential Blephasospasm. .....
2. What is the probability of another break if one retinal break is present? .....
3. What is the frequency used in Ultrasound biomicroscopy? .....
4. What is the thickness of posterior capsule of Human crystaline lens in centre? .....
5. Why negative cylinder is preferred in refraction / prescription? .....
6. Most common infective organism in neonatal conjunctivitis .....
7. Most common cause of internuclear ophthalmoplegia in young patients .....
8. Most common ocular opportunist in patients with AIDS .....
9. Most common systemic association of Angiods streak .....
10. Most important immediate management of Chemical burn to eye is .....

Rules:

- Please send your entries to the DOS office latest by 10th August, 2004.
- Prize Rs.500 / - *Courtesy:* ***Syntho Pharmaceuticals***

ANSWERS OF DOS QUIZ NO. 9

1. Downbeat Nystagmus is associated with which disease ..... Cervicomedullary junction (Arnold Chiari malformation)
2. Perineural invasion is seen in which lacrimal gland tumour..... Adenoid Cystic Carcinoma
3. What is the treatment of choice of DVD ..... Superior Rectus Recession ± Faden
4. What are the contraindications for Brimonidine ..... Parkinsonism, Infants
5. What is the inheritence of choriodermia ..... X-linked recessive
6. Most radio resistant structure of the eye is ..... Sclera
7. Indication of systemic steroid in Herpetic eye disease ..... Immunocompromised, Infants, Zoster Ophthalmicus
8. Indications of treatment in Ocular Toxoplasmosis ..... Immunocompromised, Severe Vitritis, threatening major vessel, disc or macula
9. Which stereopsis test does not require special spectacles ..... Langs
10. Infantile esotropia is associated with which type of nystagmus..... Manifest Latent

FORTHCOMING EVENTS

INTERNATIONAL

XXII Congress of the ESCRS
Temple House, Road
Blackrock, Co Dublin, Ireland
Date : 18-22 September, 2004
Venue : Paris, France
Contact: ESCRS
Tel : +353-1-209-1100 Fax : +353-1-209-1112
Email : escrs@agenda-comm.ie
Web : www.escrs.org

American Academy of Ophthalmology
23-26th October, 2004
New Orleans, LA, USA
American Academy of Ophthalmology
Tel : + 1-415-561-8500 Ext. 304
Fax : + 1-415-561-8583
Web : www.aao.org

The 20th Asia Pacific Academy of Ophthalmology Congress
27-31st March, 2005
Kuala Lumpur, Malaysia
The 20th Asia Pacific Academy of Ophthalmology Congress
Tel : +603-7956-3113 Fax : +603-7960-8297
Email : scretariat@apao2005.com.my
Web : www.apao2005.com.my

5th International Glaucoma Symposium
20th March, 2005 – 2nd April, 2005
Cape Tow, South Africa
Contact : Kenes International
Tel : +41-22-908-04-88 Fax : +41-22-7322850
Email : glaucoma@kenes.com
Website : www.kenes.com/glaucoma

ASCRS/ASOA Meeting Congress
16-20th April, 2005
Washington, DC
Contact : ASCRS
Tel : +1-703-591-2220 Fax : +1-703-591-0614
Web : www.ascrs.org

NATIONAL

63rd All India Ophthalmological Society Conference
13-16th January, 2005
Contact : Dr. B. K. Tripathy, Organising Secretary,
Bimal Tripathy Lane, Mahatab Road,
Cuttack – 753001, Orissa
Ph : 0671-2310111, 2332483 Fax : 0671-2330111
E-mail : bimaltripathy@sify.com

Monthly Meetings Calendar For The Year 2004-2005
1st August, 2004 (Sunday) **Army Hospital (R&R)**
28th Auguest, 2004 (Saturday) Sir Ganga Ram Hospital
25th September, 2004 (Saturday) Hindu Rao Hospital
30th October, 2004 (Saturday) R.P. Centre for Ophthalmic Sciences
21st November, 2004 (Sunday) **DOS Midterm Conference**
27th November, 2004 (Saturday) Dr. Shroff's Charity Eye Hospital
18th December, 2004 (Saturday) Venu Eye Hospital & Research Centre
29th January, 2005 (Saturday) Safdarjung Hospital
26th February, 2005 (Saturday) M.A.M.C. (GNEC)
26th March, 2005 (Saturday) Mohan Eye Institute

DOS Midterm Conference
21st November, 2004
Contact : Dr. Jeewan S. Titiyal, Secretary DOS
R.No. 476, 4th Floor,
Dr. R.P. Centre for Ophthalmic Sciences
AIIMS, Ansari Nagar, New Delhi – 110029
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DELHI OPHTHALMOLOGICAL SOCIETY

(LIFE MEMBERSHIP FORM)

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S/D/W/o _____ Date of Birth _____

Qualifications _____ Registration No. _____

Sub Speciality (if any) _____

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Clinic/Hospital/Practice _____

_____ Phone _____

Residence _____

_____ Phone _____

Correspondence _____

_____ Phone _____

Email _____ Fax No. _____

Proposed by

Dr. _____ Membership No. _____ Signature _____

Seconded by

Dr. _____ Membership No. _____ Signature _____

[Must submit a photocopy of the MBBS/MD/DO Certificate for our records.]

I agree to become a life member of the Delhi Ophthalmological Society and shall abide by the Rules and Regulations of the Society.

(Please Note : Life membership fee Rs. 3100/- payable by DD for outstation members. Local Cheques acceptable, payable to Delhi Ophthalmological Society)

Please find enclosed Rs._____in words _____ by Cash/

Cheque/DD No._____ Dated_____ Drawn on_____

Signature of Applicant with Date

Three specimen signatures for I.D. Card.

FOR OFFICIAL USE ONLY

Dr._____has been admitted as Life Member of the Delhi Ophthalmological Society by the General Body in their meeting held on_____

His/her membership No. is _____. Fee received by Cash/Cheque/DD No._____ dated_____ drawn on _____.

(Secretary DOS)

INSTRUCTIONS

1. The Society reserve all rights to accepts or reject the application.

2. No reasons shall be given for any application rejected by the Society.

3. No application for membership will be accepted unless it is complete in all respects and accompanied by a Demand Draft of Rs. 3100/- in favour of "Delhi Ophthalmological Society" payable at New Delhi.

4. Every new member is entitled to received Society's Bulletin (DOS Times) and Annual proceedings of the Society free.

5. Every new member will initially be admitted provisionally and shall be deemed to have become a full member only after formal ratification by the General Body and issue of Ratification order by the Society. Only then he or she will be eligible to vote, or apply for any Fellowship/Award, propose or contest for any election of the Society.

6. Application for the membership along with the Bank Draft for the membership fee should be addressed to Dr. Jeewan S. Titiyal, Secretary, Delhi Ophthalmological Society, R.No. 476, 4th Floor, Dr. R.P. Centre for Ophthalmic Sciences, AIIMS, Ansari Nagar, New Delhi – 110029.

7. Licence Size Coloured Photograph is to be pasted on the form in the space provided and two Stamp/ Licences Size Coloured photographs are required to be sent along with this form for issue of Laminated Photo Identity Card (to be issued only after the Membership ratification).