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Capsular Bag Distension Syndrome
Postoperative Blindness
Delayed Visual Maturation
Intravitreal Injection Technique
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Mid-Term Conference
Delhi Ophthalmological Society
14th-15th November 2009
India Habitat Centre, Lodhi Road, New Delhi

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Editorial

My Dear Friends and Colleagues,

I thank all of you for giving me the opportunity to serve the ophthalmic fraternity.

DOS is such a great association of over 5000 highly qualified professionals from all over the country. Together, we have extreme power. Our power is benign, and meant to be exercised in the service of the humanity. However, it can be and must be used to safeguard and protect the interests of the Indian Ophthalmologist.

My friends, I have an agenda, a vision for my tenure.

I wish to see all the members of DOS connected and networked though DOS.

We live in a nearly virtual world. We buy our tickets online and think nothing of it. As if it is the most commonly, boringly, ordinary thing to do. Our banks are online and we use credit cards, as if we have been using them for ever. Google is our new temple and facebook our new mohalla. Ladies and gentlemen, this is the much fabled “The Fourth Dimension”.

But here at DOS. Let me say, we have a long way to go. Out of the 5000 members, we have valid email addresses of only 1200.

Dear friends we need to get on to this virtual highway and we need to do it at once.

DOS has its own website: dosonline.org. With all of you registered and online, the website can and will take off and zoom. We can have it all. Discussion forums, online CME, online library. We can make our own practice protocols and our own social networking. The DOS facebook. With such force and presence, we can ensure that our members get the best deals from the industry.

There is a lot that we can achieve and we will find out as we move on and start doing it.

You might have heard the song: First step, “Tell her she’s the one you’ve been dreaming of”

Your first step Dear friends! send me your valid emails and register on dosonline.org.

With love and regards and lots of both.

Your friend,

Thanking you,

Dr. Amit Khosla
Secretary,
Delhi Ophthalmological Society

Editor-in-chief
Amit Khosla MD, DNB

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It is indeed great to be part of this great organization with such wonderful colleagues. In an endeavor to do something unique, something big and more purposeful I am constantly in touch with our mentors and senior colleagues.

In the midterm we plan some exclusive ophthalmic events. We plan to have programs which gives the general ophthalmologist the confidence, skill and ability to handle newer technological advances. Also I feel that quality of ophthalmic care should be improved by regular CME’s. In the midterm conference, we would like to ensure that the best speakers with authority on their subjects, are selected and there is minimum repetitive deliberation. We would like to see a good amalgamation of institutional expertise with a fine blend of experienced faculty and everybody can participate and carry home some important message.

We would also like to ensure that all the important topics are covered with an emphasis on current concepts including a few topics of futuristic importance. We have to ensure that our DOS Times & DOS Journal raise the bar of International Standard and come out in reckoning as the best News Letter & Journal. We have great talent and would like to see that the talents are absorbed and assimilated in the best form. Please feel free to share your ideas with me and my Executive.

In Annual conference, we plan to have a significant international presence. And I’d love to have your suggestions.

Regarding the DOS house, we are already in touch with AIOS & exploring other options for a suitable proposal. We would ensure that DOS interest is kept foremost and not compromised in any way. A financial sub-committee has also been formed with the Treasurer and Secretary to help streamline expenses and suggest methodologies to improve revenues. We have analysed the publishing and distribution costs of the DOS Times & DOS Journal and hope that credible means to reduce these would be found.

I must appreciate our Secretary’s energy and zeal and would also encourage all Executive members to work in the same spirit. This way we can multiply our energy 20-fold. I’d also love to see that any member interested in serving DOS, in anyway should come forward and give his proposal.

After all DOS belongs to us all.

Long Live DOS.

Dr. Sharad Lakhotia
President, DOS

Please Note ...
New Secretariat Office

This is to inform all the DOS Members that new Secretariat office is at the following address:

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Ph.: 011-65705229, E-mail: dosonlin@vsnl.net, Website: www.dosonline.org
Please correspond at the above address only.
Zonular compromise complicates every step of cataract surgery and poses a serious challenge in terms of safety and visual outcome. When the crystalline lens albeit displaced from its position still lies in the pupillary area, it is called as subluxated. It is termed as “luxated or dislocated” when it is completely displaced from the pupillary area.

Ectopia lentis is the term used to describe congenital dislocations.1 Lens displacements may be traumatic, heritable, and spontaneous. (Table 1)

The adoption of new devices and techniques that minimize the stress on compromised zonules have gained acceptance over older approaches like iridectomy, laser iridotomy and intracapsular cataract extraction. The contemporary techniques of management shall be discussed at length.

Anatomy

The lens is suspended in its anatomic position by ciliary zonules (zonules of Zinn or suspensory ligament of Zinn) that consist of fibers that run from ciliary body and fuse into the outer layer of the lens capsule around the equatorial zone. The bundles that insert into the anterior capsule are stronger than those that insert in the posterior capsule. The zonules are inserted till 1.5mm anterior and posterior ones 1mm posterior to equator. Each zonule measures 5 to 30μm in diameter and is composed of bundles of microfibrils. Biochemically they are composed of fibrillin, a protein product of the gene linked to Marfan’s syndrome.

Preoperative Evaluation

A detailed ocular examination is mandatory. Both near and distant BCVA should be determined, keeping in mind that the patient may best see with an aphakic correction if the lens is markedly subluxated. The exact degree of zonular loss, location of defect and presence or absence of vitreous in the anterior chamber should be noted. An inferior subluxation often indicates 360 degrees of zonular insufficiency and combined with the effect of gravity is more difficult to manage. Ultrasound biomicroscopy and anterior segment OCT, besides being useful for angle evaluation, are indicated for zonular assessment in patients where the pupil fails to dilate. Gonioscopy is performed to note any developmental defects, pseudoexfoliative material and deformities secondary to trauma or as a sequelae to subluxation. The fundus examination is done to look for lattice degeneration, cyclitic membranes, retinal detachment or posttraumatic pathology. Retinal detachments occur in 10% eyes with Marfan’s syndrome and Homocystinuria. Bscan ultrasonography is indicated in opaque ocular media. (Figure 1)

Indications for surgery

A subluxated lens itself is not sufficient reason to pursue surgery especially in the absence of pupillary-block glaucoma, corneal...
decompensation, inflammation or intractable visual disability. A stable induced refractive error may preferably be corrected with glasses or contact lenses. However, the decision to operate may be undertaken in following conditions:-

- A visually significant and progressive subluxation in children along with amblyopia that cannot be treated by conventional means such as glasses, contact lens, and/or patching.

- In older children and adults where poor visually acuity is attributed to subluxation not amenable to refractive correction or there is an anterior or posterior luxation risk.

- Lens induced uveitis.

- Significant cataract.

**Operative procedure**

The extent of subluxation determines the choice of operative procedure. The principles of the surgery include preventing worsening of subluxation and maintaining capsular stability—short term as well as long term. The Capsular tension ring (CTR) has helped us in achieving both these goals. (Table 2) Implantation of a capsular tension ring, or CTR, stabilizes a loose lens and allows the surgeon to place the IOL in the most beneficial place—the capsular bag. It reduces the chances of Vitreous herniation to the anterior chamber; a taut capsule gives countertraction to all traction maneuvers, making them easier to perform; capsular support for an “in the bag” implant is obtained; and, most important, the capsular bag maintains its shape, avoiding capsular fibrosis syndrome and IOL decentration.

In 1991, the CTR (capsular tension ring) was introduced by Dr. Hara and subsequent studies demonstrated that CTR could provide both intraoperative and postoperative stabilization of capsular bag and IOL. These PMMA rings can be inserted anytime after the capsulorrhesis has been completed. In patients with profound and progressive zonular loss, Osher described the technique of suturing CTR to the scleral wall by straddling the CTR with a 10.0 prolene suture, double armed with CIF-4 needles. This procedure involved the risk of rupturing the bag. The modified

<table>
<thead>
<tr>
<th>Causes</th>
<th>Features</th>
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<tbody>
<tr>
<td>Traumatic</td>
<td>Responsible for &gt;50% lens displacements</td>
</tr>
<tr>
<td>Homocystinuria: Inborn error of sulfur containing aminoacids, Sodium nitroprusside test of urine is the screening test. Autosomal recessive. Tall, slender, genu valgum, flat feet, kyphoscoliosis, joint laxity, deformed sternum, generalized osteoporosis with vertebral collapse, high arched palate, malar flush, light, fair &amp; dry skin, fair and sparse hair. Mental retardation &gt; 50% of cases; thromboembolic phenomena in veins, middle-sized arteries, premature death in 40% patients. Ectopia lentis: downwards &amp; nasal; progressive. Angle anomalies donot occur. Glaucoma and retinal detachment may occur.</td>
<td></td>
</tr>
<tr>
<td>Weill- Marchesani syndrome: Dominant and recessive inheritance. Brachymorphy, short stature, spade like hands and feet, large thoraces, reduced joint motility. No vascular, cutaneous, mental or urinary abnormality. Microspherophakia, lens displacement later in life.</td>
<td></td>
</tr>
<tr>
<td>Other disorders: Hyperlysinemia, Ehlers-Danlos syndrome, Sturge Weber syndrome, Crouzon’s syndrome, chondrodysplastic, dwarfism, oxycephaly, polydactyly, sulfite oxidase deficiency.</td>
<td></td>
</tr>
<tr>
<td>Associated ocular anomalies</td>
<td>Ectopia lentis et pupillae, aniridia, isolated anomaly.</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>Old age, high myopia, endophthalmitis, treatment of retinal detachment by diathermy, Eale’s disease, chalcosis, buphthalmos, megalocornea, coloboma of iris/choroids, perforation of corneal ulcer and growth of intraocular tumor.</td>
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CTR (MCTR)/ Cionni ring, designed by Dr Robert Cionni, incorporates a unique fixation hook to provide scleral fixation without violating the integrity of the capsular bag. The MCTR consists of an open, flexible PMMA filament with a fixation hook that loops anteriorly and in a second plane wrapping around the capsulorrhexis edge. At the free end of the hook is an eyelet through which a suture can be passed for scleral fixation. Depending on the extent of subluxation, single or double loop models can be chosen. (Figure 4) The MCTR has shown to provide a good centration of the capsular bag.

A peribulbar anaesthesia is preferred. Smallest possible incision depending on the surgeon’s ease should be used. Phacoemulsification has an edge over manual small incision surgery (MSICS) but care should be taken in more than 1800 subluxation where MSICS may be easier. All the steps are performed keeping in mind the site of subluxation (Table 3). In phacoemulsification for subluxated cataracts, it is advisable to place the incision in the meridian with intact zonules to avoid damage to the zonular fibres with the movement of the phacotip.

After the initial incision, a high molecular weight viscoelastic is injected over the area of zonular dialysis to tamponade the vitreous and maintain a deep non-collapsible anterior chamber. Capsulorrhexis in subluxated cataract may severely test the skill of the surgeon. Initial relaxing capsulotomy is difficult because of the lack of circumferential traction forces. It is advisable to begin the capsulorrhexis where the zonules are intact and the anterior capsule offers sufficient resistance. A capsulorrhexis foreceps is preferred over a capsulotomy needle. The capsulotomy should be large enough to allow easy manipulation of nucleus. A 5.5 - 6 mm for phacoemulsification and 6.5 mm for MSICS is usually adequate. Iris hooks can be a very handy instrument in such cases. It can help in pulling up the capsulorrhexis and in preventing posterior movement of the lens during phacoemulsification. The CTR insertion and placement is much easier when the capsulorrhexis margin is lifted up by the hook.

**Insertion of CTR & MCTR**

CTR/MCTR can be inserted into the capsular bag at any point after the capsulorrhexis. It is preferable to insert a CTR after capsulorrhexis and a good hydroprocedure as it reduces the risk of intraoperative herniation of the vitreous into the anterior chamber due to partial reformation of the capsular zonular anatomic barrier. It also redistributes the traction forces to the

### Table 2

<table>
<thead>
<tr>
<th>Degree of Zonular dehiscence</th>
<th>Procedure chosen</th>
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<tbody>
<tr>
<td>Superior upto 4 clock hours (100°)</td>
<td>First choice: CTR with IOL implantation</td>
</tr>
<tr>
<td>Inferior upto 3 clock hours (90°)</td>
<td>Second choice: PMMA IOL implantation with haptic being used to stretch the bag</td>
</tr>
<tr>
<td>Anywhere &gt;3 to 6 clock hours (&gt;90° to 180°)</td>
<td>CTR with IOL implantation</td>
</tr>
<tr>
<td>Anywhere &gt;6 to &lt;9 clock hours (&gt;180° to &lt;270°)</td>
<td>Modified CTR with single loop</td>
</tr>
<tr>
<td>Anywhere &gt;9 clock hours (270° or more)/generalized weakness of zonules</td>
<td>Modified CTR with double loop with IOL implantation</td>
</tr>
<tr>
<td>9 or more clock hours (270° or more)/generalized weakness of zonules</td>
<td>Intracapsular cataract extraction with scleral fixated IOL/Iris fixated IOL/ anterior chamber IOL</td>
</tr>
</tbody>
</table>
Techniques of CTR insertion

The CTR is inserted using forceps or a specially designed injector. A ‘fish-tail’ technique can also be used to insert the CTR where both its ends are held with the forceps in a crossed manner to create a central closed loop which is placed adjacent to the area of dehiscence in the capsular bag first. This is followed by release of

Table 3

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<tr>
<td>• Incisions should be preferably away from area of zonular weakness</td>
</tr>
<tr>
<td>• Use high molecular weight viscoelastic</td>
</tr>
<tr>
<td>• Capsulorrhexis should be initiated in an area remote from the dialysis</td>
</tr>
<tr>
<td>• Capsulorrhexis is more easily performed with forceps than with cystitome &amp; should be made “off-center” in an eye with significant lens subluxation</td>
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</table>
each end at a time and manipulation of the entire device into the bag (Figure 5).

MCTR needs to be fixed to the sclera using either an ab-externo or an ab interno approach with single or double armed 9,0 prolene suture on CIF-4 needles.

The authors prefer an ab-externo approach. A partial thickness scleral flap is dissected at the fixation site (Figure 6). The CIF-4 needle is passed 1.5mm posterior to the limbus through the scleral bed to emerge from the main wound and tied to the fixation hook (Figure 7 & 8). In case of larger subluxation, where double loop device is used, two scleral flaps are prepared 180 degrees apart and prolene is passed as earlier and tied to the other fixation hook. The MCTR is inserted with smooth forceps through the main incision and dialed into the capsular bag (Figure 9). The Y-hook can be used to “dial” the MCTR until the eyelet is centered adjacent to its proposed site of fixation. An anchoring knot is placed adjacent to the exiting 9,0 prolene suture using 10,0 nylon and the prolene is tied to it. The curved needle on 10,0 nylon is then used to take a bite from undersurface of the scleral flap which when pulled covers the mesh of sutures (Figure 10,11).

Alternatively, using a double-armed 9,0 prolene suture, two passes are made from each scleral bed, which is tied to the fixation hook and the two prolene ends emerging 1.5 mm apart and posterior to limbus are tied to each other. The knot is then rotated to be buried into the sclera and covered by conjunctiva.

When using an ab-interno approach the prolene is first tied to
the fixation hook eyelet and then the CIF-4 straight needle is passed through the main wound to emerge from the the scleral bed. The emerging prolene sutures are dealt as described earlier. Fibrin glue can be used to reposition the scleral flap and the conjunctiva as in scleral fixation.9

**Insertion before nuclear extraction**

In this case a space is created between the peripheral capsular bag and remaining lenticular material with viscoelastic so as to prevent entrapment of cortex under the CTR. If the CTR is placed before phacoemulsification, a “safety-suture” (10,0 Prolene) is looped through the leading eyelet. This suture is left trailing out of the incision and can be used to retrieve the CTR in the event of a posterior capsular rent or if the CTR is difficult to place, this suture can be used to help “coax” the leading haptic around the capsular bag periphery.

**Insertion after nuclear extraction**

Once capsulorrhexis has been completed, if one plans to extract the nucleus prior to capsular tension ring implantation, if there is moderate subluxation, the capsular bag should be stabilized with iris retractors placed through limbal stab incisions. (Figure 12)

Hydrodissection is then performed gently, yet thoroughly, to maximally free the nucleus and thereby decrease zonular stress during manipulation of the nucleus. If the nucleus is soft, supracapsular phacoemulsification will virtually eliminate zonular stress.

Phacoemulsification should be performed using low vacuum and aspiration settings in order to keep the bottle height and flow rate at a minimum.9 Chop techniques are preferred for the dense nuclei to minimize zonular stress during phacoemulsification.

Cortical viscodissection prior to aspiration will also limit the stress on remaining zonules.10 The cortex should be stripped along a vector tangential to the capsular bag periphery to decrease the risk of further damaging the zonules.

The cortical entrapment can be prevented, by injecting the viscoelastic just under the surface of the residual anterior capsular rim before inserting the CTR or MCTR. This will create a space for the ring and dissect the residual cortex away from the peripheral capsule.

Once the CTR/MCTR has been placed appropriately, the posterior chamber intraocular lens (IOL) is inserted in the bag. It is easier to inject a foldable IOL in comparison to a PMMA lens but either can be used. It is safer to place the IOL haptics in the meridian of the zonular disinsertion whenever possible.

If vitreous presents at any time during the procedure, it should be completely removed from the anterior chamber. Kenalog (Alcon) (triamcinolone suspension) can be used to identify vitreous in the anterior chamber.

**Contraindications of CTR/MCTR**

- Complete continuous capsulorrhexis is not attained
- Posterior capsular tear occurs since the expansile forces may cause the capsular bag to rupture.
- Extensive generalized zonular weakness.
- MCTR is not to be used in patients with scleral disorders.

It must be noted that despite all its advantages use of CTR is fraught with certain risks in cases with severe or progressive zonular dehiscence since it might lead to IOL decentration, pseudophakodonesis and rarely the disastrous occurrence of complete dislocation of the bag, CTR and the IOL into the vitreous11.

In conclusion, CTR and MCTR have revolutionized the approach to subluxated cataracts. Except in very extensive subluxations or where the integrity of the capsulorrhexis/ capsular bag is breached, it is now possible to save and recenter the capsular bag, and implant a PCIOL within it.

**References**

Capsular Bag Distension Syndrome

Shishir Agrawal, MS DNB FRCS, Jaya Agrawal, MS DNB MNAMS FRCS, T.P. Agrawal, MS

Capsular bag distension syndrome was first described by Davison in 1990.1 The advent of capsulorhexis or continuous tear anterior capsulotomy had been a major advance in cataract surgery. This made phacoemulsification in the capsular bag efficient and safe. It assures correct intraocular lens fixation and centration within the bag. However, it may sometimes give rise to a complication termed as capsular bag distension syndrome, referring to blockage of the anterior capsulorhexis opening by in-the-bag intraocular lens and entrapment of fluid within the capsular bag resulting in its distension, in the immediate post-phacoemulsification period most commonly.

Clinical Features

Capsular bag distension syndrome occurs following the continuous circular capsulorhexis margin getting occluded by the intraocular lens optic, the space between the intraocular lens and the capsular bag getting filled up with fluid; this has particulate debris and flare, and the fluid might be slightly turbid or a turbid suspension. There is a forward displacement of the posterior chamber intraocular lens and ballooning backward of the posterior capsule. The anterior vaulting of the intraocular lens causes an anterior bowing of the iris associated with a variable amount of shallowing of the anterior chamber, and results in an induced myopia. It typically occurs in the early postoperative period.1-3 It may sometimes cause raised intraocular pressure. Capsular bag distension syndrome has also been termed as capsular block syndrome.4 Other synonyms for it are viscoelastic entrapment syndrome, capsular bag hyperdistension, capsulorrhesis block syndrome, and capsular bag syndrome.

Capsular bag distension syndrome occurs typically in immediate post-phacoemulsification period.1-8 It may sometimes occur late, with liquefied aftercataract formation related to retained lens material and proliferation of lens epithelial cells.9-12 In late postoperative period, the liquefied milky white aftercataract forms in the same space, the closed chamber formed due to the fibrosed anterior capsular rim adhering to the intraocular lens. These cases ultimately require intervention.9-12 Capsular bag distension syndrome has been shown to occur with all types of intraocular lenses now. It occurs with a flexible lens, which has no positioning holes. Initially, it was found in cases with multipiece uniplanar PMMA intraocular lens. It has been shown to occur with silicone and acrylic lenses. It has been reported to occur from a reversed optic of the IOL.13 Variations of the capsular bag distension syndrome formation have been described as occurrence from ciliary sulcus implantation of intraocular lens without or with optic capture,14-17 sometimes associated with secondary angle-closure glaucoma.18 Complete capsular bag distension syndrome formation following adhering back of the anterior capsule that has not been removed is described.19

It is probably quite common postoperatively, and goes unnoticed as it is symptomless so often and due to lack of awareness/understanding of the condition. Ultrasonography confirms anterior chamber shallowing, anterior bowing of the iris, tight apposition of the intraocular lens to the iris, and posterior distension of the posterior capsule.2,7,8,20

Etiopathogenesis

A thin layer of fibrotic transparent “glue” pasted onto the anterior optic surface of the lens, probably representing epithelial generated materials/glue-like inflammatory-epithelial fibrinoid membrane, causes the anterior capsule to stick to it. Tight adhesion of anterior capsular remnants to anterior optic/ fibrotic glue occurs.1 The source of fluid is unclear, however, capsular bag distension syndrome is thought to occur due to viscoelastics in all probability.2-8 Retained viscoelastic, especially that behind the lens optic has been considered responsible. Before intraocular implantation, the capsular bag is expanded with viscoelastic, which remains behind the lens despite efforts to aspirate it. Retained viscoelastic supports the remaining lenticular epithelial cells, which create the colloidal suspensate. This creates an osmotic gradient across the capsular membrane. Hence, capsular bag distension is not seen intraoperatively, but occurs later, when the viscoelastic has had time to draw fluid from the surroundings. There are also other theories on the origin of the capsular bag fluid. The distended capsular bag is thought to have epithelial cells and cortical debris suspended in a fluid comprising lens epithelial protein, cellular breakdown products, balanced salt solution and water, and the fluid is considered to be derived by oncotic pressure created by retained lens epithelial cells and their proteinaceous byproducts, and possibly by retained cortex.1,2 Fluid analysis of the capsular bag aspirate has shown sodium hyaluronate in it. The lenticular protein alpha-2 crystalline has been found in the material from capsulorhexis.5,9 The cases are self-limiting and normally resolve on their own.1-8

Management

The early capsular bag distension syndrome settles in a few days, there being a spontaneous resolution.1,8 A complete removal of viscoelastic peroperatively from the eye, with particular attention to the space behind the intraocular lens, is required to prevent the occurrence of capsular bag distension syndrome. Capsular bag distension syndrome, not resolving on its own, is best treated by a peripheral anterior Nd:YAG laser capsulotomy. Nd:YAG capsulotomy – anterior or posterior – will be required, and is the preferred mode of treatment, if capsular bag distension syndrome causes problem or in late cases.1-5,7,8,10,11,13,14,16-19 Anterior capsular puncture beyond the intraocular lens optic is usually all that is needed and is sufficient to ameliorate the condition – the capsular bag shrinks. However, an anterior capsulotomy may not be possible if the pupil does not dilate beyond the intraocular lens optic. Posterior capsulotomy is reserved for such cases, not routinely advisable to maintain the compartmentalization of

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anterior and posterior segments. Again, posterior capsulotomy may be difficult as the posterior capsule balloons backwards and is difficult to focus by Abraham lens. Also, turbid fluid will make the media hazy. Peripheral posterior capsulotomy is helpful here.\textsuperscript{21} The patient asked to look to one side, the periphery of the posterior capsule, which is less posteriorly bowed, is focused and disrupted with ease in difficult cases. Intraoperative peripheral anterior capsulotomy (IPAC) has been advocated that can prevent a capsular bag distension syndrome formation.\textsuperscript{22} However, as the incidence of capsular bag distension syndrome is very low, it is rarely routinely performed/advocated. If the rhexis is small, prophylactic puncture of the bag or IPAC peroperatively may be done. Aspiration from the capsular bag is another treatment option.\textsuperscript{6,9} However, being an invasive procedure, it carries the risk of infection, etc. Posterior capsular opacification also occurs more frequently in cases of capsular bag distension syndrome, as there is no barrier to the migration of epithelial cells.\textsuperscript{23}

A Typical Case Report

A 40-year-old male presented with blurred vision following an uneventful phacoemulsification with implantation of a foldable AcrySof lens two weeks back. There was shallowing of the anterior chamber and induction of myopia. His visual acuity was 6/60 with \(-2.75\) DS. A dilated pupil slitlamp examination revealed an anteriorly pushed in-the-bag intraocular lens, the optic occluding the relatively small – 4 mm – rhexis margin. The posterior capsule was focused far backwards with difficulty (Figures 1 and 2). Nd:YAG laser anterior capsular puncture beyond the intraocular lens optic resolved the condition. The normal pseudophakic anatomy restored, myopia reduced to \(-1.50\) DS, and blurred vision resolved.

Unusual Cases

We had described a case of complete capsular bag distension syndrome in a patient who had undergone a manual extracapsular cataract extraction.\textsuperscript{19} In this case, the complete capsular bag was retained inside the eye – the anterior capsule was only partially cut and not removed; it got sealed back and the complete capsular bag anatomy having been restored, capsular bag distended, the anterior and posterior capsules ballooning far anteriorly and posteriorly, respectively, the intraocular lens lying in the centre. In fact when first seen without pupillary dilatation, it appeared that the patient had an advanced cataract, the capsular bag being biconvex and the intraocular lens partially obscured by the milky fluid. When the milky fluid accumulated in the capsular bag, the vision deteriorated and the patient reported, necessitating intervention. It is important to remove the anterior capsule during surgery, the capsular bag distension remaining a distinct possibility.

We saw another case of ‘incomplete capsular bag distension syndrome’ in a case post-Nd:YAG capsulotomy.\textsuperscript{24} This was in a patient where the two capsules were present behind the intraocular lens, which was implanted in the sulcus. The central part was yagged but the peripheral capsular leaves adhered together at their edges giving rise to an incomplete capsular bag distension.

Conclusion

We would want to highlight the fact that in our scenario, we can see cases of capsular bag distension syndrome even after manual extracapsular cataract extraction and this should be kept in mind.

References


Figures: 1 & 2. Capsular bag distension syndrome. The intraocular lens lies against the anterior capsule, and the capsular bag has distended. Note the gap between the 2 capsules (the anterior, A, and the posterior, B, capsules indicated by the slit)


Postoperative blindness as a result of anaesthesia administered for non-ocular surgery, although rare, is well documented. Previously almost unheard of, this devastating perioperative complication is increasingly being reported, more commonly in cases involving lumbar spine surgery. Of late, it has generated a heightened attention and concern among anaesthesiologists, spine surgeons and ophthalmologists.

With the healthcare expansion encompassing greater population quantitatively, more patients are being subjected to longer, more complicated surgical procedures. Reports of postoperative visual loss (PVL) are on the rise owing to the above in addition to better reporting techniques. Etiology of PVL is ‘presumably’ multifactorial but a definite cause-effect relationship continues to elude physicians. Researchers are currently involved in collecting much needed prospective data to substantiate the basis of PVL.

This overview attempts to analyze, correlate and discuss the causes and consequences of PVL along with the theoretical explanation of mechanisms involved, management strategies, preventive measures and its impact on general population at large.

Epidemiology of PVL

Eye injury after non-ocular surgery is rare with an incidence of 0.06%. Of the various eye injuries (corneal abrasion, red eye, direct trauma, eyelid haematoma, PVL), corneal abrasion and blindness are the most and the least common complication, respectively. Symptomatic visual defects is reported to occur as infrequently as 1 in 60,965 anaesthetic for non-ocular surgery but the incidence may be as high as 0.5 - 4.5% in selective patient population [cardiopulmonary bypass (CPB), spine surgery]. Specific cause of injury could be determined in 21% of cases and only 39% of patients had partial recovery in vision when diagnosis got established.

Most common cause of PVL is ischemic optic neuropathy (ION). Majority of anterior-ION (AION) cases occurred during CPB (53%) and spinal surgery in prone position (12%), whereas majority of posterior-ION (PION) cases were reported following radical neck dissection, nose and sinus surgery (48%), spine surgery (16%) and CPB (11%).

In 1992, as per American Society of Anaesthesiologists (ASA) closed claim project, ocular injury accounted for 3% of claims and payment frequency being as high as 70% as opposed to 56% for others. A survey involving 801 anaesthesiologists, conducted by anaesthesia patient safety foundation, blindness due to anesthetic techniques ranked 11th among a total of 53 patient safety concerns. Owing to a perceived increase in incidence of PVL, ASA committee for professional liability has established a PVL registry in 1999 (mode of reporting - anonymous/goal - 100 cases of PVL) to systematically study possible risk factors.

Etiopathogenesis

Neither the mechanism nor definitive risk factors for PVL have been identified. The etiology is not substantiated in most cases of PVL, but is probably multifactorial. A number of risk factors have been implicated. Some factors render the anterior optic nerve susceptible to ischaemia (predisposing factors), while some other may lead to final insult (precipitating factors) (Table1).

The set of factors may differ in different patients. Although pressure on the eyeball is an important cause temporally related to the PVL, it may occur in an unrelated manner to host of ischemic and non-ischemic problems. (Table 2)

Patient Positioning and Head-rest

Prone position is inherently risky and difficult to manage because in order to make operative site accessible to the surgeon, very often, the head and the extremities assume dependent position (lower than the level of heart). In addition, the patient and specially the eyes become inaccessible and difficult to monitor.

The devices used as head rests (e.g. bed pillows, metal horse-shoe frame, T-shaped frame, pin head holder, foam rest, Mayfield pins) during prone position are neither checked nor approved in a qualitative manner for the purpose of support to head and face. Once positioned, there is absolutely no way to monitor face/head during aggressive surgical manipulation (Screwing, drilling, tapping) and there may develop a catastrophic pressure on eyeballs specially if the anesthesiologist is not particularly vigilant. Height of the nasal bridge is vital as a low bridge allows medial aspect of eyes to experience greater contact and pressure with the foam cushion and as the peri-orbital area becomes edematous during the course of surgery, the medial part of eyes presses more into the nasal bridge.

Prone positioning is generally not related to any adversity in healthy patients, however, serious damage may occur in patients having retinal vascular abnormalities, macular degeneration, ocular hypertension and glaucoma. Prone position with 15º head down tilt along with some abdomen / thorax compression may contribute to increased central venous pressure (CVP) and intraocular pressure (IOP), facial oedema, decreased eye perfusion, thereby retarding venous drainage through ophthalmic veins. Additionally, keeping a head-down tilt for prolonged period could also decrease venous outflow from cranium causing local capillary bed stasis in the eyes. The effect appears to get enhanced in obese patients.

In anaesthetized patients, prone positioning is known to decrease ocular perfusion pressure (OPP) despite the maintenance of normo-tension owing to an increase in the IOP. General anaesthesia (GA) has been shown to decrease IOP in supine subjects. Conceivably, the balancing effects of GA as opposed to prone positioning on IOP intraoperatively may contribute to the net OPP.
Prone positioning with neck in neutral position and neck extension-flexion limited to 15° from horizontal on a pinhead holder along with normo-tension appears to be ideal to avoid ocular problems. Anaesthesiologists are well aware of the need to avoid external pressure on the eyes once patient is positioned prone. There are various methods available now to continuously monitor eyes; palpation directly or through conventional foam rest and neck prone view foam cushion system (eyes directly monitored by the mirror images).

Prone positioning during spinal surgery and external pressure on the eyes caused by improper head rest leading to globe compression seems to be most plausible basis of PVL. This PVL is secondary to PION, CRVO/CRAO and cortical blindness. Various other contributory factors are - prolonged surgical duration, low haematocrit, hypotensive anaesthesia, may also lead to ischemia of visual pathway in susceptible patients.

PION has occurred even in patients whose eyes were free from external compressions of any sort (head in may field pins). The registry showed that patient who developed PION underwent prone surgery of longer duration (8 hrs) than the patients who developed CRAO (5.5 hrs).

<table>
<thead>
<tr>
<th>Table 1: Risk Factors</th>
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<td><strong>Patient factors</strong></td>
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<tr>
<td>Age</td>
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<tr>
<td>Chronic Anemia</td>
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<td>Hypertension</td>
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<td>Obesity</td>
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<td>Atherosclerotic CVS diseases</td>
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<td>Diabetes mellitus</td>
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<td>Systemic lupus erythematoses</td>
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<td>Aspergillosis fumigatus</td>
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<th>Table 2: Causes of PVL</th>
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<td><strong>Ischaemic PVL</strong></td>
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<td>Ischaemic optic neuropathy</td>
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<tr>
<td>Anterior ischaemic optic neuropathy (AION)</td>
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<td>Posterior ischaemic optic neuropathy (PION)</td>
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<td>Central Retinal Artery occlusion (CRAO)</td>
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<td>Central retinal vein occlusion (CRVO)</td>
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<td>Cortical blindness, Hypertensive, Embolic</td>
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**Anemia, Blood loss & hypotension**

Ischemic optic neuropathy is stated to be the most common cause of PVL complicating anaesthesia after major surgery. Anemia, perioperative hypotension and major blood loss seems to be the prime contributor apart from the direct pressure on eye.

A review of literature reported 6 patients who developed ION after GA. It revealed that all of them had hemoglobin less than 8.0gm% recorded at least once. In addition, all 6 patients had episodes of decreased arterial pressure (24-46% of preoperative value) for extended period of time. Brown et al, commented, “Although, severe anemia may not cause ION alone, even a short period of hypotension in an already anemic patients may predispose the patient to an ION -induced PVL”.

Controlled hypotension during spinal surgery is in use within accepted limits of autoregulation. Despite this, the incidence of ION is on the rise. One possibility is probably the changing practices of blood transfusion. In the past, intraoperative hemoglobin less than 10.0gm% warranted transfusion. Since 1986, when conservative recommendations of blood transfusion came into being (due to risk of HIV transfusion), stricter regulation of blood transfusion is the norm. Now, American Association of
The onset of PVL usually occurs immediately on awakening but may be delayed up to seven days. Other confounding reason, such as, patient may not report the problem (believing it to be postoperative phenomena), misdiagnosis (confused state, delirium), and personnel not accepting the problem, may further complicate the clinical presentation. Judicious use of the following investigation may delineate the actual problem

Blood loss is an inevitable part of surgery, which is associated with hypotension. If non-blood products are administered to counteract hypotension it incurs a net loss of O$_2$ inspite of a normalized blood pressure. This may translate into an ischemic insult. Additionally, too much non-blood fluids may cause swelling and constriction of blood flow, compounding to further problems.

In case of decrease in blood pressure, hemoglobin and oxygen concentration, optic nerve is starved of O$_2$ and parts of the nerve that are particularly susceptible include watershed areas in the posterior and intermediate parts of the eye. A hematocrit of 30% has the greatest O$_2$ transport to tissue and so lower levels for surgery is not advisable.

Lee et al reported a patient, who developed PION in absence of anemia, hypotension and intraoperative blood loss. He concluded that we may not precisely pinpoint etiologic factors nor do we have the knowledge to prevent them. Unique vascular anatomy and ocular hemodynamics, atherosclerosis of the vessel that feed the eye, auto-regulatory dysfunction, emboli, vasospasm, interstitial oedema seems to have a role to play. Interestingly, massive blood transfusion has also been implicated for causation of PVL.

**Diagnosis of PVL**

The onset of PVL usually occurs immediately on awakening but may be delayed up to seven days. Other confounding reason, such as, patient may not report the problem (believing it to be postoperative phenomena), misdiagnosis (confused state, delirium), and personnel not accepting the problem, may further complicate the clinical presentation. Judicious use of the following investigation may delineate the actual problem

- **Fundoscopic examination**
- **Visual perimetry (visual acuity assessment)**
- **Intraocular pressure**
- **Special tests: fluorescin angiography / CT-scan / MRI-scan / visual evoked potential (VEP)**

Perimetry is the most important visual function test and it usually with reflects relative or absolute visual defects, the most common being inferior nasal quadrant followed by inferior half and central scotoma. Optic disc oedema and splinter hemorrhage at disc margin may also be present. Generalized patchy pallor takes over once disc oedema subsides. Fluorescin angiography shows lack of blood flow in the disc. Visual evoked potentials can be elicited in both eyes even though visual deficit occurs in one eye. This may suggesting sub-clinical damage to the non-affect eye.

In the setting of PION, patient may not be adequately responsive, pupillary signs inconsistent, and visual field accessible. In addition, fundoscopic examination is likely to be inconclusive. Also that PVL may appear any time after awakening to a week later, serial fundoscopic examination are desirable.

The differential diagnosis of PVL is difficult especially when there is an overlap. Following are common situations that may lead to PVL:

**Ischemic AION** - Optic disc oedema on initial examination and occasional improvement in vision is possible. Typically occurs in one eye but the other eye gets afflicted after variable period. Visual acuity may improve in 30% of patients despite persistent pallor of disc.

**Ischemic PION** - Normal fundoscopy initially but followed by delayed optic disc oedema. Vision seldom improves otherwise CT-scan of orbit may show enlargement of intraorbital optic nerve.

**CRAO** - Cherry red spot with retinal oedema is seen. Carotid artery disease may be present. Visual improvement may take place spontaneously.

**CRVO** - Cotton wool spots, retinal haemorrhages in all four quadrants, dilated tortuous retinal veins. Permanent visual loss may occur.

**Cortical blindness** - Normal fundoscopy and pupillary light reflex. Vision improvement is frequent. Abnormalities (as per CT/MRI examinations) of parietal or occipital lobe confirm diagnosis.

**Atypical situations**

**Intravitreal Gas**

Intraocular gases (purified air, perflouropropane) are currently utilized as tamponading agents in vitreo-retinal surgery. These gases may persist in eye for as long as 3 months post surgery. If nitrous oxide anesthesia is used during this period, the intraocular gas bubble expands and may lead to sight threatening increase in IOP.

The expanding bubble may displace lens iris diaphragm anteriorly closing the anterior chamber angle and eventually adding pressure and leading to decreased blood flow to the retina and optic nerve head.
Transurethral Resection of Prostate Syndrome (TURP): One of the most alarming complications of “TURP syndrome” is transient blindness. The vision becomes foggy, one sees halos around the objects and pupils are typically dilated and unresponsive. The reason put forward is retinal dysfunction secondary to glycine toxicity.

Functional Endoscopic Sinus Surgery (FESS): Irreversible blindness due to electrocoagulation undertaken to gain control of delayed bleeding after FESS is a well-known but fortunately rare complication. Several anatomic structures in the nasal area lies in close proximity to one another, including: anterior cranial fossa, orbit, etc. Intranasal ethmoidectomy is considered ‘blinder’ and the ‘most dangerous’ of all endoscopic nasal surgeries. The area between posterior ethmoidal and sphenoid sinus is of critical significance because it is here where anatomic variation can predispose to inadvertent injury. Two mechanisms by which visual loss may occur during FESS are:

- Direct injury to the optic nerve, carotid artery or ethmoidal artery (anterior/posterior)
- Retroorbital or extraocular muscle hematoma

Radical Neck Surgery: internal jugular vein ligation performed during radical neck dissection, can cause a rapid and severe increase in venous pressure of head and orbit often resulting in massive facial and orbital oedema. Therefore, monitoring of gross facial and orbital features during surgery is of utmost importance.

Drugs: Patients on anticoagulants, phosphodiesterase group-5 inhibitors (sildenafil, vardenafil, tadalafil) prescribed for erectile dysfunction, and intranasal α-agonists has been reported to cause AION. Patients on the above stated drugs are predisposed to PVL during prospective surgery (if any) under anesthesia. Local anesthetic (viz: bupivacaine, dyclomine, lidocaine, tetracaine) allergy/toxicity may result in blurring of vision, which may sometimes be construed as PVL.

Venous Air Embolism (VAE): Typically, VAE causes cortical blindness. Following surgeries are infamous for such an occurrence: Open heart surgery involving cardiopulmonary bypass, lower segment cesarean section, laryngectomy, neurosurgery and neck dissection.

Management of PVL

It is suggested that early diagnosis of PVL pave way for significantly improved outcome by virtue of initiation of appropriate therapy. The role of immediate ophthalmologic consultation is vital to above said.

Irrespective of cause, optimizing oxygen delivery to the optic nerves as promptly as possible- is the cornerstone of management. This includes, normalization of blood pressure (restore circulation in the anterior or posterior optic nerves); lowering of IOP (mannitol, acetazolamide, topical timolol maleate); aggressive fluid and blood administration to achieve euvolaemia in patients who are hypotensive and hypovolumic after surgery.

In addition, correct diagnosis aid in initiation of specific treatment, such as, arteritic AION requires rapid treatment with steroids; prompt search for carotid artery disease in case CRAO is suspected followed by intervention; maintenance of normal cerebral perfusion pressure and hematocrit avoids exaggeration of deficit in case of cortical blindness and hyperbaric O₂ therapy is desirable when air embolism is the problem. Alternatively, urokinase/PGE₂ administration, stellate ganglion block may be of help in cases of CRAO.

The management of PION remains controversial. Although systemic steroids result in some improvement, patients with PION often show improvement without any treatment. Despite spontaneous recovery, efforts should be directed at correcting anemia and persistent hypotension as quickly as possible to compliment the recovery process.

Prevention

As patients with ION have not shown to be at higher risk for transient ischemic attacks/episodes, and myocardial infarction than unaffected patients, “optic nerve is now considered to be a critically sensitive watershed area”.

Despite the best of diagnosis and management methods, therapy is not uniformly successful and the results appear to depend on severity of the presenting symptoms. Following steps may help prevent PVL:

- Avoidance of external pressure on the eyeballs.
- Maintain IOP (eucarbia, euvoolemia, head neutral/ head up position during spine surgery)
- Limit periods of extreme controlled hypotension to warranted part of surgery and titrate an increase in blood pressure with surgical blood loss
- Staging for unusually long surgeries requiring multiple approaches
- Individualize the percentage of blood pressure drop with patients resting blood pressure to decide the target deliberate hypotension instead of using same defined pressure for all cases.
- In addition to the above, special considerations [augmented intraoperatives monitoring (central venous pressure, direct arterial blood pressure, ETCO₂, blood gases)] should be given to inpatients with following pre-existing illness:
  - Hypertension
  - Diabetes mellitus
  - Chronic smokers
  - Coronary artery disease
- Documentation of periodic eye checks prior to the surgery and intermittently checking patients face intraoperatively to ensure absence of pressure against the eye and peri orbital area.
- Monitor the position of abdomen and pelvis during surgery with the patient in prone position to rule out excessive vena caval pressure (with consequent rise in IOP)
- Timely increase in blood pressure, volume replacement and transfusion is desirable and cannot be over emphasized.
**Prognosis**

Preventive measures and directed treatment may not be always successful and the result appears to depend largely on the severity of the presenting symptoms. Patients with severe initial deficit tend to have poor prognosis for final visual outcome. However, there is no evidence to suggest the preoperative ophthalmic examination could be beneficial in predicting, anesthetized subjects who may experience postoperative visual loss.

Anecdotal evidence suggests that in cases of non-postsurgical partial vision may get restored if they are treated aggressively (soon after diagnosis) with vasopressors, volume replacement and blood transfusion aiming at acutely raising and sustaining the blood pressure. Presently, however, this concept cannot be extended to ION in post surgical population.

**Conclusion**

While, future research aimed at identifying individual susceptibility followed by implementation of preventive measures is of vital importance, continual monitoring of anesthetized patients and prompt action in case of eye-related adversity will go a long way in obviating postoperative blindness.
In this era, huge strides have been made in all spheres of medical science including neonatology, helping us to salvage many lives that otherwise was unheard of in earlier times. However, this has also thrown up a host of pathology that was either was not very evident or did not present to the care providers on time. Delayed visual maturation is one of these.

The term ‘delayed visual maturation’ (DVM) was coined by Illingworth in 1961 and can be applied either in a broad sense to all infants who appear to be blind yet subsequently develop some vision, or, more specifically, to a group with normal ocular and systemic clinical examination whose visual behavior markedly improves by four to six months of age and whose visual acuity is subsequently normal. Without a consensus on usage there is therefore some confusion in the literature.

**Visual Milestones**

Normally, a visual following response should be present, and the baby should smile responsively to a parent by two months of age. At four months, a child should reach for an object. Visual evoked potentials and forced preferential looking (FPL) data show rapid improvement of grating resolution during the first months of life. In case the onset of smiling is delayed till after the age of eight weeks, and then too is prompted by handling or a voice and not by sight, the visual development is delayed. Such a child appears visually inattentive even by two to four months of age.

**Risk Factors**

Cortical Visual Impairment, Delayed Visual Maturation, and Cortical Blindness have been classified together as Neurological Visual Impairment (NVI). The risk factors for these conditions are similar with differentiation in the presentation according to the area of the brain affected.

- Asphyxia
- Brain maldevelopment
- Head injury
- Periventricular haemorrhage
- Infection e.g meningitis, encephalitis
- Seizure disorders
- Hydrocephalus, uraemia
- Premature infants with severe intercurrent illness

**Types of Delayed Visual Maturation**

**Type 1 DVM**

In infants with isolated delayed maturation of vision, general and neurological development is normal, and the only problem is that they appear to see less well than expected for their chronologic age. They have normal ocular examination and no systemic abnormalities. They generally have an excellent visual prognosis and show normal fixation and visual attentiveness by approximately six to twelve months of age, without specific treatment. However, several studies have emphasized that upon follow-up examination, these children may have delays in achieving developmental milestones speaking, sitting and walking, compared to unaffected siblings.

Fielder et al. have slightly modified this classification to 1A and 1B to include children without or with perinatal problems respectively, considering perinatal factors as negative prognostical indicators.

**Type 2 DVM**

In which there is neurological abnormality and/or learning disability and in which visual function is often, but not invariably, permanently impaired to some degree;

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**Figure A and B: Tools for Vision Stimulation**
Children who were born premature, with severe intercurrent illness early in life (hydrocephalus, brain malformations, infantile spasm, seizure disorders, hypoglycemia, hypocalcemia, Aicardi syndrome, tuberous sclerosis), mental retardation with or without seizures, belong to this group. Their vision usually improves in the same way as in group 1, with residual defects related to their illness such as residual visual defects, problems with visual perception, or hand-eye coordination. Those patients who have DVM in association with neurologic defects and/or seizures show slower visual development and less complete visual recovery.

**Type 3**

The children belonging to this category have ocular disease that occurred early in life and frequently have nystagmus. Children with albinism, bilateral cataracts and optic nerve hypoplasia belong to this group. Their vision is much worse than would be expected from the primary disease alone. Therefore, there is DVM in addition to their structural eye defect. Their vision improves to their final level more slowly and less fully than group 1, but faster and more completely than group 2.

Fielder and Mayer\(^1\) further expanded the classification by limiting Type 5 to albinism and idiopathic congenital nystagmus, and separating severe ocular disorders into type 4.

**Clinical Features**

**Type 1 DVM**

Initially the infant appears blind and does not fixate or follow a light or a large bright object. No visual response will be obtained on preferential looking tests. The eyes may be divergent (less commonly, convergent) or ‘roving’. Pupil reactions to light are normal. Ocular examination is normal, except for a few cases where a grey coloration of the optic disc is noted at presentation.

**Oculomotor findings**

Although the DVM infant does not voluntarily fix and follow, *binocular full-field optokinetic nystagmus (OKN) can be readily elicited*. However, *monocular OKN is markedly asymmetrical* with no nasotemporal following. Visual orientation improves, often rapidly over one or two weeks, between three and five months of age. Usually over the next few months completely normal eye movement recordings are attained. Monocular OKN becomes symmetrical, provided strabismus does not develop.

On spinning the infant, the vestibulo-ocular response is recordable\(^1\) even in the blind stage in infants with DVM type 1. In the absence of prematurity, *lack of nystagmus induced by spinning the infant should alert the clinician to the presence of neurological deficit other than DVM type 1*.

**Type 2 DVM**

The clinical picture is more complex; recovery is often slow and incomplete. Many affected infants have perinatal hypoxic injury and so associated abnormalities.

Full field binocular OKN may be abnormal\(^6\)

*Nystagmus may be present; in such a case, a neurological cause such as *intra ventricular hemorrhage* or *raised intracranial tension* must be sought*\(^1\).

**Type 3 DVM**

These infants also show a slower and mostly incomplete visual recovery as compared to type 1. *It has been noted that a characteristic congenital nystagmus develops one or two weeks before behavioral improvement in vision.*

**Type 4 and 5**

Depend mostly on fundoscopy and electrophysiology to identify associated sensory defects.

*DVM Type 4 and 5 are not commonly used terms.*

**Electrophysiological Findings**

There is wide variation in the normal waveform even in the visually attentive normal infant. It is therefore very difficult to establish that is outside the normal range. Despite the variations, it can be said that VEP is abnormal in the blind phase but may become normal later\(^7,\) \(^8\).

**Pathophysiology of DVM**

Hypoxic changes in the thalamus and dorsal brainstem can lead to this picture. Brain damage in the frontal or parietal cortex\(^1\) can lead to DVM; even patients with DVM type 1 without overt perinatal problems may in many cases have mild brain damage. These changes may or may not appear on an MRI scan; we cannot be sure that there are no subresolutional changes. Some authors believe a delay in myelination is responsible for the DVM. A combination of delayed myelination and delayed dendritic and synaptic formation is favoured by most, and this could easily result from hypoxic-ischaemic brain damage\(^5\).

**Neuroimaging**

MRI may reveal areas of hypoxic stress or non specific demyelination but there is no consistent pattern correlated with DVM type 1. MRI in Type 2 may show features of additional neurological insult.

**Prognosis**

If there has been no improvement in visual responsiveness by six months of age (corrected for any prematurity) then the diagnosis is not likely to be DVM type 1. *Most DVM infants show a marked improvement in vision by six months of age.* The six-month-old baby who appears blind (and who has a normal ERG and age-matched VEP) may yet achieve good vision, but is more likely to have a significant persistent neurological deficit. By definition, all patients with type 1 DVM achieve normal vision\(^7\). Type 2 and 3 do show some improvement but this is much slower and mostly though not always incomplete.

**Intervention**

Although vision improves on its own in most cases of DVM, vision stimulation\(^7\) is proven to help speed this up a little. For vision stimulation to be effective it needs to happen in everyday real life situations, not only in therapy sessions. Identifying colors in an activity, visually tracking their family members as they move across the room, and identifying the shape of everyday objects are examples.
Conclusion
The importance of early identification of serious visual disability is important for both rehabilitation reasons and genetic counseling. However, an incorrect diagnosis of visual impairment in an infant can be devastating to the family.

A thorough history and examination including fundoscopy can help in making the diagnosis. It is important to alleviate anxiety and reassure; vision stimulation needs to be started.

References
Balance between lipophilic and hydrophilic nature of topically applied drug is an important factor for corneal penetration. Corneal epithelium allows fat soluble drugs to pass through while stroma allows water soluble ones.

Topical anesthetics & Benzalkonium chloride (BKC) disrupts corneal epithelial integrity, thereby improving corneal penetration of topically applied water soluble drugs. Intraocular inflammation (e.g. endophthalmitis) breaks down the blood-retina & blood-aqueous barriers, thereby improving the diffusion of systemically taken drugs into the eye.

Melanin present in iris (uveal tissue) binds the lipid soluble drugs e.g. mydriatic-cycloplegic thereby delaying the onset of mydriasis in pigmented eyes but at the same time prolonging their effect. Normal pH & osmolarity of tear film is 7.5 & 300 mOsm/l respectively. Very high or low pH drugs are irritating when applied on the eye.

Contact time

 Longer contact time of topically applied drug to the corneal epithelium increases its absorption through the cornea. Various means to prolong it include-

- **Suspensions**: stay longer and Smaller particle size (optimally 2 ¼m) minimizes irritation & improves the dissolution.
- **Ointments**: however some ointments may bind the drug so strongly that it gets released in insufficient concentrations. Moreover, ointments blur the vision, hence are generally recommended to be used at night only.
- **Viscous carriers**: e.g. HPMC, polyvinyl pyrrolidone prolong the contact time and reduce the systemic absorption.
- **Gels and gel forming solutions**
- **Collagen shields applied over the cornea (e.g. from porcine sclera as in Biocor) can soak the drug and releases it gradually.**

Cyclodextrins

They are oligo-saccharides with hydrophobic central cavity but hydrophilic coating thereby improving the bioavailability of drugcyclodextrin complexes.

Liposomes

Can be used as drug carriers as they have an aqueous centre with phospholipid coating. Drug can be bound to either its lipophilic or hydrophilic component.

Topically applied corticosteroids, pilocarpine, atropine & chloramphenical penetrate cornea very well after topical administration unlike most antibiotics, antifungals, epinephrine & β-blockers which penetrate poorly under normal circumstances and need surfactant (e.g. BKC), corneal epithelial disintegrity or very frequent instillations.

Tips on topical anti-allergic therapy

It comprises of following categories of drugs:

- **Purely antihistaminic- pheniramine (0.3%), chlorpheniramine maleate**
- **Multi-action antihistaminics (these also in addition have the mast cell membrane stabilization effect) - Azelastin Hcl, Ketotifen fumarate (0.025%), Olopatidine Hcl (0.1 & 0.2%) & Levocabastine Hcl.**
- **Decongestants (vasoconstrictors) - Naphazoline Hcl (0.05% & 0.1%), Phenylephrine Hcl (0.12%), Antazoline Hcl (0.05%), Tetrahydrozoline (Tetryzoline, 0.4%).**
- **Mast cell stabilizers- Cromolyn sodium (sodium cromoglycate, 2-4%, Nedocromil sodium & Pemirolast potassium.**
- **NSAIDs- q.v.**
- **Corticosteroids- q.v.**
- **Zinc preparations by acting as antagonistic to calcium can help in mast cell membrane stabilization.**
- **4% Cromolyn drops are far superior to 2% although none is effective in severe allergies. Its full effect may take weeks of uninterrupted therapy.**
- **Topical vasoconstrictors should be avoided in patients with occludable angles. Prolonged use or overuse can cause rebound congestion and systemic effects like hypertension and arrhythmias especially in elderly patients. Blurred vision can also result because of slight mydriasis.**

Tips on Topical Antifungal therapy

- Efficacy of topical antifungal therapy is very limited and confines only to superficial ulcers as their penetration into corneal stroma is poor.
- **Natamycin (5% suspension) is considered the first choice as it is a broad spectrum antifungal and is especially effective against filamentous fungi e.g. Fusarium.**
- **Debridement of ulcer (may be needed 2-3 times) is very important in fungal ulcers as there is no good & effective antifungal available till today. Debridement also allows better penetration of antifungal into deeper corneal layers.**

Topical antifungal drugs:

- **Amphotericin-B (0.15%) - Mix 50 mg powder with 33 ml of d/w. Refrigerate in amber coloured or opaque bottle & shake well before use.**
• Ketoconazole (2%) - Crush & mix 200 mg table in 10 ml of artificial tear drops.

• Miconazole (1%) - Use IV solution directly as drops. Miconazole penetrates the corneal stroma better than other antifungals.

**Tips on topical antiviral therapy**

• Only Acyclovir (3% ointment) is available in India commercially as topical antiviral preparation.

• Acyclovir is a prodrug & is converted to active form by viral particles within the infected cells. Hence, it is very less toxic to the uninfected cells unlike Idoxiuridine which was the only antiviral available earlier.

• Topical steroids in viral keratitis are used only for viral stromal keratitis & must not be used if epithelial defects are present (i.e. fluorescein stain positive) and it may need to be given over many months.

• Topical antiviral treatment should be continued for 1-2 weeks after complete epithelial healing so as to minimize the chances of recurrence which itself takes 1-2 weeks, although healing starts with in 2-3 days.

• Topical antiviral therapy is given in herpetic stromal keratitis for prophylaxis.

• Herpetic epithelial keratitis is self healing disease (in 2-3 weeks time) and topical therapy only shortens the course of disease while recurrence rate of epithelial disease and incidence of stromal involvement is not reduced.

**Tips on topical dry eye therapy**

Preservative free solutions are preferred for dry eye because patient may need them for lifetime & also because prolonged use of preservatives can cause epithelial toxicity and disruption of the tear film.

**Tear substitutes:**

• Cellulose polymers- they get adsorb at corneal aqueous interface thereby stabilizing the fluid layer e.g.
  - Hypromellose (hydroxypropyl methylcellulose, HPMC 0.3% & 0.5% solution).
  - Carmellose (carboxymethyl cellulose, CMC 0.5% & 1% solution).
  - Carbomer 0.3% solution.

• Hydrophlic polymers- they have lower viscosity & have good film forming properties e.g.
  - Polyvinyl alcohol (1% & 1.4% solution)
  - Polyvinyl pyrrolidone (5% polyvidone)
  - Viscoelastic agents - e.g.
  - Sodium hyaluronate (0.1%)

<table>
<thead>
<tr>
<th>Organism</th>
<th>Drug of first choice (ocular use)</th>
<th>Next line drugs</th>
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<tr>
<td><strong>Gram +ve organisms</strong></td>
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<tr>
<td>Penicillin resistant Staph</td>
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<td>Nocardia</td>
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<td><strong>Gram -ve organisms</strong></td>
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<td>Acinetobacter</td>
<td>Carbenicillin + Aminoglycoside (synergistic)</td>
<td>Chloramphenical</td>
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<tr>
<td>Hemophilus</td>
<td>Ceftriaxone, FQ</td>
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<tr>
<td>Bacteroides</td>
<td>Penicillin G</td>
<td>Clinda, 3rd Gen cephalo</td>
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Lubricating ointments (water soluble) - e.g.
- White petroleum jelly (mineral oil)

Non water soluble ointments break up the tear film and are recommended only for night use except in very severe dry eyes.

Although no ideal tear substitute is available, a well tolerable tear substitute should have -

- Electrolytes resembling that of normal tears i.e. sodium, potassium, calcium & bicarbonates.
- Osmolarity below 300 mOsm/l (i.e. between 200-280) to compensate for higher osmolarity in tear film of dry eye patient.
- pH between 7.4-7.7.
- Preservative free. Chlorbutanol (0.5%) is safest among the preservatives.

Topical Cyclosporin (0.5% and 0.1% emulsion) also can help in moderate to severe dry eyes by providing actual curative effect as it inhibits inflammatory cytokine production & apoptosis.

Topical corticosteroids sometimes help in severe dry eyes with significant ocular inflammation causing irritation. It is given only as a short term pulse therapy.

Topical fortified Acetyl-cysteine- 2-5% solution can sometimes be used to open disulfide linkages in the viscid mucus thereby making it thin (mucolytic). However it has a foul smell of rotten eggs.

**Tips on topical antiglaucoma therapy**

Beta-blockers are contraindicated in asthma & other COPDs, bradycardia & cardiac failure. One has to be cautions in diabetes as these may mask the symptoms of hypoglycemia. Beta-1 blocker (Betaxolol) is also relatively contraindicated in these conditions and is preferably avoided.

Other side effects of Beta blockers include hypotension, impotence; mood disorders esp. depression, lipid & carbohydrate metabolism disturbances.

Two drops of 0.5% Timolol is equivalent to 10mg of systemic dose of beta blocker!

Alpha-2 agonists & Prostaglandins have no contraindication in these cardiopulmonary patients.

Alpha-2 agonists should be avoided in narrow angle as mild pupillary dilatation can precipitate an acute attack.

Local effects like burning, redness and allergic reactions are most common with alpha-2 agonists.

Topical carbonic anhydrase inhibitors can cause severe systemic side effects if absorbed systemically and are contraindicated in Sulfonamide allergy.

Side effects of Prostaglandins include conjunctival congestion & irritation & increase in iris pigmentation. They should be avoided in uveitis patients & asthmatics.

Pilocarpine can cause ciliary spasm with browaches & low grade uveitis. It should be avoided in patients with uveitis, cataract, young patients (<40 years of age) & neovascular glaucoma. It is relatively contraindicated in high myopes & all other patients with history of or predisposition to retinal detachment.

Combination of 2 antiglaucoma agents increases compliance and is probably equally effective as when these 2 drugs are used separately. Combination solution also decreases dose of preservative administered.

**Tips on topical NSAID therapy**

- Diclofenac & Ketorolac in addition have a significant analgesic effect and decrease in corneal sensitivity on topical use.
- 0.3% flurbiprofen for preventing intraoperative miosis.
- 0.1% diclofenac for postoperative inflammation (e.g. after cataract surgery)
- 0.5% ketorolac for allergic conjunctivitis.
Tips on topical mydriatic / Cycloplegic therapy

Atropine drops (1% i.e. 10 mg/ml) - one drop of atropine is equivalent to systemic therapeutic dosage of atropine. Thus if absorbed systemically, even a single drop can cause systemic side effects. Hence it is preferable to use atropine ointment rather than drops.

Full cycloplegia with atropine is achieved only after 2-3 days of tid dosage and lasts for 1-2 weeks.

Atropine is the most effective cycloplegic followed by Cyclopentolate (1%) & then Homatropine (2%) & Tropicamide (0.5-1%). Cycloplegia by tropicamide is very short lasting (half to one hour only).

Cyclopentolate should not be prescribed in uveitis cases as it has a chemotactic effect on leucocytes.

Mydriasis is quickest with Tropicamide among all parasympathomimetics (in 20 minutes) and is also shortest lasting (for 4-6 hours only), while it lasts for 24-48 hours with homatropine & cyclopentolate and it is 1-2 weeks for atropine.

Even 2.5% Phenylephrine (selective Alpha-1 agonist) induces maximum mydriasis along with anticholinergics like tropicamide. Phenylephrine in small concentration (0.125%) is also used as a potent conjunctival vasoconstrictor.

Local Anesthetics (LA)

These fall into 2 groups-

Amides

- Lignocaine (2% as injection & 4% as eye-drops).
- Bupivacaine 0.5%) is available only for injection
- Benoxinate (0.4%) is no more available commercially.

Esters- Proparacaine (0.5%)

No cross-allergy exists between esters & amides. Proparacaine is very less irritant unlike lignocaine & causes quick anesthesia within 20-30 seconds lasting for 15-20 minutes.

Lignocaine causes intense burning on instillation and takes 40-60 seconds for anesthesia to start.

All local anesthetics can cause corneal epithelial disintegrity, an effect which helps in increasing corneal penetration e.g. of mydriatic-cycloplegic to allow for faster dilatation of pupils.

LA should never be prescribed for home use as these not only make cornea vulnerable to injury but also are toxic to corneal epithelium & also cause delayed healing.

Viscoelastics

Viscoelastics (VEs) are broadly classified into 2 groups- Cohesives e.g. Hyaluronic acid & Dispersives e.g. Chondroitin sulfate (20%) & Methyl cellulose (2%).

Remove the VEs thoroughly after intraocular surgery especially if hyaluronic acid has been used as they can raise IOP postoperatively significantly.

Cohesives are primarily used for:
- During capsulorresection for maintaining anterior chamber
- For preventing iris prolapse during surgery.
- During IOL insertion to fill the capsular bag.

Dispersives are primarily used for:
- Corneal endothelium protection during phaco surgery by coating endothelium.
- For lubricating the IOL Injector system.

Tips on topical Antibiotic therapy

2nd generation Fluoroquinolones/FQ (Norfloxacin, Ciprofloxacin & Ofloxacin) have excellent activity against gram –ve organisms but less active against gram +ve organisms like Staph & poor activity against Strep including Pneumococcus. 3rd gen FQ (Lomefloxacin & Sparfloxacin) have better coverage of gram +ve organism. But 4th gen FQ (Moxifloxacin & Gatifloxacin) provide still better activity against gram +ve organisms including Pneumococcus. Cipro & Oflox (2nd gen.) & Gati/Moxi are also active against Mycobacteria (including Atypical ones).

All Fluoroquinolone drops are 0.3% except Moxifloxacin which is 0.5% & is supplied preservative free (because there is no carbon in Moxi molecule without which no microorganism can survive). Moxi drops are also safe in neonates.

4th Gen FQ achieve high aqueous concn. on instillation providing Mutation prevention concentrations (MPC or RPC i.e. Resistance prevention concn) rather than just the Mutation inhibition concern (MIC). They also have delayed epithelial healing effect (important in lasik surgery). Ofloxa has better corneal penetration & does not form precipitates on eye surface unlike Cipro.

Lomefl is given with initial loading dose of 1 drop every 5 min for 20 min followed by BD dosage.

Aminoglycosides are effective against Staph, Enterobacteriaceae & Pseudomonas (but not against Strep & anaerobes)

Tobramycin is less bound to tear proteins & hence is better available for ocular absorption than Gentamycin although both are equally efficacious.

Neomycin (used only topically) is the most frequent allergy causing agent.

Polymyxin-B (used topically only) is effective against Pseudomonas also. It is present in Neosporin ointment (5,000 IU/gm only) along with Bacitracin & Neomycin.

Bacitracin (only for topical use) is highly effective against Staph.

Vancomycin is especially useful against methicillin resistant Staph. Vancomycin & cefazolin have poor penetration into aqueous when given topically unlike FQ.

Chloramphenicol is especially useful for uncomplicated superficial bacterial infections because of its widest spectrum & least chances of inducing drug resistance. Bone marrow aplasia is rarely also possible on prolonged topical use.
Antibiotic drops given for prophylaxis against infections after cataract surgery should be abruptly stopped after 7-10 days & not tapered like topical steroids to prevent development of resistance.

Chronic canaliculitis is caused by Actinomyces israeli (Streptothrix). Treatment is opening of canaliculus on conjunctival side. Penicillin-G is the drug of choice.

**Fortified antibiotic drops**

- Conc. Tobramycin & Gentamycin (1.33%) - 5ml of 0.3% drops + 2 ml inj. (80mg)
- Conc Amikacin (2%) - 2 ml of 50 mg/ml Amika inj + 5 ml of 1% Aminogen drops
- Cefazolin, Ceftazidime & Ceftriaxone (5%) - To 500 mg powder, add 10 ml Tear substitute drops
- Vancomycin (5%) - To 500 mg powders, add 10ml diluent.
- Cloxacillin (5%) - To 500 mg powder, add 10ml diluent.
- Penicillin-G (1lacU/ml) - To vial of 10LacU, add 10ml diluent.

**Tips on topical corticosteroid therapy**

Corticosteroids (CSd) are used for their anti-inflammatory, anti-immunological, anti-angiogenic (this effect is unrelated to gluco & mineralocorticoid activity of individual CSd) effects.

Anti-inflammatory potency of CSd is different on systemic use than from their ocular application e.g. while dexe & betamethasone are more potent inflammatory steroids than prednisolone when given systemically, prednisolone (pred) acetate is far more potent than dexamethasone sodium phosphate when given topically because of better corneal penetration.

**Topical CSd**

All acetate preparations (e.g. Prednisolone, flurometholone, and hydrocortisone) are in suspension form; hence these must be shaken well before use.

Phosphate salts of Dexe/Betamethasone are less effective than acetate or alcohol salts. Phosphate salts are the ones primarily used in most of their eyedrop preparations.

Dexe/Betamethasone is normally used in 0.1% concern; however commercial preparations are now available in diluted forms e.g. 0.05% in Lowdex, 0.01% in Decolite & Solodex-J. These are supposed to have retained the anti-inflammatory potency but with diluted side effects.

Cyclodextrin preparations of Dexamethasone are (e.g. in Gate-Dx & Adrop-Dx) now commercially available. Cyclodextrin molecule significantly improves the Dexe penetration through the cornea (drugs need biphasic solubility to pass easily through the cornea as is provided by complexing with Cyclodextrin).

Out of all the commercially available CSd drops, prednisolone acetate (1% suspension) is the most efficacious anti-inflammatory drug. Probably Cyclodextrin preparations of Dexamethasone are also as efficacious.

CSd drop preparations that are less likely to cause secondary glaucoma include:

- Medrysone (1%) - is no more available now, is less efficacious & has poor corneal penetration.
- Loteprednol etabonate (e.g. Lotepred, Loteflam) is rapidly deactivated by cellular esterase enzyme inside the anterior chamber & on the surface of the eye.
- Flurometholone (0.1% & 0.25%) Anti-inflammatory efficacy of steroids is probably related to their ocular hypertensive effect.

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Retinal drug delivery is a challenging area in the field of ophthalmic drug delivery. A variety of approaches have been described for drug delivery into the vitreous cavity.1

Intravitreal injection was first reported by Ohm in 1911 as a technique to introduce air for retinal tamponade and repair of retinal detachment. Intravitreal administration of pharmacotherapies dates to the mid-1940s with the use of penicillin to treat endophthalmitis.2 Since that time, use of the intravitreal injection technique has progressively increased, with its usage being focused primarily on the treatment of retinal detachment, endophthalmitis, and cytomegalovirus retinitis.

The increasing efficacy and safety of intravitreal injections, in conjunction with the development of further pharmacotherapies, has led to a recent rapid increase in the use of this technique for the administration of various pharmacotherapies e.g., bevacizumab, ranibizumab, pegaptanib sodium and intravitreal triamcinolone acetone. An intravitreal drug application has been suggested to achieve therapeutic levels locally, with prolonged effective concentrations.

“All injections should be performed under standardized sterile conditions.”

Intravitreal drug delivery

The advantage of intravitreal drug administrations is an immediate therapeutic effect at the intended tissue. With the widespread use of intravitreal injections, there has been an increased interest regarding the best application technique.

Clinical experience with intravitreal injection has provided physicians with an outline of avoidable risks.

All injections should be performed under standardized sterile conditions. While surgeons/ophthalmologists in some countries recommend applying the injection in an operating room, those in other countries perform the injection in a conventional examining room under sterile conditions.3

Preoperative preparation

A major concern associated with preoperative discontinuation of anticoagulation therapy is the increased risk of thromboembolic or cerebrovascular events.4-6

Hemorrhagic events after intravitreal injections may relate to the procedure itself, the applied drug as well as the anticoagulation status of the patient. Small-incision techniques may limit the risks of hemorrhages in anticoagulated patient. Approximately 10% of all patients with exudative AMD receive warfarin anticoagulation due to concomitant medical conditions. These patients may undergo intravitreal injections without significant risk of hemorrhagic complications while maintaining their anticoagulation therapy.

Preoperative prophylaxis of endophthalmitis

It is mandatory to treat any active external infection, including significant blepharitis, before each intravitreal injection. It has been demonstrated in rabbits, that 51 ± 4 + colony-forming units of S. aureus are required to induce endophthalmitis, when injected into the anterior chamber, compared with 19 ± 8 + colony-forming units when injected into the vitreous. The primary goal of any endophthalmitis prophylaxis is to minimize the present bacterial flora around the surgical entry site. This can be achieved with the topical application of povidone–iodine, eyelid hygiene, proper isolation of the surgical site and possibly postoperative antibiotics.7-11

The key step to avoid an endophthalmitis is a sufficient disinfection of the skin, eye lashes and conjunctiva. Povidone–iodine reduces significantly the risk of postoperative endophthalmitis. The lids and lashes are usually disinfected by a povidone-iodine (10%) scrub. A sterile speculum is placed between the lids and 2-3 drops of povidone-iodine (5%) are then applied 2 several minutes apart over the ocular surface. The various methods of applying povidone–iodine preoperatively have been studied. Two drops of 5% povidone–iodine placed on the eye reduced conjunctival bacterial flora significantly by approximately 91% and reduced the prevalence of endophthalmitis significantly after intraocular surgery from 0.24% to 0.06%. Several investigators have also advocated irrigating the conjunctival sac with 1% or 5% povidone–iodine to decrease conjunctival colonization; however it remains unknown whether the application of povidone-iodine drops versus a flush has an impact to prevent an endophthalmitis. A surprising finding is the significant increase in bacterial flora with lid scrubs alone compared with a control. There is no consensus among experts whether topical antibiotics should be used preoperatively. Preinjection antibiotic drops may be applied according to the label of the applied drug, although there is no evidence supporting its use before intravitreal injections.

Anesthesia

Satisfactory pain relief may be achieved with topical lidocaine. The drug may be applied with 2% eye drops, gel or subconjunctivally. The effective relief of pain with lidocaine for intravitreal injection is independent of its mode of application (gel vs. subconjunctival...
injection). However, lidocaine gel usually causes less chemosis compared with subconjunctival anaesthesia pain sensation may be controlled by applying a sterile cotton swab soaked in sterile 4% lidocaine eye drops to the injection area12.

The procedure and recommended technique

Injection site: The injection site should be located 3.5 to 4 mm posterior to the limbus. The injection site may differ in repeated injections by approximately one clock hour. This avoids a double penetration through the same site, inducing a persisting scleral hole with consecutive leaking or vitreous incarceration ‘vitreous wick’.

Angle of incision: The angle of the incision through the sclera may be directed in an oblique, tunneled fashion, as rectangular radial incisions may remain open inducing vitreous or drug reflux under the conjunctiva as well as severe chemosis and even hypotony in vitrectomized eyes.13 We have observed persistent unsealed sclerotomies following radial injections using a 30-gauge needle, requiring secondary suturing to seal the penetrating scleral wound.14

Depth of the insertion of the needle: The depth of the insertion may vary between 5 to 7mm, so that the tip of the needle is placed in the mid vitreous. The drug is then gently applied into the vitreous cavity.

Needle diameter: The diameter of the needle should be smaller than 25-gauge to reduce the risk of injury to ocular structures or wound leakage. Injections with crystalline triamcinolone acetonide are frequently applied with 27-gauge needles, while most liquid injections are performed with 30-gauge needles. Experimental studies examined the force required to penetrate the sclera with 27, 30 or even 31-gauge needles on a tuberculin syringe and measured almost twice as much force to penetrate the sclera using the 27-gauge needles. Larger needles may not necessarily induce more pain to the patient, however apparently may induce more trauma, reflux and subconjunctival hemorrhages.

Needle sharpness: In addition, a reduced sharpness of the needle, as found in some prefilled syringes, induced also a deeper inbounding and visible indentation of the eye wall possibly causing more discomfort to the patients’ eye.

Injected volume: The injected volume should be limited up to 0.15 ml without a routine paracentesis releasing an elevated ocular pressure. Administration of tTPA, anti-VEGF and SF6-gas as triple injection for the management of subretinal hemorrhage requires frequently a paracentesis to release the elevated eye pressure.15-17

Ocular complications

A survey on published studies and case series including more than 15,000 intravitreal injections in 4,400 eyes determined after triamcinolone acetonide injections a prevalence of endophthalmitis (including pseudo-endophthalmitis) of 0.3% per injection and 0.9% per eye.

The overall prevalence for retinal detachments was 3.9% per eye or 0.9% per injection.

The prevalence of intraocular inflammation (iritis, uveitis, vitritis, and/or anterior chamber inflammation) was 28.5% per eye and 6.3% (839/13,400) per injection.

Traumatic cataracts, intraocular hemorrhage (i.e., retinal hemorrhage, vitreous hemorrhage, or hyphema) were reported occasionally after intravitreal injection.

Temporary hypotony, defined as an IOP of <4 mmHg, may occur rarely after intravitreal injections and is primarily limited to eyes receiving antiviral therapy.

Some cases of retinal artery occlusions have also been reported after intravitreal injections.

Postoperative prophylaxis of endophthalmitis

The role of topical antibiotics to prevent postoperative endophthalmitis remains controversial. Topical antibiotics may be applied after the injection for a few days, as the break in the conjunctiva and sclera is not completely healed and water-sealed. In addition there may be a synergistic effect between topical antibiotics and povidone-iodine. There is general agreement that the risk of infectious endophthalmitis following intravitreal injection is small. The exact rate of endophthalmitis after intravitreal injection is unknown due to the paucity of large reported series. The highest reported rate to date is 0.9% for triamcinolone acetonide and 0.01% for bevacizumab.

“The injected volume should be limited up to 0.15 ml unless a routine paracentesis to lower an elevated intraocular pressure is intended.”

General guidelines

General guidelines for intravitreal injections were established for the injecting physician. Some aspects are supported by consensus agreement (use of adequate aesthetics, povidone-iodine and a lid speculum; dilate the pupil; avoid injecting patients with active eyelid or ocular infection, avoid extensive massage of eyelids, avoid prophylactic or post-injection paracentesis) while other parts have less agreement (e.g. most investigators advocate gloves, most
prefer povidone-iodine drops over flush, most use no sterile drape. There is no agreement regarding the use of pre- or post-injection topical antibiotics, as well as a specific intraocular pressure level, that should not be exceeded before the injection.

The rational for intravitreal drug application is an immediate and increased therapeutic delivery to the targeted tissue. Intravitreal drug application is a safe and effective procedure. Possible side effects e.g. elevated IOP, cataract formation, and endophthalmitis are limited. The pharmacokinetics depends on the status of the vitreous as well as the structure of the drug. Combined surgical and pharmacological approaches have become an efficient approach to deliver drugs in therapeutic levels to the posterior segment.

References

A stem cell is a multipotent cell with the capacity to self renew and to produce daughter cells capable of differentiating into multiple mature cell types. However, progenitor cells, which possess the ability to generate a more limited range of adult cell types, may also contribute to tissue repair. Thus stem cells and or progenitor cells offer new hope for treating historically incurable disease, such as glaucoma, via the selective replacement of degenerated cells to restore function.

**Objectives**

**Retinal Ganglion cell replacement**

The clinical end point for uncontrolled glaucoma is total visual field loss as a result of progressive RGC death. Despite aggressive treatment, a significant proportion of glaucoma patients experience considerable visual field reduction during their lifetime.

In fish and amphibians, retinal regeneration is an automatic process that proceeds via differentiation of ocular stem cells located in the ciliary's marginal zone. In adult mammals, retinal regeneration after injury or in neurodegenerative disease does not occur.

Degenerated retinal neurons can be replaced by a transplantation of suitable precursor cells. It has been demonstrated experimentally that neural precursor cells derived from embryonic stem cells when transplanted into eye, can migrate into the retina and express markers of mature retinal neuron. Transplanted fetal derived hippocampal progenitors also demonstrate the ability to localize to the retinal ganglion cell layer from whence they can extend neuritis into the inner plexiform layer and towards the optic nerve head.

**Optic nerve head restoration**

Stem cell therapy directed towards repairing the structure and function of the ONH has been proposed as a possible way to slow disease progression. For example, given that fibroblast cells are responsible for maintaining the ONH extracellular matrix, it is conceivable that fibroblast precursor cells could modulate the environment of the glaucomatous ONH to enhance RGC survival.

**Trabecular Meshwork Restoration**

Restoration of TM function is a potential target for stem cell transplantation. While progenitor cell population isolated form the trabecular meshwork can be expanded in culture, it remains to be established whether such a cell population is capable of improving the conventional outflow pathway in glaucoma patients.

**Conjunctival Restoration and Glaucoma Filtering Surgery**

One emerging technique to repair leaky blebs is through ocular surface grafting of tissue equivalents. Tissue equivalents can be generated by the isolation and in vitro expansion of conjunctival specimens isolated from the superior fornix, which contains a population of conjunctival progenitor cells.

**Sources of stem cells**

*Embryonic stem cell (ES Cells).* These are derived from inner cell mass of the developing blastocyst and are capable of indefinite cell renewal and proliferation in culture. They are also pleuripotent, meaning that they can generate all cell types of the body.

In the context of glaucoma, ES cells in vitro have yielded both glial and neuroglial cell types. Differentiation can be accomplished in vitro by mimicking the molecular events that occur during development via exposure of cells to signaling molecules. Thus ES cells have been differentiated into retinal precursor cells that express markers of retinal development including Pax16, Lhx 2, Rx/Rax and Six 3/6.

In addition, ES cell transplants are necessarily allogenic and therefore, carry risk of graft rejection.

**Somatic stem cells**

Somatic stem cells derived from blood, skin, bone-marrow and umbilical cord may have neuronal potential. Intraocular implantation of mesenchymal and umbilical cord derived stem cells into a degenerated photoreceptor model has demonstrated an ability to improve retinal structure and function.

This would facilitate autologous grafting and would avoid graft rejection and negates the need for immuno suppression.

**Neural Stem Cells**

These are somatic stem cells that give rise to neurons, astrocytes and oligodendrocytes in the CNS. These are commonly isolated from the sub ventricular zone of the lateral ventricle. In vitro differentiation of NS cells into mature RGCs has yet to be achieved.

**Adult Ocular Cells**

Like limbal basal stem cells a population of cells in the trabecular meshwork has been isolated and expanded in culture. Genetic analysis suggests these TM cells exhibit an undifferentiated, progenitor phenotype. Proliferative cells capable of generating neural retinal cells have also been cultured form the pigmented ciliary body and the pigmented iris epithelium. The pigmented iris epithelium shares developmental origin with the pigmented ciliary body and the neural retina. This suggests the possibility that these cells, under the correct condition, may possess the potential to generate cells for each tissue type.

**Prospects of stem cell therapy in glaucoma**

Intravitreal introduction theoretically provides the transplanted cells with direct access to the inner retina. This route may prove to be more appropriate for glaucoma directed therapy, as opposed to outer retinal therapy in retinitis pigmentosa and macular degeneration.
One of the first tasks transplanted cells must accomplish is morphological integration in the host tissue. Integration into the intact rodent retina occurs much more readily in young animals than in adults, although the cells can survive in the posterior segment of adult eyes for weeks. This lack of integration may be partially overcome by injuring the adult retina. It appears that endogenous signals from the injured retina play a key role in determining the potential for integration of engrafted cells.

Transplantation of lineage-restricted cells has shown that grafted cells can differentiate and express some RGC specific markers in vivo. It may not be necessary to create fully differentiated RGCs in vitro for transplantation as it is possible that some of the required signals may remain endogenous to the adult retina, allowing less mature cells to be used.

Whether the glaucomatous retina can provide the necessary cues to guide the migration, differentiated, and integration of transplanted cells remains to be established.

Potential Hurdles

Rejection

It is a major complication of allogenic transplantation. Preferentially, autologous transplantation of the recipients own somatic stem cells would be a desirable future therapy.

Reactive Gliosis

Reactive gliosis following neural injury obstructs endogenous neurite regrowth and can impede the migration and integration of engrafted stem cells.

Axonal guidance and myelination

An approach would be to enhance the permissiveness of the host retina and optic nerve to neurite extension. One possible mechanism involves the down regulation of growth inhibitory molecules such as myelin associated protein (MAG) or nogo.

There is difficulty in assessing visual improvement in animal models.

There is continued disease progression.

Attention DOS Members

All DOS members less than 35 years are invited to write for DOS Times the best five articles will be selected the first authors will be invites as speakers at the Annual Conference on 17th and 18th April 2010 at Hotel Ashok.

Attention DOS Member

DOS Times is not published in the month of May & June each year.
Optical coherence tomography (OCT) is a novel, three-dimensional, non-contact imaging technology that allows detailed cross-sectional imaging (tomography) of the eye\(^1\). Ophthalmic OCT was initially developed for posterior segment imaging\(^2,3\) and used 830nm wavelength superluminescent diode (SLD) light source. The Anterior Segment OCT uses longer wavelength (1310 nm), thus allowing cross-sectional imaging and detailed visualization of the anterior chamber angle. A scan speed which is 40 times faster than previous OCT systems allows real-time imaging to be performed. In the last one decade, OCT has become one of the most common applied imaging technologies for retinal conditions\(^4,5\). However, it is only recently that the OCT technology has been adapted for similar imaging of the anterior segment.

**Development of Anterior Segment Optical Coherence Tomography (ASOCT)**

OCT was invented by David Huang and colleagues at a laboratory at the Massachusetts Institute of Technology (MIT) in 1991\(^6\). In 1994, Joseph Izatt and colleagues first demonstrated corneal and anterior segment OCT imaging\(^7\). Most of the initial work focused on corneal imaging\(^8,9,10\). The modern version of the Anterior Segment OCT was first developed by David Huang and Joseph Izatt, which was first announced at the First International Workshop on Corneal and Anterior Segment Optical Coherence Tomography in 2001. It had 3 key features:

- 1310 nm wavelength light
- Telecentric transverse scanning
- RSOD (rapid scanning optical delay) technology in the reference arm.

The system had a scan speed of 4000 A-scans/sec and 17 μm axial resolution (in tissue).

**Why the Need for Anterior Segment OCT?**

OCT systems utilizing 830 nm wavelength light was unsuitable for optimal imaging of the anterior chamber angle. Light of 830nm cannot penetrate the sclera. This prevents the visualization of anterior chamber angle structures. OCT using 1310nm wavelength is better suited for anterior chamber imaging for 2 reasons (See Figure 1). First, there is lower amount of scattering of light at this wavelength\(^11\) by opaque structures like sclera and limbus, allowing better visualization of sclera, the iris and the anterior chamber angle. (According to Rayleigh’s law, scattering of light is inversely proportional to fourth power of wavelength). Secondly, when light of 1310 nm is used, less than 7% of the light incident on the cornea is able to reach the retina\(^12\), compared to 93% transmission for 830 nm light. Thus, 20 times more power can be safely used for anterior segment imaging. Also since lesser amount of light reaches retina, it allows better retinal protection. In addition, light of 1310 nm wavelength penetrates six times deeper than light of 830nm, allowing better visualization of deeper angle structures.

The Zeiss Visante Anterior Segment OCT system was approved by the Food and Drug Administration (FDA) in 2005. The Visante OCT uses wavelength of 1300nm, and has a speed of 2000 A-scans/sec and 4-8 frames/sec. Such high scanning speed allows motion artifacts to be excluded.

Another ASOCT system available is the SL-OCT (slit lamp-mounted OCT) which is manufactured by Heidelberg Engineering (Germany). It also employs 1310nm wavelength, but the scanning speed is much slower (1 frame/sec).

**Principle**

OCT is based on the principle of low-coherence interferometry (Figure 2). Its principle is similar to that of ultrasound imaging, except that in OCT a beam of infrared light is used to obtain...
images, rather than an ultrasound wave. Light from a broadband, near-infrared source (SLD) is combined with a visible aiming beam and coupled into Michelson interferometer. Light is split into two fibers using a 2x2 coupler. One arm leads to the reference mirror and the other focused into the tissue of interest. By changing the length of the reference arm, reflection sites at various depths in the tissue can be sampled. An optical detector in the final arm of the Michelson interferometer detects the interference between the reference and tissue signals.

Utilization of Anterior Segment OCT for Glaucoma Imaging

Glaucoma provides a large domain of application of Anterior Segment OCT (ASOCT). Anterior Segment OCT can be used for both imaging of angle structures as well as quantitative measurements of the angle.

Imaging of Angle Structures

The ASOCT allows for detailed imaging of anatomy of cornea, iris and sclera. Structures in the anterior chamber angle can be clearly delineated, such as the scleral spur and the angle recess. However structures in the posterior chamber are not well delineated due to attenuation of the light beam of OCT by the pigmented epithelium of iris.

Quantitative Measurement of Angle Structures

The scleral spur is used as the landmark for measuring anterior chamber angle parameters. It marks the posterior boundary of trabecular meshwork. It is easily identified on ASOCT images and is seen as a highly reflective structure. The parameters for measurement of the angle are

- **Angle opening distance at 500μm (AOD 500)**: Defined as the perpendicular distance between the iris and trabecular meshwork (TM) at a point 500μm away from the scleral spur. The scleral spur is identified first and a point 500μm anterior to the scleral spur is marked. From here, a line is drawn perpendicular to the plane of the TM to the opposing iris. The distance between the last two points is defined as AOD500 (See Figure 4) This parameter was first described by Pavlin and colleagues for Ultrasound Biomicroscopy (UBM).

- **Angle opening distance at 750μm (AOD750)**: Angle opening distance is measured 750μm away from the scleral spur instead of at 500μm, as discussed above. It was suggested by Radhakrishnan et al.

- **Angle Recess area at 500,750 μm (ARA 500, ARA 750)**: ARA was first described by Ishikawa and co-workers for UBM. It is a triangular area, boundaries of which are AOD 500 or AOD 750 (base), angle recess (apex), the iris surface and the inner corneoscleral wall (sides of the triangle). The ARA may theoretically be a better parameter than AOD as it takes into account the contour of the iris surface rather than a single point on iris as is the case with AOD (See Figure 3)

- **Trabeculo-iris contact length (TICL)**: Defined as the linear length of contact between the iris and the trabecular meshwork, beginning at the scleral spur in an anatomically apposed or synechially closed angle. It was proposed by Radhakrishnan et al.

All quantitative measurements of the ACA require identification of scleral spur as the first step. However, according to a recent study by Sakata et al, it was not possible to identify the scleral spur in 28% of the eyes on images taken on Visante OCT. This study also found that it is more difficult to identify the scleral spur in patients with narrow angles than in patients with open angles. Another study reported that interobserver variability in the identification of scleral spur can lead to 50% variation in measurement of angle area (ARA, TISA) and 10% variation in linear measurements (AOD).

Applications of ASO CT in Glaucoma

- Imaging of the anterior chamber angle (Figure 5)
- Large scale screening for Angle Closure, Angle Closure Glaucoma
- Evaluation of structural causes of angle closure, effect of intervention such as laser iridotomy (Figure 6,7)
Applications in Angle Closure Glaucoma

The ASOCT permits objective measurement of the angle. The ASOCT is provided with calipers which can be used to exactly measure the angle recess in degrees (Figure 5).

Since it is a real time imaging device, it also allows the dynamic relationship between different structures, such as peripheral iris and TM to be studied. It also permits the assessment of dynamic relationship between structures such as iris and lens to be studied. This is important in cases of pupillary block glaucoma and pigment.

- Pachymetry values can be obtained (Figure 11)
- Angle assessment in the presence of corneal opacities (Figure 12, 13)
- Imaging of trabeculectomy blebs
- Tube position in Glaucoma Drainage Implants (Figure 14)
- Assessment of Cysts, Tumours of Iris and Ciliary Body (Figure 10)
dispersion syndrome. Illumination-induced changes in anterior chamber angle can also be studied.

AS-OCT can also find application as a screening tool for angle closure. ASOCT possesses certain advantages over gonioscopy (discussed below). Because it is non-contact and image acquisition is rapid, it can be used for screening a large number of patients.

ASOCT also allows the evaluation of efficacy of various treatment such as laser peripheral iridotomy (LPI), effect of cataract surgery on anterior chamber angle. LPI is evidence based primary intervention to relieve pupillary-block (Figure 6,7). See et al obtained ASOCT images of nasal, inferior, and temporal angles before and after laser iridotomy. There was a significant increase in AOD 500 in all three angles after laser iridotomy. TISA showed a significant increase only in the temporal angles while ARA 500 remained unchanged. Memarzadeh et al studied the change in anterior segment morphology by ASOCT and gonioscopy before and after LPI. They studied ten eyes of 10 patients with occludable angles. They noted a significant increase in AOD500, ARA500, TISA 500 and TISA 750 after PI. On OCT images, the convex iris configuration flattened after LPI.

Radhakrishnan et al, in a study of 31 eyes, have proposed a cut-off value of 0.19 mm for AOD500 for detection of occludable angles. This value was found to have a sensitivity of 100% and specificity of 87.5%. This translates into a cut-off value of 21° for the angle (in degrees). Thus, values smaller than this may be considered occludable. It must however be noted that angle values measured by scleral spur-based methods are larger than recess-based measurements which are larger than gonioscopic estimates.

Pachymetry

The ASOCT allows cross-sectional visualization and measurement of central corneal thickness (Figure 11). Accurate pachymetry mapping can also be done. This is important in pre-operative LASIK workup. This is also important in post-operative evaluation of LASIK flaps, as it can be used to measure flap thickness as well as residual stromal bed thickness (RSBT).

Zhao et al compared pachymetry values obtained by OCT with that obtained by ultrasound pachymetry and concluded that CCT readings of ASOCT were lower than that obtained by the other technique. In contrast, Leung and associates found in their study of 50 subjects that retinal OCT (Carl Zeiss Meditec) overestimated the CCT measured by ultrasound (Pachette 500) by a mean of 23 microns. These findings may have important implications in management of glaucoma and in refractive surgery. Previous pachymetry studies using retinal OCT show that OCT pachymetry correlated well with ultrasound pachymetry but tended to underestimate ultrasound values.

Imaging of Trabeculectomy Blebs

ASOCT may be used to provide information about trabeculectomy blebs. It enables us to clearly visualize the bleb wall, cavity, flap and ostium. In addition it demonstrates certain other bleb characteristics- bleb structure, location of scleral flap, presence of cystic spaces, and semi-quantitative characteristics- total bleb height, size of bleb cavity, bleb wall thickness, scleral flap thickness, tangential and radial dimensions of the bleb.

OCT is particularly helpful in demonstrating level of failure in failed bleb. It can demonstrate ostial closure, flap fibrosis or episcleral fibrosis as the cause of bleb failure. It can thus help in initiating appropriate management in rescuing a failing bleb.
Singh et al studied the ASOCT characteristics of trabeculectomy blebs in 78 eyes of 55 patients. They found that conjunctival-episcleral thickening in the bleb wall was the hallmark of blebs in which IOP was successfully controlled. In contrast, majority of failed blebs showed thin conjunctiva which was comparable to conjunctiva of virgin eye. Thus, flow of aqueous through the conjunctiva-episclera in a functioning bleb may be more easily demonstrable on OCT than on slit lamp examination. In 3 out of 21 (14.3%) failed blebs, the occlusion of inner ostium was shown on ASOCT to be responsible for bleb failure. In 5 failed blebs (23.8%) the level of obstruction was found at the level between boundary of sclera and subconjunctival space. In such cases, obstruction was presumed to be at the level of episclera, in which cases needling of bleb may prove therapeutic. It was thus concluded that imaging of internal ostium and trabeculectomy blebs in the early postoperative period by ASOCT may be helpful in guiding early measures to optimize aqueous flow through the bleb.

Angle Assessment in the Presence of Corneal Opacity
The ASOCT may be used to image the angle and iris even in presence of corneal opacity (See Figure 12,13). It may help provide a view of the angle when gonioscopic visualization of angle is impossible because of corneal edema or opacity. Memarzadeh et al evaluated four eyes of four patients with secondary glaucoma after penetrating keratoplasty with the anterior segment OCT23. They concluded that ASOCT was similar to UBM in allowing visualization of angle through opaque cornea.

Tube Implants
ASOCT provides high resolution images of the glaucoma drainage implant to assess it’s position, patency, drainage and intra-luminal stent suture (Figure 14). It can also demonstrate tube-cornea or tube-iris touch24. Such details may be difficult to see on slit-lamp examination, especially if the corneal clarity is reduced or if the intraluminal portion of the shunt is short25.

Technique of Angle Scanning
The angle can be imaged with the low resolution and high resolution scan with Visante OCT (Carl Zeiss Meditec Inc, Dublin, Calif). Low resolution scan allows imaging of the entire chamber and allows measurements such as anterior chamber depth, angle-to-angle width, iris thickness to be made. High resolution scans allow greater resolution of angle structures. The scleral spur can be identified clearly in high resolution scans. Both the low and the high resolution scans provide useful and complementary information. It is thus recommended to use both type of scans for angle imaging. However, for quantitative measurements of angle parameters, high resolution scans should be used.

Procedure for ASOCT Imaging (OPTOVUE)
- Explain the procedure to the patient
- Always clean all patient contact surfaces (forehead and chin rest etc)
- Ensure that correct CAM Lens is attached (CAM-L for angle scanning)
- Align the eye to the proper eye position (canthus) indicator mark on the chin rest

Figure 14: ASOCT image showing contact of tube with corneal endothelium

- Illuminate the external eye structures using the Red LED (Light Emitting Diode) illumination light
- Use the Live IR image on the LCD monitor to correctly orient the marker to the desired structure
- Always monitor the pupil size as change in pupil size due to external illumination can affect angle assessment
- Dark room conditions are needed to perform angle assessment

External Fixation
External fixation target is attached to the instrument and consists of a flashing red LED on a swivel arm. This may be used for other protocols but should preferably be avoided during angle assessment protocols. This is because the illumination of LED is bright enough to cause pupillary constriction. Other means of achieving fixation are:
- Verbal commands by the operator
- Temporary fixation target may be provided (eg operator’s finger)

Interpretation of OCT Image
Unprocessed OCT image is distorted by refraction at the air-cornea and cornea-aqueous interfaces due to the differential speed of light in air, cornea and aqueous. This distortion is removed using computer processing of the image. This is called “DEWARPING”. Dewarping calculation is used in pachymetry map. Processing of images is also done by the instrument before angle assessment.

Factors Which Influence Visualization of Angle Structures
1) Pigmentation of Iris
   a. Lightly pigmented iris: Light coloured iris (blue, gray) allows penetration of the 1310nm wavelength light. In such eyes, outline of ciliary body may be identifiable
   b. Heavily pigmented iris: Heavily pigmented iris provides a barrier to visualization of deeper angle structures. In such eyes, ciliary body is not identifiable
2) Iris thickness: Thicker iris obstructs more light. Constriction of pupil by pilocarpine may cause thinning of iris and thus improve visualization of angle structures.

3) Poor image quality, motion artifacts may contribute to difficulty in identifying key anatomic landmarks.

**Comparison of ASOCT with Gonioscopy**

The traditional method and reference diagnostic standard for angle assessment is visualization of angle structures by indirect Gonioscopy. However, there are certain limitations:

- Gonioscopy is subject to variability even in the hands of experienced practitioners. ASOCT shows less interoperator variability.
- It is a learned skill. Identification of angle structures may be difficult for beginners, especially in lightly pigmented eyes. ASOCT does not require technical expertise and the technique can be learnt easily.
- Inadvertent pressure on eye by the goniolens can cause distortion of angle structures whereas ASOCT is a noncontact technique.
- Slit lamp illumination required for performing Gonioscopy may cause artifactual widening of the angle due to light impinging the pupil. ASOCT uses infrared light and thus the angle can be viewed in its natural state.

**Correlation between Gonioscopy and ASOCT**

Nolan and colleagues examined fifty-four eyes of 29 patients with gonioscopy and ASOCT. In 28 of 29 eyes, temporal angle was identified as occludable on gonioscopy. OCT demonstrated narrow or closed angles in 24 of 28 eyes (85.7%). Inferior angle was identified as occludable on gonioscopy in 46 eyes, of which OCT identified 40 as narrow or closed (87%). In total, OCT correctly identified 81 (87%) of 93 occludable angle quadrants as occludable or closed. Other studies which compared gonioscopy for diagnosis of appositional angle closure with ASOCT have shown ASOCT to over-classify individuals as having closed angles. The most important reason for this may be that the definitions of angle closure using gonioscopy and ASOCT differ. Closed angle on gonioscopy is defined as non-visibility of posterior TM, whereas the definition of angle closure on ASOCT is any contact between peripheral iris and angle wall anterior to scleral spur.

Sakata and colleagues compared gonioscopy with anterior segment OCT in detecting angle closure in different quadrants of the anterior chamber angle. The study included five hundred and two subjects. The overall agreement between gonioscopy and anterior segment OCT in detecting angle closure in different quadrants was fair (κ = 0.40, 95% confidence interval, 0.35-0.45). The superior quadrant had the highest frequency of angle closure followed by inferior quadrant, both by gonioscopy and ASOCT. With gonioscopy, the frequency of persons with closed angles in the superior quadrant was significantly higher than in any other quadrant (P<0.03). With ASOCT, the frequency of persons with closed ACA in superior and inferior quadrant was significantly greater than nasal and temporal angles (P<0.001). Comparing the two techniques, frequency of closed superior and inferior quadrant was significantly greater using ASOCT, whereas the frequency of closed temporal angle was greater using gonioscopy. This difference may be attributable to technical difficulties in performing the two techniques. Viewing the temporal angle may be difficult using gonioscopy whereas imaging of superior and inferior angle (because of obstruction by upper, lower lid) is difficult with ASOCT.

(Figure 15,16) Gonioscopy of patient with PAC, ASOCT of same patient.

**Comparison of ASOCT and UBM**

Cross-sectional imaging of the anterior chamber angle can be performed by both the anterior segment OCT and the UBM. Both can be used to image through an opaque cornea. However, there are several advantages of OCT over UBM such as:

- ASOCT has a higher image resolution than the UBM. With high resolution corneal software, resolution of ASOCT can reach 8μ (UBM; lateral resolution =50μ, axial resolution =25μ)
- OCT uses light energy whereas UBM uses sound energy.
- OCT is a non-contact technique while the UBM uses an immersion bath. Non-contact nature of OCT leads to enhanced patient comfort and safety.
- Anterior segment OCT does not require technical expertise. It can hence be performed even by a technician. UBM, requires a highly skilled operator to perform it.
• ASOCT allows more rapid image acquisition (8 frames/sec) than UBM. Examination time is shorter with ASOCT.
• Better assessment of angle structures with OCT as there is minimal distortion of the angle, compared to UBM. Placement of an immersion bath can lead to some distortion of the angle.
• Newer angle measurement parameters (TISA, TICL) are available with the ASOCT.
• Important landmarks such as scleral spur are more distinct on ASOCT images than on UBM.
• Patient does not have to lie supine as in UBM. Lying supine may theoretically cause iris-lens diaphragm to fall back. ASOCT is performed with patient seated and is hence, more physiological.
• With ASOCT, all 4 angle quadrants can be measured simultaneously whereas UBM can image only one angle at a time.

Radhakrishnan et al performed a study to compare OCT and UBM angle parameters with gonioscopic grading by glaucoma specialists21. Thirty-one eyes of 28 subjects were examined. They found excellent correlation between Gonioscopy and OCT and UBM. OCT showed 100% sensitivity and 97.5% specificity in detecting gonioscopically occludable angles. Dada et al31 also reported excellent correlation between OCT and UBM in measurement of nasal angle (r=0.84; P<0.0001), temporal angle (r=0.86, P<0.0001), ACD (r=0.97, P<0.0001) and CCT (r=0.91, P<0.0001). There was no statistical significant difference between the mean ACD, CCT and angle parameters measured by the OCT or UBM. However, the ASOCT images showed sharper definition of the scleral spur than the UBM but could not image the ciliary body due to attenuation of light by overlying iris.

**Limitations of ASOCT**

Limited visualization of structures posterior to the iris (which is possible with UBM, with which imaging of ciliary body and peripheral retina is possible). Thus the UBM allows detection of anterior rotation of ciliary body, iridociliary cysts, cycloidalysis etc.

Imaging of superior, inferior angles is difficult due to obstruction by eyelids. While retracting the lids, one should be careful not to exert accidental pressure on the globe as this may distort angle structures.

Identification of scleral spur may be subject to inter-observer variability. It may be difficult to identify the scleral spur in narrow/closed angles.

In conclusion, Anterior segment OCT is a rapid, non-contact, non-invasive technology which allows detailed evaluation of the anterior chamber angle and the angle structures. Since it operates in the infrared spectrum, it allows the angle to be viewed in its natural state. Widespread use of anterior segment OCT may permit accurate diagnosis of angle closure cases even by an ophthalmologist untrained in gonioscopy. It may thus help pick up more cases of angle closure, which constitutes 50% of all glaucoma cases worldwide.39,40,41,42,47. Ease of image acquisition and non-contact nature may make it a desirable tool for large-scale screening of patients with narrow angles. Furthermore, it's a potentially valuable tool in glaucoma research and may help us understand better the natural history and patho-physiologic mechanisms behind different types of glaucoma. However, there are certain limitations of ASOCT such as limited visualization of structures posterior to the iris and difficulty in identification of key landmarks (eg scleral spur) in closed angles.

**References**


The internet is a wonderful new universe. Mysterious and full of surprises. Seek and thou shall find holds true here, more than anywhere else. Look for crap and you will wallow in the dirt to your hearts content, but look for gold and for diamonds [meaning good relevant stuff] and you will find them too, albeit with a little more effort.

I wish to share with you some of the good stuff, that I found. Some of which you may already know, even though I will try my best to surprise you.

Need less to say, every thing is about ophthalmology.

Ophthalmology excites us, it gives us as much pleasure as!! well it gives us a great amount of pleasure.

The internet is bursting with information about almost everything. Even our beloved Ophthalmology. Literally bursting to its seams. My aim is not to overload you, but to try and delight you.

Have you done a FM100 Hue Test for colour vision. I think very few institutes have it. I hadn't seen one before, except in text books and here suddenly I found myself doing it on my computer screen. I'll tell you where.

This is where.

http://www.colormunki.com/article/hue_test

I reached this site by accident[this is not an ophthalmology site as such] and took this test. The hue test is really exhausting. Compared to this our Ishihara is as simple as “patting a baby’s behind”.

Try it. They ask you to register, but that is easy. It is free.

Another such place to enjoy ophthalmology is this website. www.alconinstitute.com

This is a Spanish website by Alcon and the few guys who meet me from alcon in Gurgaon were unaware of it.

Now what is so exciting about it, and why am I raving about a website from spain and one that is being maintained by a pharma company.

There are reasons.

We are strangulated by info overload. We are drowned in the sea of magazines, and journals and books, haunted by over conferencing and we want a breather.

But there is no where else to go, no where else for us. We are ophthalmologists and this is our world.

We want it to be different, but we want the same.
Alcon eye institute is just that. Same of the different, or have it your way; different of the same.

“The more things change, the more they remain the same”

This is a website dedicated to the student in all of us. The resident and the teacher and the practitioner.

There are animations galore. They are beautifully created and it is a shame that all of us have not seen them.

For example this animation on “Sulcus fixated IOL”. Agreed, you have seen actual videos maybe, but the sheer beauty of this animation is fascinating.

And this animation on the radiotherapy of Choroidal Melanoma. This is not just education, This is education with artistry.

And then there are simulators.

These simulators have excellent graphics and are highly educative. It is like attending a workshop. There is this simulator on
Retinoscopy [there is a much better tutorial on retinoscopy elsewhere on the web about which I will talk some time later]. Now, nothing can teach a guy retinoscopy, except actually doing it, but this simulator does try its best.

And a simulator on visual field defects in different neuroophthalmologic conditions and one on ocular movements.

And one on Phacodynamics too. **Phaco is God. It is omnipresent.**

Real cool. I tell you.

**Nestled in a quiet corner, at this address** [http://www.cybersight.org/bins/content_page.asp?cid=1-3](http://www.cybersight.org/bins/content_page.asp?cid=1-3) is the **Strabismus Minute**.

Not easy to find maybe, but definitely worth the effort. A more interesting and absorbing CME on Strabismus will be hard to find.

Short, independent tutorials, beautifully illustrated and the language so lucid that it is almost like reading your favourite novel. The following quote will illustrate the great sense of humour, that is instilled in the whole of "Strabismus Minute".

* **Throughout these pages you will find the comment**: "There are only two kinds of strabismus – congenital esotropia and all the rest."

When ever I need a brush up on my shaky squint, I go back to the strabismus minute. Reading it gives me as much pleasure as reading "P G Wodehouse", my favourite author of comedy fiction.

**Strabismus minute is authored by the highly reputed** "**Editor-in-Chief**: Eugene M. Helveston, M.D and **Senior Editorial Consultant**: Gunter K. von Noorden, M.D.

I will be back with more exciting things in my next post.

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**Figure 7**: Screen grab of a page from the strabismus minute.
Delhi Ophthalmological Society
Monthly Clinical Meeting, July 2009

Dr. Rajendra Prasad Centre for Ophthalmic Sciences

Venue: Jawahar Lal Auditorium, All India Institute of Medical Sciences, Ansari Nagar, New Delhi -110029

Date and Time: 26th July, 2009 (Sunday)

Annual General Body Meeting: 9:00 a.m. (Tea & Coffee)

First 20 Early Bird Prizes

Clinical Cases:
1. Masquerade Syndrome : Naginder
2. Congenital Cystic Eye : Mridula Mehta

Clinical Talk:
Macular Hole Surgery Revisited : Atul Kumar

Mini Symposium: How to do it?

Chairman: Prof. Supriyo Ghose  Co-Chairman: Prof. Rajvardhan Azad
1. Revision Trabeculectomy : Viney Gupta
2. Cut & Paste Pterygium Surgery : Tushar Agarwal
3. Double Bubble DALK : Namrata Sharma
4. Scleral Fixated IOL’s : Rajesh Sinha

To be followed by Lunch sponsored by: M/s. Syntho Pharmaceuticals Pvt. Ltd.

Delhi Ophthalmological Society
Monthly Clinical Meeting, August 2009

Venue: Conference Hall Dr. Shroff’s Charity Eye Hospital, Daryaganj, New Delhi- 110002

Date and Time: Sunday, 23 August 2009, 11:00 AM

Clinical Cases:
1. Refractive surgery in Accommodative Esotropia : Manisha Acharya
   Discussant : Suma Ganesh
2. Management of Cataract with Corneal ectasia : Monica Gandhi
   Discussant : Umang Mathur

Clinical Talk:
‘Oculoplasty Then And Now’: The New Horizons : Sima Das

Mini Symposium: Quality Assurance in Eye Care

Chairman: Dr. Noshir Shroff  Co-Chairman: Dr. Suneeta Dubey
1. Quality assurance in health Care : Arti Verma
2. Our path towards achieving quality : Suneeta Dubey
3. To err is human…but certainly not quality : Sandeep Buttan

10 Early Bird Prize (Free Movie Tickets to PVR Cinemas)
The meeting would be followed by Lunch.
Successful Treatment of Stevens-Johnson Syndrome with Steroid Pulse Therapy at Disease Onset

Yayoi Araki, Chie Sotozono, Tsutomu Inatomi, Mayumi Ueta, Norihiko Yokoi, Eiichiro Ueda, Saburo Kishimoto, Shigeru Kinoshita


PURPOSE
To evaluate the visual prognosis of patients with Stevens-Johnson syndrome (SJS) and its severe variant, toxic epidermal necrolysis (TEN), followed by general and topical high-dose corticosteroids administration from disease onset.

DESIGN
Prospective, observational case series.

METHODS
Between May 1, 2003 and June 30, 2005, we enrolled 5 patients with SJS or TEN with ocular complications at the acute stage. Intravenous pulse therapy with methylprednisolone (steroid pulse therapy; 500 or 1000 mg/day for 3 to 4 days) was initiated within 4 days from disease onset. Topically, 0.1% betamethasone was applied over 5 times daily for at least 2 weeks. Visual acuity (VA) and slit-lamp microscopic appearance 1 year from disease onset were evaluated.

RESULTS
At the first examination, corneal or conjunctival epithelial defects and pseudomembranous conjunctivitis were present in all cases. Skin eruptions dramatically improved after steroid pulse therapy. Although ocular inflammation increased for several days, pseudomembranes disappeared and corneal and conjunctival epithelium regenerated within 6 weeks. At the chronic stage, all eyes had clear corneas with the palisades of Vogt (POV), implying the presence of corneal epithelial stem cells. Best-corrected VA was 20/20 or better in all eyes. Five eyes showed superficial punctate keratopathy. No eye had cicatricial changes except for 1 with slight fornix shortening. No significant adverse effects of steroid occurred during all clinical courses.

CONCLUSIONS
Steroid pulse therapy at disease onset is of great therapeutic importance in preventing ocular complications. Topical betamethasone also shows great promise for preventing corneal epithelial stem cell loss in the limbal region and cicatricial changes.

Online Journal Available
Many New Journals at DOS Library

Dear DOS Members,

We are pleased to announce that DOS has subscribed to online access of the following 18 journals. We are also in the process of adding a few more journals. These journals can be accessed at the DOS library situated at Room No. 2225, 2nd Floor, New Building, Sir Ganga Ram Hospital, Rajinder Nagar, New Delhi-60. The timings are from 10.00 A.M. to 5.00 P.M. on weekdays and 10.00 A.M. - 2.00 P.M. on Saturday. The library will remain closed on Gazetted Holidays. Members are requested to utilise the available facilities i.e. Computer with Video Editing & Conversion facility VHS to VCD, Journals Viewing, Books and Journals etc. The DOS members can get the full text articles of the current issues as well as many back issues of these subscribed journals. You need to send the request for the article needed. We will email you full text.

E-mail ID: dos_library@rediffmail.com

- Acta Ophthalmologica
- Acta Ophthalmologica Scandinavica Supplement
- British Journal of Ophthalmology
- Contemporary Ophthalmology
- Current Opinion in Ophthalmology
- Evidence-Based Ophthalmology
- Journal of Glaucoma
- Journal of Pediatric Ophthalmology & Strabismus
- Ophthalmic Surgery & Lasers
- Ophthalmology Management
- RETINAL Cases & Brief Reports
- Acta Ophthalmologica Scandinavica
- Archives of Ophthalmology
- Clinical & Experimental Ophthalmology
- Cornea
- Evidence-Based Eye Care
- International Ophthalmology Clinics
- Journal of Neuro-Ophthalmology
- Journal of Refractive Surgery
- Ophthalmic Surgery, Lasers & Imaging
- Retina
- Techniques in Ophthalmology
Forthcoming Events : National

August 2009
29th-30th, NEW DELHI
Annual Conference Intra Ocular Implant & Refractive Society, India
Contact Person & Address
Dr. Charu Khurana & Dr. Vikas Menon
Organising Secretary
Centre For Sight
B-5/24, Safdarjung Enclave, New Delhi-29
Tel: +91-011-41644000, 41653401-07
Email: iirsidelhi@gmail.com

August 2009
30th, CHANDIGARH
XXII Annual Conference of Chandigarh Ophthalmological Society
Contact Person & Address
Dr. Jaspreet Sukhiha, Organising Secretary
Advanced Eye Centre, PGI, Chandigarh
Tel: +91-0172-2756111 (M) 09876118740
Email: secretarycos@yahoo.co.in
Website: http://www.cosonline.co.in

October 2009
2nd, NEW DELHI
14th Dr. R.K. Seth Memorial Symposium on Surgical Advances in Cataract
Contact Person & Address
Dr. Gaurav Kakkar, Organising Secretary
Venu Eye Institute & Research Centre,
1/31, Sheikh Sarai, Institutional Area,
Phase-II, New Delhi-17
Tel: +91-011-29251155/56 Fax: 011-29252370
Email: education@venueyeinstitute.org

October 2009
2nd-4th, NAINITAL, UTTARAKHAND
Uttara-Eyecon-2009
6th Annual Conference of Uttarakhand State Ophthalmological Society
Contact Person & Address
Dr. Anurag Garg, Organising Secretary
Prakash Eye Hospital & Laser Centre
Doctor Colony, Civil Lines, Rudrapur
Tel: 05944-246946, Fax: 242394 (M) 09837180286
Email: uttaraeyecon2009@gmail.com
Website: http://www.cosonline.co.in

November 2009
14-15, GORAKHPUR, U.P.
44th Annual Conference of UP State Ophthalmological Society
Venue: B.R.D. Medical College Campus, Gorakhpur, U.P.
Contact Person & Address
Dr. Satish Sharma, Chairman, Organising Committee
Deptt. of Ophthalmology, B.R.D. Medical College, Gorakhpur, U.P.
Mobile: +09415313296
Email: ghoshorgsec09@gmail.com

25-28, PALAMPUR, HIMACHAL PRADESH
XVIII Annual Conference of Vitreo Retinal Society of India
Contact Person & Address
Dr. Sangeet Mittal, Organising Secretary
VRSI 2009, Third Eye Hospital,
701-L, Mall Road, Model Town, Jalandhar, Punjab
Mobile: +09988988844
Email: sangeet.mittal@gmail.com
Website: http://www.vrsi.in

January 2010
21-24, KOLKATA
AIOC 2010: Joint Meeting of the 68th AIOS Annual Conference & 15th Afro-Asian Congress of Ophthalmology
Contact Person & Address
Dr. Ashis Kumar Bhattacharya Organising Secretary
IMA House, Room No. 8, 53,
Creek Row, Kolkata - 700014
Tel: 033-22371679, 033-22366350,
Mobile: +09831019779
Email: oswb@vsnl.com

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Forthcoming Events: International

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12-16 BARCELONA, SPAIN
XXVII Congress of the ESCRS
Phone: +35312091100, Fax: 35312091112
Email: escrs@escrs.org, Web Site: http://www.escrs.org

October, 2009
15-17 SINGAPORE
Singapore International Convention & Exhibition Centre
NHG Eye Institute, Level 1, TTSH Medical Centre,
11 Jalan Tan Tock Seng, Singapore
Phone: 065-6357 7735 / Fax: 065-6357 7649
E-mail: tei@nhg.com.sg

24-27 SAN FRANCISCO, USA
Annual Meeting American Academy of Ophthalmology
Contact
American Academy of Ophthalmology,
P.O. Box 7424, San Francisco, CA 94120-7424
Phone: 415.561.8500 Fax: 415.461.8533

November, 2009
21-23 INDIA
World ROP Congress 2009
Hotel Le Meridien,
Contact Person & Address
Prof. Rajvardhan Azad,
Executive Chairman, Organizing Committee
World ROP Congress Secretariat
Room No. 486, Dr. Rajendra Prasad Centre for Ophthalmic Sciences, All India Institute of Medical Sciences, Ansari Nagar, New Delhi
Phone: +91-11-26593187 / Fax: +91-11-26852919
E-mail: world@gmail.com,
Website: http://worldrop2009.org

Missed DOS Times Copy
If your have missed your copy of DOS Times.
Please Contact:
Secretary DOS: Dr. Amit Khosla
Room No. 2225, 2nd Floor, New Building,
Sir Ganga Ram Hospital, Rajinder Nagar, New Delhi - 110 060
Ph.: 91-11-65705229 • E-mail: dosonlin@vsnl.net,
Website: www.dosonline.org
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 _____________________________________________ Mobile No. __________________

Proposed by
Dr. ______________________________________ Membership No. ________ Signature___________________

Seconded by
Dr. ______________________________________ Membership No. ________ Signature___________________

[Must submit a photocopy of the MBBS/MD/DO & State Medical Council / MCI 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__________________________

Three specimen signatures for I.D. Card.

Signature of Applicant with Date

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 accept 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 receive 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. Amit Khosla, Secretary, Delhi Ophthalmological Society, Room No. 2225, 2nd Floor, New Building, Sir Ganga Ram Hospital, Rajinder Nagar, New Delhi - 110 060
7. Licence Size Coloured Photograph is to be pasted on the form in the space provided and two Stamp/Licence 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).
8. Applications for ‘Delhi Life Member’ should either reside or practice in Delhi. The proof of residence may be in the form Passport/Licence/Voters Identity Card/Ration Card/Electricity Bill/MTNL (Landline) Telephone Bill.

Proceeding Protocol for Monthly Clinical Meetings

1. The Host (usually the ophthalmic chief of the Hosting Institution) will welcome the DOS and request the President and Secretary of the DOS to come to the Dais and start the Meeting.
2. The President and the Secretary will take up their seats on the side of the Dais, which is opposite to the Lectern. (They would continue to be in the same position throughout the Meeting, including the Mini Symposium.) The Chairman of the Symposium will be invited to a Third seat next to the President on the same table, after the ‘Clinical Talk.’ The Speakers, who if they form a Panel would be seated on the same side as the Lectern.
3. The President will declare the Meeting open.
4. The President and the Secretary will then conduct the meeting.
5. The case presentations (2 in no.) will form the first part of the clinical meeting. Each presenter will be allotted 10 min. time for his/her presentation. This will be followed by discussion with the audience on both the cases (Total time allotted is 15 min.). The case presentation will be followed by a Clinical Talk of 15 min. duration. This will be followed by discussion with the audience on the topic for 10 min.
6. After the first part of the meeting is over, the President will introduce the subject of the Mini Symposium (which will be of 1 hour duration) and invite the Chairperson of the Symposium to the Dais to conduct the Symposium. All the speakers may be invited to assume their seats on the Dais at this time or one by one after they have presented their Talks (at the discretion of the chair person of the symposium).
7. After the Symposium is over, the President will thank the Speakers and the Chair person and request Secretary to make any Announcements, including the Prizes etc.
8. By the time, the Clinical Meeting is to be declared closed by the President, the Host or his representative would be at the Lectern to (take the floor immediately after the Meeting is closed) thank people, firms who had helped him in hosting the Meeting and invite the Members of the DOS for Refreshments.

9. Venue: The monthly clinical meeting will definitely be held in the premises of the allotted institution.
10. Day: The meeting shall be held on the last Saturday/Sunday of the month, whichever the institution deems feasible.
11. Presenter: The presenting faculty/resident/fellows should be from the same institute for clinical case presentations and the clinical talk.
12. One person will be allowed only one-presentation for the award-winning session in the same academic year.
13. Exchange of dates: In case two institutions want to exchange the date of the meeting, it can be done with mutual agreement by the heads of the department and with information to the Secretary’s office, well in advance.
14. Mini Symposium: It shall be organized by the institution but other DOS members can be invited to participate, if required. There should not be more than 3 speakers in the mini symposium.
15. To qualify for the retention in the monthly meeting calendar, a minimum attendance of 70 members is required (inclusive of the members of the host institute).
16. For the Best Clinical Meeting award i.e. Bodhraj Sabharwal Trophy, the overall assessment of the meeting will be made purely on the overall marks by outside delegates and for Dr. Minoo Shroff Trophy the award will be given to the most popular meeting (based on total attendance including outside and inside delegates as per the attendance register).
17. The attendance will be marked in the register which will be at a separate counter and will be managed by the DOS Staff. At the close of the clinical meeting, the attendance register will be signed by the Secretary and the President on the same day.
18. Meetings in the month of May and June may be opened from the next year if there are applications for the same.
19. No alcoholic drinks will be served during or after the meeting; only refreshments/snacks/lunch will be served.
Applications are invited for DOS Fellowship for partial financial assistance to attend conference(s).

**Conferences**

**International**: Two fellowships per year (two fellowships can be awarded at a time if committee feels that papers are very good)
- Maximum of Rs. 25,000/- per fellowship will be sanctioned

**National**: Three fellowships per year (only for AIOS)
- Maximum of Rs. 5,000/- per fellowship will be sanctioned

**Eligibility**
- DOS Life Members (Delhi Members only)
- 75 or More DCRS Points
- Accepted paper for oral presentation, poster, video or instruction course.

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

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

**Quality of Paper**
The applicant has to submit abstract along with full text to the DOS Fellowship Committee. The committee will review the paper for its scientific and academic standard. The paper should be certified by the head of the department / institution, that the work has been carried out in the institution. In case of individual practitioner he or she should mention the place of study and give undertaking that work is genuine. The fellowship committee while scrutinizing the paper may seek further clarification from the applicant before satisfying itself about the quality and authenticity of the paper. Only Single best paper has to be submitted by the applicant for review (6 copies). Quality of the paper will carry 50% weightage while deciding the final points.

**Poster and Video**
The applicant will need to submit poster and video for review.

**Credit to DOS**
The presenter will acknowledge DOS partial financial assistance in the abstract book / proceedings.
The author will present his or her paper in the immediate next DOS conference and it will be published in DJO/DOS Times.

**Points Awarded**

1) **Age of the Applicant**
   - < 35 years: 10
   - 36 to 45 years: 07
   - 45 years plus: 05

2) **Type of Presentation**
   - Instructor/ Co-instructor of Course: 12
   - Free Paper (Oral) / Video: 07
   - Poster: 05

3) **Institutional Affiliation**
   - Academic Institution: 15
   - Private Practitioner: 20

4) **DCRS Rating in the immediate previous year**
   - 75-150: 05
   - > 150: 08
   - < 75: not eligible for fellowship

**Documents**
- Proof for age. Date of Birth Certificate
- Original / attested copy of letter of acceptance of paper for oral presentation / video / poster or instruction course.
- Details of announcement of the conference
- Details of both International & National Conferences attended in previous three years.
- Copy of letter from other national or international agency / agencies committing to bear partial cost of conference if any.
- At least one original document should be provided, that is ticket, boarding pass or registration certificate along with attendance certificate of the conference.
- Fellowship Money will be reimbursed only after submission of all the required documents and verified by the committee.
- Undertaking from the applicant stating that above given information's are true.
- If found guilty the candidate is liable to be barred for future fellowships.

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

**Dr. Amit Khosla**  
Room No. 2225, 2nd Floor,  
New Building, Sir Ganga Ram Hospital  
Rajinder Nagar, New Delhi - 110 060  
Ph.: 91-11-65705229  
**E-mail**: dosonlin@vsnl.net, **Website**: www.dosonline.org
## Advertisement Tariff: DOS Times & DJO

(July, 2009 to April, 2010)

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- Ten issues: 20 % discount
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  - Three pages ten issues additional 15% discount
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  - Four pages color ten issues Flat 50% discount

If any company booked the advertisement in DOS Times + Delhi Journal Ophthalmology, we will be given 5% extra discount.

Please send your bookings at the earliest accompanied by Demand Draft in the favour of “Delhi Ophthalmological Society” payable at New Delhi.

**SPECIFICATION of DOS Times & Delhi Journal of Ophthalmology:**

- **Size of Advertisement page:** 7.5” × 10.25”
- **Frequency of DOS Times:** Monthly (10 Issues in a year) (July – April)
- **Frequency of Delhi Journal Ophthalmology:** Quarterly (4 Issues in a year) (July – April)
- **Model of Printing:** Offset
- **Advertisement Material:** CDR/PSD open file with fonts & proof
- **Mailing and Contact:**
  - **Delhi Ophthalmological Society**
  - Room No. 2225, 2nd Floor, New Building
  - Sir Ganga Ram Hospital,
  - Rajinder Nagar, New Delhi - 110060.
  - **Phone:** 011-65705229
  - **Email:** dosonlin@vsnl.net, dosrecords@gmail.com
Assessment of Poor Vision

Does the child see? 1

Visual / Social contact to Examiner’s face / cartoon / silent toys 2

No

Yes

Follows silent toys moving 3

Yes

Check Preference of fixation with either eye 4

Yes

If Squint Present

No

Induced 10 pd B.D. test 5

No

Also resents closure of preferred eye 6

Yes

Non preferred eye Amblyopic

Yes

Strabismic Amblyopia

Quantification of Visual Acuity 7

6-18mths

Teller Acuity

1-3 yrs

Cardiff Acuity

3 yrs +

Picture Cards

4 yrs +

Leiter Optotypes

Also check with pinhole if V.A. Less 7

Improves

Refractive error

Glasses

Improve fully

No

- Anetropic
- Anisometropic Amblyopia 9

Not improved

Look for Media opacity 10

Retinal diseases

Optic Nerve anomalies

No

Organic Amblyopia
Assessment of Poor Vision

1. This basic question needs to be addressed commonly. There should not be any conclusion made in haste. As the child may be sleepy or inattentive at the outset.

2. Keen observation of the child’s response to Examiner’s face or a cartoon or some coloured noiseless toy is made. A relatively quiet room with subdued room light is preferred. Child may smile or just fixate. Observe the type of fixation: steady or unsteady.

3. Silent toys are moved to attract child’s attention and make observation as above. Sound toys may be used to wake him up or hold attention intermittently.

4. Preference of fixation by either eye equally: alternately or cross fixation implies equal vision in the two eyes. This is easy in the presence of manifest squint. If not it can be induced by interposing a 10pd base down (B.D). prism.

5. Blinking response to lights switched on and suppression of an after nystagmus after rotating the child held in your arms indicates ability to hold fixation.

6. A very useful test is to observe the resentment to closure of one eye. If present it indicates poor vision in the other eye.

7. Visual acuity may be quantitatively assessed, although not essential. If available age appropriate tests should be used. Teller Acuity is based on the behavioral pattern of the child to prefer a resolvable pattern rather than a blank in the two alternative forced choice challenge. Over 18 months his attention may be more varied and Teller not proper. Cardiff acuity cards are based on the vanishing optotype principle, i.e. they are visible at a particular distance or nearer. They are useful up to 3 years. Older children may co-operate for picture optotype types, like Allen cards, Lea symbols or Wright picture cards. Still older children may be assessed with Illiterate E or Landolt C or a Sjogren hand test for direction identification. Still later letter-identification test like Snellen or ETDRS can be done.

8. In case the visual acuity is subnormal on above tests, pin hole is used. If vision improves through pin hole. It implies a refractive error. In higher refractive errors in addition to pin hole a correcting lens may be required for full improvement.

9. If the vision does not improve despite full correction of glasses if may be due to amblyopia, provided no other organic cause responsible for poor vision is seen. Unilateral or unequal uncorrected refractive errors causes anisometropic amblyopia. While bilateral uncorrected refractive error causes Ametropic amblyopia.

10. Other causes of poor vision should be looked for before diagnosing Amblyopia. It may be media opacities, retinal diseases or optic nerve anomalies or neuropathy.

11. Role of VER and ERG is useful in confirming cause of poor vision. A subnormal VER or extinguished response or a prolonged latency is associated with optic nerve anomalies or neuropathy.

12. Special Notes:

A. Presence of nystagmus indicates poor vision. If nystagmus increases on covering one eye it has latent component also. For such cases Binocular vision is much better than uniocular vision. For uniocular vision the other eye should be covered with a +4,+6 D lens and not an opaque occluder.

In case of a null positon, vision should be assessed in that position also, in addition to primary position.

B. Amblyopia: is unilateral or bilateral diminution of vision for which no organic cause can be detected on physical examination (no media opacity, no retinal or optic nerve disease) or the diminution of vision persists despite removal of that cause or despite use of corrective glasses. And which is corrected fully if treated in time. Usually a two line difference in vision is taken for diagnosis, but for recovery it has to be equal and also free alternation in each eye.

Author
Pradeep Sharma MD
Dr. R.P. Centre for Ophthalmic Sciences, AIIMS, Ansari Nagar, New Delhi
Registration Form

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Institutional Affiliation

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Member Past President DOS or AOS Membership No. Non Member

Registration Fee enclosed Rs. Total Rupees in words

By Demand Draft / Local Cheque No. Dated

Drawn on Bank in favour of

“Delhi Ophthalmological Society” payable at New Delhi (Out Station Delegates to pay by Demand Drafts only)

Pre-Registration is open, send your application on or before 25th October, 2009 to the Conference Secretariat.

Last date for received the Registration by Mails - 25 October, 2009

Kindly mail your Registration Form with Demand Draft / Cheque to:

Conference Secretariat:

Dr. Amit Khosla, Organising Secretary
Room No. 2225, 2nd Floor, New Building, Sir Ganga Ram Hospital,
Rajinder Nagar, New Delhi - 110 060

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<th>Registration Fee</th>
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<tr>
<td>Status</td>
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<tr>
<td>MEMBERS</td>
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<td>NON MEMBERS</td>
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FOR OFFICE USE ONLY

RECEIPT No. REGN. No.
Dear DOS Members,

Please use the under mentioned form to update your membership details. This will ensure that you receive regular correspondence including DOS Times and Delhi Journal of Ophthalmology. We will improve the distribution of DOS Times & Delhi Journal of Ophthalmology & other information.

Your Mobile No. & email id is a must. We will be informing to you by SMS or Email whatever DOS Times or Delhi Journal of Ophthalmology is dispatched.

Please include the following along with the filled up form (Only for Delhi Members):

1. One Recent Passport / Licence Size Coloured Photograph is to be pasted on the form in the space provided.

2. For ‘Delhi Life Member’ please include the proof of residence. The proof of residence may be in the form Passport/License/ Voters Identity Card/Ration Card/Electricity Bill/MTNL (Landline) Telephone Bill.

You are requested to correct/update the given data & return back to us immediately at the following address or email or post; this form is available on www.dosonline.org.

INSTRUCTIONS

• For your protection, members are REQUIRED to sign the bottom of the Update form.

• Information cannot be updated without a member’s signature.

• Fill out only information you are adding or modifying.

• Please return the signed “Membership Contact Information - Update Form” to:

Dr. Amit Khosla, Secretary
Delhi Ophthalmological Society
Room No. 2225, 2nd Floor, New Building,
Sir Ganga Ram Hospital, Rajinder Nagar
New Delhi - 110060, Ph: 011-65705229
Email: dosrecords@gmail.com or admin@dosonline.org

P.T.O
Your Correspondence Information available with DOS Secretariat, if you have any change in your available information, please fill up the new form & send to us immediately

Please update the information, using BLOCK LETTERS ONLY. All filled are mandated

DOS Membership Number ________________

Your Name: Dr. ____________________________________________________________

S/D/W/o ___________________________ Date of Birth ________ / ________ / ________

Qualifications ____________________ Registration No. ___________ Sub Speciality (if any) ______________________

Clinic/Hospital/Practice ADDRESS ____________________________________________________________

City ___________________________ State______________________________Pincode __________________

Clinic/Hospital/Practice Phone (with STD Code) __________________________________________________

Correspondence Address ________________________________________________________________

_______________________________________________ Phone (with STD Code) ______________________

Email ID (Mandatory) _______________________________ Remarks : __________________________________

Mobile (Mandatory) ____________________________

Document Enclosed. (Tick only): (Only Delhi Members)

1. One Recent Passport / Stamp Size Coloured Photograph (essential)
2. Photocopy of Passport
3. Photocopy of License
4. Photocopy of Voters Identity Card
5. Photocopy of Ration Card
6. Photocopy of Electricity Bill
7. Photocopy of MTNL (Landline) Telephone Bill

Doctor Signature: _______________________

Dated: ___________________