EDITORIAL

Dear Colleagues,

As the festive season comes to an end the season of conferences begin with our very own DOS Midterm meeting. You all must have received the Midterm brochure by now & I am sure each & every one is eagerly looking forward for the meet just as I am. This issue of DOS Times comes with a mix of old & new topics which all of us would like to go through once in a while. DOS Times is quite popular among students of Ophthalmology because of the fact that important topics of academic and those of clinical importance in day to day practice are dealt in a simple and concise manner.

I would also like to invite non-institutional DOS members to actively participate in clinical talk & case presentation in our monthly meetings.

See you all on 21st November, 2004 at India Habitat Centre.

Dr. Jeewan S. Titiyal

DOS MONTHLY CLINICAL MEETING FOR NOVEMBER, 2004

Venue : **Conference Hall, Dr. Shroff's Charity Eye Hospital,**
Daryaganj, New Delhi
Date & Time : 27-11-04 **(Saturday)** at 2:30 PM

Case Presentation

1. Unusual complication of a dislocated pseudophakos .....: Dr. Umang Mathur (10 Min)
2. Sterile endophthalmitis due to endotoxins .....: Dr. Suneeta Dubey (10 Min)

Clinical Talk

- Update on ARMD .....: Dr. Manisha Aggarwal (20Min)

Mini Symposium : Recent Advances

Chairman : Dr. B. Patnaik
Co-Chairman : Dr. Noshir M. Shroff, Prof. J.C. Das

1. Adjustable suture techniques in strabismus surgery.....: Dr. Suma Ganesh (15 Min)
2. Tube implants in glaucoma .....: Dr. Suneeta Dubey (15 Min)
3. Newer phaco technologies .....: Dr. Umang Mathur (15 Min)

Discussion - 15 Min

Tribute to Prof. (Dr.) L.P. Agarwal

This is to pay my gratitude for the deep feelings and sympathy at this hour of grief of mine and my family which includes my only daughter and son-in-law. Dr. Agarwal's end came suddenly in the evening of 24th September, 2004. He was working till end. It was a sudden end. This is a saintly death. No time was left for medical aid.

Dr. Agarwal is well known for his vast contribution in Ophthalmology in developing it and in sending the message far and wide. He founded a world known center, Dr. R. P. Centre at the All India Institute of Medical Sciences, New Delhi where many sub-specialties were developed adding to Scientific Research and teaching advances and new dimensions were added to Ophthalmology. The students were made to work very hard, as a result they showed brilliance in all aspects including other sub-specialties like Pathology, Pharmacology and Bio-Chemistry etc. wherever they went, here or abroad. They are spread throughout the world.

He was a visionary and could see many things ahead of his times. He was associated with many Institutions and Organisations of Spain, U.K., U.S.A., Africa, Pakistan, Sri Lanka, Japan either as a member, editor, or visitor. He remained a Member of International Council of Ophthalmology, Life Fellow of International Academy of Ophthalmology.

He served as chairman of Steering Committee of WHO for development of Global Programme of Prevention of Blindness as well as Regional Committees of WHO for Prevention of Blindness.

His contribution in developing a Programme for Prevention of Blindness and alleviation of blindness as advisor in the Ministry of Health which has been named as "National Programme for Prevention of Visual Impairment and Control of Blindness", was adopted throughout the country and abroad.

He penned about 22 books in various disciplines of Ophthalmology and more than 350 research papers in National and International Journals. They are quoted in books, journals and seminars. He was associated with various journals, National and International, as editor or member like "Ophthalmogica" and "Vision".

Dr. Lalit P. Agarwal was Ophthalmology and Ophthalmology was his.

He founded a Federation of Ophthalmology roping in many Ophthalmologists throughout the country and enthused in them the zeal for public work and training of the doctors and paramedical staff.

Since some time past he was devoting himself to producing paramedical staff throughout the country through the Federation of Ophthalmic Research and Education Centres. The course includes not only Optometry and refraction but many advances in Modern Ophthalmology besides basic Pharmacology, Anatomy, Physiology and Microbiology. He developed in the students an allround personality in dealing with public, theatre work, enterpreneurship, accounts and computer education. This is an example of vision far ahead of the time. He is termed the "father of Modern Ophthalmology" in Asia and other countries. He will be remembered as such. He was a man of action throughout his life and a great visionary.

It will be an appropriate tribute to him and for the peace of his soul if his mission is carried forward.

Thanking you,

Dr. (Mrs.) Savitri Agarwal
W/o Dr. (Prof.). L.P. Agarwal
And family
Dr. Kavita A. Sharma (Daughter)
Mr. J.C. Sharma (Son-in-law)

Phaconit-without Surge

Roop MD, **Sangeeta** MS

Whenever there is break of occlusion during phacoemulsification sudden outflow of fluid occurs from anterior chamber (AC). If not compensated by adequate inflow in the AC, it leads to momentary collapse of AC. This is what we call surge. Importance of surge is ever increasing because of use of higher vacuum and less phaco power to consume nuclear pieces in conventional phaco. Now with availability of phaco systems in which not much heat is generated at phaco tip (AMO Sovereign Whitestar, STAAR Sonic, Alcon Neosonix and Aqualase and Dodick Nd-YAG laser photolysis)[1] and IOLs which can be inserted through sub 2mm incisions (Acri.Smart, Thin optX (Ultrachoice), Medennium Smart IOL, Microl)[2], sleeveless phaco or phaconit or bimanual phaco or micro incision cataract surgery is being practiced more frequently now. However, it is the presence of surge, which is preventing most of the ophthalmic surgeons to convert to phaconit. In the present article we attempt to analyze causes of surge in phaconit and find innovative ways to prevent it on the basis of our experience of phaconit.

Compliance of Tubing

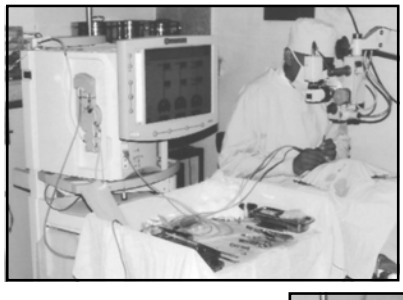
The compliance of tubing is one of the most well recognized cause of surge. By compliance of tubing we mean that when occlusion occurs at the phaco tip the vacuum rises in the aspiration tubing. Due to the compliance, tubing, walls collapse partially and as occlusion breaks these come to their normal shape causing sudden outflow of fluid from AC, which is for a short period of time much more than the maximum flow-rate we have set in the machine. To make tubings less compliant thicker walled tubings are now being used which allow us to use higher vacuums. To further reduce surge due to tubing's compliance we employ an innovative way. We have changed the way we place our phaco system in relation to the instrument trolley and the patient's head in such a way that we can work with half of the original length of the tubing supplied by the manufacturer. Using the half the length of the aspiration tubing we can reduce the compliance of the tubing to less than half. It allows us to increase the maximum vacuum settings by at least 50 mm / Hg without any corresponding increase in the surge. It has proven to be the most simple and least cumbersome way to reduce surge without increasing any cost. The most important thing is that this innovation would work with all machines, all types of pumps, all sets of parameters, all types of cataracts and all kinds of techniques used for phaco.

Roop Netralaya
Run by : Meerut Laser and Eye Care Centre Pvt. Ltd.
Opp. N.A.S. College, E.K. Road, Meerut-250 001

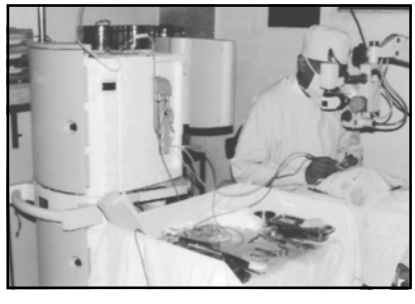
This simple innovation not only helps us in reducing surge, it also makes the whole system more efficient. As the effect of changes occurring in the AC needs to be transmitted to the sensor located in the console through the tubing and the corresponding changes in pump's speed takes some time for its effect to come in AC. The longer the tubing the more the time lag. Smaller tubing reduces this time lag and the sensor and pump located in the console become more responsive to surgeon's commands by the foot-switch. All the positive features of a particular system controlling fluidics become more effective. By reducing the length of aspiration tubing even the fluidics of lower end machines becomes more effective.

The Kick of the Pump

Another important cause, which has not been adequately emphasized, is the kick of the pump. By kick we mean the initial thrust that pump makes to overcome the inertia of static state. Just to understand the importance of this phenomenon as far as surge is concerned, put the test chamber over the handpiece. Now fill the test chamber with fluid and go to foot pedal position 2. Now pinch the aspiration tubing near its end at the peristalsis pump. Allow the vacuum to build to the maximum set limit. When the maximum vacuum is reached and pump stops release the tubing. One has to remain in foot pedal position 2 throughout. As the tubing is released the test chamber collapses. This part of surge is caused by the kick of the pump as we have avoided the part of the surge caused by tubing by pinching the tubing at the end of its aspirating part. Now one can vary the flow rate and repeat the experiment and realize that



Compliance of tubing



Reducing the surge by decreasing the length of the tubing

higher the flow rate greater the kick is. This means that the kick of the pump is proportionate to the flow rate set and is responsible for much of the post occlusion surge we see in our phaco surgery.

To avoid the surge caused by the kick of the pump during phaconit we make use of the occlusion mode of our phaco system (The Sovereign Whitestar). The occlusion mode allows the surgeon to have two sets of parameters, which automatically change, at a preset level of vacuum called the threshold. During our conventional phaco surgery we keep the flow rate at 24cc /min which changes to 30cc/min at the threshold vacuum of 100mm of Hg. The highest vacuum is set at 350mm of Hg. At these parameters routine phaco is done speedily without any surge and same parameters are used for chopping and also for consuming the chopped nuclear pieces. But same parameters cannot be used safely in phaconit even with half the size of aspiration tubing. For phaconit we use different parameters for chopping nucleus into 6 pieces and different parameters to consume chopped nuclear pieces. For chopping, the flow rate is kept at 12cc/min which changes to 24cc/min at threshold vacuum of 100mm of Hg.The maximum vacuum is kept at 300mm of Hg.This gives good hold for chopping while not increasing the rise time significantly. As during chopping the occlusion breaks gradually there is no surge even at 300mm of Hg vaccume. For consuming the nuclear pieces the flow rate is kept at 24cc/min to give good followability. The threshold vacuum is set at 250mm Hg at which level the flow rate changes to only 8cc/min. The highest vacuum is 300mm Hg.Till 250mm Hg the rise time is fast then nuclear pieces start being sucked and as the flow rate drops most of the nuclear pieces get sucked before highest vacuum is reached or pump stops. In this way surge due to kick of the pump is all together avoided. Even if the pump stops due to reaching highest vacuum the kick is mild due to low flow rate of just 8cc/min.

Phaco Power and Surge

Another observation we have made and which is not yet discussed in literature is the relationship of phaco power with surge. To appreciate it, while consuming relatively softer nuclear pieces, keep all other parameters same and consume one piece with 40% phaco power and another similar piece with 10% phaco power and one can find that post occlusion surge is more when higher power is used. The explanation for this observation is that with low phaco power larger pieces of nucleus get sucked into the tubing and hence offer greater resistance to flow till they are cleared from the other end of the tubing thus causing less surge. While when we use higher phaco power the nuclear pieces are emulsified into smaller pieces quickly and they offer less resistance to fluid flow in the tubing causing more surge. To take advantage of this phenomenon during phaconit we set the phaco power at 30% continuous, which is used just to impale the nuclear fragment and which at threshold vacuum of 100mm Hg changes to 3 long pulses per second with whitestar on with 33% on duty cycle. Thus even pulses are composed of micro pulses with the time interval between micro pulses being double of each micropulse.As phaco power is also linearly controlled, the foot pedal is depressed to the minimal level in position 3 to further minimize the use of phaco power.

Discussion

To reduce surge in phaconit other modalities used are use of air pump[3], use of Star cruise control device[4] and use of posterior segment system[5] to work as air injector.

The use of air pump allows more fluid to flow in AC to avoid surge. It is an effective way to control surge but it involves use of one extra gadget. The AC at times becomes very deep due to high positive pressure and it may be deleterious in patients with compromised optic nerve perfusion and patients with weak zonules. With the use of air pump the tunnel needs to be slightly long reducing the maneuverability of instruments in AC and also distorts the cornea reducing the visibility. Another disadvantage that we have experienced is that when pump is put off due to positive pressure in the bottle the fluid comes back in the tubing of the pump causing frequent breakdown of the pump. Due to these problems the air pump has not become very popular.

Another alternative is the use of Star Cruise Control devise. Its cost and disposable nature are obvious disadvantages. Apart from this as it works on the principle of reducing the smallest aspirating internal lumen to 0.3mm it reduces the actual outflow from AC causing the reduction in followability to a significant extent. One can do a simple experiment to understand this. By collecting the fluid aspirated at the outflow end of the tubing, we measured the fluid using 20 gauze phaco tip and using 0.3mm I/A tip. When we set the flow rate as 40cc/min the actual fluid collected with I/A tip is less by about 66% as compared with the phaco tip. As the actual outflow from AC is responsible for the followability during surgery the smaller internal aspirating lumen decreases the followability during surgery for the same settings of flow rate set in the machine.

Now even the use of posterior segment machine - Accurus Surgical System has been described for injecting air into the bottle in place of separate air pump but the availability of this machine with cataract surgeons is a limitation.

Thus reducing the length of the aspirating tubing to the minimum required, judicious use of occlusion mode in our existing machines and minimum use of phaco power are the most cost-effective and least cumbersome way to reduce surge in phaconit.

Till today we were trying to reduce surge by reducing the compliance of tubings by making the changes in the wall of the tubing, but nobody thought of reducing the length of the tubing to reduce the total compliance of the

tubing. The simplicity, the cost effectiveness and the unquestionable effectiveness of this innovative concept can be used to improve our existing machines. This also gives an idea to phaco machine manufacturers to design consoles in such a way that the length of aspiration tubing can be minimized. It is helpful even in conventional phaco and surgeons with lower end machines can also increase their maximum vaccume settings by reducing the length of aspiration tubing.

Our observations also emphasize the importance of kick of the pump in causing post occlusion surge. We have also suggested the use of occlusion mode of our machines to avoid surge caused by the kick of the pump. May better software be developed in future to take care of this kick of the pump.

References

1. Bovet J,Chiou A,Mehta CK.Phaconit Bimanual Phacoemulsification.Journal of Intraocular Implant and Refractive Society,India2004;1(3):7-11
2. PandeySK,WernerL,MamalisN,OlsonRJ.Ultrasmall Incision Intraocular Lenses.Journal of Intraocular Implant and Refractive Society,India 2004;1(3):29-33
3. Agarwal A, Agarwal S, Agarwal A,et al. Antichamber collapser. J Cataract Refract Surg 2002;28: 1085-1086
4. Chang F C. 400 mm Hg High-Vacuum Bimanual Phaco Attainable with the Staar Cruise Control Device. J Cataract Refract Surg 2004;30:932-933
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CASE REPORT

Central Retinal Vein Occlusion (CRVO) In a Young Patient

D.N. Saksena, M.S.

Introduction

Central Retinal Vein Occlusion (CRVO) is a condition in which central retinal central retinal vein is compressed by arteriosclerotic central retinal artery where the two shares common sheath (adventia) i.e. just behind lamina cribirosa and at arteriovenous crossings. Here a case of CRVO in young patient is reported.

A 25 year old male patient presented in eye clinic with complaints of disturbance of vision in right eye of approximately one week duration. There was no history of pain in right eye and previous episodes of such complaints. Ocular examination revealed a best corrected visual acuity of 6/12 in right eye and 6/6 in left eye, normal papillary reactions and intraocular pressure 17.3 mm Hg in both eyes. Fundus examination showed generalized tortusity of retinal veins and occasional flame shaped fresh hemorrhages along retinal veins. No retinal or macular oedema except foveal reflex was indistinct. In view of these symptoms and findings of transient visual obscurations, provisional diagnosis of impending retinal venous occlusion was thought off and patient was put of tablet Aspirin (100 mg.) one tablet daily. The relevant laboratory investigations were requested like bleeding time, clotting time, Hb, TLC, DLC. ESR, Platelet count, Mauntex test, X-ray chest. Patient was asked to come for follow up after one week. In the mean time results of requested investigations were received and all were within normal limits. On follow up visit, patient reported further detoriation in vision in right eye. On examination visual acuity in right eye was 6/36 and left eye 6/6. Fundus exam showed extensive flame shaped dot and blot hemorrhages in all quadrant of retina with occasional soft exudates. There was moderate degree of macular oedema and mild disc swelling. This was typical picture of central retina vein occlusion (CRVO) clinically. In view of extensive hemorrhages Flourescein Angiography was delayed to a later date.

In view of possible causes of CRVO in young patient, presenting symptoms, fundus findings and associated gross diminution of visual acuity in right eye, patient was prescribed tablet Prednisolone (10 mg.) one tablet q.i.d. for one week and tablet aspirin (100 mg.) one tablet daily for three weeks. On subsequent follow up, patient was looking happy and on examination his vision in right eye improved to 6/9 and left eye 6/6. Fundus examination showed almost 90% resolution of retinal hemorrhages and macular and disc oedema. Flourescein angiography revealed prolongation of retinal circulation time with compromised retinal capillary permeability and minimal non perfusion areas in retina. This also suggests that CRVO is of non ischaemic type. In the next follow up visit after one month, it was found that patient's vision was maintained and stabilized at 6/9 in right eye with no recurrence and fundus picture further improved. There was no neovascularization of iris and therefore laser photocoagulation was not required as patient responded well to medical treatment.

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Discussion

Central retinal vein occlusion (CRVO) is caused by compression of central retinal vein by atherosclerotic central retinal artery where the two share the common adventia i.e. just behind lamina cribirosa and at arterio venous crossings. This leads to turbulence, thrombus formation and endothelial damage. Typical picture of CRVO consist of dilated tortuous retinal veins, intra retinal hemorrhages, dot and blot and flame shaped fiery looking fundus picture along with retinal and disc oedema. Predisposing factors in elderly are age (6th and 7th decade), associated systemic diseases like hypertension, diabetes mellitus and also associated primary open angle glaucoma. In young patients like this one, causes are blood dyscrasia by causing either hypercellularity (Leukaemias, polycythaemia) or change in plasma proteins (macroglobunaemias), sickle cell disease, periphlibitis (Bechets, diseases, sarcoidosis). This patient was young and all investigations were normal, therefore no specific cause could be identified.

CRVO is of two types.

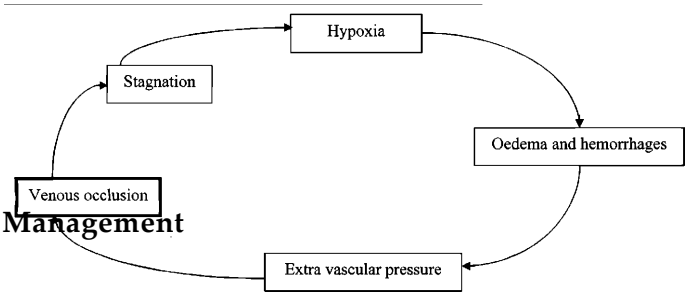
a. Non ischaemic

b. Ischaemic

Non ischaemic type of CRVO can convert to ischaemic type so visual prognosis should be guarded as vision in latter type is much compromised.

Effects of Venous Occlusion on Retinal

Circulation



Management

Regarding management of CRVO in non ischaemic type, no treatment is effective but in this case medical treatment did help and laser photocoagulation is helpful only if iris revascularization is present according to CRVO study group. In this particular case improvement in visual acuity is either due to spontaneous resolution due to development of collateral circulation or as effectiveness of medical treatment.

Differences between non-ischaemic and ischaemic type of CRVO

Sl.No.	Non ischaemic	Ischaemic
1.	Also known as Venous Stasis Retinopathy or Incomplete CVRO	Hemorrhagic or complete CRVO
2.	More common 75%	Less Common
3.	Visual acuity moderately reduced and final visual out come good.	Final visual acuity not good usually less than 6/60
4.	RAPD- Minimal	Maximum due to retinal ischaemia
5.	Rubiosis Iridis- Not present	Present
6.	Fundus minimal soft exudates	Maximal soft exudates & extensive hemorrhages.

Conclusion

I thought of reporting this case as patient was young and CRVO is uncommon in young patients and secondly all investigations were normal still patient responded very well to medical treatment and finally this patient presented as case of impending CRVO and went on to develop as full blown case of CRVO in spite of starting treatment well in time.

Reference :

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Corrigendum

In the last issue of DOS Times (Oct, 2004 Vol. 10 No. 4) the article titled "Chemical Injuries of the eye" by Dr. Tishu Saxena et. al. had a table of Ropper- Hall (Ballen) Classification, which was not complete. We are printing the complete table for the benefit of our readers. The error is regretted.

Editor DOS Times

(Table 1) **Ropper -Hall (Ballen) Classification**

Grade	Prognosis	Cornea	Conjunctival limbus
I	Good	Corneal epithelial haze	No limbal ischaemia
II	Good	Corneal haze, iris details visible	No limbal ischaemia
III	Guarded	Total epithelial loss, stromal haze, iris details obscured	1/3-1/2 limbal ischaemia
IV	Poor	Cornea opaque ,iris and pupil obscured	>1/2 limbal ischaemia

Roper- Hall MJ. Thermal and chemical burns. Tras Ophthalmol Soc UK 1965; 85: 631-61.

Dark Adaptometry

Parul Sony, MD, **Vandana Kori,** BSc, **Pradeep Vankatesh,** MD.

Exposure of an eye to a bright light reduces the visual sensitivity of retina. In dark the retinal sensitivity to light shows a marked increase. Out of rods and cones, rods (containing rhodopsin pigment) are extremely sensitive to low levels of illumination and are responsible for dark-adapted vision or the **scotopic vision** and the cones are responsible for the **photopic vision** or the light-adapted vision.

The scotopic vision is characterized by

- A high retinal sensitivity to light (1000 times higher than sensitivity in light adapted state),
- Low resolution visual acuity.
- Absence of color perception.

The photopic vision is characterized by presence of

- Low sensitivity to light
- A high resolution visual acuity
- Presence of color perception.

Light exposure results in depletion of photopigment by bleaching. Rhodopsin is decomposed in bright light, making the rods nonfunctional. Dark adaptation requires biochemical regeneration of rhodopsin pigment requiring expenditure of metabolic energy. It has been shown that the recovery of visual sensitivity in dark corresponds well with curves of rhodopsin regeneration. As rods are absent in the foveal region scotopic vision shows a relative foveal depression of visual sensitivity. The visual sensitivity is maximum 7^{o} away from fixation where the density of rods is maximum.

Light minimum or absolute threshold is the minimum intensity of light that an eye can see and perceive under a dark-adapted state. To measure threshold perception in a dark adapted eye initially a relatively bright light is projected and then the duration of its exposure is adjusted on the basis of repeated trial so that an observer can detect only 50% of its presentation. It can be calculated by obtaining mean of threshold of appearance and threshold of disappearance in an eye, dark adapted for at least 40 minutes. The duration of the flash is altered in proportion to the change in luminance. Generally an intense light need not remain for a long duration as a weaker light requires.

Dark adaptation refers to the ability of the visual system, (both rods and cones mechanisms) to recover sensitivity following exposure to light. Whenever a subject is taken from a bright light to a dark room the retinal sensitivity changes and this change/ or recovery follows a characteristic pattern known as course of dark adaptation. The recovery is faster in the cones as compared to rods. However, the absolute level of sensitivity is greatest in the rods.

Instruments called **photometers or adaptometers,** which can control and vary the amount of light to a definite and known extent are used to record the course of dark adaptation. Most primitive model that was initially used was photometer of Richard Forster. Hemispherical adaptometers are used nowadays (Goldman-Weeker by Haag Streit). Goldmann-Weeker adaptometer has various advantages such as it can be used both for binocular/ uniocular testing, it can test entire retina/ only macula/ or any specific peripheral part of retina. It allows determination of absolute threshold of sensitivity; and has good objectivity.

The absolute threshold values of dark adaptation depend on following variables:-

- State of adaptation (preadaptation)
- Stimulus variables (wavelength)
- Methodological variables
- Variability of the sensitivity of the retina depending on retinal location

Pupillary size affects the amount of illumination entering into the eye and therefore has important impact on the measurement of dark adaptation. It is best to fix the pupil with pilocarpine (3 drops of 1% pilocarpine at 3 minutes interval) and measure it both prior to and after the completion of the test. Depending on the pupillary size the correction factor (by Reeve et al) is used to know the exact intensity of light used.

Temperature of illumination should also be kept constant. Therefore the changes in light intensity are made by filters or diaphragm and not by a rheostat that can alter the temperature of the light.

Dark adaptation takes about 15-30 minutes. It has two mechanisms

1. Neural adaptation which is fast and represents reversal of neural light adaptation of photoreceptors and other retinal cells.
2. Photochemical adaptation is a slow phenomenon involving pigment regeneration.

Technique

- Pupil is fixed with pilocarpine
- Preadaptation / light adaptation of eye is performed by 10 minutes of pre-exposure of the eye to a diffusely

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illuminated hemisphere of the adaptometer with a standard and constant illumination of 1500 miliambert. This bleachs the photoreceptor pigments.

- The light is turned of and threshold sensitivity is determined immediately by presenting a series of flashes of light 7° - 10 ° below fixation. The intensity of the flashes is controlled by neutral density filter. The readings are repeated every few minutes till a point is reached when no further rise in sensitivity is recognized. The results are plotted as log units of brightness against time. A biphasic sensitivity curve is obtained. The initial rapid segment represents a cone function and the second slower segment represents the rod function. The kink on the curve where the rod segment begins is called rod-cone break (alpha point).

Normally the whole process of dark adaptation requires 15-30 minutes. The alpha-point occurs after 7-10 minutes of dark adaptation. The threshold measured on the cone and rod plateaus are the absolute thresholds of the cone and rod mechanism.

Dark Adaptation curve varies with the location of the retina where the test is performed. When performed at 5^0 a typical biphasic curve is obtained (Figure 1). At the start the threshold is very high, then it falls rapidly in the initial phase and gradually in the next phase and reaches its lowest value over 30 minutes.

0^0 curve: If the flash of light is focused on foveola only cone plate is obtained as rods are absent at foveola.

Clinical applications

1. Disorders of pigment degeneration
   a. Vitamin A deficiency: Threshold for dark adaptation is increased for both rods and cones with a decrease in serum Vitamin A levels. Time required for dark adaptation is also increased.
   b. Fundus albipunctatus: This is autosomal recessive hereditary disorder where the regeneration time for visual pigment is prolonged. It requires several hours for dark adaptation instead of the usual 30 minutes. The fundus has scattered white dots and the serum Vitamin levels are normal.
2. Disorders of neural adaptation
   a. Oguchi's disease requires hours for dark adaptation. Though the rate of rodhopsin regeneration is normal, the rod arm is prolonged.
   b. Congenital stationary night blindness (CSNB) is characterized by absence of any rod adaptation.

Chloroquine toxicity: Rod limb of the dark adaptation curve is unaffected however the cones are selectively destroyed thus cone limb is absent. **Retinitis pigmentosa** Type II (due to damaged photoreceptor not the visual pigment cycle) shows a prolonged cone -rod break time, normal final rod threshold.

Abnormal dark adaptation curves in tapetoretinal degeneration (Figure2)

- Type I normal
- Type II biphasic curve with cone arm normal and rods segment is delayed.
- Type III rod adaptation never develops
- Type IV monophasic curve and represents the adaptation of foveal cones
- Type V the adaptation of foveal cone is defective

Apart from these other conditions like high myopia, glaucoma and extreme miosis may also result in abnormal dark adaptation function.

Visual field in dark-adapted eye gives an overall survey of functioning of rods. It shows a central scotoma at the fovea. The sensitivity is greatest between 10-200 eccentric to fovea and it again declines in the periphery especially superiorly.

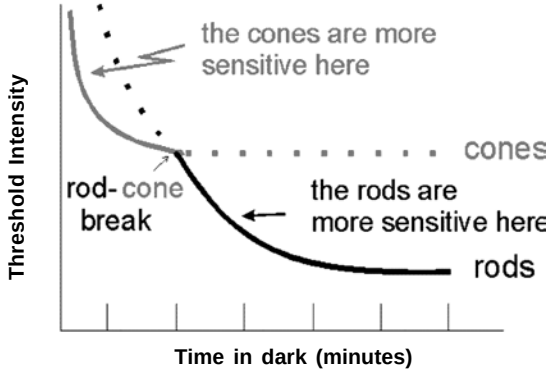

Figure 1: The normal dark adaptation curve

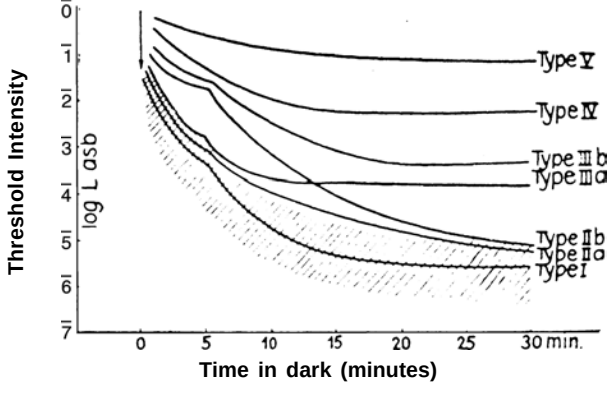

Figure 2: Typical adaptation curves in tapetoretinal degeneration

Suggested Readings

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Diplopia Workup

Harinder Singh Sethi, MD, DNB,FRCS, **Rohit Saxena,** MD

Double vision, also called diplopia, causes a person to see two images of a single object. It is often the first manifestation of many systemic disorders, especially muscular or neurologic processes. The cause of diplopia must be resolved to diagnose and manage it appropriately.

The diplopia can be uniocular or binocular.Binocular diplopia can be differentiated from uniocular diplopia by covering either eye; monocular diplopia persists in one eye despite covering the other eye while binocular diplopia disappears. Causes of uniocular diplopia are a severe corneal deformity or marked astigmatism (keratoconus), a mass or swelling in the eyelid, Dry eye,more than one pupil or opening in the iris, refractive anomalies within the eye (early cataracts or partially displaced lenses as in Marfan syndrome), and retinal abnormalities (macular scarring and distortion). It can be differentiated from the binocular diplopia by following methods a)Monocular occlusion :Uniocular diplopia may be present in each eye (eg cortical diplopia) or in one eye only. Uniocular diplopia persists on covering the other eye and disappears on covering the affected eye.b) Visual acuity with and without pin-hole : Refractive monocular diplopia will normally disappear through a pinhole. c) Amsler chart : Metamorphopsia if present, indicates an association with macula & pathology.

Binocular diplopia is encountered almost exclusively in adults or in those with mature visual systems because children may not be able to express this symptom and the immature visual system deals with diplopia by suppressing the poorer image, possibly resulting in irreversible amblyopia. Children with obvious and marked ocular malalignment from strabismus are comfortable and content because the visual image from the deviating eye is suppressed and not noticed. In contrast, adults who have mature visual processing pathways cannot easily ignore the second image, this manifests as diplopia.

Causes of binocular diplopia are :

Damage to III, IV,VI cranial nerves controlling the extraocular muscles - Nerves can be involved by infection, multiple sclerosis, stroke, head trauma or a brain tumor, Diabetes and hyertension

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Myasthenia gravis.

Graves' disease

Trauma to the eye muscles

Evaluation of a Patient with Diplopia.

A. History

The patient typically presents with a history of double vision, where 1 object appears as 2 objects.

The most important symptom to be elicited is whether the diplopia is horizontal (2 images are side by side) or vertical (2 images are above each other). Oblique diplopia (2 images are separated both horizontally and vertically) should be considered as a manifestation of vertical diplopia.

The patient should be enquired about onset (abrupt or slow), severity, duration, location, associated symptoms, and aggravating and relieving factors. This evaluation is as important as performing appropriate examinations and ordering special tests. Determine if diplopia worsens when the muscles are fatigued (eg, at the end of the day, after strenuous use as in Myasthenia gravis). On presentation of any acute onset diplopia, one of the most difficult but important decisions to make is whether it is of recent onset or due to the decompensation of a long standing deviation. This is especially important in cases of congenital superior oblique palsies. A detailed history of systemic diseases (eg diabetes, vascular disease, or hypertension; headache and other neurologic complaints), as well as a past surgical and medical history should be taken.

History of recent trauma to the face and the head should be ruled out. Blunt injury to the cheek can result in a blow-out fracture of the orbit with hematoma or entrapment of the soft tissues and extraocular muscles, restricting upward and downward eye movement. Blunt head injury is associated with nonspecific sixth cranial nerve (abducens) weakness. Evaluate old photographs to determine if the head posture is long-standing or of recent onset.

B. Examination of a patient with diplopia

Abnormal head posture (AHP)

It often gives clues towards the underlying etiology. It is developed to compensate for an incomitancy by moving the eyes into a position of comfortable binocular vision. The presence of an AHP normally indicates that

the patient has the capacity for good binocular functions. Head posture has three components:

a) Chin elevation or depression (vertical),

b) Face turn to right or left side (horizontal)

c) Head tilt to right or left shoulder (torsional).

The patient prefers a head posture at which the ocular deviation is the least, and image can be fused. For example, the head posture in a case of left superior oblique (LSO) palsy will have chin depression, face turn to the right, and head tilt to the right shoulder. It can be explained as follows :- LSO being a depressor, chin depression occurs, it being an intorter, a head tilt towards the opposite shoulder occurs. And a face turn to the right brings the eyes in abduction so that the vertical movements can be executed by the vertical recti. Thus the head posture ensures that the eye is out of the field of action of the paralytic muscle. Rarely a head posture which causes the maximal deviation is chosen so that the peripheral image can be easily suppressed or ignored.

Lid position

Ptosis of the upper eyelid indicates possible third-nerve lesions, while eyelid retraction suggests thyroid ophthalmopathy.

Pupils

Pupil asymmetry is a sinister sign when associated with diplopia because it indicates involvement of the third cranial nerve (oculomotor nerve).

Cover test

The cover test should be performed, at least initially, using an accommodative target for fixation. Where there is an incomitancy, the angle of deviation normally differs depending on the fixating eye, with the secondary deviation being larger than the primary. The primary deviation is the angle when fixating with the unaffected eye and the secondary deviation is the angle when fixating with the affected eye. The cover test should be repeated with and without any AHP. Normally, the angle of deviation will reduce with the head posture and a previously manifest deviation will become latent. It is necessary to determine whether the diplopia and manifest deviation are constant or intermittent, and whether the angle of deviation varies with fixation distance.

Ocular motility

Complete ocular motility examination should be done. The alternate cover test should be performed in the different directions of gaze to reveal the full angle of the deviation. Subjective responses can be improved by the wearing of red and green goggles. Suppression is less likely and the patient can report on the direction and separation of the images. The use of a vertical bar-light as a fixation target allows for the assessment of torsion, and can be plotted as a 'diplopia chart'.

Mechanical or myogenic versus neurogenic:

Observation of the smoothness of eye movements can help differentiate between a mechanical and neurogenic defect. In a neurogenic lesion, as the eyes move into the direction of the defect, the under-acting eye will move smoothly but more slowly than the normal eye. In a mechanical deviation, the eye movements will be smooth and symmetrical until meeting the obstruction, at which point there will be an abrupt slowing of the defective eye.

Another clue to differentiating a neurogenic from a mechanical lesion is by observing the diplopia. If the distal image changes in opposite directions of gaze (e.g. the image seen by the right eye is inferior on down gaze and superior on up gaze), then this indicates a mechanical deviation, and is described as a crossing of diplopia.

A neurogenic lesion generally results in an under-action of the eye movement. In a mechanical restriction, the eye is often tethered, such that it cannot move into the stipulated direction. This mechanical tethering can put pressure on the globe, causing an increase in intra-ocular pressure (IOP) on an attempted movement in the direction of the restriction.

Further the neurogenic lesion can be differentiated from the myogenic one by the forced duction test (FDT) and electromyography. In a mechanical defect, the electromyographic activity will be normal or increased on attempting to look in the direction of the restriction, whereas the activity will be reduced with a neurogenic lesion.

Investigations of a case of diplopia

Ocular investigations

Near point of convergence Measuring the near point of convergence is of clear value in patients complaining of asthenopic symptoms. Assessment of convergence can also provide useful information in differentiating a supranuclear from an infranuclear defect.

Measurement of the angle of deviation

Measurement of the deviation in different directions of gaze helps to confirm the direction of maximum misalignment of the visual axes. In true incomitancies, the angle of deviation differs depending on which eye is fixating. It can be measured with prism cover test. It should be measured in 9 gazes with each eye fixing. In order to use the prism cover test to measure the angle

when fixating with the right eye, for example, the strength of prism should be adjusted until the movement of the left eye is nulled. When fixating with the right eye, the angle of the left eye is being measured, and when fixating with the left eye, the angle of the right eye is being measured. As the secondary angle of a deviation is the greater, measurement of the angle of deviation fixating each eye in turn can help determine the eye with the defect.

Maddox double rod test

Maddox rods at the same orientation in front of each eye (normally vertically orientated to produce a horizontal streak) can be used to assess the angle of torsion. If the streaks seen by each eye are not parallel, then the rods can be rotated until the streaks become parallel and horizontal, thus giving a measure of the rotation required and hence the torsion. This test is maximally dissociating and can produce erroneous results (possibly as a result of small angles of head tilt).

Bielschowsky's head tilt test

Due to the development of the muscle sequelae, the eye movement pattern in a longstanding SO palsy in one eye can be difficult to differentiate from a SR palsy of the other eye. A positive Bielschowsky head tilt test can confirm the culprit as the SO, but a negative result is inconclusive. Normally, as the head is tilted towards the right shoulder, for example, the right SR and right SO work in partnership to intort the eye, the opposing vertical actions of the two muscles cancelling out. If a patient has a SO palsy, as the head is tilted towards the affected side, the SR acts unopposed, so it not only intorts the eye but also elevates it. To perform the test, seat the patient upright, maintaining steady fixation straight ahead at a distance of 3m, so that fixation doesn't favour either the SO or SR. Tilt the head towards the eye with the suspected SO palsy (the hypertropic eye) and if the vertical angle of the deviation increases, the defective muscle is confirmed as the SO).

Parks used this information to devise a three step test for differentiating the four vertically acting extra-ocular muscles. Parks 3-step test helps to elucidate which of the 4 extraocular muscles responsible for vertical eye movements may be weak, thereby causing vertical diplopia. Although first appearing impossibly complex, this test follows a logical progression to progressively eliminate groups of muscles from the 4 pairs.

First, determine which eye appears higher with the head in a normal position, with the head turned to the left and to the right, and with the head tilted left and tilted right. Then, answer the questions in the following steps:

Step 1: Which eye is higher in primary gaze? This reduces the possibilities of muscles from 4 pairs to 2 pairs. For example, if the right eye is higher, the weakness resides either in the muscles depressing the right eye (right superior oblique muscle and right inferior rectus muscle) or in the elevators of the left eye (left superior rectus muscle and left inferior oblique muscle).

Step 2: Is the deviation greater with left head turn or with right head turn? Now, only one pair remains. If the right eye deviates most when the head is turned to the right (both eyes are turning to the left), then only the right superior oblique muscle or the left superior rectus muscle remains.

Step 3: Is the deviation greatest with tilting the head to the left or to the right? Called the Bielschowsky head tilt, it relies on the torsional balancing reflexes provoked by head tilt. The higher eye extorts (because of the inferior oblique muscle), while the lower eye intorts (because of the superior oblique muscle).

By combining steps 1-3, only one muscle remains. However, the astute clinician can reduce this process by recognizing that the superior oblique muscle is by far the one muscle most likely to be responsible. A head tilt to the same side as the involved muscle exacerbates the problem. Alternately, the eye that is highest in adduction "points at" the muscle that is affected.

Fusional Reserves

Measurement of fusional reserves can be of diagnostic value when differentiating a long standing vertical muscle palsy from one of recent onset. Congenital SO palsies, for example, can have vertical fusional reserves in excess of 10D, whereas a recent onset deviation will usually have a normal vertical fusion range (4D - 6D). Vertical fusion ranges can also increase over a long period of gradual change in the direction of the visual axes, such as in dysthyroid eye disease.

Past Pointing

Past pointing can be used to differentiate a recent onset cause from a long-standing condition. On occlusion of the unaffected eye, the patient is asked to rapidly look at, and point towards, an object in the field of action of the palsied muscle. In recent onset incomitancies, the input required to look at the object will be greater than normal, so the object will be perceived as more peripheral than its actual position, and the patient will tend to point towards a more eccentric location.

Diplopia Chart

A vertical bar of light is viewed through red and green goggles at a fixed distance from the eye. The bar light is moved into each direction of gaze, and the patient describes the image separation and appearance. The image separation can be measured. By convention, the red filter is always placed before the right eye. The

symbol `$' is used to describe the two lines as superimposed. When interpreting a diplopia chart, it should always be remembered that the most distal image belongs to the under-acting eye. The position of the image is the reverse of the position of the eye. Ideally, the distance of fixation and image separation should also be recorded.

Field of binocular single vision (BSV)

The field of BSV is a test used to describe the areas of BSV, and hence diplopia. It is of particular value in the management of symptomatic incomitancy. It is very simple to do using a kinetic perimeter, or to approximate from ocular motility. The patient is seated at the perimeter, with the chin central to fixation. The target is moved outwards until the patient recognizes diplopia, and the point is marked. The target is then moved further until one image disappears, normally due to occlusion by facial contours, and this point is marked. The inner ring describes the area of BSV, the outer ring describes the limits of the binocular field of fixation.

Hess chart/ Lees screen

The patient should be seated squarely facing the screen being plotted, with the head centred on the central fixation spot. Ideally, plot the centre position first, then the 15° fixation points, and finally as many of the 30° points as can be seen without moving the head. There are a number of basic rules for interpreting a Hess plot:

1. The smaller field belongs to the eye with the defect.
2. Neurogenic pareses will show the muscle sequelae to a greater or lesser extent (dependent on the duration of the condition and the eye used for fixation). The largest underaction is normally in the direction of action of the paretic muscle and the largest over-action is normally the contralateral synergist.
3. Mechanical defects show a compressed field. There is not normally an obvious over-action of the direct antagonist, nor under-action of the contralateral antagonist, so the effects of the defect are limited to the direction of action of the mechanical restriction. The most obvious feature of a mechanical defect is normally the marked overaction of the contralateral synergist.

Systemic investigations

They are indicated to detect or confirm the underlying suspected cause of pathological binocular diplopia as indicated by the history and detailed work up of the patient.

Magnetic resonance imaging (MRI) or computed tomography (CT) scan of head and orbit may be required to find the cause of oculomotor nerve palsies, to check for signs of trauma, tumor or blood vessel malformations etc.

Tensilon test should be done in cases suspected to be due to Mysthenia gravis

Thyroid profile in cases of diplopia due to thyroid ophthalmopathy

To summarize, many patients complaining of diplopia will have conditions that can be appropriately managed in practice while other cases will need referral . Those for whom the condition is more sinister and require neuro-ophthalmologic investigation are, fortunately, uncommon. In an adult or a child, recent, acute onset diplopia due to a concomitant or incomitant deviation with an uncertain aetiology should be referred urgently for a neuro-ophthalmologic investigation.The consequences of inappropriate action, however, can be life threatening. The long-standing deviations which are cosmetically unacceptable may be assisted by surgery or prisms.

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Vitreous Hemorrhage

Cyrus M. Shroff, MD

Vitreous hemorrhage is defined as the presence of extravasated blood within the space outlined by the internal limiting membrane of the retina posteriorly and laterally, the non-pigmented epithelium of the ciliary body antero-laterally and the lens zonular fibers and posterior lens capsule anteriorly

Symptoms:

Patients present with sudden painless diminution of vision, floaters, photopsia, and the perception of shadow and cobwebs.

Signs:

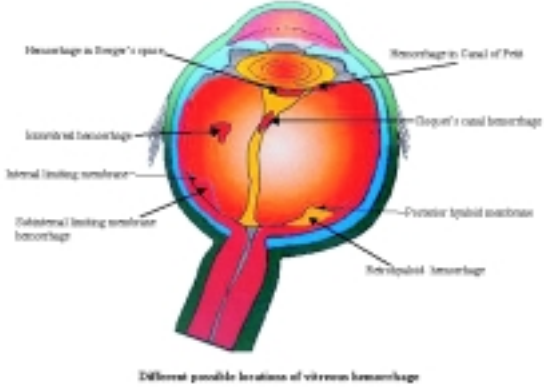
1. Slit lamp examination: - Red blood cells can be appreciated when the light beam is focused posterior to the lens. Mild afferent pupillary defect may be present.
2. Ophthalmoscopy:- In severe vitreous hemorrhage, the red fundus reflex may be absent, and there may be no fundus view. In mild vitreous hemorrhage, blood obscures part of the retina and retinal vessels.

   Chronic vitreous hemorrhage has a yellow ochre appearance secondary to the break down of hemoglobin.

   Depending on the etiology there may be other abnormalities.

Pathologic Mechanisms.

There are three main pathologic mechanisms for vitreous hemorrhages.



Different possible locations of vitreous hemorrhage

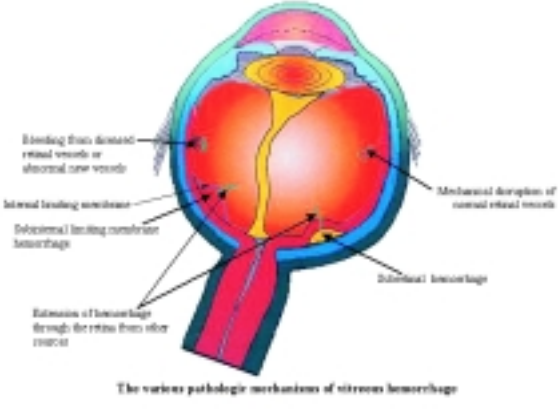
1. Bleeding from diseased retinal vessels or abnormal new vessels, as in proliferative diabetic retinopathy, proliferative retinopathy secondary to retinal vein occlusion, Eales' disease, sickle cell retinopathy and retinopathy of prematurity. The common pathologic mechanism in this group of diseases is retinal ischaemia, which results in formation of new vessels caused by the production of angiogenic factors. Occasionally vitreous hemorrhage can occur from inflamed obstructed retinal vessels as in severe vasculitis or acute central retinal vein occlusion.
2. Tearing of retinal vessels caused either by formation of retinal break or posterior vitreous detachment due to adherence of cortical vitreous to retinal vessels.
3. Less frequently it is caused by extension of hemorrhage through the retina from other sources like from subretinal hemorrhage as in age related macular degeneration and choroidal melanoma.

Infrequently vitreous hemorrhage occurs in coagulation disorders and in patients on anticoagulant therapy.

Etiology

Children:

1. Trauma
   a. Birth Trauma
   b. Shaken Baby Syndrome
   c. Traumatic Child abuse
2. Retinopathy of Prematurity
3. Retinal Break
4. Retinal Detachment
5. Retinoblastoma



The various pathologic mechanisms of vitreous hemorrhage

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New Delhi

6. Congenital Retinoschisis
7. Pars Planitis
8. PHPV
9. TERSON'S

Adult :

A. Bleeding from diseased retinal vessels or abnormal new vessels
   Diabetic Retinopathy
   Eales' Disease
a. Neovascular Disease
b. Acute Severe Vasculitis
   Retinal Vein Occlusion
   Central
   a. Neovascularization
   b. Acute Central Vein Occlusion
      Branch
   Sickle Cell Retinopathy
   Retinal Capillary Angioma
   Hypertensive Retinopathy
   Radiation Retinopathy
   Macroaneurysm
B. Rupture of Normal Retinal vessels.
   Retinal breaks
   Retinal Detachment
   Posterior Vitreous Detachment
   Trauma
C. Extension of hemorrhage from other sources
   Age related macular degeneration with CNVM
   Intraocular Tumour - Choroidal Melanoma
D. Miscellaneous
   Terson's Syndrome
   Bleeding …..

Workup

1. History: History of ocular or systemic disease especially diabetes, hypertension, trauma, retinal break or detachment in the other eye, family history of detachment.
2. Complete ocular examination including slit lamp examination to check for neovascularization of iris, intraocular pressure measurement and a dilated fundus examination of both eyes using indirect ophthalmoscope. This may be supplemented by slit lamp biomicroscopy using 78D or 90D lens or fundus contact lens. Contralateral eye examination is often diagnostic e.g. diabetes, age related macular degeneration, Eales' retinopathy of prematurity. Presence of peripheral retinal lesions in the other eye should alert one to possibility of retinal break. In cases of spontaneous vitreous hemorrhage without obvious vascular disease it is important to do an indirect ophthalmoscopic examination with scleral depression. Retinal breaks are commonly located superiorly in cases of dense vitreous hemorrhage. In traumatic vitreous hemorrhage (especially open globe injuries) scleral depression is avoided for the first 3-4 weeks.
3. Ultrasonography : When there is no fundal glow a Bscan Ultrasound helps in a diagnosis of vitreous hemorrhage as well as in detection of any associated posterior vitreous detachment, retinal detachment (traction or rhegmatogenous), intraocular tumor, retinal break (if large), scleral rupture . In fresh, mild hemorrhage, dots and short lines are displayed on B-Scan, and a chain of low amplitude spikes is found on A-Scan. The more dense the hemorrhage, the more opacities are seen on B-Scan and the higher is the reflectivity on A-scan. If the blood organizes, larger interfaces are found which have higher reflectivity on A-Scan and may be confused with retinal detachment. Kinetic echography shows undulating after movements on B-Scan which are to be differentiated from the less mobile retinal and choroidal detachment.
4. Fluorescein angiography may aid in defining the etiology although the quality of the angiogram may depend on the density of the hemorrhage. Angiography is especially useful in diagnosis of proliferative retinopathies, wherein abnormal new vessels show leakage of the dye into the vitreous cavity, and in age related macular degeneration, where there is subretinal leak. Fluorescein angiography of the contra lateral eye is also an important diagnostic aid.

Differential Diagnosis

1. Vitritis : The onset is rarely as sudden as in vitreous hemorrhage. History is very important. Pain or redness at onset can alert one to vitritis. History of any surgical intervention is important. The possibility of trivial undiagnosed trauma should be specifically asked for and explored. Detailed history of systemic illnesses must be taken if vitritis is suspected. There may also be antecedent signs of anterior or posterior uveitis. Pars planitis should be looked for. On slit lamp examination there may be presence of vitreous cells in the anterior vitreous. If

unexplained 'vitritis' is seen, masquerade syndromes like intra-ocular lymphoma should be ruled out.

2. Retinal Detachment: may occur without a vitreous hemorrhage, yet the symptoms may be identical. The fundus view may be difficult in a fresh retinal detachment with dense vitreous hemorrhage. However, the retina can usually be viewed with indirect ophthalmoscopy by an experienced observor. Ultrasonography is indicated in case of doubt.
3. Very advanced 'brown' or 'black' cataract can sometimes give an impression of vitreous hemorrhage on indirect ophthalmoscopy. The ability to see some retinal detail despite the advanced cataract can often help to differentiate the two conditions. If a doubt persists a combined A & B Scan is helpful.

Natural History and Prognosis

The natural history and prognosis of vitreous hemorrhage depends on the underlying disease. In general, patients with diseases that have no tendency for recurrent bleeding, such as avulsion of a vessel associated with a retinal tear or posterior vitreous detachment have good prognosis for resolution of the vitreous hemorrhage and restoration of vision. Clearance of blood from the vitreous is a slow process, with a time constant in the order of 1% per day. Hemorrhage in the vitreous gel remains suspended in a lamellar fashion until the vitreous liquifies and the blood sinks to the bottom of the vitreous cavity where it is absorbed.

Patients with diabetic retinopathy as the underlying disease process have a relatively poorer prognosis for spontaneous clearing of vitreous hemorrhages and restoration of vision especially after recurrent bleeds. Among patients with vitreous hemorrhage secondary to retinal vein obstruction, branch vein occlusion patients have the best visual prognosis, hemi-central vein occlusion patient inter-mediate and central retinal vein occlusion worst. Natural history of vitreous hemorrhage secondary to Eales Disease and retinal arterial macro aneurysms is generally good while that following age related macular degeneration and sickle cells retinopathy is poor.

Visual recovery following trauma is unpredictable and depends on the nature and extent of injury.

Management

Once the diagnosis of vitreous hemorrhage has been made, the patient must be referred to an ophthalmologist with special interest in vitreo-retinal disease and well-versed with indirect ophthalmoscopy. Unlike retinal detachment, vitreous hemorrhage is usually not a surgical emergency. However, prompt examination especially for a first time bleed is important as surgical intervention may be required if there is retinal detachment. IF other causes like AMD with CNVM, intra-ocular tumour are suspected these must be specifically investigated for and treated promptly. In most other cases one can safely wait for a few weeks for the hemorrhage to clear up spontaneously. The patient must be reassured and the plan of management explained. The treatment modalities are:

A. Conservative Management: Bed rest with head end of the bed elevated. Eye movements maintain RBC in the vitreous cavity in diffuse suspension. If the eye movements are diminished, the blood gravitates to the bottom of the space. Sometimes bilateral patching is advised as this accelerates the settling of the blood cells. Settling of blood enables visualization of the superior retina for examination and treatment. If dense vitreous hemorrhage persists, and the etiology remains unknown, the patient is followed with a B-scan ultrasound every 1-3 weeks to rule out a retinal detachment.

   Eliminate asprin, nonsteroidal anti-inflammatory drugs and other anticlotting agents unless they are medically necessary.

B. Photocoagulation or cryotherapy: Laser photocoagulation or cryotherapy is used to seal retinal breaks. Photocoagulation is done as soon as media is clear enough to permit it. Indirect ophthalmoscopic laser delivery especially with 810-nm diode laser is very useful when it may not be possible to achieve adequate laser burns with the green laser. As these retinal breaks are associated with vitreous traction they should be surrounded completely by 3-4 rows of confluent laser burns. Patents must be informed that if retinal detachment occurs before firm chorio-retinal adhesion is formed, surgery in the form of scleral buckling or vitrectomy may be required.

   If there are large retinal tears and the media is not clear enough for adequate prophylactic treatment the option of early surgery has to be seriously considered.

   In the case of vascular retinopathies it is usually safer to wait for longer periods for the hemorrhage to clear before starting laser photocoagulation. The other eye must have a fundus fluorescein angiography to detect proliferative vascular disease and receive immediate laser photocoagulation if new vessels are present.

   Some surgeons advocate peripheral retinal cryotherapy for non-resolving vitreous hemorrhage. Peripheral retinal cryotherapy accelerates the resorption rate of vitreous hemorrhage by causing a breakdown of the blood retinal barriers and an increase in tissue plasminogen activator and has been

used as a treatment modality for non-resolving vitreous hemorrhage. However this should be used with caution in cases where organisation and contraction of the vitreous gel can precipitate a traction retinal detachment. This is especially true in cases where there is no PVD or very limited PVD.

C. Posterior Hyaloidotomy: Hemorrhage located between the internal limiting membrane and the retina may cause permanent macular changes before spontaneous resolution occurs. In few cases posterior hyaloidotomy may be performed using a Nd-YAG laser which disrupts the internal limiting membrane and releases blood cells into the vitreous cavity.

D. Pars Plana Vitrectomy: Pars plana vitreous surgery has revolutionised the management of vitreous hemorrhage, dramatically improving the prognosis in a number of cases and enabling quicker visual rehabilitation in may others. Vitrectomy or surgical removal of blood is usually performed for:-

Urgent:

1. Vitreous hemorrhage accompanied by RD
2. Neovascular AMD with vitreous hemorrhage and subretinal hemorrhage.
3. Certain cases of trauma.

Early: Vitreous hemorrhage secondary to vascular retinopathies:

1. Bilateral hemorrhage.
2. Associated with early iris neovascularization
3. Associated with hemolytic "ghost cell" glaucoma
4. With severe progressive fibrovascular proliferation
5. Dense premacular hemorrhage.

Late/Elective :

Chronic vitreous hemorrhage with no traction on retina or other associated problems, with good vision in the other eye may be observed almost indefinitely if the patient is not handicapped, or for medical reasons, is not a good candidate for surgery.

Summary :-

Vitreous hemorrhage is a common presenting sign in patients with a vitreo-retinal disorder. It is alarming to the patient as it produces dramatic visual symptoms and often marked visual loss. Though, most of the time, vitreous hemorrhage is not a surgical emergency the patient should be examined and investigated very carefully to detect those who do require early intervention. Appropriate management ensures good prognosis in most cases of vitreous hemorrhage.

Monthly Meetings Calendar For The Year 2004-2005

1st August, 2004 (Sunday) Army Hospital (R&R)
29th Auguest, 2004 (Sunday) Sir Ganga Ram Hospital
6th November, 2004 (Saturday) *Rescheduled :* 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)
27th March, 2005 (Sunday) Mohan Eye Institute
2nd & 3rd April, 2005 (Saturday & Sunday) Annual DOS Conference

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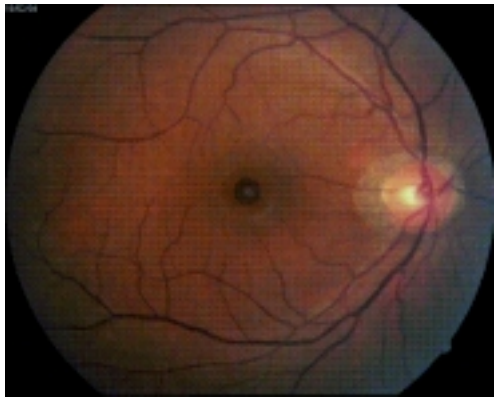
Issues in Diagnosis and Treatment of Macular Holes

Atul Kumar MD, **Gunjan Prakash** MD

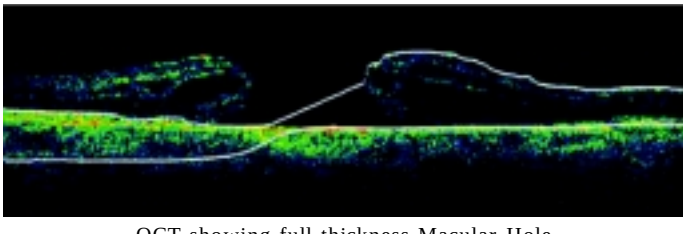
Macular hole surgery was first described in 1991. Until that time, there was no known treatment for the condition, and we would customarily tell patients that nothing could be done to save their vision. In the early 1990's, Kelly and Mandel demonstrated that by separating the posterior hyaloid, performing an air-fluid exchange and positioning the intravitreal gas bubble on the macular hole with the patient in a facedown position, they could successfully close the macular hole. Our knowledge of macular holes has evolved since that time, but several issues still remain open to question.



Colour photograph showing full thickness Macular Hole

Diagnosis

Diagnosis of macular holes is easiest using slit-lamp biomicroscopy of the fovea. A specific diagnostic test, the Watzke-Allen, involves passing a slit beam over the central fovea and identifying a break in the line. This test, however, is not very reliable, so we must depend upon many ancillary tests to help us diagnose macular holes. The best ancillary test now available is Optical Coherence Tomography (OCT).



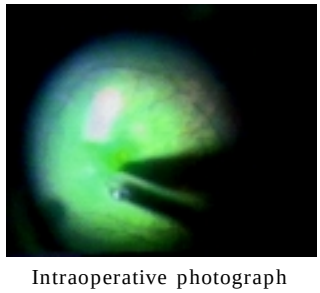
OCT showing full thickness Macular Hole

When should we operate on patients with macular holes? Interestingly, the first surgery attempted was for impending macular holes. De Bustros and colleagues hypothesized that we may be more successful treating or preventing holes before rather than after they form. Therefore, the first clinical trial focused on Stage 1 macular holes. As that trial evolved, however, the technique for matching Stage 2 and Stage 3 macular holes was developed. We found that operating on Stage 1 holes was as likely to cause them to progress to Stage 2 or 3 as to cause improvement. We had a high success rate in closure when operating on Stage 2 or 3 macular holes. Consequently, we now observe Stage 1 holes. With Stage 2 holes, we tend to operate in order to provide early intervention and success. The days of waiting unit a patient's vision deteriorates to 20/100 or 20/200 to repair their macular holes are behind us.

Of course, chronic Stage 3 and Stage 4 holes have a lower rate of anatomic closure, and visual outcomes tend to be poorer. A patient who has had a macular hole for 5 or 6 years is unlikely to benefit from surgery. The patients with a good surgical prognosis tend to be those with Stage 2 holes or Stage 3 holes with relatively recent changes in visual acuity.

Peeling the Internal Limiting Membrane

Other important issues include the advisability of peeling the internal limiting membrane (ILM), the type of intraocular tamponade, and managing complications. The success rate has gradually increased in macular hole surgery. Looking at anatomic closure, we can now see success rates that exceed 90%. Peeling the internal limiting membrane may play a critical role in the improvement of success rate.



Intraoperative photograph depicting ICG enhanced ILM being peeled

Intraoperative photograph depicting completely peeled ILM from Posterior Pole

Vitreoretina Services
Dr. R. P. Centre for Ophthalmic Sciences
AIIMS, New Delhi - 110 029

Several studies have yielded varied results. Our study at RP Centre found benefit in ILM peeling, but other research has suggested it is less critical. Working with a group of 18 eyes, we were able to have anatomic closure in 83.33% in the year 2000-2001. Preoperative visual acuity was also a factor in the outcomes.

There are a variety of techniques of ILM peeling. After vitrectomy has already been done, and the posterior hyaloid has been removed from the retinal surface, we inject ICG dye into the vitreous cavity over the macular hole, taking care not to put any in the macular hole itself. The solution is diluted to 0.5 %, which is weaker than the solution used in the anterior segment. We then aspirate the ICG dye out of the vitreous cavity. An optimal starting point for ILM-peel is chosen within the arcade vessels but remote from the fovea. The site is chosen to lie outside the maculopapular bundle. Tano's Diamond Dusted Membrane Scraper (Synergetics, Inc., USA) is used to raise a small ILM flap. In this way, engaging the neurosensory retina is avoided. The ILM flap is then grasped with end-opening forceps (Grieshaber, Alcon Laboratories, Inc. 6201 South Freeway, Fort Worth, TX 76134, USA) and a "rhexis" (smooth-edged continuous tear) is created by slowly tearing the ILM in a circular motion, concentric with the fovea, keeping the direction of force always following the natural course of the nerve fibres (Figure No. 1). Most of the times the ILM can be removed as single piece, but if the tear is incomplete, the ILM can simply be-grasped at the new edge, and the rhexis resumed. This is how the process works.

Some researchers noticed that some of the patients were developing poor visual acuity after ILM peeling. Some patients experienced toxicity in the discrete area of RPE dropout in the macular hole. Tissue culture studies were carried where RPE cells were grown and then treated with ICG. No histological damage occurred. However, when viability of the cells was measured using a mitochondrial dehydrogenase assay, it was found a definite decrease in the viability of the RPE cells when they were exposed to ICG. And the combination of the light exposure to the ICG enhanced the effect. So it is hypothesized that ICG might actually be a photosensitizer like Visudyne. Now use of lower doses of ICG and lower light levels is advised, believing this will be safer way to take advantage of ICG without causing harm to the macula.

Internal Tamponade

Whether gas or air should be used for internal tamponade is still open to question. Most ophthalmologists use either SF_6 or C_3F_8; both are adequate provided the patient maintains a face-down position. Some, in particular McCuen and colleagues, report very good results using a silicone oil tamponade. The advantage of a silicone oil tamponade is that patients do not have to be as compulsive about maintaining their face-down positioning in the postoperative period, and visual rehabilitation occurs more immediately, the disadvantages are that patients need a second operation to have the oil removed.

On the other hand, a gas internal tamponade leads to cataracts, which will result in a second operation anyway. In a study on a series of patients comparing oil to gas, the closure rate was basically the same with the two groups. The overall closure rate was 89% with oil and 91% with gas. Perhaps the rate was somewhat higher with gas because of the somewhat higher surface tension. But the groups differed in visual acuity. Close to 40% of patients has 20/100 or better visual acuity with gas, whereas only 25% has 20/100 or better vision with oil. Because of the difference in visual acuity, it is currently recommend to use a gas tamponade.

Reducing Complications

Another question to resolve is the recommended length of the postoperative face-down positioning. We generally advise patients to be extremely compulsive about maintaining this position for 1 week. Finally, we must consider the issue of cataract management in these eyes. These patients develop acceleration of cataract/nucleus sclerosis. In order to maximize their visual recovery, patients will need cataract surgery within 1 or 2 years of the macular hole surgery. Cataract surgery on a vitrectomized eye introduces more difficulty than the usual procedure. Highly experienced cataract surgeons say they prefer to operate on these eyes earlier rather than later. Waiting too long allows the nucleus to get very hard, and it is technically difficult to phacoemulsify harder nuclei in the absence of vitreous.

Overall, we have found macular hole surgery to be one of the most exciting innovations in our specialty. This condition that was considered totally inoperable previously now is treated with a success rate of over 90%. This is a very dramatic turnaround. Patients have high expectations from the results, and we owe it to them to try to optimize their visual outcome.

Suggested Readings:

1. ICG enhanced maculorhexis in macular hole surgery. *Ind J Ophthalmol* 2002
2. Visual outcome of macular hole surgery with ILM peeling. *Jpn J Ophthalmol* 2002
3. Clinicopathologic correlation of a macular hole treated by cortical vitreous peeling and gas tamponade. *Ophthalmology* 1994
4. A Multicentered Clinical Study of Serum as Adjuvant Therapy for Surgical Treatment of Macular Holes. *Arch Ophthalmol*. 1999

Corneal Reinnervation after LASIK: Prospective 3-Year Longitudinal Study.

Calvillo MP, McLaren JW, Hodge DO, Bourne WM.

Invest Ophthalmol Vis Sci. 2004 Nov;45(11):3991-6.

The Study measured the return of innervation to the cornea during 3 years after LASIK. Seventeen corneas of 11 patients who had undergone LASIK to correct myopia from -2.0 D to -11.0 D were examined by confocal microscopy before surgery, and at 1, 3, 6, 12, 24, and 36 months after surgery. In all available scans, the number of nerve fiber bundles and their density (visible length of nerve per frame area), orientation (mean angle), and depth in the cornea were measured. The number and density of subbasal nerves decreased >90% in the first month after LASIK. By 6 months these nerves began to recover, and by 2 years they reached densities not significantly different from those before LASIK. Between 2 and 3 years they decreased again, so that at 3 years the numbers remained <60% of the pre-LASIK numbers ($P < 0.001$). In the stromal flap most nerve fiber bundles were also lost after LASIK, and these began recovering by the third month, but by the third year they did not reach their original numbers ($P < 0.001$). In the stromal bed (posterior to the LASIK flap interface), there were no significant changes in nerve number or density. As the subbasal nerves returned, their mean orientation did not change from the predominantly vertical orientation before LASIK. Nerve orientation in the stromal flap and the stromal bed also did not change. The study concluded that both subbasal and stromal corneal nerves in LASIK flaps recovered slowly and did not return to preoperative densities by 3 years after LASIK. The numbers of subbasal nerves appeared to decrease between 2 and 3 years after LASIK.

Concurrent use of 5% natamycin and 2% econazole for the management of fungal keratitis.

Cornea. 2004 Nov;23(8):793-6.

Prajna NV, Nirmalan PK, Mahalakshmi R, Lalitha P, Srinivasan M.

PURPOSE:: To determine if concurrent use of 5% natamycin and 2% econazole offers greater benefits than monotherapy with 5% natamycin for the management of fungal keratitis. METHODS:: Subjects presenting to the cornea service were treated with 5% natamycin and 2% econazole used concurrently. We compared the results with a historical control of patients treated with 5% natamycin in the same calendar year. The same clinical and examination protocol including inclusion and exclusion criteria was used for both groups. RESULTS:: We compared results of 47 subjects on concurrent use of 5% natamycin and 2% econazole with all 53 subjects who had received 5% natamycin in a previous study (historical controls). Baseline characteristics were similar between the 2 groups. There were no significant differences ($P = 0.9$) between the 2 arms for success (defined as a healed or healing ulcer). CONCLUSIONS:: Concurrent use of 5% natamycin and 2% econazole does not appear to offer additional benefits over monotherapy with 5% natamycin for the management of fungal keratitis.es remains predominantly vertical.

Intraocular pressure, safety and quality of life in glaucoma patients switching to latanoprost from adjunctive and monotherapy treatments.

Eur J Ophthalmol. 2004 Sep-Oct;14(5):407-15.

Haverkamp F, Wuensch S, Fuchs M, Stewart WC.

The authors evaluated efficacy, safety and quality of life in ocular hypertensive or open-angle glaucoma patients changed to latanoprost from previous therapy. The study was a prospective, multicenter, active-controlled design in which qualified patients had their previous therapy substituted for latanoprost and were followed for at least three months. In 1068 patients, latanoprost was continued 92% throughout the 36-month observation period. Latanoprost treatment reduced the intraocular pressure (1OP)($p < 0.001$) when compared to previous monotherapies including: beta-blockers (-4.0 +/- 3.7 mmHg, 42%), alpha-antagonists (-3.9 +/- 3.0 mmHg, 14%), miotics (-3.8 +/- 3.5 mmHg, 2%), or carbonic anhydrase inhibitors (CAI) (-3.8 +/- 3.6 mmHg, n = 16%), and adjunctive therapy including: beta-blocker and CAI (-3.7 +/- 3.1 mmHg, n = 12%), alpha-agonist (-3.7 +/- 3.4 mmHg, n = 5%), or pilocarpine (-3.4 +/- 3.7 mmHg, n = 6%), or CAI and alpha-agonist (-4.6 +/- 6.4 mm Hg, n = 2%)($p < 0.0017$). The most common adverse event with latanoprost was ocular allergy (1.5% incidence). Patients showed a preference for latanoprost for many systemic and ocular quality of life measures on a non-validated

questionnaire ($p < 0.05$). The authors concluded that in a clinical setting, patients who have their mono- and adjunctive therapy treatment substituted for latanoprost may on average experience reduced IOP, decreased side effects and increased quality of life measures.

Cost effectiveness of photodynamic therapy with verteporfin for age related macular degeneration: the UK case.

Br J Ophthalmol. 2004 Sep;88(9):1107-12.

Smith DH, Fenn P, Drummond M.

AIM : To estimate the potential cost effectiveness of photodynamic therapy (PDT) with verteporfin in the UK setting. METHODS: Using data from a variety of sources a Markov model was built to produce estimates of the cost effectiveness (incremental cost per quality adjusted life year (QALY) and incremental cost per vision year gained) of PDT for two cohorts of patients (one with starting visual acuity (VA) of 20/40 and one at 20/100) with predominantly classic choroidal neovascular disease over a 2 year and 5 year time horizon. A government perspective and a treatment cost only perspective were considered. Probabilistic and one way sensitivity analyses were undertaken. RESULTS: From the government perspective, over the 2 year period, the expected incremental cost effectiveness ratios range from 286 000 (starting VA 20/100) to 76 000 UK pounds (starting VA 20/40) per QALY gained and from 14 000 (20/100) to 34 000 UK pounds (20/40) per vision year gained. A 5 year perspective yields incremental ratios less than 5000 UK pounds for vision years gained and from 9000 (20/40) to 30 000 UK pounds (20/100) for QALYs gained. Without societal or NHS cost offsets included, the 2 year incremental cost per vision year gained ranges from 20 000 (20/100) to 40 000 UK pounds (20/40), and the 2 year incremental cost per QALY gained ranges from 412 000 (20/100) to 90 000 UK pounds (20/40). The 5 year time frame shows expected costs of 7000 (20/40) to 10 000 UK pounds (20/100) per vision year gained and from 38 000 (20/40) to 69 000 UK pounds (20/100) per QALY gained. CONCLUSION: This evaluation suggests that early treatment (that is, treating eyes at less severe stages of disease) with PDT leads to increased efficiency. When considering only the cost of therapy, treating people at lower levels of visual acuity would probably not be considered cost effective. However, a broad perspective that incorporates other NHS treatment costs and social care costs suggests that over a long period of time, PDT may yield reasonable value for money.

Cost utility of photodynamic therapy for predominantly classic neovascular age related macular degeneration.

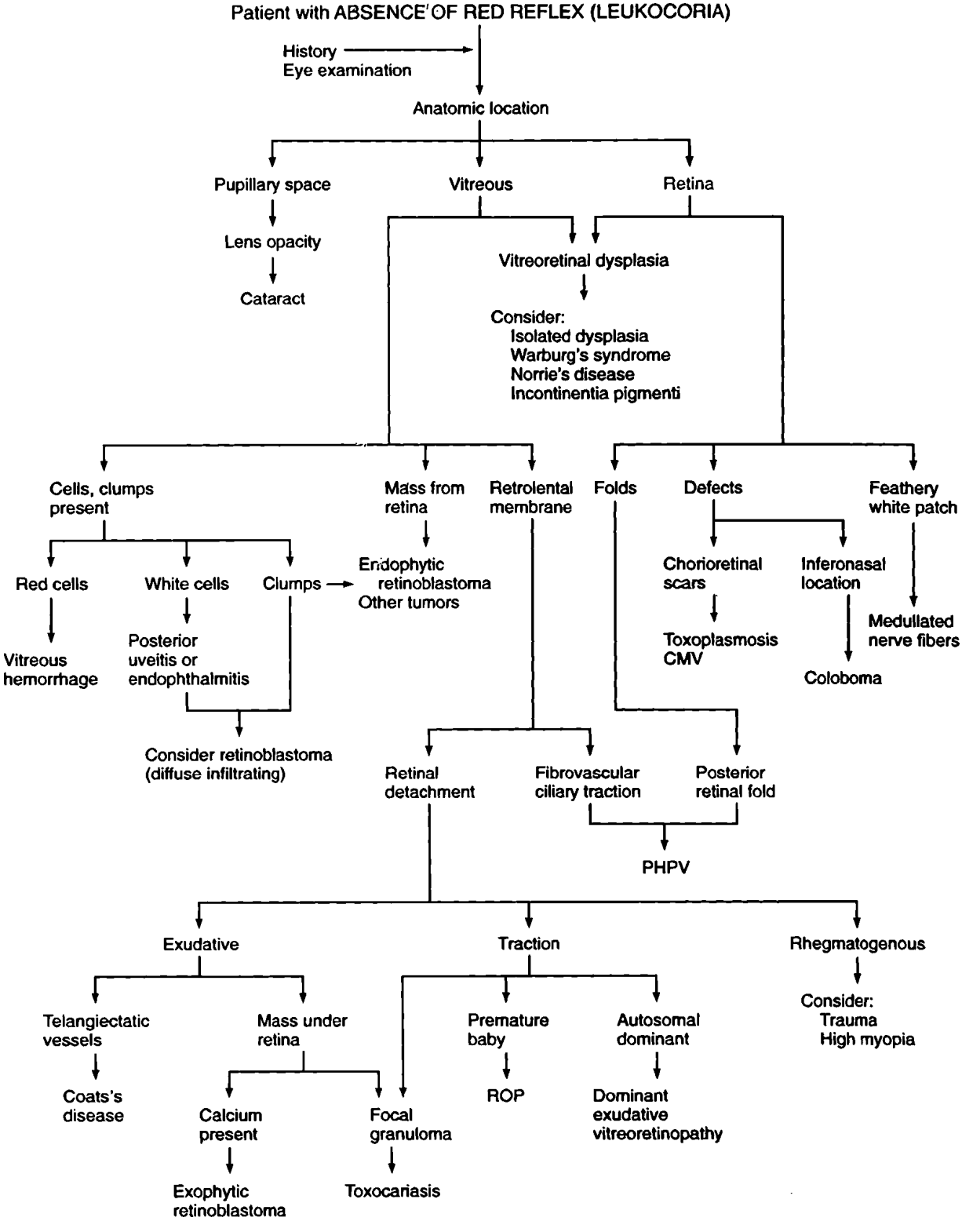
Br J Ophthalmol. 2004 Aug;88(8):982-7.

Hopley C, Salkeld G, Mitchell P.

Centre for Vision Research, University of Sydney, Department of Ophthalmology, Westmead Millennium Institute, Australia.

Age related macular degeneration (AMD) is the leading cause of severe vision impairment and blindness in older people throughout the developed world and currently affects around 420 000 UK citizens. Choroidal neovascularisation (CNV) is treatable with photodynamic therapy (PDT) but is expensive at over pound 1200 per treatment. The aim of this study was to assess the cost utility of PDT for better eye, predominantly classic, subfoveal choroidal neovascular lesions secondary to AMD. Cost utility analysis (CUA) was conducted to estimate the cost effectiveness of PDT for scenarios involving reasonable (6/12) and poor (6/60) visual acuity. The models incorporated data from the Treatment of Age-related Macular Degeneration with PDT (TAP) Study and patient based utilities. The incremental CUA was based on decision analytical models, comparing treatment to a placebo comparator. Extensive one way sensitivity analysis of parameters was conducted to determine the robustness of the model. A discount rate of 6% was used for costs and quality adjusted life years (QALY). RESULTS: Model 1: in people with reasonable initial visual acuity, the cost utility of treating applicable neovascular AMD lesions was pound 31 607 per QALY saved, with a sensitivity analysis range from pound 25 285 to pound 37 928. Model 2: in people with poor initial visual acuity, the cost utility was pound 63 214 per QALY saved, with a sensitivity analysis range from pound 54 183 to pound 75 856. CONCLUSIONS: PDT treatment is the only available treatment for some forms of neovascular ("wet") AMD. Under these assumptions, PDT can be considered moderately cost effective for those with reasonable visual acuity but less cost effective for those with initial poor visual acuity. These findings have implications for ophthalmic practice and healthcare planning.

Approach to Leukocoria



Harish Pathak, MD, **Vijay B. Wagh,** MD, **M.S. Bajaj,** MD
Dr. R. P. Centre for Ophthalmic Sciences
AIIMS, New Delhi - 110 029

FORTHCOMING EVENTS

INTERNATIONAL

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 Town, 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

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
Ph : 91-011-26589549, 265888852-65 Ext. 3146
Fax : 91-011-26588919
Email : dosonlin@vsnl.net
Website : www.dosonline.org

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

Annual DOS Conference
2nd & 3rd April, 2005
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
Ph : 91-011-26589549, 265888852-65 Ext. 3146
Fax : 91-011-26588919
Email : dosonlin@vsnl.net

!! Congratulations !!

1. Prof. R.B. Vajpayee, Dr. Namrata Sharma & Dr. Tushar Agarwal for being awarded Best Scientific Video Presentation in American Academy of Ophthalmology, New Orleans, USA, 2004.
2. Dr. Mohita Sharma & Dr. Angshuman Goswami, Tirupati Eye Centre, Noida for recieving Awadh Dubey Award for paper presentation in 39th Annual U.P. State Ophthalmological Society in October, 2004 at Agra.
3. Dr. Neeraj Sanduja for recieving Dr. Prem Chandra award for Best Free Paper Presentation in Annual Conference of North Zone Ophthalmological Society, 2004 & Rashtriya Gaurav Award for Year 2004.

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.

DOS QUIZ NO. 15

1. *Vertical saccade is controlled by the _____*
2. *_____variety of Adenoid cystic carcinoma of the lacrimal gland is associated with the worst prognosis.*
3. *Blood staining of the cornea is caused by the presence of _____within the cornea*
4. *Bitots spots is caused by goblet cell hyperplasia - True/False*
5. *Wedl cells occur in_____type of cataract.*
6. *Rosenthal fibres are seen in_____*
7. *Sympathetic ophthalmitis is prevented by removal of the exciting eye within_____weeks of injury*
8. *Term retinoblastoma was coined by_____*
9. *Ocular bobbing is associated with haemorrhages in _____ area of the brain*
10. *KF ring starts at_____clock hour.*

Rules:

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

ANSWERS OF DOS QUIZ NO. 13

1.	What is the frequency of acoustic waves used in ophthalmology?	***10 MHz***
2.	What is the normal thickness of optic nerve in imaging.....?	***2.4 to 3.4 mm***
3.	Who described first external dacryo cystorhinostomy.....?	***Toti***
4.	Who is the Editor of Journal Survey of Ophthalmology.....?	***B. Schwartz***
5.	Most Common location of juvenile retinoschisis is.....?	***Inferotemporal***
6.	Most common organism causing post operative endophthalmitis?	***Staph. Epidermidis***
7.	Classical visual field finding in hysteria is..... ?	***Symmetrical field constriction***
8.	Toutan giant cell is seen in .....?	***Juvenile xanthogranuloma***
9.	Abraham lens has a focusing button of .....?	***66D***
10.	Angle between optical and visual axis is known as .....?	***Alpha Angle***

DOS Credit Rating System Report Card

DCRS July 2004 – Army Hospital (R&R)

Total No. of Delegates	83
Delegates from Out side (N)	75
Delegates from Army Hospital (n)	8
Overall assessment by outside delegates (M)	610.5
Assessment of case presentation-I (Dr. Lt. Col. R. Maggon) by outside delegates	549
Assessment of case presentation-II (Dr. Lt. Col. (Mrs.) Madhu Bhaduria) by outside delegates	541.5
Assessment of clinical talk (Dr. Col. Ajay Banajee) by outside delegates	572.5
Rejected Form Army Hospital (n)	2
Rejected Form Out side (N)	2

DCRS August, 2004 – Sir Ganga Ram Hospital

Total no. of Delegates (Valid DCRS forms)	86
Delegates from Out side (N)	76
Delegates from Sir Ganga Ram Hospital (n)	10
Overall assessment by outside delegates (M)	552
Assessment of case presentation-I (Deepti Manocha) by outside delegates	475
Assessment of case presentation-II (Dr. Piyush Kapoor) by outside delegates	498
Assessment of clinical talk (Prof. H.K. Tewari) by outside delegates	571
Total no. of invalid DCRS forms	NIL

DCRS September, 2004 – Hindu Rao Hospital

Total No. of Delegates	45
Delegates from Out side (N)	32
Delegates from Hindu Rao Hospital (n)	13
Overall assessment by outside delegates (M)	225.5
Assessment of case presentation-I (Dr. Vikas Anand / Dr. Ruchi Goel) by outside delegates	214.5
Assessment of case presentation-II (Dr. Bithi Chowdhury) by outside delegates	216
Assessment of clinical talk (Dr. Ruchi Goel) by outside delegates	229
Rejected Form Hindu Rao Hospital (n)	NIL
Rejected Form Out side (N)	1

DCRS October, 2004 – Dr. R.P. Centre for Ophthalmic Sciences

Total No. of Delegates	57
Delegates from Out side (N)	38
Delegates from Dr. R.P. Centre (n)	19
Overall assessment by outside delegates (M)	272.5
Assessment of case presentation-I (Dr. Balasubramanya R.) by outside delegates	261
Assessment of case presentation-II (Dr. Arun Singhvi) by outside delegates	264
Assessment of clinical talk (Dr. Rajesh Sinha) by outside delegates	300
Rejected Form Dr. R.P. Centre (n)	2
Rejected Form Out side (N)	NIL

SCIENTIFIC PROGRAMME
Mid Term Conference of DOS : Crisis Management

Forenoon Session : 9:00 AM - 11:00 AM

Emergencies in Ophthalmology

1. CRAO
2. Post Operative Endophthalmitis
3. Traumatic Optic Neuropathy
4. Acute Angle Closure Glaucoma
5. Orbital Cellulitis
6. Cornea Scleral Perforation with Lens Rupture
7. Impending or Perforated Corneal Ulcer
8. Acute Lime Injury

Forenoon Session: 11:30 AM - 1:30 PM

Difficult Intra Operative surgical situations: How to manage them?

1. Accidental Needle Perforation in RD Surgery
   a) With Subretinal Hemorrhage
   b) With incarceration of retina
2. Accidental Slippage of Muscle in Squint Surgery
3. Phaco in Torn Capsulorhexis
4. Intra- op Zonular Dialysis
5. Posterior Capsular Rent
6. Damaged / Wrong IOL
7. Nucleus / IOL Drop
8. LASIK Incomplete Flaps and Buttonholes

Afternoon Session : 2:30 PM - 4:30 PM

Diagnostic & Management Dilemmas

Corneal & Refractive cases:

1. Post Mito-C Scleral Necrosis
2. Cystoid Cicatrix following P.K.
3. Unusual presentation of Scleral abscess

Neuro-Ophthalmology Cases:

1. Disc Oedema diagnostic deliema
   Three Cases

Oculoplasty Cases

1. Unusual Presentation of lacrimal gland tumour
2. Three Cases

Vitreo Retina / Uvea Cases

1. Three Cases

DELHI OPHTHALMOLOGICAL SOCIETY

(LIFE MEMBERSHIP FORM)

Name (In Block Letters) _____

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

Qualifications _____ Registration No. _____

Sub Speciality (if any) _____

ADDRESS

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).

!!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.