Vision Assessment in Children: A Simplified Approach

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Measuring vision in children is a special skill requiring time, patience and understanding on the examiner's part and motivation & attention of patient.

Methods should be adapted to the child's age, abilities, knowledge and experience. Young children are not able to describe their vision or explain their visual symptoms. Through observation, and with information from the mother or guardian, functional vision can be evaluated.

Normal Visual Development and Functions

In normal visual development, children start by observing their mother's face. Experiences of space, distances, and hand-eye coordination are important steps in stimulating the development of all visual functions during the critical period from birth to five years. Deprivation disturbs this development and may lead to nystagmus and amblyopia.

Normal visual development:

- Fix and follow objects by 3 months
- Blink response 2-5 months
- Reach for objects by 6 months
- Binocular vision 3-7 months

Normal age related visual acuity estimate:

Vision at birth	6/120
4 months	6/60
6 months	6/36
1 year	6/18
2 years	6/6

(Varies depending upon test method employed, but by the age of 2 yrs is 6/6)

Major visual functions, which can be tested from a very young age, include:

- pupil reaction
- fixation
- motility
- visual acuity
- fusion
- colour vision

The most important visual function is visual acuity.

Key Points for Measuring a Child's Vision

Prepare for the assessment and assemble all the testing materials in advance so that the assessment can proceed without interruption.

Create rapport

Speak in a language he or she will understand.

If you only talk to the mother, the child will lose interest.

Make the child comfortable so that you have his or her full co-operation.

If the child is sick, crying or hungry it may be better to postpone the assessment, even for half an hour.

Small children may do better if they sit on their mother's lap. Explain the testing procedures to the mother and let her conduct the test while you observe the child.

Occlusion of a young child in order to carry out monocular visual acuity assessment is probably achieved by sitting the child in parent's lap and asking the parent to cover each eye in turn, taking care not to let the child peek between the fingers or to press too firmly, distort the cornea and lead to artificially reduced visual acuity.

Because older children may peek around the handheld occluder, sometimes you have to take a drastic step of occluding the eye with a strip of 2-inch Micropore tape.

Gain attention

To get the child's attention, try using an interesting object. This could be a torch or a coloured light, achieved by shining a torch through coloured plastic or cloth. Large objects with high-contrast forms activate visual pathways more effectively than light without forms.

Sounds can also be used such as a small toy that makes sound. In this case, auditory attention is used first to reinforce the visual attention, but this is obviously not appropriate if testing a deaf child.

When attention is achieved (which is sometimes only for very short periods) observe the child's behavior. Does he look towards the object, is there nystagmus, does she reach for the object?

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Test fixation

Fixation of each eye can be evaluated as (1) Central (2) Steady, and (3) Maintained, or CSM,

Cover one eye and observe the corneal reflex with a torch in the other eye. The corneal reflex should be central; if not, eccentric fixation is present.

If eccentric fixation is present it usually means a visual acuity 20/200 or worse.

If you notice nystagmus, the fixation is unsteady. If possible describe the type, form and amplitude of nystagmus, so that you will be able to compare changes (improvements) during follow-up visits.

The eye is then uncovered. Look for how long the child is able to maintain fixation. In case of less vision in one eye or strabismic patient who strongly prefers the eye just uncovered will switch fixation to that eye meaning unmaintained fixation

Identify the preferred fixating eye

In cases of squint it is easy to detect the preferred eye, as it is mostly in a straight position.

If there is no squint you can use the vertical prism test to induce a vertical deviation. If there is no obvious dominance the preferred fixating eye is always the one behind the prism.

A simpler test is to close one eye at a time and compare the reaction of the child. The child will be less co-operative while covering the dominant eye.

Test visual acuity

In infants and children visual functions at near distances are more important than visual functions at greater distances. Also, it helps in building confidence. Therefore visual acuity should always be first measured at a near distance

The vision must be tested using one eye at a time, and binocularly, with both eyes open. If there is suspicion of poor vision in one eye then that eye should be examined first. While testing vision binocularly look for abnormal head postures.

It is always useful to measure visual acuity binocularly because this reflects how the child is seeing in normal viewing conditions. It is well recognized that children with latent nystagmus may dramatically see better binocularly than with either eye individually. Furthermore, in the binocular state, compensatory face positions for nystagmus with a null zone or abnormal head posture from paralytic strabismus will be appreciated. In fact, occlusion of one eye may eliminate a compensatory face position in some cases of paralytic strabismus. This finding may help to distinguish an ocular from a non-ocular cause of abnormal head posture.

Even young children are quite capable of remembering letters from one eye to the next, so different letters of approximately equal difficulty should be used for each visual acuity determination.

All patients, no mater what their age, should be encouraged to guess letters, especially if they read a line of letters quickly and accurately, and then stop. Forcing patients to their visual acuity threshold will make the measurement more accurate.

Patients should not be allowed to lean forward.

Single letters are easier to identify than letters arranged in groups; with single letters there is less contour interaction and the crowding phenomenon is reduced. Pointing to one letter in a line of letters with a finger also reduces contour interaction and therefore makes task easier. It should be avoided and if done should be noted on the record card.

WHO recommendation for vision assessment in children

Visual acuity needs to be measured at distance and at near using tests with the same optotypes.

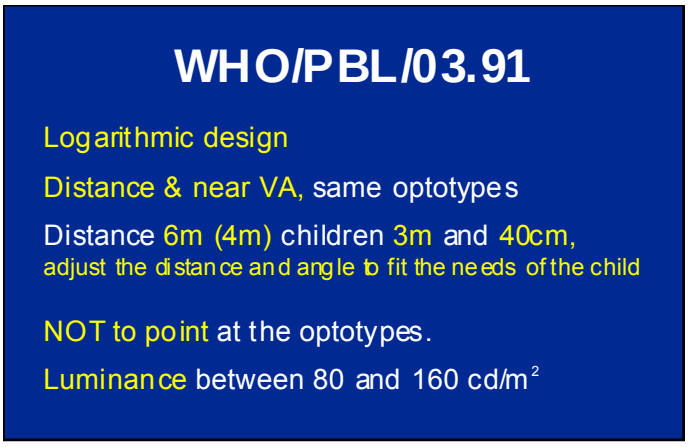
Recommended distance for measurements at distance is 6 meters

For children the test distance is 3 meters. Near vision is assessed at a distance of 40cm (16 inches).

Optotypes are not to be pointed at but line-by-line isolation and pointing at the line to be read are acceptable techniques to help the person/child to know on what line to read. Pointing at optotypes may facilitate fixation so much that visual acuity with pointing may be twice the value measured without pointing.

Luminance on the surface of the test should be between 80 and 160 candelas per square meter (cd/m2). This luminance level is available in the lightboxes made for measurement of visual acuity.

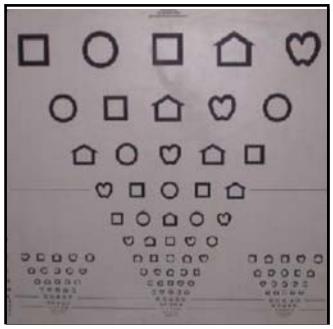
Logarithmic design has been the recommended



structure of visual acuity tests

When visual acuity cannot be measured with optotype tests, grating acuity tests are used. The resulting grating acuity values must not be reported as optotype acuity values.

Preferential looking: LEA gratings



Lea symbols

Age Factors for Measuring Visual Acuity

Infants: birth to 18 months

Birth to three months of the age

Object awareness: lighted objects, human face is the most consistent early stimulus to which infants respond

- Child recognizes mother face 6 weeks
- Follows light (mainly in vertical & horizontal meridian)

Kay pictures

3-6 months

- Colourful objects without detail
- A standard assessment strategy is to determine whether each eye can fixate on an object, maintain fixation, and then follow the object into various gaze positions (F &F). Failure to perform these maneuvers indicates significant visual impairment. The assessment should be performed binocularly and then monocularly. If poor fixation and following is noted binocularly after 3 months of age, a significant bilateral eye or brain abnormality is suspected, and referral for more formal vision assessment is advisable. It is important to ensure that the child is awake and alert, because disinterest or poor cooperation can mimic a poor vision response.
- A child with poor vision will react strongly to occlusion of the eye with better vision. The anxiety and avoidance maneuvers precipitated by the occlusion provide evidence of poor visual acuity in the uncovered eye.

6 months-1 year

- Small objects with detail (puppet, doll)
- CSM method

Indirect method (should be done in all cases and all ages)

- Pupillary responses
- Red reflex (bruckner's test)
- Fundus examination
- Refraction

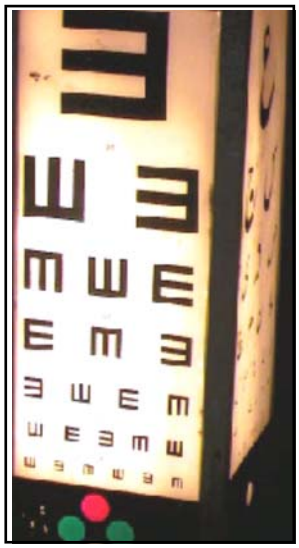
OKN response

The optokinetic nystagmus, or "OKN response," is evidence of gross vision and can be elicited by an optokinetic drum or tape. Absence of an OKN response does not necessarily imply a visual deficit, as it needs attention.

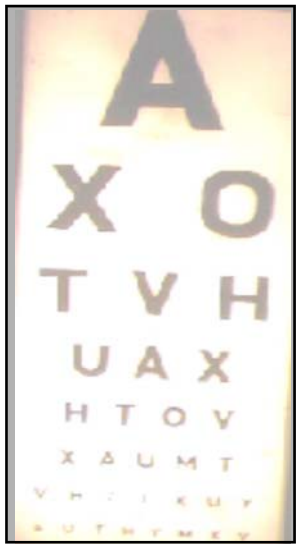
Preferential Looking Techniques

Pre-verbal children cannot describe what they see, so they are tested using preferential looking techniques. Children will automatically look towards shapes and lines rather than a plain grey surface.

In the youngest children (0-24 months) grating acuity can be used. This can be tested with the Teller acuity cards, Cardiff acuity cards & Lea (paddles) Gratings. These techniques are expensive and are clinically useful in the hands of experienced examiners.



Snellen chart showing illiterate E



Snellen chart

Pattern-Evoked Potentials

Pattern-evoked potentials (PEVP) testing has demonstrated that infant visual acuity reaches normal levels at 6 months of age. This electrophysiologic test can be used clinically to record and monitor visual acuity in young children. Expensive, time consuming, limited availability, not standardized, little clinical relevance

Toddlers: 18 months to 3 years

Various tests are available for measuring visual

acuity in older children. Allen pictures, Lea symbols, Kay pictures all are simple tests to record visual acuity. The children are first given few symbols to memorize so they can recognize them on the charts when tested in the clinic. Lea symbols are according to WHO & ICO guidelines. Lea symbols will set you back by 50$ for distance chart and $25 for near. Kay picture will cost 10,000 Rupees

Teller acuity cards or other preferential test can also be used.

Preschool: 3-5 years

Different picture tests, as mentioned above such as LH symbols (LEA symbols) and Allen cards can be used. Tests for children who know alphabets/ numbers include wall charts containing Snellen letters, Snellen numbers, and the HOTV test (a letter-matching test involving these 4 letters). The tumbling E test, Landolt rings can be used in other children.

School age: 5 plus years

That's child's play!

Use any: Snellen chart, ETDRS/Lighthouse chart, log MAR

Conclusion

Measuring vision in children is a comprehensive task. For diagnostic purposes, follow- up and evaluation of visual impairments, all elements should be considered and compared with the visual function of daily life. It is negligence on the part of ophthalmologist if they do not record visual acuity, the most important function. The above discussion is to give simplified and inexpensive way of assessing vision in children which would be helpful to all ophthalmologists alike.

Complications of Contact Lens Wear

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Contact lenses usage has increased considerably over the past few years. Factors responsible for this trend are improvement in the quality of lenses, their easier availability and intensive marketing by contact lens manufacturers. However, along with the usage, the contact lens complications have also increased. Prevalence of contact lens related complications has been estimated to be 5%. Corneal complications are the commonest followed by conjunctival. Lid complications are rare.

Pathophysiology

The factors causing complications related to contact lens wear can be divided into four major categories:-

1. Hypoxia and hypercapnia
2. Allergy and toxicity
3. Mechanical effects
4. Osmotic effects

The first two factors are responsible for the majority of complications encountered in clinical practice.

Hypoxia And Hypercapnia

Contact lens wear decreases the gas exchange at the corneal surface. Oxygen transmissibility (dK/L), which is lens material permeability (dK) divided by lens thickness (L), is the most important variable in determining relative oxygen delivery to the corneal surface with contact lens wear. Tear exchange under the contact lens also influences corneal oxygen tension. Rigid gas permeable lenses (smaller diameter) of the same or lower oxygen transmissibility may result in less corneal edema than larger diameter soft lenses because of better tear exchange.

Inadequate oxygenation leads to a decrease in the corneal epithelial mitosis rate causing decreased thickness of epithelium, formation of microcysts and increased fragility. The junctional integrity of the epithelial cells is compromised and may leads to punctate epithelial keratopathy, epithelial abrasions, and increased risk of microbial keratitis. Lactate accumulation in the stroma, from anaerobic metabolism, causes increased stromal thickness and disruption of the regular pattern of collagen lamellae, leading to striae and descemet's folds. Acidosis also leads to endothelial blebs and eventually to endothelial cell polymegethism over a period of time. Hypoxia also leads to corneal hypoesthesia and neovascularization of both the epithelium and stroma. Stromal vascularization may evolve to interstitial keratitis, deep opacities, or rarely, intrastromal hemorrhage. In some cases of long-term wear, the cornea becomes accustomed to the new oxygen tension, and stromal edema is replaced by stromal thinning.

Allergy and toxicity

Contact lens intolerance may result from a number of conditions that are allergic in nature. Contact lens solutions and particularly the preservatives within them may induce allergic responses like conjunctivitis, punctate epithelial keratopathy and superior limbic keratoconjunctivitis in susceptible individuals. Reaction to protein deposits on contact lenses may produce giant papillary conjunctivitis.

Mechanical forces

Complications include injuries sustained by improper placement or removal of a lens, or those related to contact lens fitting and wear. Steep-fitting rigid lenses may induce corneal distortion or leave a surface imprint. In severe cases, the corneal surface becomes warped. Surface wrinkling may be induced by tight-fitting soft lenses.

Osmotic effects

Increased tear evaporation and decreased reflex tearing as a result of contact lens wear may lead to the development of punctate epithelial keratopathy. Surface desiccation impairs ocular lubrication by the tear film. This renders the epithelium at risk for mechanical injuries such as abrasions and erosions.

Eyelid Complications

Meibomian Gland Dysfunction

Contact lens wearers have been observed to have more meibomian gland dysfunction than matched controls. Meibomian gland dysfunction in a symptomatic contact lens patient should be treated with moist heat and, in severe cases, with oral tetracycline derivatives. In refractory cases, discontinuation of contact lens wear is necessary to break the cycle perpetuated by inflammation.

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Blepharoptosis

Rigid contact lens wearers may develop ptosis from levator aponeurosis disinsertion from years of repeated stretching of the lid during lens removal. A second proposed mechanism is that the repeated trauma of the lens edge rubbing against the palpebral conjunctiva produces chronic inflammation and edema in the soft tissues of the lid. Because all or part of the ptosis may resolve with discontinuation of contact lens wear, it is recommended that patients stop wearing their lenses for a period of time prior to surgical correction of the ptosis. Reinsertion of the levator aponeurosis to the superior tarsal border is necessary for adequate functional repair.

Upper Lid Mass

In rare cases, rigid contact lenses can migrate through the conjunctiva at the superior tarsal border of the upper eyelid to present many years later as an upper eyelid mass. The presence of a ring-shaped protrusion with a hole in the center located on the conjunctival side of a lid mass suggests the diagnosis. The clinical presentation is that of an eyelid tumor or chalazion. Computed tomography scanning will not reveal the lens if present; therefore, one must inquire about a previously lost contact lens in rigid contact lens wearers with a lid mass.

The lens may also migrate into the orbit to cause an orbital mass.

Conjunctival Complications

Conjunctival injection occurs more frequently in contact lens wearers, particularly if the fit is inadequate. Soft contact lens wearers are more likely to present with a hypersensitivity or toxicity-related disorder than rigid lens wearer.

Mucus Formation

There is increased formation of mucus in contact lens users. This is because of continuous friction and chemicals on the conjunctival surface. On a microscopic level, the number of goblet cells is increased over that of normal controls. However, overproduction of mucus does not usually result in loss of the ability to wear contact lenses.

Dry Eye

As discussed above, contact lenses have been associated with an increased risk of meibomian gland dysfunction with its attendant decrease in tear surface integrity. In addition to the loss of surface lubrication, the loss of lipid layer integrity allows increased aqueous evaporation. This loss is particularly exacerbated by a high-water-content, extended-wear hydrogel lens. The high requirement for hydration of these lenses increases water absorption from the surrounding tear lake. Hydrogel lenses, and to a greater extent rigid lenses, cause corneal hypoesthesia, which decreases basal and reflex tear secretion. The combination of lipid, aqueous, and mucin abnormalities caused by contact lens wear puts the wearer at higher risk for dry eye. Treatment should be directed toward symptomatic patients with regard to specific abnormalities identified on careful examination. In most cases, the problem is multifactorial and may not respond to artificial tear therapy alone. Contact lens cessation allows more rapid resolution of dry eye symptoms. Once resolved, lenses may then be worn with protective measures advised to decrease recurrences.

Concretions

Chronic contact lens wear in the presence of significant daily exposure of the eyes to particulate matter promotes the phagocytosis of some of this material by cells of the conjunctival epithelium, primarily in the inferior fornix. In most people, concretions are benign but if symptomatic they may need to be removed under topical anesthesia.

Toxic Conjunctivitis

The toxic effect of various contact lens-related chemicals on the ocular surface cause bulbar and papillary conjunctival injection, usually more pronounced inferiorly. This usually happens if the hydrogel lenses are not rinsed sufficiently after disinfection or enzyme treatment. This condition responds very well to discontinuation of lens wear and avoidance of the inciting agent. Cool compresses and preservative-free artificial tears will provide symptomatic relief during recovery. Severe cases may require a short course of corticosteroid drops. Reinforcing the basic elements of proper contact lens care is important as well.

Allergic Conjunctivitis

Allergic conjunctivitis secondary to contact lens wear is characterized by severe itching, swelling and the presence of diffuse papillary conjunctivitis. Certain contact lens solution components, particularly preservatives, have been implicated as allergens in this condition.

Changing to a different solution may be tried in the absence of an identifiable allergen. Symptomatic treatment is the same as that for toxic conjunctivitis. Topical vasoconstrictors and antihistamines are used during the acute phase to control the itching. Topical ketorolac can also be used for the symptomatic relief of itching but does not improve local congestion as well as topical vasoconstrictors do. Oral antihistamines and rarely topical corticosteroids may be needed for severe cases. Topical sodium cromoglycate (or newer antihistaminic+mast cell stabilizers like ketotifen and azelastine) may be used for chronic recurrent cases.

Fig .1(a): Giant Papillary Conjunctivitis (GPC)

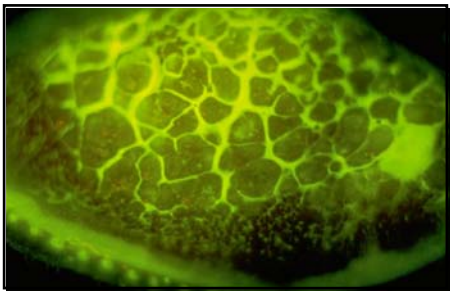

Fig. 1(b): GPC (with Fluorescein)

Giant Papillary Conjunctivitis

Giant papillary conjunctivitis (GPC) derives its name from the large papillae (> 1mm) that form on the upper tarsal conjunctiva in response to contact lens wear and the chronic presence of other ocular foreign bodies. All contact lens types have been associated with this complication. The classic symptom triad of itching, excess mucus, and contact lens intolerance coupled with giant papillae of the upper tarsal conjunctiva are suggestive of the diagnosis. As the disease process progresses, the papillae coalesce and become flattened. Ptosis may also develop. The condition must be differentiated from vernal keratoconjunctivitis because they have similar clinical features.

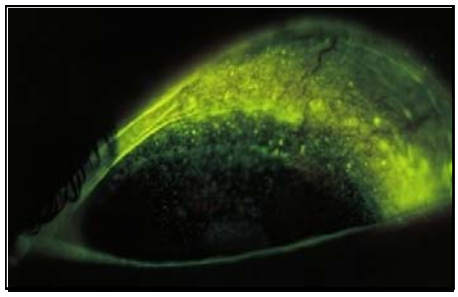

Fig .2: Contact Lens-Associated Superior Limbic Keratoconj-unctivitis (Fluorescein Stained)

The pathophysiology of GPC is believed to involve conjunctival reaction to protein deposits on the lens surface, but mechanical trauma may also contribute.

Rigid lens wearers are less likely than hydrogel lens wearers to develop GPC because rigid lenses accumulate less protein and are more completely cleaned.

Treatment consists of discontinuing lens wear and initiating pharmacologic therapy with topical sodium cromoglycate four times a day (or newer antihistaminic+ mast cell stabilizers like ketotifen and azelastine). Topical corticosteroids are not of much benefit in this disorder but may be tried in severe cases. Topical antihistamines and vasoconstrictors are important during the acute phase, which lasts 3 to 5 days in the absence of contact lenses. After resolution of itching, hyperemia, and mucus production, contact lenses can be refit and topical sodium cromoglycate used. Patients may be advised to change over to rigid gas-permeable (RGP) lenses from hydrogel lenses because of their tendency to bind less protein. Some authors recommend changing over to a new, soft lens with reinforcement of appropriate lens care and frequent replacement protocol, or the daily use of a disposable soft lens. Chronic recurrent GPC may require discontinuation of lens wear.

Contact Lens-Associated Superior Limbic Keratoconjunctivitis

Contact lens–induced superior limbic keratoconjunctivitis (CL-SLK) is an immunologic reaction in the peripheral conjunctiva produced by contact lens wear that is similar to that seen in Theodore superior limbic keratoconjunctivitis (SLK). It is characterized by conjunctival thickening, erythema, and a variable amount of fluorescein staining of the superior bulbar conjunctiva.

The keratinized epithelium loses many of its goblet cells and is invaded by neutrophils. The typical symptoms of CL-SLK are foreign body sensation, photophobia, tearing, burning, occasional itching, and reduced visual acuity due to punctate epitheliopathy .

Although similar in name, CL-SLK is a separate and distinct entity from Theodore SLK. CL-SLK can be differentiated by a lack of filaments, minimal tarsal papillary reaction, impaired vision, and lack of association with thyroid disease.

CL-SLK may be caused by excessive lens movement or sensitivity to thimerosal. Treatment consists of discontinuing contact lens wear until the epithelium returns to normal and the symptoms resolve. Refitting with better fitting lenses, using preservative-free solutions with a hydrogen peroxide disinfecting system or switching to rigid gas-permeable (RGP) contact lenses may permit a resumption of contact lens wear.

Corneal Complications

Corneal complications are discussed with respect to the layer primarily affected:-

Epithelium

1) Mechanical Epithelial Defects

A) Abrasions- Indicate chronic epithelial stress due to the contact lens usage. They can also result from manipulation of a contact lens during insertion and removal, debris trapped under a contact lens or with chipped or torn lenses. Epithelial defects can allow bacteria to penetrate the cornea, resulting in a stromal infection. However, if treated properly, abrasions usually heal quite rapidly with simple lubrication or patching.

B) Punctate Epithelial Erosions- occur commonly with contact lens wear and have several causes.

i) Rigid Gas Permeable Lens-3 staining patterns are seen-

a) Central- An excessively flat-fitting lens, bearing on the cornea centrally, may produce a central punctate staining. Keratoconus, where the lens rubs on the tip of the cone, is a typical example.

b) Peripheral- An excessively steep lens can produce peripheral punctate staining patterns, often in a superior arcuate shape.

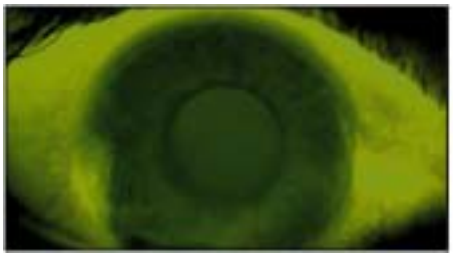

Fig. 3: 3 and 9 O'clock Staining (Fluorescein stained)

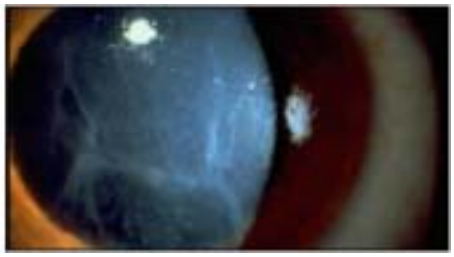

Fig. 4: Contact Lens Solution Toxicity

c) 3- and 9-o'clock staining- Is the most common staining pattern. It occurs between the lens and the limbus in the interpalpebral fissure (at the 3- and 9-o'clock positions). This condition is caused by the contact lens lifting the lid away from the cornea and poor tear stability with subsequent drying of the cornea. This is often exacerbated by an incomplete blink. A small amount of 3- and 9-o'clock staining is benign, but persistent epithelial erosions can lead to dellen formation, neovascularization, Salzmann-type elevated lesions and pseudopterygium formation. This type of punctate staining is alleviated by decreasing the distance from the lens to the limbus with a larger lens, reducing edge lift with a thinner-edged lens, or refitting with a lens that rests under the upper lid (alignment fit).

ii) Soft contact lens-Punctate staining by soft lenses is not as common as with rigid lenses. Soft lenses that cause excessive desiccation can cause an inferior central or inferior arcuate pattern. These patients may have minor symptoms of mild irritation or slightly decreased vision. Refitting with a higher water content lens or RGP lens can eliminate the problem.

2) Chemical Epithelial Defects

Contact lens solutions can produce a range of epithelial defects- from marked erosions to less extensive punctate defects. Surfactant cleaning solutions that are left on the lens after cleaning may cause immediate pain, redness, photophobia, and tearing upon lens insertion. These symptoms typically disappear after 1 or 2 days of lens discontinuation.

3) Hypoxia

As discussed earlier, contact lens wear can cause hypoxia. The hypoxia could be acute or chronic. The etiology of hypoxia may be due to low oxygen permeability, improper lens fitting or deposits over the lens surface.

a) Acute hypoxia-

Mild, acute hypoxia produces epithelial edema and temporary blurred vision. If severe, it can cause epithelial cell death and desquamation. Typically, the conjunctiva is hyperemic and the epithelium has fine punctate defects, producing temporary decreased vision and photophobia.

b) Chronic hypoxia- Produces a variety of more subtle effects:-

i) *Epithelial microcysts*- Are transparent epithelial inclusions of degenerated epithelium, 10-15 ìm in size. They begin in the deep epithelium, and slowly migrate anteriorly. Upon reaching the surface, they rupture, creating depressions that pool with fluorescein. Epithelial microcysts do not produce any significant symptoms other than a mild decrease in vision. It takes several weeks for the microcysts to disappear after discontinuation of the contact lenses.

ii) *Neovascularization*- One of the hallmarks of chronic corneal hypoxia is superficial neovascularization, especially along the superior limbus. Neovascularization of up to 1.5 mm from the limbus is not visually significant and is generally well tolerated but should be recognized as a sign of hypoxia and may be a harbinger of more significant problems. Rarely, deep stromal neovascularization can occur. Changing to lenses that are thinner or contain materials with greater oxygen permeability, have greater lens movement, and by decreasing wear time (especially eliminating overnight wear) can greatly reduce the risk of progression.

iii) Decreased corneal sensitivity- Chronic hypoxia has been implicated as a cause of the decreased corneal sensitivity that occurs with prolonged contact lens wear and may be partly the reason why some patients have increased comfort with long-term wear and why they often have decreased comfort with a change from polymethyl methacrylate (PMMA) to gas-permeable contact lenses.

The physiology of the corneal epithelium also is altered by contact lens wear. The barrier function of the epithelium is reduced and the permeability to fluorescein is doubled after as little as 2 weeks of soft contact lens wear. Similarly, rigid contact lenses can alter the epithelial permeability.

4) Superficial Immunologic Reactions

Superficial toxic or immune reactions can result from a variety of chemicals in contact lens solutions. The typical

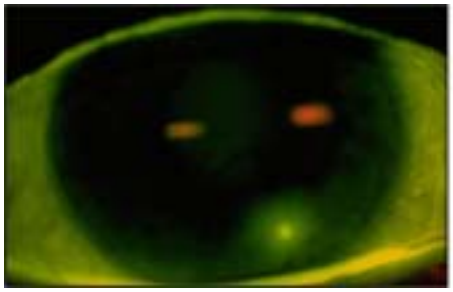

Fig. 5: Sterile infiltrate (Fluorescein stained)

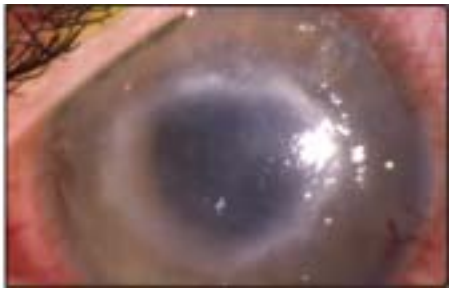

Fig. 6: Acanthamoeba Keratitis (ring shaped infiltrate)

response is a fine punctate keratopathy, conjunctival injection, tearing, itching, and occasionally chemosis. The preservative, thimerosal, was notorious for these reactions and is no longer used in these products. The symptoms resolve over a few days after the chemical disinfecting solution is discontinued. Changing to a preservative-free hydrogen peroxide based disinfection system is recommended.

Stroma

1) Sterile Infiltrates

Contact lens wear can induce a distinctive sterile keratitis, presenting as a sudden onset of an anterior stromal or subepithelial polymorphonuclear leukocyte and mononuclear cell infiltrate typically in the periphery of the cornea. The infiltrates usually are small (0.1-2 mm) and may be single or in groups. The infiltrates may be round, oval or arcuate and may underlie either an intact epithelium or an epithelial defect.

The etiology of these sterile infiltrates may involve an immune-mediated reaction to bacterial toxins from colonized contact lenses. Staphylococcal organisms have been isolated from contact lens wearers that have sterile infiltrates. The infiltrates tend to resolve with no loss of vision leaving behind only a faint scar in the anterior stroma after a short course of topical steroids or simple elimination of contact lens wear.

Sterile infiltrates should be distinguished from infectious infiltrates. Sterile infiltrates tend to be multiple, peripheral, associated with less pain, minimal anterior chamber inflammation, and with less of an epithelial defect than infectious ulcers. However, if doubt exists, they should be managed as presumed infectious ulcers.

2) Infectious Keratitis

Microbial keratitis is a relatively uncommon but most dreaded complication of contact lens wear. The various defense mechanisms of the eye are adversely affected by contact lens wear.

Contact lenses wear may cause breaks in the epithelium (punctate erosions, abrasions), which allow direct access of pathogens to the stroma. The epithelium of contact lens wearers is thinner, less sensitive, and relatively hypoxic. These factors reduce the ability of the epithelium to repair itself and act as a barrier against invading organisms. Breaks in the corneal epithelium probably are important predisposing factors for bacterial keratitis. However, they are not a necessary precondition. Extended wear protocol, in general, has a higher risk of infection as compared to the daily wear protocol.

A variety of both gram-positive and gram-negative organisms have been isolated from corneal infections. However, the most commonly cultured pathogens have been *Pseudomonas aeruginosa, Staphylococcus aureus,* and *Staphylococcus epidermidis. Acanthamoeba* keratitis has been associated with improper contact lens care and hygiene.

The symptoms of bacterial keratitis usually present acutely and include pain, photophobia, tearing, purulent discharge, and reduced vision. Initially, a stromal infiltrate develops under an epithelial defect in the presence of anterior chamber reaction and conjunctival injection. This may progresses to stromal and epithelial edema, anterior chamber reaction, hypopyon, and eventually, stromal necrosis.

The management remains essentially the same as for any infectious keratitis. A definitive diagnosis of bacterial infection is made with a positive culture. Corneal scrapings for smear and culture and sensitivity should be done for every patient. However, even with the best techniques, cultures often are negative and treatment must be empirical. The mainstay of treatment has consisted of broad-spectrum topical antibiotics (eg, combination of cefazolin 50 mg/mL and tobramycin 14 mg/mL) administered at half hourly intervals initially. The dose is reduced depending on the clinical response. Ciprofloxacin 0.3% and ofloxacin 0.3% may be as effective in treating bacterial keratitis as the traditional combination of fortified antibiotics. Fortunately, with prompt antibiotic therapy most bacterial corneal infections can be cured with little sequelae.

3) Tight Lens (Acute Red Eye) Syndrome

A contact lens occasionally can become tightly adherent to the eye and produce marked, diffuse stromal inflammation and an anterior chamber reaction. The resultant pain, photophobia, injection, and tearing are typically acute and severe. The epithelium has punctate staining, diffuse or peripheral infiltrates in the anterior stroma. The symptoms resolve with removal of the contact

lens. The infiltrates may take a few days to disappear. A short course of topical steroids will speed the resolution of the symptoms.

4) Corneal Warpage

Prolonged contact lens wear may produce gradual and unpredictable changes in the contour of the cornea. Astigmatism or the general steepness may be either increased or decreased. Typically, corneal warpage produces an irregular astigmatism, which reduces the best spectacle correction. Corneal warpage commonly is seen with hard lenses but can also occur with soft contact lens wear. Corneas usually regain a stable and regular shape after discontinuation of the contact lenses, but it may take weeks or even months.

Corneal Endothelium

Contact lens wear may compromise the corneal endothelium. Wearers have a greater variation in endothelial cell size (polymegethism) and an increased frequency of nonhexagonal cells (polymorphism) than do nonwearers. Along with alteration in endothelial cell morphology, a small decrease in endothelial cell density has also been found in long-term contact lens wearers of soft, PMMA and RGP lenses.

Endothelial bleb response is a transient change in the appearance of the endothelium occurring between 10-30 minutes. This is apparently to change in Ph due to hypoxia. On specular reflection of slit lamp the bleb appears as a very small, circumscribed, irregularly shaped black zone obscuring the cellular mosaic pattern. Refitting with high oxygen permeable lenses minimizes the bleb formation.

Summary

Contact lens usage is becoming increasingly prevalent. It is the primary responsibility of the contact lens prescriber to educate each lens wearer with regard to the potential side effects of contact lens wear. These effects may range from incidental findings without any apparent functional significance to severely painful and sight-threatening pathology. Fortunately, a large majority of contact lens wearers enjoy the benefits of comfort and excellent vision without experiencing any significant ill effects. The more we learn about the complications of contact lens wear, the more we can help our patients treat and avoid them.

Pictures Courtesy:

1) Photography Department, Venu Eye Institute & Research Centre
2) Bausch & Lomb

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Diabetic Maculopathy Management

Ramandeep Singh MS, **Vishali Gupta** MS, **Amod Gupta** MS

Diabetic macular edema (DME) is the most common cause of a decrease in visual acuity among diabetic patients.[1] Patients can develop DME any time during the course of diabetic retinopathy. As the severity of overall retinopathy increases, the proportion of eyes with macular edema also increases: 3% in eyes with mild nonproliferative diabetic retinopathy (NPDR), 38% with moderate to severe NPDR, and 71% with proliferative diabetic retinopathy (PDR).[2] Recommended treatments for DME, such as laser photocoagulation, target only the advanced stages of disease. The Early Treatment Diabetic Retinopathy Study (ETDRS)[3] demonstrated that focal laser photocoagulation for clinically significant macular edema reduces the risk of moderate visual loss. The current management strategy for DME involves early detection and optimal glycemic control to slow the progression of disease. Several new pharmacological therapies have also been tried to treat early stages of DME.

Management

Prevention and systemic control

Since DM has multifactorial origin, control of the metabolic abnormalities of diabetes has a major impact on the development of diabetic microvascular complications. The Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS) showed that optimal metabolic control could reduce the incidence and progression of DR.[4, 5] Elevated HbAIC was associated with persistent bilateral CSME following laser photocoagulation.[6] Tight blood pressure control in type 2 diabetes led to reduction in micro vascular complications, including need for retinal laser photocoagulation.[4]

Treatment of dyslipidemia with oral Atrovastatin led to improved outcome due to reduction of both hard exudates and subfoveal migration in CSME.[7] Correction of anemia has led to reversal of the retinopathy changes in several studies.[8-10]

Diffuse macular edema is exacerbated and ameliorated due to certain systemic factors such as fluid retention due to cardiovascular diseases or nephropathy[11-13] and severe hypertension. Treatment of these systemic abnormalities by diuresis, dialysis, cardiac drugs and antihypertensive have been shown to induce resolution of the systemic fluid as well as macular edema.[14, 15]

Thus, optimal metabolic control should be an important treatment goal and should be implemented early and maintained. In a pilot study, it has been shown that with multifactorial control of various risk factors diabetes such as HbAIC, blood pressure, lipid profile, anemia and 24 hrs proteinuria, even before laser photocoagulation can lead to reduction in macular edema on OCT and a trend towards improvement in visual acuity.[16](Figure 1)

The recommended values for HbA1c, blood pressure, and Low-density lipoprotein are <7%, <130/< 80 mmHg and <100 mg/dl, respectively.[17]

Treatment

Once sight-threatening DME has been detected, the treatment options recommended are systemic control, laser photocoagulation, pars plana vitrectomy and pharmacological agents such as anti VEGF drugs and PKC inhibitors.

Laser photocoagulation

The goal of macular laser photocoagulation for DME is to limit vascular leakage through focal laser burns of leaking microaneurysms or grid laser burns in areas of

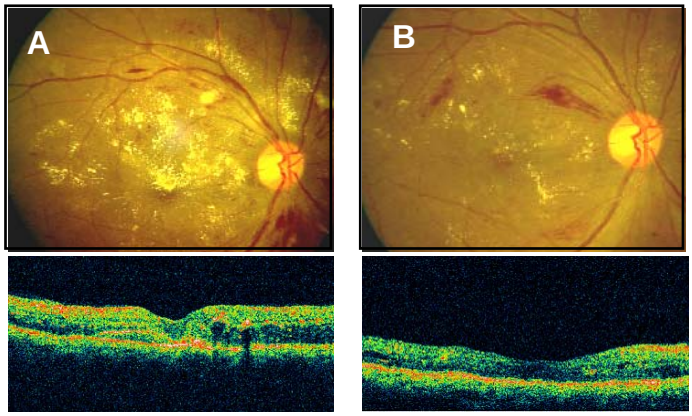

Fig. 1: A) Right eye fundus of a 44-year-old type 2 diabetic male with dyslipidemia and increased 24 hours albuminuria at presentation. OCT line scan shows increased retinal thickness, **B)** After six weeks of control i.e. anti-lipid lowering drugs and angiotensin receptor blockers (Losartan) for proteinuria, even before laser photocoagulation, fundus picture and OCT shows decrease in macular edema.

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Post Graduate Institute of Medical Education and Research, Chandigarh

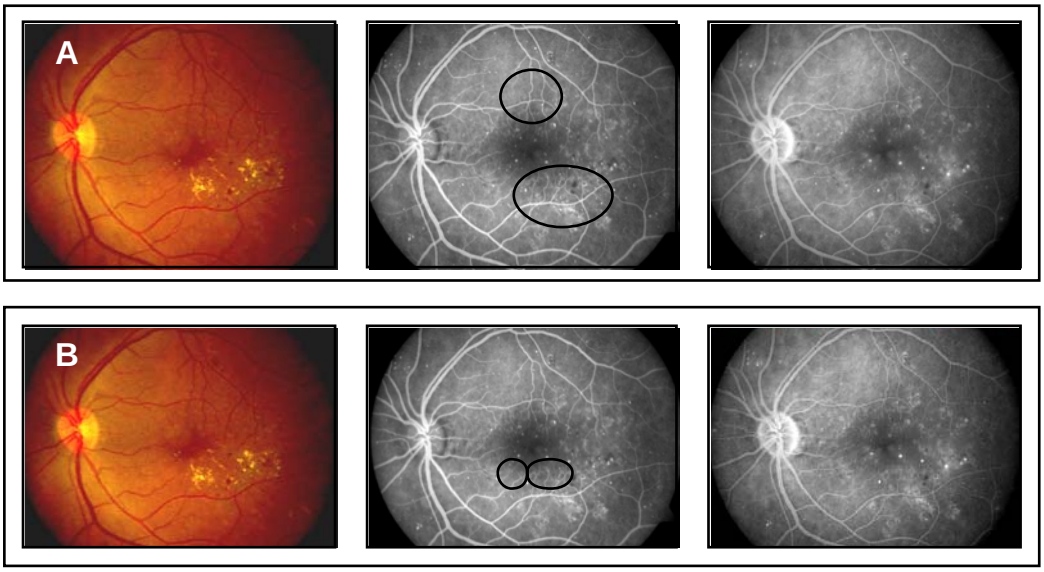

Fig. 2: Treatable lesions: (a) Focal leaks greater than 500u from the center of the macula causing retinal thickness/ or hard exudates, **b)** Focal leaks within 300- 500 u from the center of the macula thought to be causing retinal thickness/ or hard exudates

diffuse breakdown of the blood-retinal barrier. The ETDRS compared outcomes in eyes assigned to either deferral of macular laser photocoagulation or immediate treatment for clinically significant DME.[1] Results showed that laser photocoagulation reduced the risk of vision loss by 50% in patients with CSME.[3]

Argon green (514.4 nm) and frequency doubled Nd: YAG lasers (532nm) are the lasers of choice in the management of DME. Green light is absorbed well by melanin and hemoglobin. The closure is believed to be mediated by the thermal effect following absorption of laser radiation by chromophobes in the inner retinal layer.[18] Over next few months, after the closure of leakage areas, the excess fluid begins to reabsorb into the surrounding tissues and eventually the hard exudates will be reabsorbed.

ETDRS gave the treatment strategy of laser photocoagulation for DME and this has been followed most widely worldwide.[1, 19] The strategy is to photocoagulate all leaking microaneurysms further than 500µ from the center of the macula and to place a grid of 100-200 µm burns in areas of diffuse capillary leakage and in areas of capillary nonperfusion. Treatment of the macula ideally is guided by the FFA, which helps to detect areas of focal leakage, diffuse leakage from dilated capillary bed and areas of capillary non-perfusion. Local laser treatment for CSME consists of direct focal treatment, grid laser treatment to diffuse leaks, or a combination (modified grid) of direct and grid laser treatment. (Figure 2)

Technique

Focal laser photocoagulation

All focal leaks located between 500µm to 3000 µm are treated directly with 50 -100 µm spots at 0.1 second duration to produce grayish whitening of the microaneurysm. Focal lesion located within 300-500 µm

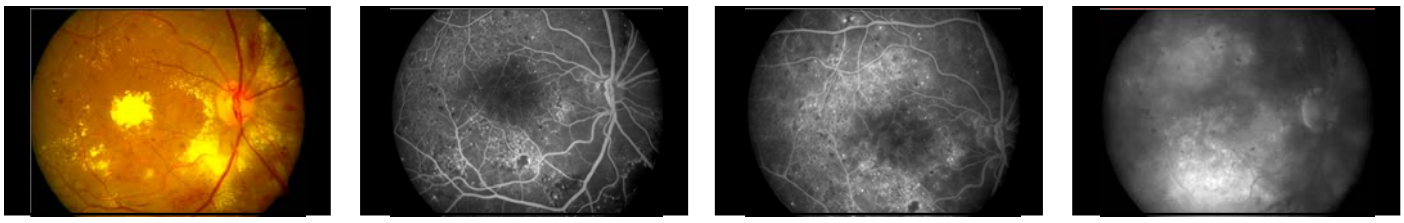

Fig. 2(c): Areas of diffuse leakage from extensive number of microaneurysms and capillary leak

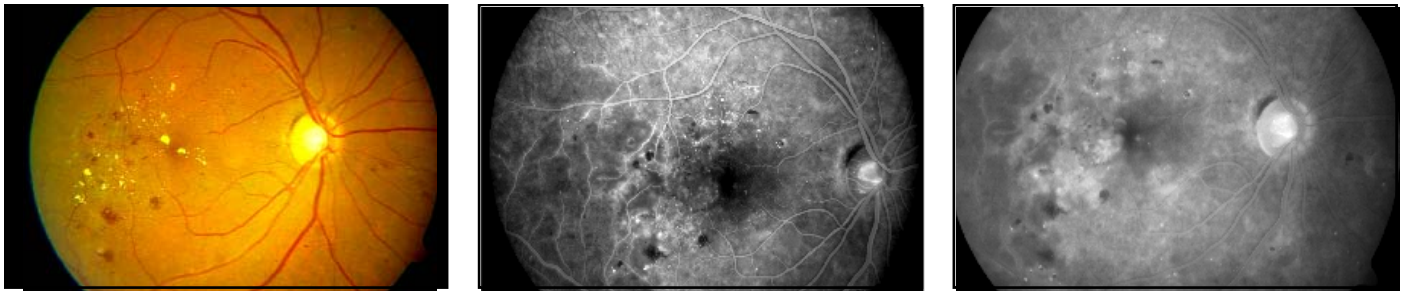

Fig. 2(d): Avascular zones other than FAZ, not previously treated

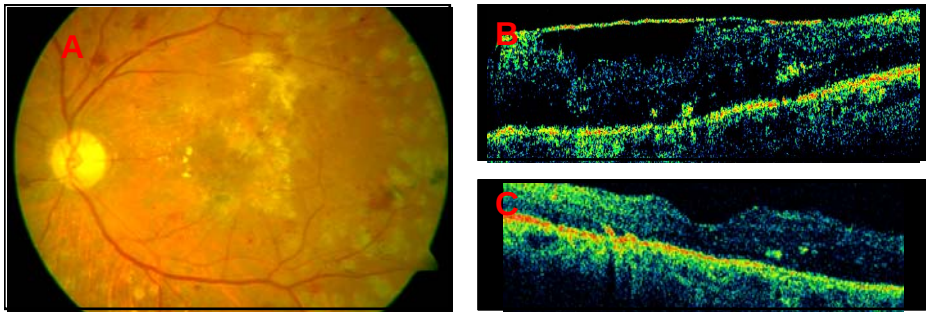

Fig.3: A 60-year-old diabetic with 20/400 vision with PRP and grid laser scars in the left eye **(A)**, OCT shows taut posterior hyaloid with increased retinal thickness **(B)**. Patient underwent Pars plana Vitrectomy (PPV) with TPHM removal. Four weeks after surgery OCT shows normal foveal contour **(C)**.

of the PAZ be treated if retreatment is required.

Grid laser photocoagulation

All areas of diffuse leakage extending from arcade to arcade are treated with 50 – 200 μm spot size placed one burn width apart, at 0.1 second duration. The laser burns must be atleast 500 μm away from the foveal center and 500 μm away from the disc margins. Avascular zones, other than the normal avascular foveal zone are also treated.

A repeat flourescein angiography is to be done at 3 months. Laser photocoagulation for any persisting focal or diffuse leakage is to be done. Most patients need 1 to 3 sessions. DME requiring more than 3 treatments becomes recalcitrant and require alternative treatment with pharmacological agents.

Vitrectomy

Indications of vitrectomy in DME includes

1. Diffuse macular edema with taut posterior hyaloid membrane (TPHM)
2. Recalcitrant diffuse macular edema without TPHM

OCT has helped us in differentiating tractional and non- tractional causes of DME. Vitrectomy was found to be useful in eyes with diffuse macular edema with vitreomacular traction due to taut posterior hyaloid membrane .[20] (Figure 3)

Vitrectomy is also being done in cases with recalcitrant DME. Studies have shown beneficial effects of posterior hyaloid removal, [21,22] internal limiting membrane peeling [23, 24], and removal of submacular hard exudates apart from vitrectomy alone.[25-27]

The idea behind the use of above procedures is based on the fact that vitreous contains various factors that are pathogonomic in causing diabetic retinopathy and DME. Removal of vitreous gel will protect against formation of macular edema and progression to proliferative disease because of removal of vitreous scaffold and angiogenic factors by improving the oxygenation of the eye. Similarly, vitreous surgery may improve perifoveal microcirculation in eyes with diffuse macular edema. The removal of various local factors like growth factors i.e. vascular endothelial growth factor (VGEF), angiotensin and inflammatory cytokines (IL-6) [28,29] helps to retard progression of DME. Ikeda et al[30] studied the pathogenesis between an attached posterior hyaloid membrane and cystoid macular edema and suggested that the removal of the barrier between vitreous cavity and retina might lead to improved fluid diffusion from the retinal tissue. Internal limiting membrane (ILM) is also considered to be the barrier of diffusion from retinal tissues, hence its removal in DME is considered to be a step towards improving diffusion.[23-24] Removal of massive hard exudates from the fovea has lead to mixed response in terms of improvement of vision in low vision patients.[25-27]

Pharmacological treatment

Despite the presence of current treatment strategies of DME, vision loss due to DME still occurs at an alarming rate. Laser photocoagulation is a late and destructive treatment that does not take the etiology of disease into account.

Pharmacological agents can be used to alter the metabolic pathway at various levels, so that the diabetes

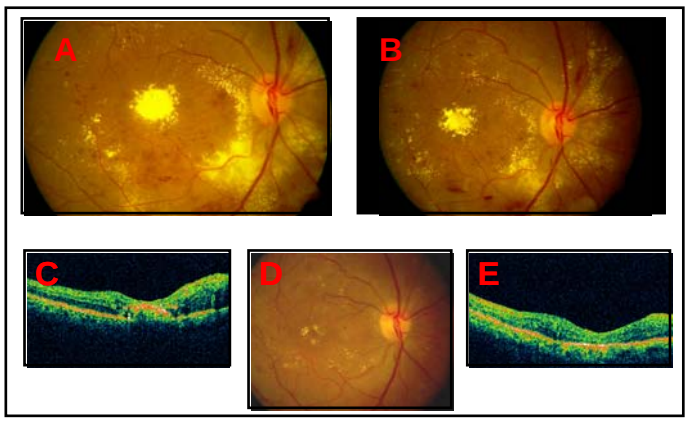

Fig. 4: A 60-year-old diabetic with 20/400 vision with subretinal hard exudates in the right eye at the presentation (A), 3 months after grid laser and systemic control including anti lipid lowering drugs, fundus photograph shows incomplete resolution of hard exudates (B). Patient was planned for PPV. Pre surgery OCT shows subretinal hard exudates with retinal thickening (C), Patient underwent PPV, posterior hyaloid removal and internal limiting membrane peeling, fundus picture shows resolution of subfoveal hard exudates at 4 weeks (D), with OCT showing normal foveal contour (E).

complications like retinopathy, neuropathy and nephropathy can be prevented. Most of the diabetes related complications like macular edema and neovascularization occur secondary to the release of the growth factors in response to retinal ischemia from alterations in the structure and cellular composition of the microvasculature.[31,32]

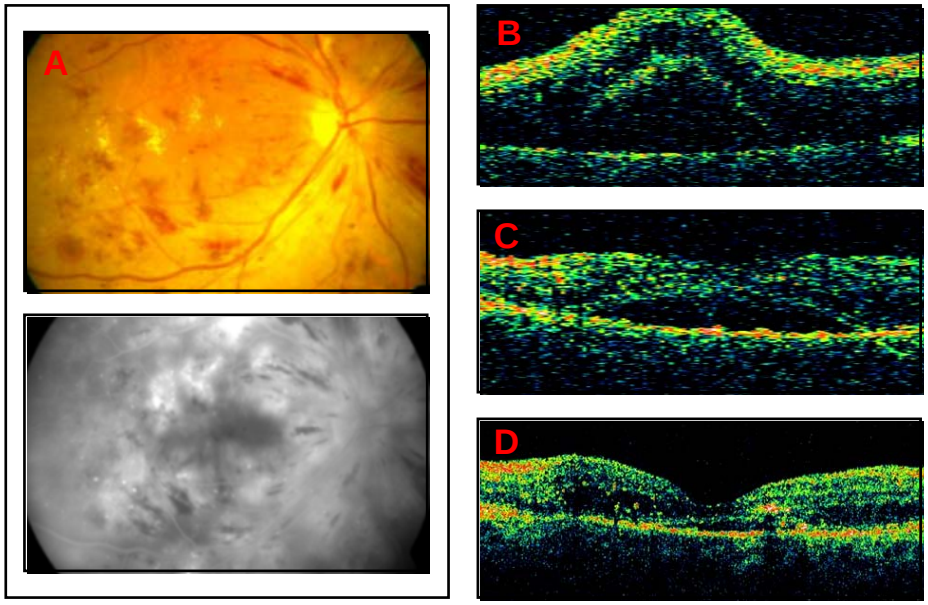

Fig. 5: A 52-year old diabetic with recalcitrant diffuse macular edema on fundus picture and fundus flourescein angiogram (A), OCT shows central foveal thickness of 589 µ (B). Patient was administered 4 mg of intravitreal Triamcinolone acetonide injection followed by focal laser at 2 weeks. Four weeks post injection; OCT shows decrease in thickness to 389µ (C), at 8 weeks, the thickness decreased to 169 µ (D).

Direct growth factor modulators

Vascular endothelial growth factor is produced by the pigment epithelial cells, pericytes and endothelial cells of the retina in response to hypoxia.[31] VEGF may also induce inflammation by inducing intracellular adhesion molecule-1 (ICAM-1) expression and leukocyte adhesion.[33]

Corticosteroids, a class of substances with anti-inflammatory properties, have been demonstrated to inhibit the expression of the VEGF gene. A study by Nauck et al[34] demonstrated that corticosteroids abolished the induction of VEGF by the pro-inflammatory mediators PDGF and platelet-activating factor (PAF) in a time and dose-dependent manner. Thus, corticosteroids down regulate VEGF production and possibly reduce breakdown of the blood-retinal barrier. Similarly, steroids have anti-angiogenic properties possibly due to attenuation of the effects of VEGF. Both of these steroid effects are utilized as intravitreal or posterior subtenon injection to cause temporary reduction in diabetic macular edema and neovascularization in various studies.[35,36]

Among the anti VEGF drugs, the intravitreal administered aptamer VEGF inhibitor, pegaptanib sodium (Macugen, Eyetech pharmaceuticals, Inc, New York, NY) has announced results of phase 2 study diabetic macular edema (DME), which showed improvement in visual acuity and decrease in retinal thickness in human clinical trials.[37] The intravitreal humanized anti-VEGF antibody fragment, ranibizumab (Lucentis, Genentech, Inc, South San Francisco, CA) and bevacizumab (Avastin, Genentech, Inc, South San Francisco, CA), intravitreal humanized murine monoclonal anti-VEGF antibody have proved to be useful for age-related macular degenration.[38,-40] These anti-VEGF drugs can be of use for diabetic macula edema and trials are awaited.

Protein kinase C activity and diacylglycerol (DAG), an activator of PKC, are increased after exposure of vascular tissues to elevated glucose.[42-43] PKC-ß has been shown to have an important role in regulating endothelial cell permeability[44] and is an important signalling component for VEGF.[45] PKC412, a nonspecific kinase inhibitor and Ruboxistaurin (LY333531), a specific inhibitor of PKC-ß1 and -ß2, did not show promising results in human trials.[46, 47] The results of PKC-Diabetic macular edema study are still awaited.

Indirect growth factor modulators

Aldose reductase plays an important role in polyol pathway, which generates sorbitol during hyperglycemia. Sorbitol accumulation in turn, disrupts the osmotic balance, thus destroying the retinal cells like pericytes.[48] Clinical trials of ARI (sorbinil, ponalrestat, and tolrestat) have been conducted for the treatment of DR,[49-52] with little therapeutic success.

Conclusions

Based on the large, randomized, controlled trials, aggressive blood sugar and blood pressure control along with laser photocoagulation are the primary treatment of diabetic macular edema. Role of reduction of HbA1C, anti-lipid lowering drugs, control of proteinuria and improvement of hemoglobin on diabetic macular edema is well documented. Since the mechanism of diabetic retinopathy has become better understood, more specific treatments are possible. The results of the current large trials of Triamcinolone acetonide, VEGF inhibitors and PKC inhibitors for DME are awaited.

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Monthly Meetings Calendar For The Year 2006-2007

Dr. Shroff Charity Eye Hospital 30th July, 2006 (Sunday)
Venu Eye Institute & Research Centre 27th August, 2006 (Sunday)
Centre for Sight 24th September, 2006 (Sunday)
Dr. R.P. Centre for Ophthalmic Sciences 28th October, 2006 (Saturday)
Midterm Conference of DOS 19 November, 2006 (Sunday)
Sir Ganga Ram Hospital 26th November, 2006 (Sunday)
Safdarjung Hospital 23rd December, 2006 (Saturday)
Army Hospital (R&R) 28th January, 2007 (Sunday)
Guru Nanak Eye Centre 24th February, 2007 (Saturday)
Mohan Eye Institute 25th March, 2007 (Sunday)
Annual Conference of DOS 6-8th April, 2007 (Friday, Saturday & Sunday)

Retinal Vasculitis – Approach to Diagnosis & Management

Mamta Agarwal DNB, **Jyotirmay Biswas** MS

Retinal vasculitis is a sight threatening inflammatory eye disease which involves the retinal blood vessels, predominantly retinal veins. Clinically, it presents as fluffy white perivascular infiltrates in the retina with aqueous and vitreous inflammatory cells. Fluorescein angiogram shows staining and diffuse leakage from the retinal blood vessels with or without cystoid macular odema. The etiology of the disease is varied. It may occur as an isolated ocular condition, as a manifestation of infectious or neoplastic disorders or in association with systemic inflammatory diseases. Hence, it is essential that a complete history, ocular and systemic examination and a detailed laboratory work up is done in all patients with retinal vasculitis.

Classification

The causes of Retinal vasculitis can be classified as follows

1. Systemic diseases
2. Infectious diseases
3. Ocular diseases
4. Malignancies
5. Drug induced

Systemic diseases

Sarcoidosis, Behcet's disease, Multiple sclerosis, Systemic lupus erythematosus, Wegeners granulomatosis, Polyarteritis nodosa, Relapsing polychondroitis, seronegative arthropathies, Polymyositis, Dermatomyositis, Antiphospholipid antibody syndrome, Takayasu arteritis, Crohn's disease

Infectious diseases

Tuberculosis, Toxoplasmosis, Syphilis, Herpes, Cytomegalovirus, Leptospirosis, Cat scratch disease, Candidiasis, Rickettsia, Amoebiasis, Brucellosis

Ocular diseases

Idiopathic, Intermediate uveitis, Eales' disease, Birdshot retinochoroidopathy, Frosted branch angiitis, Serpiginous choroiditis, Idiopathic retinal vasculitis, aneurysms and neuroretinitis (IRVAN)

Masquerades

Leukemia, Lymphoma, Retinoblastoma

Drug induced

Immunoglobulins, Rifabutin, Metamphetamines

Systemic diseases associated with retinal vasculitis

Behcet's disease – It is a multisystem inflammatory disease which is diagnosed by the clinical triad of recurrent orogenital ulcers, skin lesions and uveitis. Erythema nodosum, arthralgia and meningoencephalitis are also commonly seen. Ocular involvement is seen in 70% of patients. It usually presents as recurrent vasoocclusive retinopathy which affects both arterioles and veins in the posterior pole. Fundus examination shows retinal hemorrhages, yellow white retinal infiltrates, retinal odema and optic disc odema with hyperemia in the acute stage. Chronic stage of the disease shows macular ischemia, neovascularization of retina and disc, sheathed vessels and optic atrophy.

Fig.1: Fundus photograph of a patient of Eales' disease showing perivascular cuffing and retinal hemorrhages

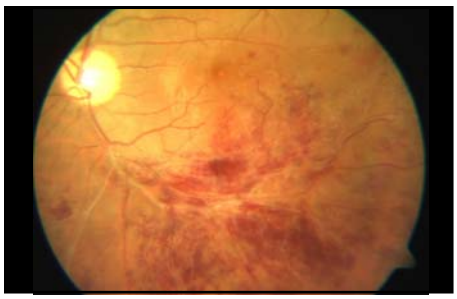

Fig. 2: Fundus photograph of a patient with Behcet's disease showing inferotemporal branch retinal vein occlusion

Sarcoidosis - This disease typically affects young adults and presents with bilateral hilar lymphadenopathy, ocular and skin lesions. Retinal vasculitis is a characteristic feature and mainly involves retinal veins. These venules are usually mid-peripheral or peripheral in location and show short segments of perivascular cuffing associated with retinal infiltrates, sarcoid nodules and snow ball vitreous opacities. Yellow perivenous exudates, classically described 'candle wax drippings' (taches de bougie) may also be seen.

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Wegeners Granulomatosis – It is a granulomatous necrotizing vasculitic condition that primarily affects upper and lower respiratory tracts and kidneys. In addition to retinal vasculitis, other clinical findings include necrotizing scleritis, peripheral ulcerative keratitis, dacryocystitis and proptosis.

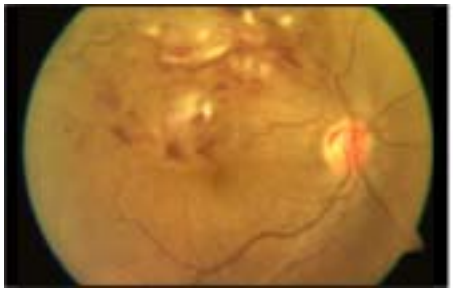


Fig. 3: Fundus photograph of a 31 yr old lady with systemic lupus erythematosus showing vaso occlusive retinopathy with dilated tortuous retinal vessels, cotton wool spots and retinal hemorrhages

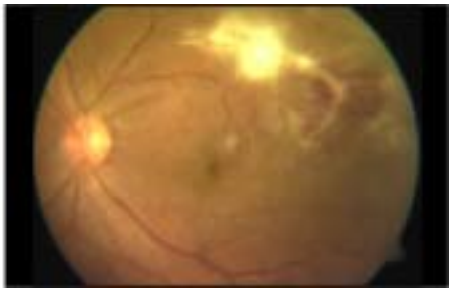


Fig.4: Acute toxoplasmic retinochoroiditis with periphlebitis, retinal hemorrhages and Kyrieleis arterialitis

Systemic lupus erythematosus - Patients present with malaise, fatigue, anorexia and low-grade fever. They may also have arthritis, facial rash, alopecia and pleurisy. Fundus examination shows multiple cotton wool spots, dilated, tortuous arterioles and intraretinal haemorrhages. Retinal vasculitis is usually uncommon but very devastating. It causes severe vaso- occlusive disease leading to retinal ischemia and proliferative retinopathy.

Infectious diseases associated with retinal vasculitis

Tuberculosis – Though choroiditis is the most common ocular feature of tuberculosis but periphlebitis is also commonly present. It occurs either by direct infection or by hypersensitivity reaction to *Mycobacterial* antigens. It is associated with vitritis and retinal hemorrhages and may lead to branch or central retinal vein occlusion leading to neovascularization and vitreous hemorrhage.

Toxoplasmosis – Toxoplasmic retinochoroiditis is commonly associated with retinal periphlebitis. Retinal veins show continuous sheathing with narrowing near acute lesions. Perivasculitis is believed to be caused by an Arthus – type reaction. Locally produced antigens diffuse into the vessel walls and react with circulating antibodies, activate complement and recruit inflammatory cells that form a perivascular cuff. Focal periarterial exudates or plaques, called *Kyrieleis arterialitis* are also seen near the active focus and these lesions do not cause vascular obstruction or leakage.

Syphilis – Syphilis can have protean ocular manifestations. Retinal vasculitis is rare and has been described in secondary and tertiary syphilis causing venous or arterial occlusion. Other clinical findings can be vitritis, chorioretinitis, neuro retinitis, optic neuritis, subretinal neovascularization and exudative retinal detachment.

Herpes virus – Retinal infections with herpes group of viruses cause necrotizing retinitis, vasculitis, and retinal hemorrhages. Acute retinal necrosis caused by herpes simplex and varicella zoster viruses is a fulminant peripheral necrotizing retinitis with severe vitritis and occlusive vasculitis affecting arterioles in the retina and choroid. Cytomegalovirus in immunocompromised patients causes fluffy white necrotic lesions along the vascular arcades of the posterior pole with retinal hemorrhages and vasculitis. The vasculitis is caused by perivascular neutrophilic infiltration of both arteries and veins.

Clinical features

History

It is very important to elicit a history of systemic complaints in a patient with retinal vasculitis in order to have a tailored investigational approach.

Orogenital ulcers, arthralgia, skin rash	Behcet's disease
Weight loss, cough, skin lesions, hilar lymphadenopathy	Sarcoidosis
Night fever, sweats, cough with expectoration	Tuberculosis
Joint pains, backache	Seronegative arthropathy
Neurological symptoms	Multiple sclerosis
Thromboembolic episodes	Antiphospholipid antibody syndrome

History related to recent infections, foreign travel, sexual habits, contact with animals should also be elicited.

Ocular diseases associated with retinal vasculitis

Intermediate uveitis is characterized by vitreitis, snowball exudates, peripheral retinal periphlebitis and pars plana exudates.

Eales' disease is an idiopathic

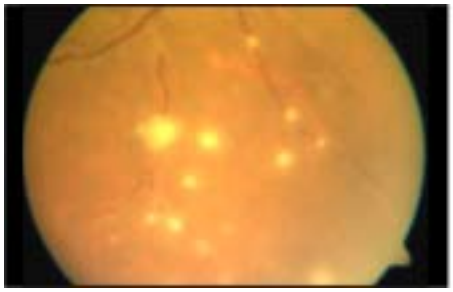


Fig.5: Fundus photograph showing multiple skip lesions of vascular cuffing (taches de bougie) in a case of sarcoidosis

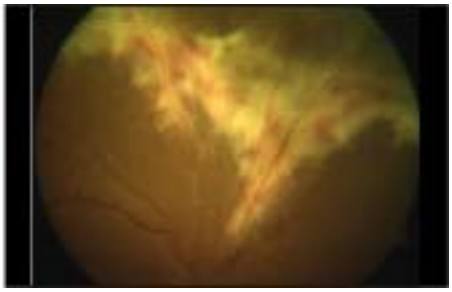


Fig.6: Fundus photograph of a patient with cytomegalovirus retinitis showing fluffy white retinal necrosis with hemorrhages and vascular sheathing

obliterative periphlebitis which commonly occurs in healthy young males between 15-40 years of age. It starts anterior to the equator and progresses posteriorly and ultimately involves multiple quadrants of the retina. This inflammation induced vascular occlusion leads to proliferative vascular retinopathy with sequelae as recurrent vitreous hemorrhage and tractional retinal detachment. The etiology of this disease is still unknown, however it is believed to be due to hypersensitivity to tuberculoprotein.

Birdshot retinochoroidopathy is a bilateral panuveitis where fundus examination shows cream colored, deep, round lesions, retinal vasculitis and cystoid macular odema.

Frosted branch angiitis is a rare vasculitis where thick inflammatory infiltrates surround the retinal arterioles and venules creating an appearance of frosted tree branches. The sheathing of the blood vessels is so extensive that the underlying vessels are obscured. Mostly, it is idiopathic, but cases have been reported in herpes, rubella, cytomegalovirus infections and malignancies.

Pathogenesis

Retinal vasculitis is presumed to be an immuno-logically mediated condition. It is an autoimmune phenomenon and various studies have shown the presence of CD4 +ve T cells within and surrounding the retinal vessels in patients with retinal vasculitis. Thus, cell mediated immunity plays a major role in the pathogenesis, however humoral immunity and immune complex formation can also be involved.

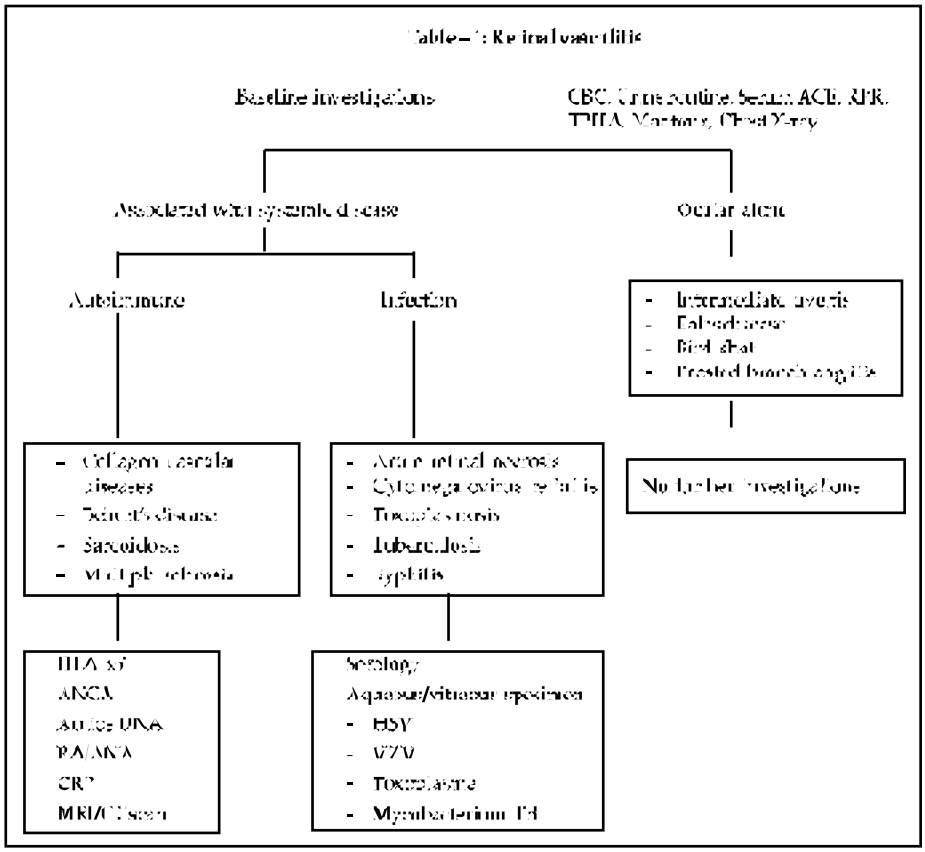
Symptoms

Patients usually complain of gradual painless loss of vision except where vasculitis involves the macula and there is sudden loss of vision. Presence of floaters is due to the inflammatory exudates in the vitreous cavity. Scotomas, photopsias, color vision alterations and metamorphopsia may also be found.

Signs

Slit lamp examination may show aqueous or vitreous cells, aqueous flare, keratic precipitates, posterior synechiae and posterior subcapsular cataract.

Fundus examination reveals fluffy white exudates around retinal vessels in the form of either continuous or skip lesions. Vascular sheathing, retinal hemorrhages, retinal odema and arterio-venous anastomoses are seen in cases where venules are the site of inflammation. Cotton wool spots which represent nerve fibre layer infarcts are mainly seen in systemic vasculitic diseases. Intra-retinal infiltrates are characteristic of infectious causes of retinal vasculitis except for Behcet's disease. Vitreous snow ball exudates and cystoid macular odema are seen in intermediate uveitis, sarcoidosis and tuberculosis. Chronic



stage of the disease shows vascular occlusion, neovascularization of the disc or retina, vitreous hemorrhage, branch retinal vein occlusion, sclerosed vessels and optic atrophy.

Involvement of retinal venules is more common in conditions like Behcet's disease, tuberculosis, sarcoidosis,, Eales' disease, multiple sclerosis, inflammatory bowel disease and sero negative arthropathies. Retinal arterioles are involved in systemic vasculitis and viral retinitis. Retinal capillary involvement occurs commonly in syphilis and Whipples disease.

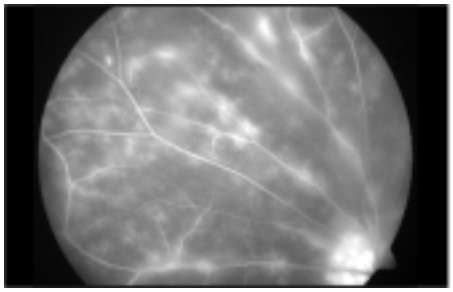


Fig .7: Fluorescein angiogram in a patient with retinal vasculitis showing diffuse vascular leakage

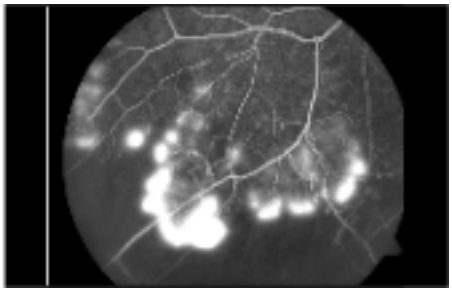


Fig. 8: Fluorescein angiogram of a patient with retinal vasculitis showing area of capillary non perfusion and neovascularization of retina

Management

Investigations

As retinal vasculitis is associated with various systemic, ocular and infectious diseases, a detailed laboratory work up is always essential. A thorough medical history and physical examination should be the basis for a focused diagnostic evaluation. The initial evaluation should include complete blood count, erythrocyte sedimentation rate, C- reactive protein, VDRL, FTA-ABS, mantoux test, angiotensin converting enzyme, rheumatoid factor (RA) and antinuclear antibody (ANA), chest x-ray.

If an infectious etiology is suspected, especially in retinal vasculitis associated with dense vitreitis, investigations should include ocular fluid cultures, serological tests and polymerase chain reaction. Serological tests are done for toxoplasma, syphilis, lyme disease and cat-scratch disease. Polymerase chain reaction in ocular fluid specimens has been extremely useful in identifying herpes simplex, varicella zoster, cytomegalovirus, *Mycobacterium tuberculosis*, and toxoplama gondii.

In patients with non-infectious systemic diseases, diagnostic tests should be focused on systemic vasculitic syndromes. Laboratory work up includes rheumatoid factor, antinuclear antibody, anti-double stranded DNA antibodies, anti-neutrophil cytoplasmic antibodies, anti-cardiolipin antibodies, complement levels, and imaging studies.

Human leukocyte antigen (HLA) testing is another useful technique in certain diseases. These HLA associations include Behcet's disease and HLA-B51, birdshot retinochoroidopathy and HLA-A29 and systemic lupus eryhthematosus and HLA-DR3. Finally, MRI and CT scan can be considered in patients with multiple sclerosis and primary central nervous system lymphoma.

Fluorescein angiography can be done in active or healed vasculitis. It shows staining and leakage of retinal veins, areas of capillary non-perfusion, retinal neovascularization, sclerosis of vessels, optic disc leakage, cystoid macular odema, vascular occlusion and macular ischemia.

Optical coherence tomography may also be done in cases of refractory macular oedema to follow them and see the response of treatment when it is not clinically apparent.

Treatment

The main goal of treatment in retinal vasculitis is suppression of intraocular inflammation in order to prevent visual loss and long-term complications. The mainstay of therapy is corticosteroids and immuno-suppressives. In case of an infective lesion, specific therapy against the infective agent with or without corticosteroids (started 2-3 days after the antiinfective therapy) may be required.

Corticosteroids may be given either systemically or by posterior subtenon's injection. Periocular steroids are useful in patients with unilateral and mild inflammation. Though this route avoids the systemic side effects, it carries a risk of raised intraocular pressure and globe perforation. Oral corticosteroids (in the dose of 1-1.5 mg/kg/day) are given in patients with moderate to severe bilateral inflammation and a marked decrease in visual acuity. Severe cases of sight threatening retinal vasculitis involving the posterior pole may require intravenous methyl prednisolone (pulse therapy for 3 days) followed by oral corticosteroids and immunosuppressives. Intravitreal steroid injections (triamcinolone acetonide) can also be given in cases of refractory macular odema.

In cases, where retinal vasculitis does not respond or shows inadequate control to oral corticosteroids, steroid sparing immunosuppressive agents are useful. These drugs are also used in patients who develop intolerable side effects to oral steroids. Various immunosuppressive agents used for treatment of retinal vasculitis include azathioprine, cyclosporine, cyclophosphamide, methotrexate, mycophenolate mofetil and infliximab. The choice of immunosuppressive agent should be individualized for each patient with a specific systemic

disease. Azathioprine, cyclosporine and infliximab are used in Behcet's disease. Alkylating agents like cyclophosphamide are often used with systemic corticosteroids in vasculitis associated with systemic autoimmune diseases like Wegeners granulomatosis and Systemic lupus erythematosus.

Topical steroids with cycloplegics are given in cases with co-existing iridocyclitis.

Other modalities of treatment in retinal vasculitis are laser photocoagulation and vitrectomy. Laser photocoagulation is indicated in patients with retinal neovascularization, with recurrent or non clearing vitreous hemorrhage and neovascular glaucoma. As photocoagulation may induce cystoid macular odema, intraocular inflammation must be adequately controlled prior to laser treatment.

Vitrectomy is useful in patients with non-clearing vitreous hemorrhage, tractional retinal detachment and epiretinal membrane removal.

Conclusion

In summary, retinal vasculitis is not only a potentially blinding intraocular inflammatory condition, it can also be the first sign of a lethal systemic disease. A detailed history, ocular and physical examination with a focused laboratory work up in a patient with retinal vasculitis can help in prompt diagnosis and appropriate management of the disease.

Suggested reading

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A New Staging System for Eales' Disease

Sandeep Saxena MS, MAMS

Eales' disease is an idiopathic retinal periphlebitis that primarily affects the peripheral retina in young adults (Figure 1). Retinal changes are characterized by periphlebitis, peripheral non-perfusion and neovascularization leading to visual loss (Figure 2).[1,2]. The disease afflicts people worldwide but for unknown reasons is more common in the Indian subcontinent. Eales' disease appears to be an immunologic reaction that may be triggered by an exogenous exposure. Mycobacterium tuberculosis DNA has been detected by polymerase chain reaction, in the vitreous of such patients.[3] However, the role of mycobacterium genome in the pathogenesis is yet to be ascertained. Author's earlier study has shown that Retinal S-antigen and Interphotoreceptor Retinoid Binding Protein plays a role in the etiopathogenesis of this condition. An extraneous agent results in the exposure of normally sequestered uveitopathogenic antigens of the immune system, leading to an immune response in the eye that may initiate the disease process.[4] Oxidative stress plays an important role in the pathogenesis of Eales' disease.[5-8] Retinal photoreceptors and platelets have been shown to be an easy target of oxidants because of high proportion of polyunsaturated fatty acids. The decreased membrane fluidity in platelets suggests alterations in the physiological events, which may result in alterations in functioning of retinal photoreceptors.[9]

The natural course of Eales' disease is quite variable with temporary and even permanent remission in some cases and relentless progression to blindness in others.

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Eales' disease is a distinct clinical entity comprising of characteristic fundoscopic and fluorescein angiographic features.[10] Charamis[11] conveniently divided the ophthalmoscopic findings into several stages, in 1965. A new classification system based on standard terminology and features has been established recently, by the author (Table). This classification system is based on both fundoscopic and fluorescein angiographic variables that have been shown to be prognostic of visual outcome.[12]

Management strategies can also be defined according

Table: Eales' disease staging system.

	Stages	*Features*
A.	Eales' disease	
	Stage 1a	Periphlebitis of small caliber vessels with superficial retinal hemorrhages.
	Stage 1b	Periphlebitis of large caliber vessels with superficial retinal hemorrhages.
	Stage 2a	Peripheral capillary nonperfusion.
	Stage 2b	Neovascularization elsewhere /Neovascularisation of the disc.
	Stage 3a	Fibrovascular proliferation.
	Stage 3b	Vitreous hemorrhage.
	Stage 4a	Traction / combined rhegmatogenous detachment.
	Stage 4b	Rubeosis iridis, neovascular glaucoma, complicated cataract, and optic atrophy.
B.	Central Eales' disease	

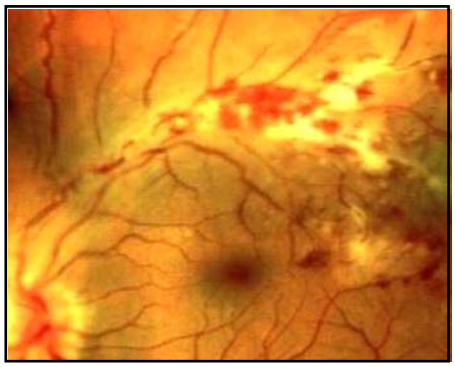

Fig .1: Eales' disease: Retinal periphlebitis

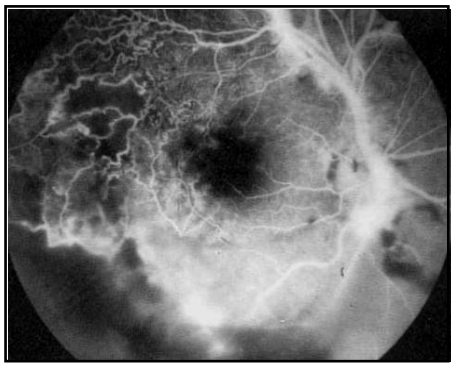

Fig. 2: Eales' disease: Fluorescein angiogram showing periphlebitis, retinal and disc neovascularisation, capillary non perfusion and vitreous hemorrhage

to the stage of the disease. Stage 1, the stage of inflammation, is amenable to medical therapy. Stage 2, the stage of ischemia and neovascularisation, requires observation/ laser photocoagulation. Stage 3, the stage of proliferation, requires laser / pars plana vitrectomy and laser. Stage 4, the stage of complications, requires sophisticated surgical management strategies.[12]

The new classification system for Eales' disease is unambiguous, consistent, simple, and is useful in assessing the severity of the disease and monitoring the effect of medical, laser and/or surgical treatment. This classification system is designed to promote the use of standard terminology and assessment with applications to patient care and research in Eales' Disease.

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Role of Patients Selection in Multifocal Intra Ocular Lens Implants

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The WHO statistics report that there are about 45 million blind and 180 million visually impaired persons worldwide. Presbyopia alone or Presbyopia with Cataract contributes to these figures apart from other ocular pathologies and is spread among all ages of population.

Phacoemulsification surgery combined with the latest in intraocular lens namely MULTIFOCAL LENSES BRINGS ABOUT THE POSSIBILITY OF GOOD QUALITY VISION for distance, near and intermediate distance too. Expectations today are not limited to just restoration of vision alone but wanting vision close to what a young normal patient has, in other words qualitative EMMETROPIA.

Infact our role as Ophthalmic Surgeons & Consultants has become much more critical in providing this modern high technology treatment to patients whose post surgical results are almost tailor made these days!

Potential Benefits for Multifocal Lens Implants

Excellent visual outcomes for near and distance vision and good intermediate vision too.

Relative spectacle independence for 90% of the patients, especially after bilateral implants with multifocals because of binocular summation.

Patient's visual symptoms to be infrequent and easily tolerable especially with the newer generation diffractive and apodised diffractive technology. With the latest technology of diffractive and apodised diffractive multifocals pupil size is no longer a major issue. This apodised diffractive lens provides equal distribution of light energy for small pupils under photopic conditions greater light energy distribution for the distance when the pupil dilates in mesopic conditions.

Apart from our honest intention to provide the best surgical outcomes some of the key considerations for Multifocal Implants are listed below.

Pre-operative Considerations

Patients who has a strong desire to be spectacle independent. This is the single most important indication for multifocal lens surgery. After all it is futile to place a multifocal lens in a patient who is reluctant to give up wearing glasses!!

Radiant Eye Foundation,
2/3, Justice Dwarkanath Road,
Kolkata

Age- By and large for the first 100 cases it would be better to operate within the age group 35 to 75. However there are exceptions to this statement.

Functional & Occupational Requirements- A detailed history on this point is most crucial. Does the patient have any of the hobbies like painting, playing the piano, playing cards or billiards or is he just the unusual avid reader?

Patients often complain of the difficulty in MULTI TASKING post IOL surgery with monofocal lens implant .This category of patients are the ones to target for.

Pre-existing Ocular Pathologies-One has to rule out the possibility of preexisting ocular diseases like ARMD,GLAUCOMA etc cases as these are visually and surgically demanding cases where the real benefits of Multifocality may not be produced or appreciated by the patients.

Degree of General Alertness-In our country this is an important issue as the educated younger generations are more demanding and conscious of the latest advancements and availability of this technology. We need to invest time to communicate with the patients.

Hypercritical & Demanding Patients-This class of patients should be strictly avoided. One should prefer to operate on those patients who are fully confident and trust their surgeon's skills and capability. In other words patients with whom there is a comfort level. (As an experienced surgeon its better to avoid proving ourselves to a cynical and suspecting patient who remains unhappy no matter how best we perform). (One does not need to break heads in counseling those patients having a 0.50 refractive error if any or the possible haloes or mild transient glare if present which lessens with time).

Strong urge for near reading without glasses -These AVID readers will be very happy with this modern apodised diffractive multifocal which has astoundingly good unaided near vision especially in bilateral lens implants.

Pre-operative Exclusion Criteria

Subjective Exclusion

Hypercritical patients

Patients with unrealistic expectations- As discussed above.

Occupational night drivers –For all these patients even a short term glare or halo will be intolerable. For patients who are chauffer driven, one should enquire about

whether they just enjoy driving back home in the evenings or like to take long drives over the weekend. These explaining patients about potential side effects and also the fact that they are easily tolerable with binocular summation and lessen with time is very crucial. After all we know that the brain perceives what it wants to!!

Medical Exclusion

More than 1.0 D of corneal astigmatism-This reduces the possibility of post op astigmatic surprises. Since we aim for emmetropia, so post op astigmatism is undesirable. Fashion the wound around the steeper meridian.

Pre-existing ocular pathology-As discussed above

Individuals with a monofocal lens- It is recommended to avoid these patients. Symmetry and binocular summation is the name of the game

History of previous Refractive Surgery-Although these patients are the most strongly motivated ones to have multifocal lens implants but they are unsuitable. If K readings are less than 40 suspect this possibility .Biometry accuracy is doubtful in these patients and clinical correlation with previous or present spectacles is important when comparing with the fellow eye.

Intra-operative Exclusion Criterias

Significant vitreous loss during surgery

Pupil trauma during surgery

Factors mentioned below can impact long-term IOL performance. All these situations can affect lens centration and support which is the key factor for target surgical outcome for all these multifocal lenses.

Zonular damage

Capsulorhexis tear

Capsular rupture

Keys for Success

Patient Selection- The right patient is to be selected

Accurate Biometry- Repetition of procedure, use of immersion technique, clinical correlation with present or past refractive error, comparison with fellow eye etc.

Power Calculation- Maximize visual outcomes by calculating for a post-operative refractive spherical equivalent from Plano to <+0.25. Use of newer formulae like SRK-T and HOLLODAY.

Surgical Technique- Round, centered CCC completely overlapping the Lens Optic, removing all viscoelastic behind the lens, proper seating of lens in capsular bag are prerequisites. In other words the surgery must be a work of art. A combination of perfect surgery and right selection of patients will fetch us a very happy patient who becomes that all important flag bearer for other eager but hesitant patients.

Multifocal phacoemulsification surgery needs a lot of spell these counselling and a certain degree of trust in the doctor patient relationship.If the surgery is not up to ones expectations it is always better not to use a multifocal lens. As of now **THIS LENS IS NOT MEANT FOR THE SULCUS!!**

The amount of our chair time spent with the patient prior to surgery greatly reduces chair time spent afterwards. However with this great technological leap forward, it is setting the bar just one step higher in the ladder of surgical quest and ambition. With the encouraging results obtained with Multifocals implants in cataract patients there may well be the answer in the future to non cataractous presbyopes who are unwilling to wear glasses.In fact many surgeons worldwide are using these high quality multifocals for clear lens extractions with very encouraging results. In one word "to conquer two targets with one shot".

Nucleotomy

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Nucleotomy forms the crux of a good phacoemulsification. For a perfect phaco-emulsification, it is essential to remove the nucleus using minimum phaco energy without damaging the surrounding structures especially the cornea and posterior capsule. The basic understanding of the anatomy of the nucleus is most important to accomplish this procedure.

Applied Anatomy

From the surgeon's viewpoint, there are mainly two important points of relevance, which are to be kept in mind before attempting a nucleotomy:

1. The lens consists of the central hard core surrounded by a comparatively softer epinucleus and the outermost layer of thin cortex. With increasing age the epinucleus gets thinner and the central hard core increases in thickness and density. The increasing density of nucleus in old age also results in a downward (more posterior) placement of the densest part of the nucleus (Fig.1). Thus the trench has to be almost 90% of the thickness of the lens if a complete crack is to be obtained.
2. The greater curvature of the lens posteriorly as compared to the anterior surface is of special relevance to the phaco surgeon. It is important to note that this curvature varies achieving almost a conical shape, as the age of patients increases and the lens gets harder. The movement of the phaco probe while trenching must follow this curvature, if adequate depth is to be achieved and capsular injury is to be avoided. The red reflex may be visible from the periphery even in a shallow trench so one should change the direction of the probe accordingly.

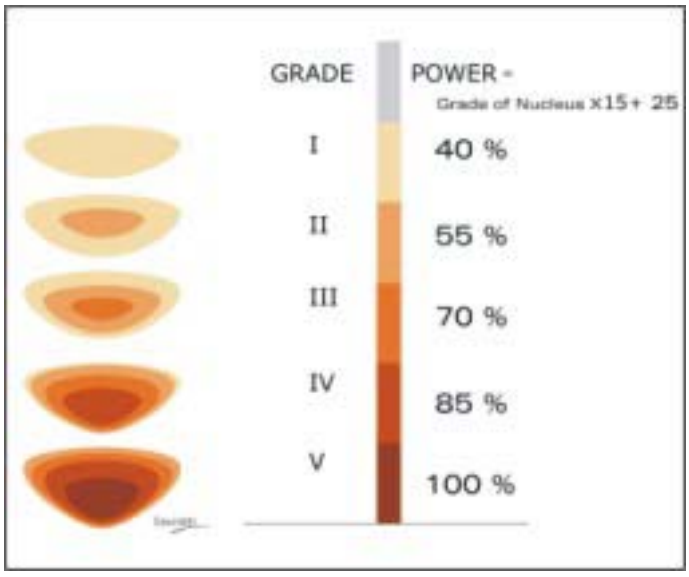

Fig.1: Grades and density of the nucleus. With increasing density the hardest part of nucleus keeps moving down and posterior curvature keeps on increasing. With increasing grade and density, more phaco power is required.

Nucleotomy

The most commonly used methods in nucleotomy are 'divide and conquer', 'stop and chop' or 'direct chop'.

Our preferred technique of nucleotomy is 'stop & chop', in which we start with trenching as in 'divide & conquer' stop after the first trench and then proceed with chopping techniques, i.e. central chop, peripheral chop and modified peripheral chop. The steps for the same are discussed as follows:

Entering the eye

The wound size should be made according to the tip being used. Usually a 2.8mm size is good enough for a comfortable smooth movement of the probe. Neither the incision should be too tight (as it can cause a wound burn) nor should it be a leaky wound which can cause an unstable chamber.

One should always enter the eye in a partially formed chamber with viscoelastic so that there is no iris or corneal touch.

While entering depress the lower lip of the tunnel and then introduce the metallic part of the tip. Now lift the upper lip of the wound and guide the sleeve in. Once the sleeve is in, lift the probe and direct the tip downwards to prevent any Descemet's detachment. The infusion may be kept on, while entering the chamber, if there is no viscoelastic in anterior chamber or the chamber is shallow. However, it may be inconvenient to the patient while doing surgery under topical anesthesia.

Baring the Nucleus (Fig.2)

Before attempting the trenching one needs to bare the nucleus by removing the anterior epinucleus plate. This

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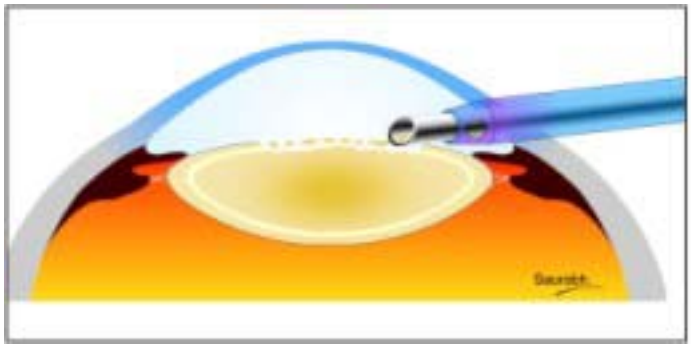


Fig.2: Removal of the anterior epinuclear plate. (Baring of the Nucleus)

Fig.3: Shaving action while trenching angulation of the tip is such that the tips is lifted up and the handle depressed using the incision as the fulcrum while moving forward.

can be done by using minimum phaco energy and moderate vacuum (60-80mmHg). The phaco probe is gently moved above the nucleus to suck away the anterior epinucleus fibres up to the capsulorhexis margin to denude the nucleus below. This defines the boundaries of the nucleus and improves the visibility of the nucleus giving a comfortable and controlled trenching maneuver with better hold.

Trenching

Trenching involves making a groove into the nucleus. This is generally performed at low vacuum settings (i.e. 20 ± 10mmHg). In this we want a good cutting action instead of good hold. Power setting will depend on the hardness of the nucleus. A practical formula that may be applied is power=(grade of nucleus x 15)+25. *In newer third generation machines the phaco can be done on hyperpulse mode* instead of the conventional continuous phaco mode. The pulses in this may be kept at 80 to 100 with a 50 to 80 percent on time. This type of energy delivery works as good as the continuous phaco mode with the advantage of minimum rise of temperature. It is better to keep the setting at the highest required levels and control the actual energy delivered by the foot pedal, using the linear mode for better control. The ideal power is that at which there is no wasting of energy and no pushing of the nucleus. Too little power or moving the probe too fast results in pushing the nucleus without cutting it leading to zonular stress.

The bottle should not be very high as it will deepen the chamber and push the lens diaphragm downward, which may cause inconvenience by making the probe more vertical and can lead to corneal burn and wound distortion causing poor visibility. The average bottle height required is about 50-60cm above, the patient head level. The bottle height should be lower in cases like high myopia, vitrectomised eyes, zonular laxity with deep chamber syndrome.

It is best to start the trench just proximal to the center and then move the probe towards the CCC margin in the cross-incisional axis. The movement of the probe & the foot control are vital during trenching. The nucleus needs to be sculpted with a shaving action which requires a minimal downward angulation without causing occlusion. The forward strikes consists of shaving action (Fig.3) with an attempt to move into the nucleus by a depth less than half the diameter of the tip. On the return stroke care should be taken that no phaco or aspiration is used. The trench is widened and deepened uniformly with multiple strokes. The nucleus needs to be rotated to bring the sub-incisional nucleus into the cross-incisional area for better access. After 180 degree nucleus rotation, trench, is performed in the opposite half. So one must be careful to continue the shaving action till the trench is of uniform depth, avoiding the tendency to go deep.

Size of a trench

This is determined by the hardness of the cataract and the size of the nucleus. The larger the nucleus, the wider and longer should the trench be. As a general rule the size of the trench should be a little short of the CCC margin (Fig.4). There is no need to go under the CCC except in hard cataracts, very soft cataracts and in small CCC (Fig.5). The trench should be about '2 tip diameter' wide in order to accommodate the sleeve. One may make a central groove and then widen both the sides by tilting the probe with bevel facing the center (Fig.6).

Most authors describe that the trench should be 2 to 3 tip-diameter deep. The visibility of red reflex is a better guide in moderate cataract. If one is unsure of the depth, it is better to withdraw the probe, fill the eye with visoelastic and then assess. If the depth seems adequate, crack the nucleus but if it does not crack easily, do not apply undue extra force as this may lead to zonular tear, on dialysis. Instead in these cases one need to further deepen the trench then attempt too crack.

Sometimes due to lack of smooth actions the trench has an uneven surface or even a large bump in the center. This is more likely if a 'Khurpi' action (Fig. 7) is used rather than shaving action. If the red reflex is visible anywhere between the bumps, then crack the nucleus. Otherwise the

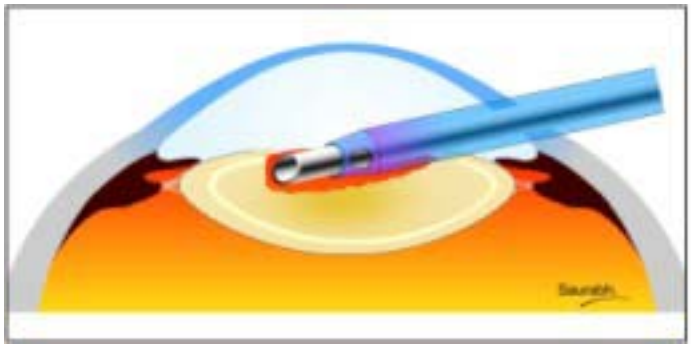


Fig.4: Length of the trench. The trench should be just short of the CCC.

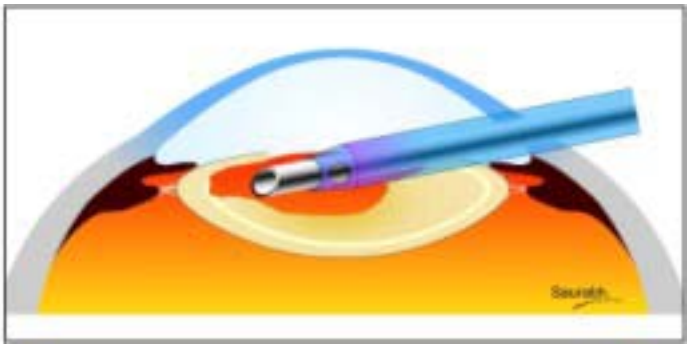


Fig.5: Relaxing nucleotomy going beyond the CCC margin in a Hard Cataract

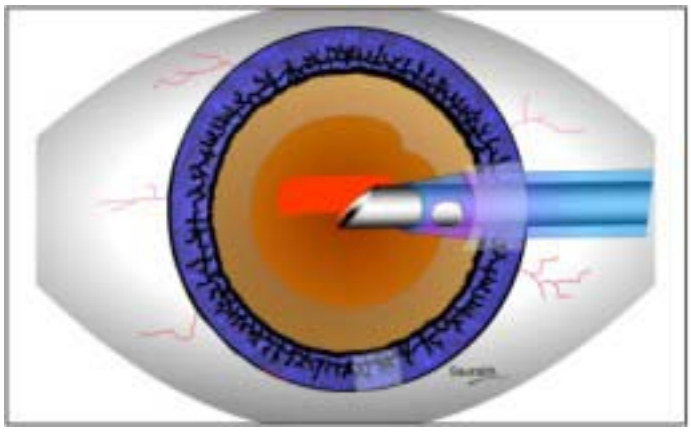


Fig.6: Increasing the width of the trench. Tilting the probe with bevel facing the center.

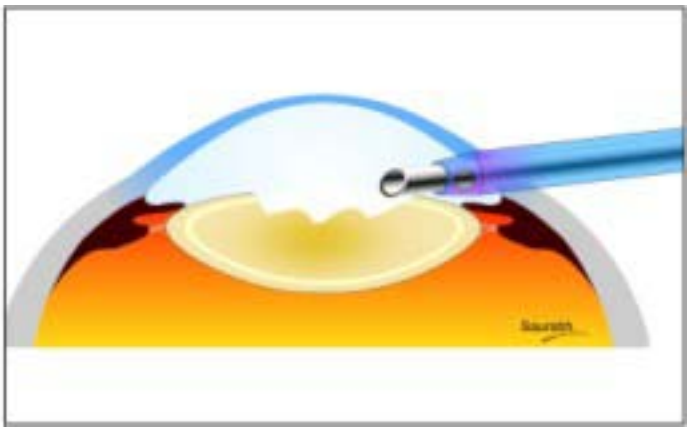


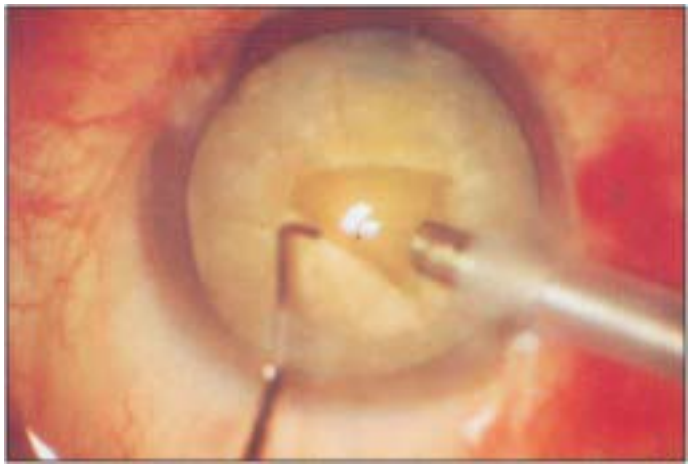
Fig.7: Repeated Khurpi action leads to uneven bumps.

bumps need to be shaved off carefully to achieve a level surface.

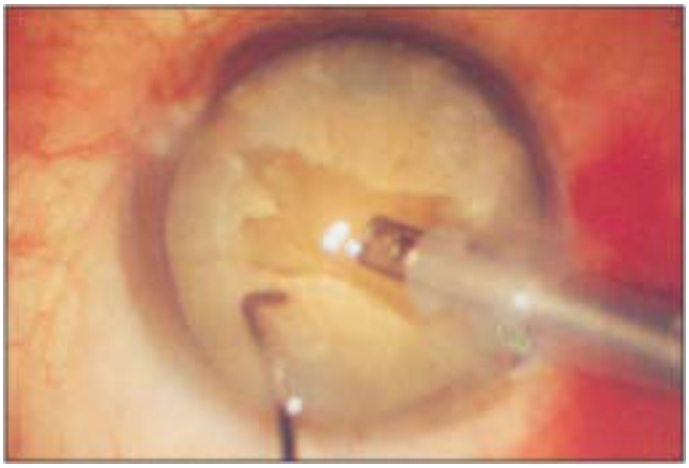
V-shaped or Victory Trench (Fig.8a-8c)

A useful modification of the trench is a 'V' trench. In this, the initial stroke is made slightly radially instead of a straight axis. The second stroke is also made radially to complete a 'V'. The trench is gradually deepened in the same shape. The nucleus is rotated and then similar strokes are made on the other side to make an 'X' pattern (Pic.-2). The trench is then deepened as normal, keeping the peripheral curvature in mind. The trench mode deep to form a vertical 'V' as in fig. 8c.

The advantage of this 'V' trench is that there is a wider space to work in. Also the tendency to get stuck in the same groove is avoided. The wider groove after rotating the nucleus accommodates the sleeve, thus the tip can reach to the deepest part of the nucleus (Pic.-1). This is our



Pic.1: 'V' Trench gives enough space to accommodate the sleeve proximally.



Pic. 2: Converting 'V' to 'X' shape.

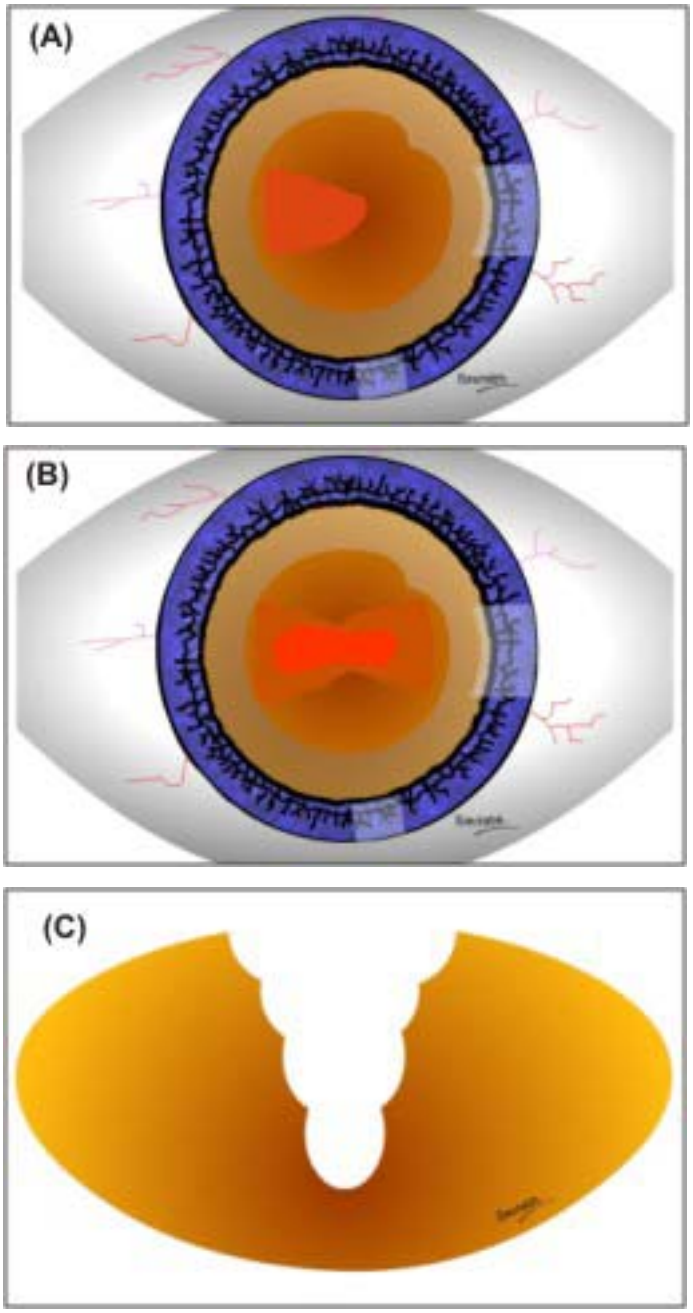
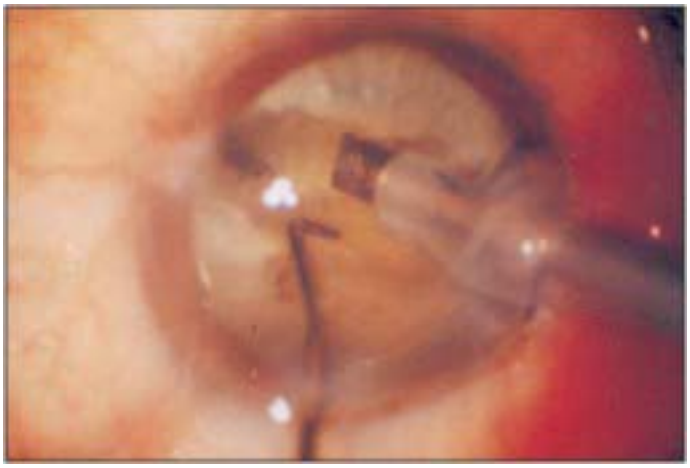


Fig.8: (a) Making a single 'V'. (b) Converting a 'V' to 'X' shape after a similar attempt 1800 opposite. (c) Depth of 'V' to reach the deepest and hardest part of the nucleus.

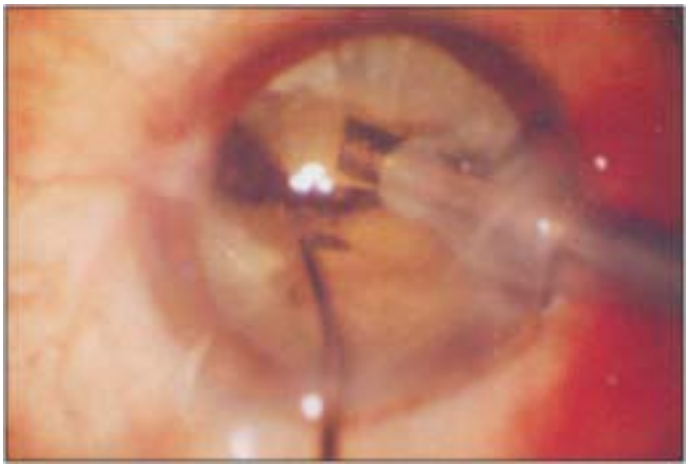
preferred mode of trenching particularly in hard and mature cataracts.

Splitting

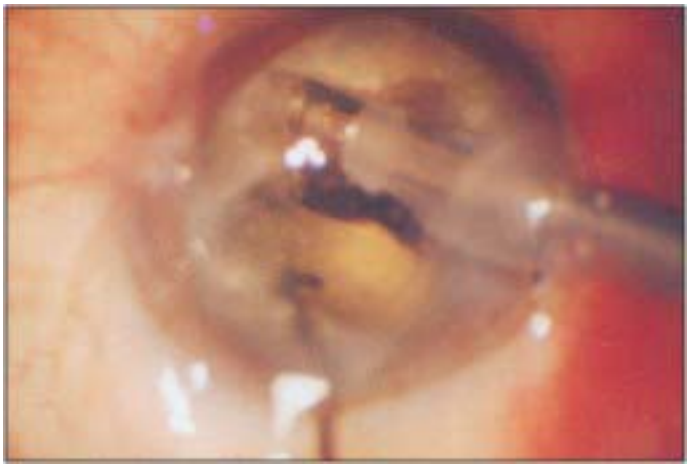
One should use two instruments (either a phaco probe and a chopper or 2 choppers) and the instruments must be placed, as deep as possible in the trench. The instruments must be close together both vertically and horizontally (Fig.9). The force is applied in opposite directions such that the center tends to depress slightly and periphery of the nucleus tends to lift upwards (Fig.9).



Splitting Pic.3: Instruments placed deep in the trench. Crack appears in the periphery.



Splitting Pic.4: Both the instruments moved in opposite directions as far as possible. Crack still not complete.



Splitting Pic.5: Nucleus rotated to bring the sub-incisional area to cross incisional side and nucleus split completed.

In cases of very hard cataracts one can initiate the split at the periphery and then move both the instruments towards the center to complete the crack. In case it does not go to the opposite periphery, one can rotate the nucleus by 180^0 degrees to complete the split (Pic.-3-5).

Another method of cracking the nucleus is to position

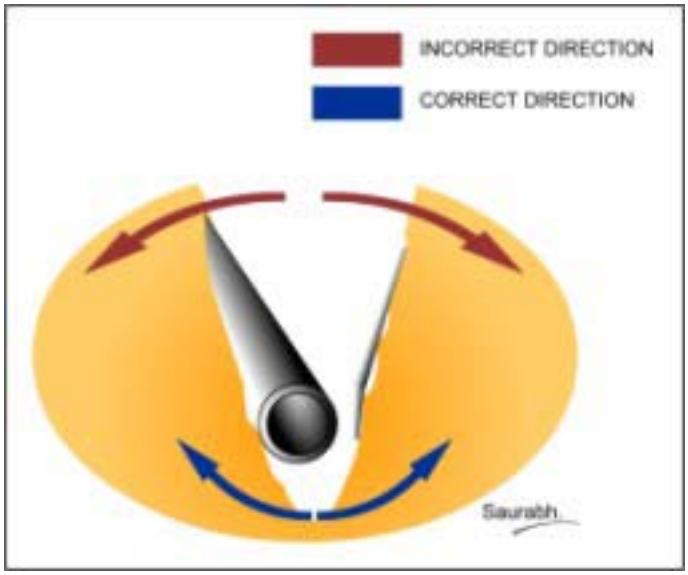


Fig.9: Splitting the nucleus: Note the vertical placement of instruments in the depth and the direction of forces.

the nucleus horizontally, embed the probe into the center of the hemi nucleus and pull with the second instrument to achieve the crack. The settings should be those of chopping and this is only applicable for cataracts harder than grade 2. One has to be sure of achieving a complete split before passing on to chopping. But at times in very hard cataracts the same cannot be achieved and inspite of a proper methodology. There remain a few bridging fibers in the center. In these cases one should not be over enthusiastic in separating these fibers and instead proceed with chopping the split ends of the nucleus.

Chopping

The term is used to denote the splitting of the nucleus into smaller pieces by a chopper. Chopping can be peripheral, central on a combination of these. The nucleus is split into two halves as already described. The probe can be easily embedded into the hard body of the nucleus and a vacuum seal is created for chopping. Since the platform is wide, one can try an alternate site if initially a good hold is not obtained. Each hemi-nucleus can be divided into three pieces in a safe and controlled manner.

For a successful chopping it is most important to achieve a good hold by creating a perfect vacuum seal.

Creation of vacuum seal/hold (Fig.10a-b)

Before embarking on this step, a phaco surgeon must have good understanding of the audible and tactile feedback of the foot pedal. One should be able to maneuver the pedal between the positions of aspiration, phaco and back to aspiration and should be able to hold on to this position for sufficient length of time to develop a vacuum hold and then chop.

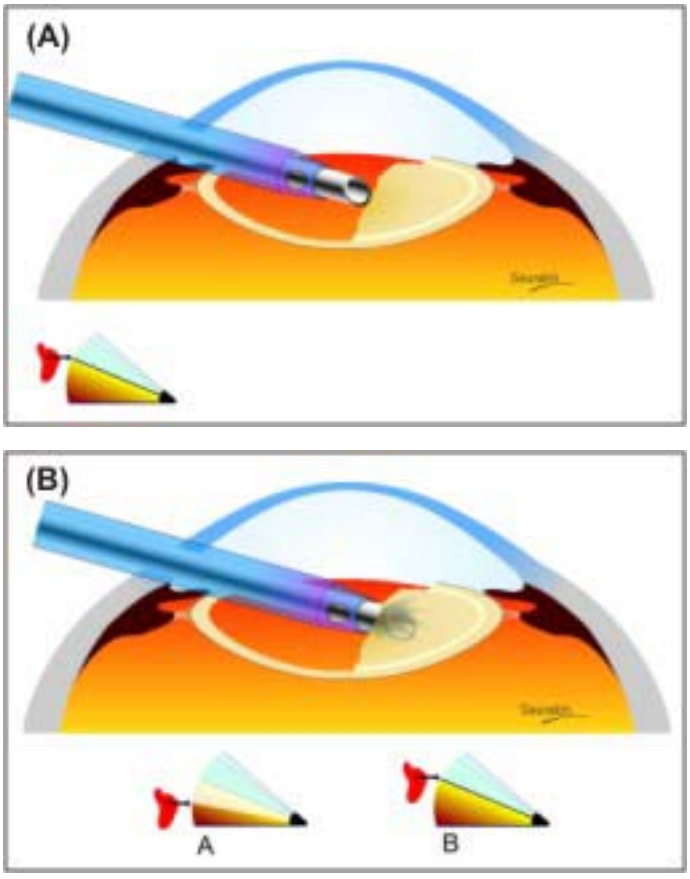


Fig.10: Creation of a vacuum seal. The probe is introduced into the eye in continuous irrigation mode. (a) The nucleus is nudged and foot pedal brought to position a (IA). (b) A short burst of phaco (position 3) is given to embed the probe into the body of the nucleus. Note that the foot pedal is brought back from position 3 to position 2 (IA) to let the vacuum build up and create a hold.

As the tactile feedback of the dentation of the footpedal is less on upward excursion, while moving from position 3 to 2, overshoot to position 1 or 0 is common. Hence it is safe to keep the machine in continuous irrigation mode to prevent AC collapse. If you have an overshoot, press the foot pedal back to position 2 and remain stable at the dentation.

The surgeon must also be familiar with the high pitched, fixed frequency sound that is emitted by the machine once the maximum preset vacuum is achieved. This indicates full vacuum and thus a good hold. Beginners can practice by pinching the tubing to occlude it with the probe outside the eye and listening for the sound.

If the impalement is adequate then on slight to and fro movement of the tip, both the tip and the nucleus piece will move as one unit. If, however, the grip is not sufficient the nucleus piece will be set free. If impalement is not achieved in one area, move to another site and attempt again. One should carefully select the area to be impaled, trying to remain within the body of the nucleus.

Causes of poor hold include keeping the foot pedal in

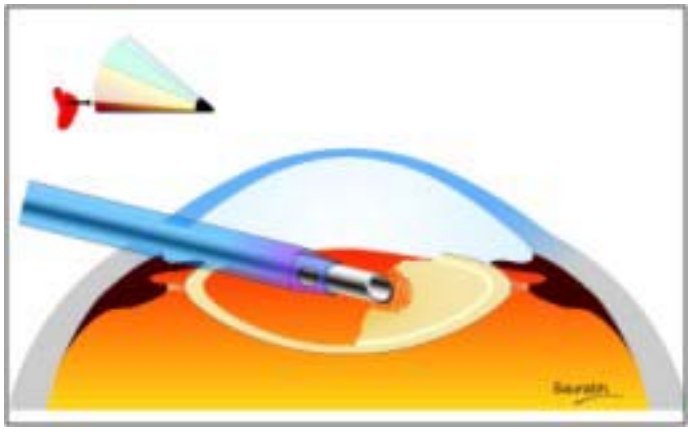


Fig.11: Maintaining the foot in position 3 leads to loosening of the grip.

position 3 for too long as this leads to emulsification of the nucleus material around the tip (Fig.11). This loosens the grip in addition to causing milking/clouding of the AC. If the site is too superficial, complete occlusion is not achieved and therefore vacuum seal is not effective. In low or inadequate vacuum settings, the probe keeps slipping and a good hold is not obtained. In very soft cataracts, creating a vacuum seal and vacuum hold is more difficult. The cataract gets sucked even without use of phaco energy and danger of going through and though is high. In such a situation vacuum and power settings should be lower.

After achieving good hold the need is to chop the nucleus fragment. The chop can be central or peripheral. Central chop (Fig. 12a,b,c) involves splitting of the nucleus from mid-periphery by embeding the chopper on the anterior surface of the nucleus and then pulling the chopper side ways. A few important point being:-

1. The chopper has to be close to the phaco tip
2. At the time of splitting the chopper has to be on the left of the phaco tip.

The vertical portion of the chopper is buried deep in the nucleus, the chop is initiated by moving the chopper to the left (Pic.-6-7). The hold is maintained and the chopper moved away from the probe till the nucleus fragments are separated.

Problems with central Chop

Since chopping is not started at full depth, sometimes a partial thickness chop may occur. To complete it, chopping has to be repeated twice or even thrice. In a peripheral chop, once the chop is initiated, it is usually full thickness.

Peripheral chop is stronger than central chop. In a hard nucleus, the chopper may not embed to the required depth. Also leathery fibers which do not split can be easily cut in the peripheral chop.

Central chop needs more space due to the rotational component in it.

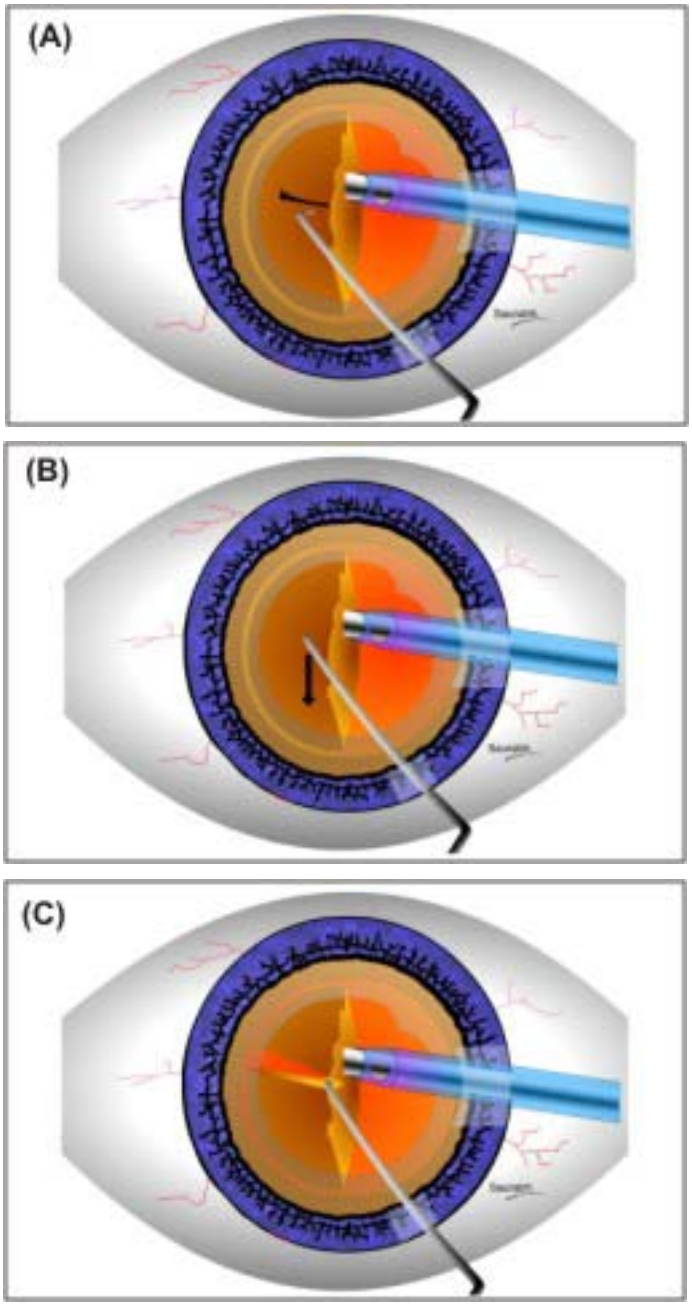
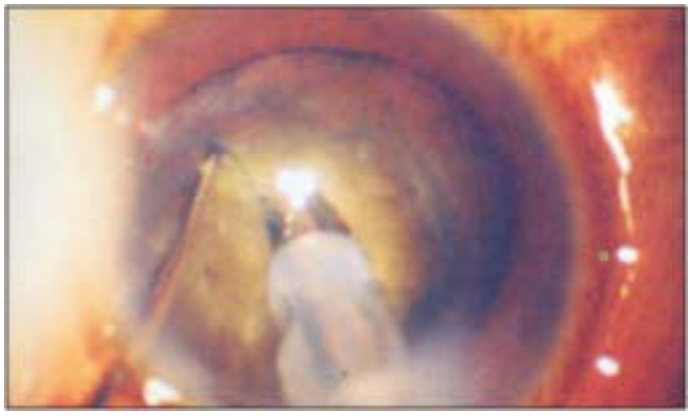


Fig.12: Central chop. (a) Embed the probe into the nucleus and create a vacuum seal. Black line denotes the ideal area to embed the chopper close to and to the left of the probe. (b) Note the direction of movement of the chopper. (c) The split starts superficially and goes deeper. One may have to move the chopper deeper to complete the crack.

Peripheral Chop (Fig. 13a-d) (Pic.-8-11)

For this the side port incision is made between 1-2 O'clock hours to the left of the main port incision. Chamber is moderately filled with visoelastic and continuous infusion is set. Chopper is placed on the anterior nucleus surface, close to rhexis margin in a horizontal position. It is slipped underneath the CCC margin and positioned just to be right of the line of the phaco probe, at the delineation line. Now with the other hand, phaco probe is impaled into the nucleus. Once the vacuum hold is obtained, the



Central Chop Pic. 6 : After stabilizing the nucleus with the phaco probe. Burying the chopper into the nucleus for central chop.

Central Chop Pic. 7: Chopper moved laterally as much as possible to extend the split.

chopper is turned vertical and pulled towards the U/S tip. Just before reaching the probe, the nucleus is split sideways.

Oblique placement of the chopper leads to a rotational effect. The ideal placing of the chopper would in the same line as the phaco probe, but this would require the entry of both chopper and the phaco probe through the main incision. Therefore, a side port incision 1-2 clock hours away instead of 3 clock hours away from the main incision helps in placing chopper close to line of the phaco probe without much increase in the rotational effect.

Problems with Peripheral Chop

Complex maneuverability is needed for this technique. The chopper must be negotiated under the CCC margin while simultaneously the hold on nucleus has to be maintained.

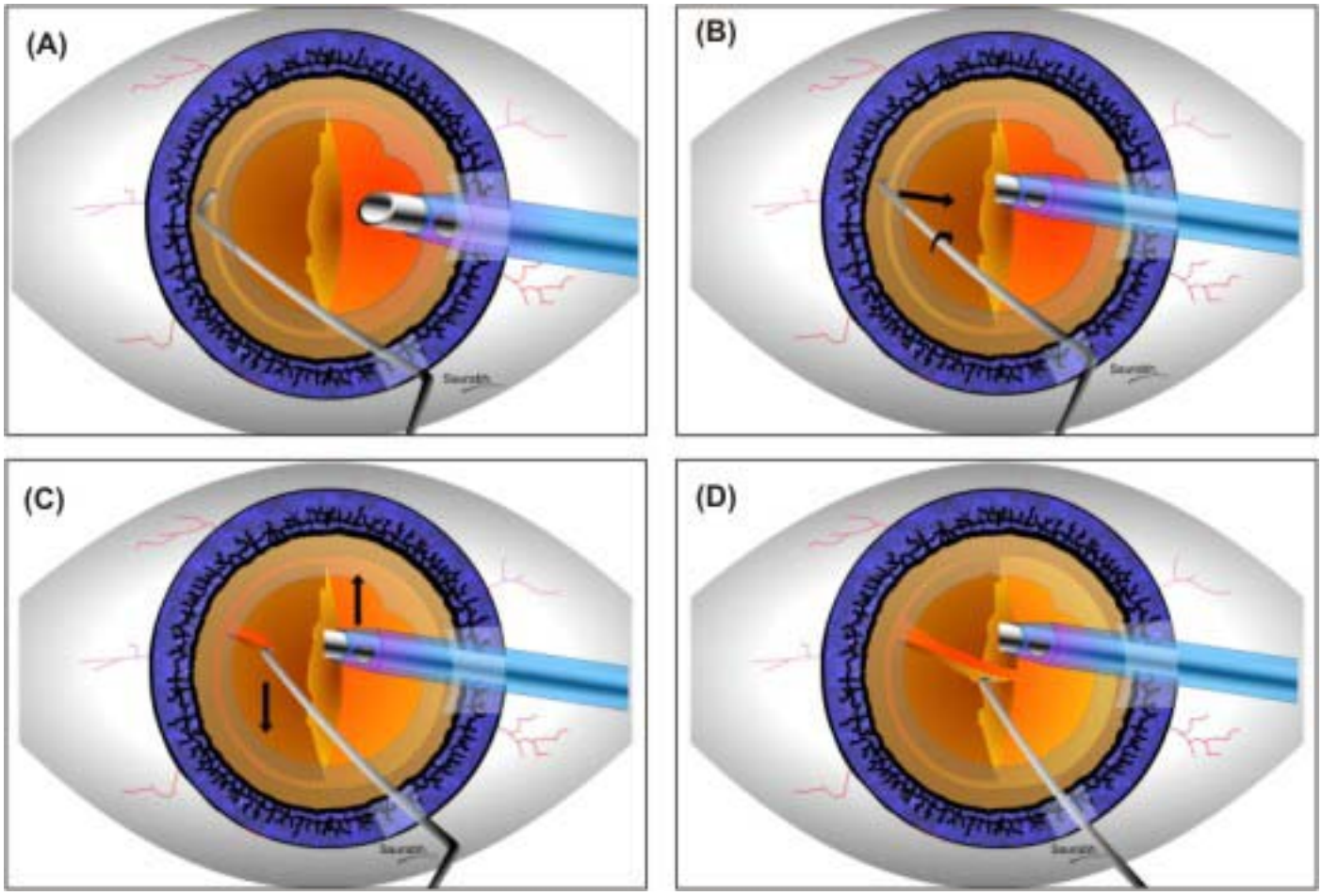
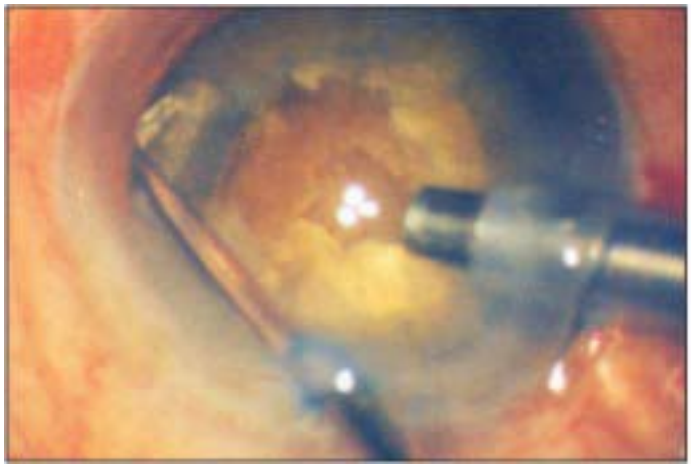
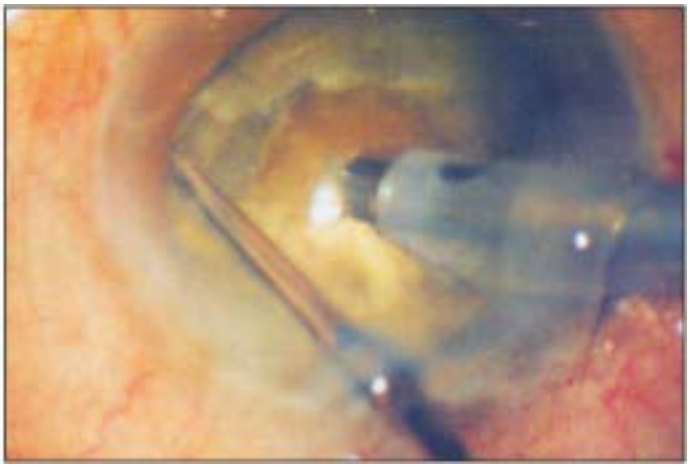


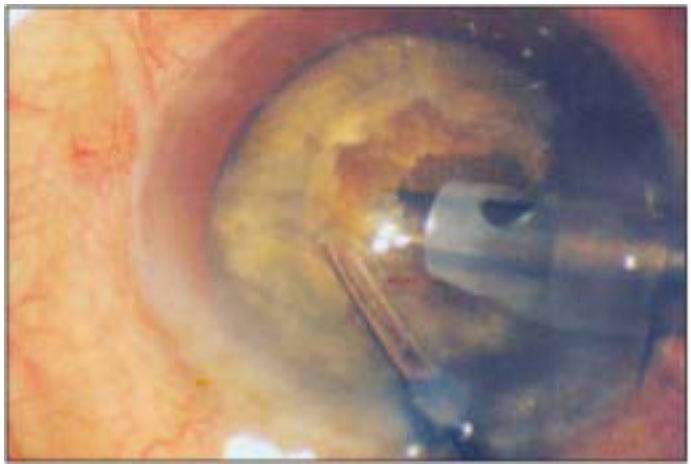
Fig.13: Peripheral chop. (a) Place the chopper underneath CCC at the delineation line. (b) Embed the tip to create the vacuum seal. Now turn chopper vertical and pull it towards the phaco tip. (c) Once the chopper reaches the tip move it sideways to complete the crack. (d) Crack will proceed from periphery to centre.



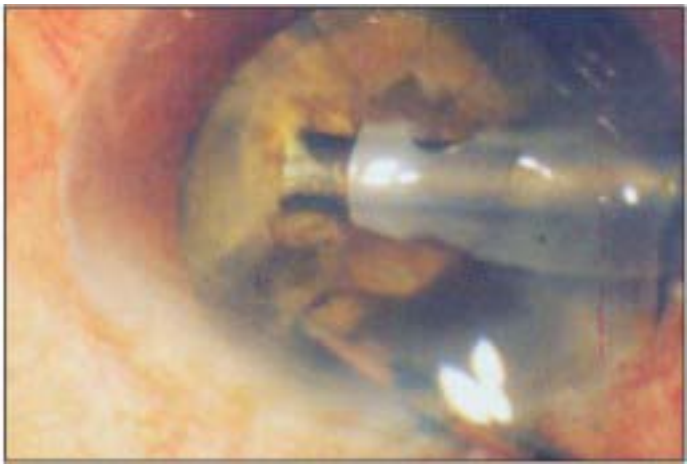
Peripheral Chop Pic. 8: Chopper under CCC at the delineation line.



Peripheral Chop Pic. 9: Vacuum seal created.



Peripheral Chop Pic.10: Chopper puled forwards towards the probe.



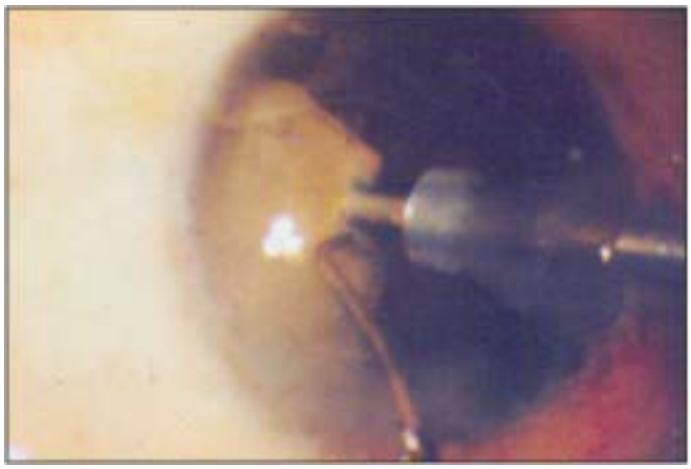
Peripheral Chop Pic.11: After it reaches the probe it is puled side ways to complete the chop.

There is danger of PCT while chopping. Use of a long chopper on a soft nucleus in an unstable AC can lead to peripheral capsular tear.

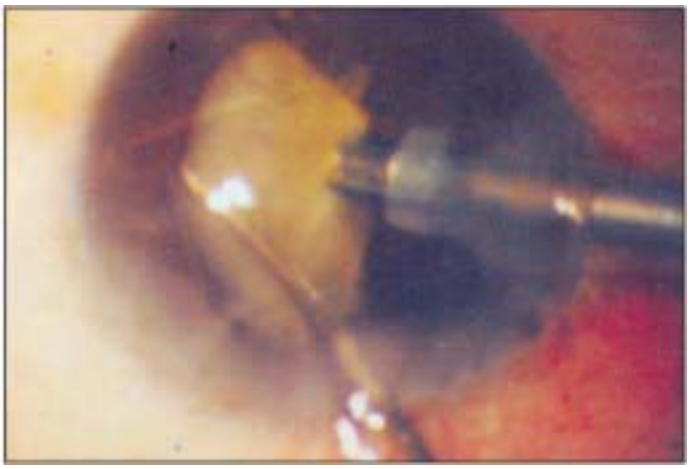
If the chopper is accidentally placed over the anterior capsule instead of underneath it and the chop is initiated, it may lead to rhexis margin tear or zonuloysis.

Modified Peripheral Chop (Fig. 14a-c) (Pic.-12-13)

Negotiating the rhexis margin is the most difficult part of the peripheral chop. Benefits of both kinds of chopping are obtained using a modified peripheral chop. In this, after a vacuum seal has been created the periphery of the nucleus is brought out of the rhexis margin thus avoiding



Modified Peripheral Chop Pic.12: Creating a vacuum seal.



Modified Peripheral Chop Pic.13: Pull the piece out of the CCC margin till the peripheral edge is visible. Position the chopper at the peripheral edge and split.

negotiating under the CCC. Following this, the method is same as in peripheral chop. However, this is only possible if:

1. The capsulorrhexis is not small (>4.5mm)
2. The nucleus is not very long
3. One half of nucleus has already been removed.

This is an extremely useful procedure for hard nuclei with leathery fibers.

Phaco Aspiration

Removal of the nucleus segments from the capsular fornix and their aspiration in the corneal safe zone is called aspiration phaco.

Phaco-aspiration must be done in the central safe zone and the pieces must be removed from the capsular fornix (CF). Removal of the first piece from the fornix is the toughest as it may be entangled with other chopped pieces, or the fibers may not be divided completely. The ideal piece to remove first is the smallest piece which has been completely separated from the rest of the nucleus.

As in chopping, take the probe close to the body of the nucleus piece and embed in it with a small burst of energy. Allow time for vacuum hold to build up and then pull the piece out of the capsular fornix to central safe zone. Attempt to keep the piece away from cornea by angulating the probe a little downwards. Then crush or divide the piece with the help of the chopper. Phaco should be off while crushing i.e. foot position 2. Care should be taken throughout the procedure to avoid contact of the chopper with the probe when the foot pedal is in phaco mode. After crushing, if the cataract is soft, it gets aspirated without the use of phaco energy. However, most of the time little phaco energy is required to remove the pieces. Crushing not only dismantles the piece, it also increases the follow ability of the pieces which now get sucked in and need little phaco energy for emulsification. This procedure is repeated many times till the whole nucleus is removed. It is preferable to remove the piece by aspiration mainly, mechanical crushing may be added on as and when needed and minimal phaco energy must be used. Phaco should only be used when pieces are gripped by phaco probe and are not getting sucked in, i.e. the probe is fully or partially occluded.

If after removal of the first piece surge is noticed, vacuum settings can be reduced as the rest of the pieces are easier to maneuver and remove. During removal of last piece vacuum settings can be further lowered, as at this stage there is no other piece in capsular fornix to prevent forward movement of posterior capsule. Even a small surge can cause a PCT particularly in a mature and hard cataract where the PC is not protected by the epinuclear plate. Beginners may fill the chamber with viscoelastic before aspirating the last piece. This gives good stability to the chamber and holds back the posterior capsule, while the last piece is being aspirated.

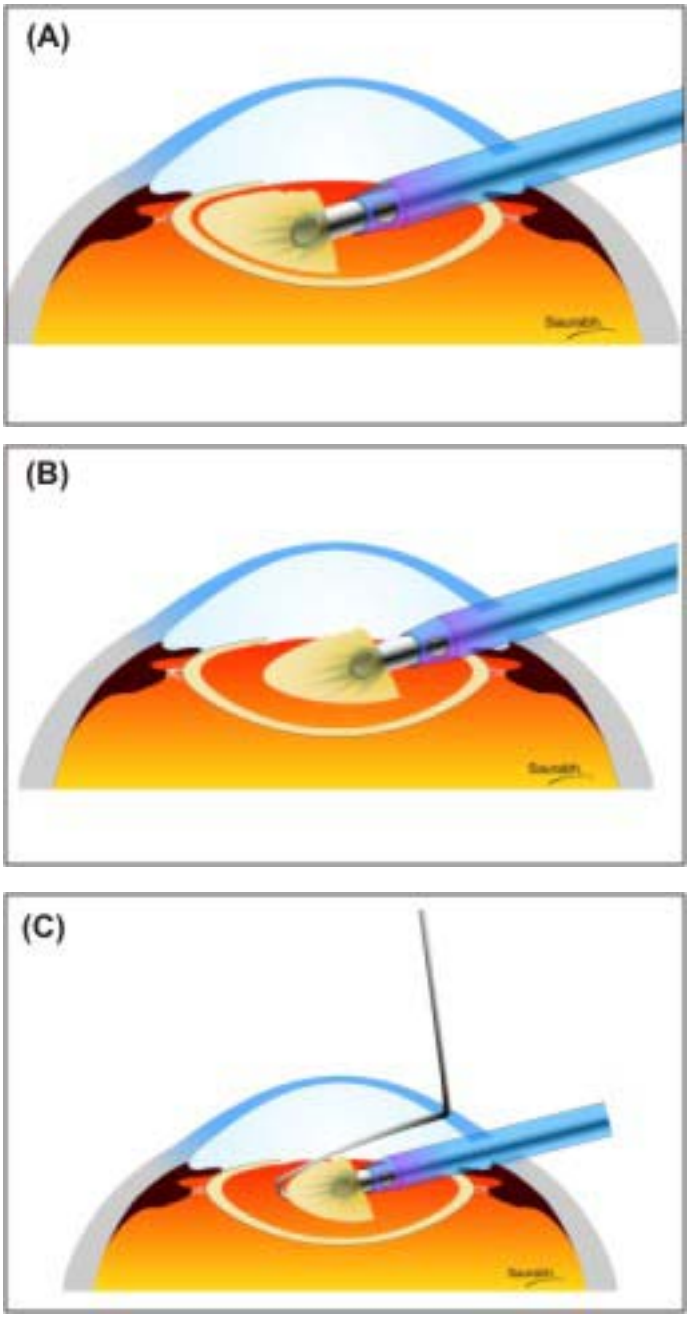


Fig.14: Modified peripheral chop (a) A vacuum seal is created. (b) Piece is brought out of the capsular fornix into the Central Safe Zone. (c) Chopper is brought from the periphery to centre to split as in a peripheral chop.

Intubation DCR

Anita Sethi, MD, FRCS, **Harbansh Lal,** MS**, Piyush Kapur,** DNB, MNAMS

An Intubation DCR is the procedure of DCR followed by insertion of a stent in the form of lacrimal intubation set. The function of the stent being to maintain the patency of the fistula created.

Indications

Int DCR maybe indicate in the following conditions

1. **Common Canalicular block**: The CC block is opened up during the surgery and may require peri-canalicular dissection. The stent is then passed to prevent re-stenosis. This may also be tried in Bi-canalicular blocks if the bock is distal(8mm)
2. **Failed DCR**: In these cases, usually the sac anatomy is distorted and there is more chance of granulation tissue blocking the opening created. The stent helps to maintain patency.
3. **Endonasal DCR**: Some surgeons prefer to use a stent to increase the success rates.
4. **Canalicular Laser DCR**: Since the opening in these cases is not very large, a stent may be preferred to ensure patency.

Applied Anatomy

The normal anatomy of the lacrimal passages, along with some variations needs to be kept in mind. The punctum which is usually 0.5mm needs to be dilated during surgery and both upppr and lower punta must be present as the available intubations sets are bicanalicular. The direction of the canaliculi, 2mm downwards and 8mm medially should be followed as the probes are passed in that direction.

Pre-op syringing and probing

Syringing: This must be carried out before the surgery to confirm the diagnosis and determine whether the intubation will be successful. In case of a CC block, there will be immediate regugitation of clear fluid from the opposite punctum. In failed DCR too there may be a similar finding or regurgitation with a few flakes of mucopus from the opposite punctum. This indicates a block at or just distal to the CC and in these cases too it is advisable to pass a stent.

Probing: In cases of CC block, there will be a soft stop on probing.In a failed DCR, there may be a hard or a soft stop. The length of the probe passed should be measured by holding with a forceps and measuring the part that was passed. A length of 8-10mm indicates that a DCR with intubation is possible. Length < 8mmindicates a proximal canalicular block in which case the procedure has a lower success rate.

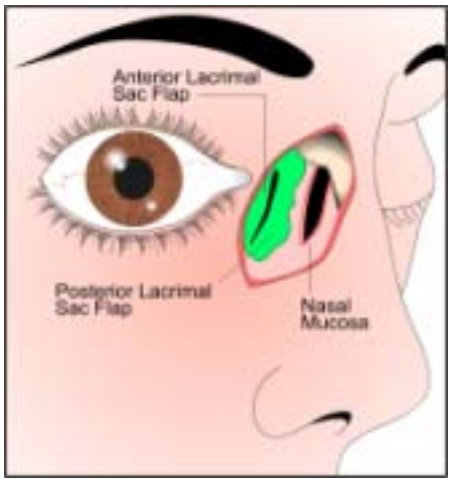


Fig.1: Diagram showing the cut open sac and nasal mucosa.

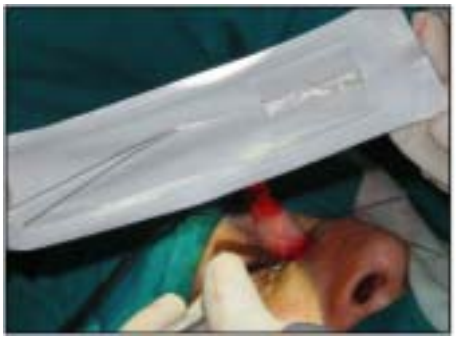


Fig.2: Lacrimal Intubation set consisting of two 17mm probes with attached silicon tubings.

Lacrimal intubation set *(Fig.1)*

The lacrimal intubation set consists of a silicon tubing attached to two probes, one for each canaliculus. The probes are available in 2 sizes, 11mm and 17mm. They may be straight or angulated .

Technique

The initial steps are similar to those of a conventional DCR. Incision is usually made 3mm from the canthus. The sac is exposed all along the length. A probe is passed to tent the sac. The sac is incised with an H shaped incision so that it opens like a book. In a CC block the probe will not be visible, there will be septa or a membrane covering the probe which will need to be cut. The membrane is dissected till the probe comes through. The probe will need to be passed through the upper canaliculus too till it comes into the open sac (Fig.2). In some case, peri-canalicular dissection may be required to enable the probe to come through. The bony opening is then created and the flaps of nasal mucosa formed. The posterior flaps, if created are now sutured with 6 0 vicryl. It is not essential to create posterior flaps

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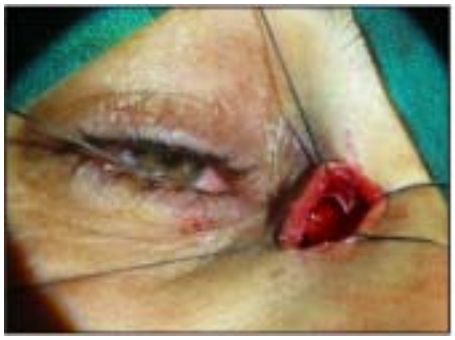


Fig.3: The probe is visible in the sac which has been opened.

Fig.4a: The probe is passed through the punctum & brought out into the sac.

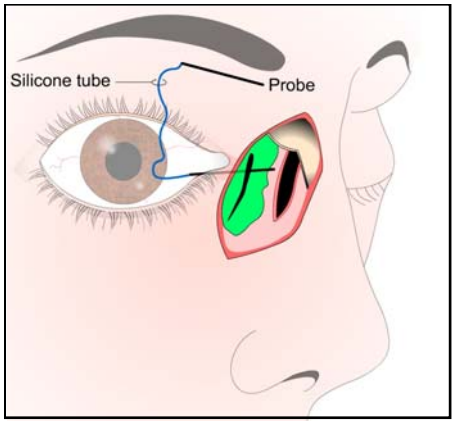


Fig.4b: Diagram showing probes being brought into sac.

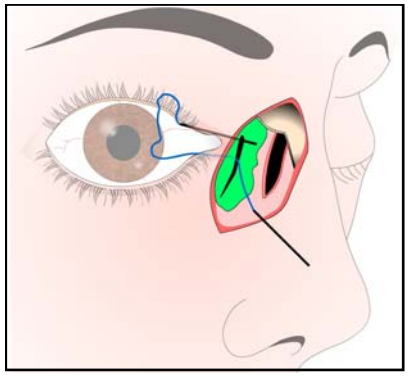


Fig.5(a&b): Tubings in sac.

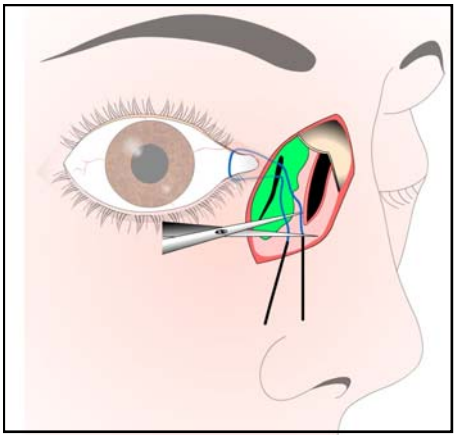


Fig.6: The probes are cut

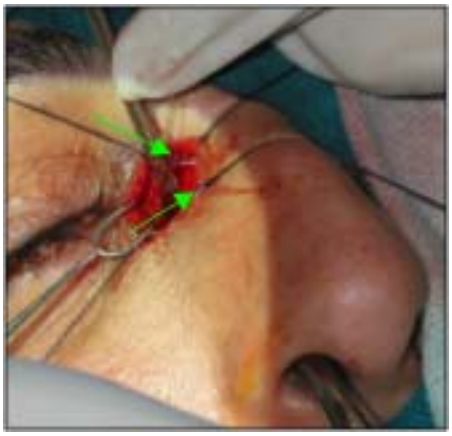
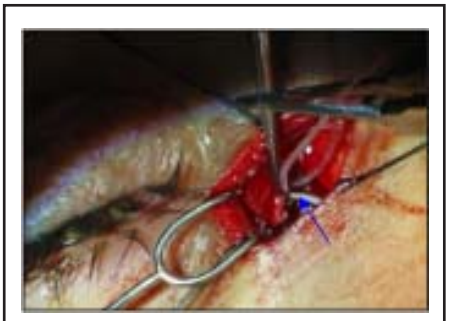


Fig.7: The hemostat is passed though the nose under the nasal flap.



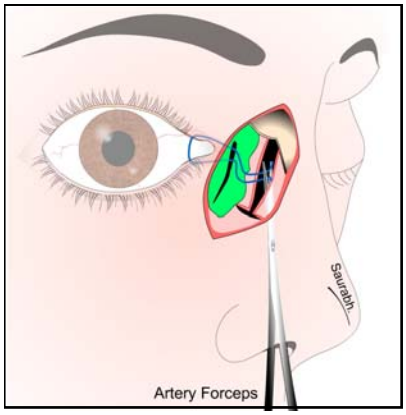


Fig.8(a&b): The silicon tubings are fed into the hemostat and brought out through the nose.

but if not made, then the posterior flap of the sac should be excised to prevent granulation tissue formation.

Passing the intubation set

The probes are lubricated with ointment (Fig.3). The probe with the attached silicon tubing is passed through the punctum and brought out into the cut sac(Fig.4a&b). The second probe is similarly passed and both the silicon tubings are brought out into the sac (Fig.5a&b). The probes are then cut (Fig.6). A curved hemostat is passed through the nose into the sac (Fig.7) and the silicon tubings are fed (Fig.8a&b) (Fig.9) so that they are both brought out through the nose. The ends are tied and left in the vestibule. While tying the knot, care is taken that the loop lies snugly in the medial canthal area (Fig.10) keeping in mind that too loose a loop can rub against the cornea and too tight can cause cheese-wiring of the punctum and canaliculus.

In cases of endonasal or Canalicular Laser DCR where the sac is not opened, the opening in the sac is visualized by the endoscope and then the intubation done.

The anterior flaps are then sutured over the tubing. The wound is closed in two layers.

Post-operative treatment

The post-operative regime is the same as that for a conventional DCR, that is antibiotic and anti-inflammatory. Syringing may be done with a fine cannula along the side of silicon tube. The tube is removed after 3-6 months by cutting the loop at the medial canthus and pulling it out from the nose.

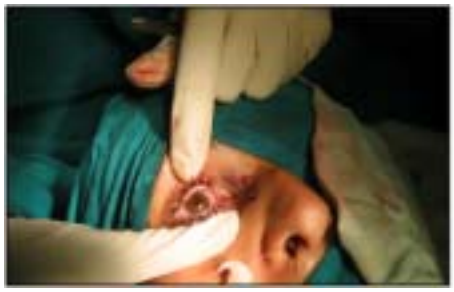


Fig.9: The silicon tubings are knotted together and ends left in vestibule.

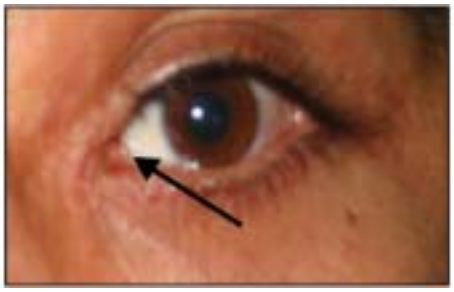


Fig.10: The loop lies in the medial canthal area.

Fig.11: Granulation tissue around lower punctum and tube causing epiphora.

Complications

1. While passing the probe into the sac, the silicon tubing may detach if too much force is used. It is important to lubricate the probe and follow the direction of the canaliculus to prevent this. In case it detaches, the tubing will have to be re-threaded onto the probe
2. Nasal mucosa injury: Attempting to pass the probe and tubing directly through the bony opening into the nose may injure the nasal mucosa unless it is visualized by an endoscope. Feeding the tubing into a curved hemostat is less traumatic.
3. Spontaneous extrusion of the tubing : Sometimes the tubing may extrude spontaneously especially if the patient sneezes repeatedly. The patient is prescribed decongestant nasal drops and anti-histaminics to prevent this.
4. Granulation around the tubing (Fig.11): This may be seen in children or in trauma cases. This is managed with anti-inflammatory drugs and if it persists may necessitate early removal of the tubing.
5. Corneal abrasion: If the loop in the eye is too long, it may rub against the cornea and cause an abrasion. The nasal end can be pulled to reduce the loop in the eye.

Cheese-wiring of the punctum and canaliculus: If the tubing is too tight it may cause cheese-wiring. The loop will then need to be loosened by pulling it gently.

What is New : Cyclodestructive Procedures

Col. M. Bhadauria MS, **Col. R.N. Kothari** MS, **Col. V. Baijal** MS, **Col. S.K. Anand** MS, **Brig. D.P. Vats** MS

Introduction & Evolution

Destruction of ciliary body has been a treatment in use for management of refractory glaucomas for past 70 years. This strategy was reserved for painful blind eyes and modalities were cyclodiathermy or cyclocryotherapy before introduction of lasers in this field. There has been a change in the indications of cycloablation in recent times due to availability of lasers and their modes of delivery. The procedures are being performed increasingly on seeing eyes with rewarding results. Weve[1] reported use of cyclodiathermy in 1933 and Vogt[2] reported penetrating diathermy in 1936 for control of intraocular pressure. Other energy sources used were therapeutic ultrasound, cycloelectrolysis and beta radiation. In 1950, Bietti[3] demonstrated that by freezing the ciliary body, the intraocular pressure could be reduced and since then this procedure has been in use for management of refractory glaucomas and still holds a place in cycloablation armamentarium. In 1961, Weeker and co-workers used Xenon arc laser to photocoagulate ciliary body but collateral tissue damage and complications were unacceptable. Beckman et al subsequently reported the transscleral use of the ruby laser and Nd: YAG laser. Ruby laser did not gain popularity due to non availability. Nd: YAG laser was later modified by Fankhauser and co-workers[4] into thermal mode for application on ciliary body. The transscleral cyclophotocoagulation (CPC) technique approved by the FDA in 1994 uses *810-nm continuous-wave diode laser*.

Indications

Cyclodestruction is definitely indicated in refractory glaucomas following failed trabeculectomy and tube shunt procedures with minimal useful vision, without visual potential and elevated intraocular pressure causing intractable pain. It can also be done in eyes with visual potential where medical condition precludes invasive surgery or patient refuses more aggressive surgery (i.e, Filter or tube) and in eyes with acute onset of neovascular glaucoma. Cyclodestruction can successfully control IOP in silicone oil-filled eyes where medical treatment fails and also following penetrating keratoplasty. As a primary procedure it's use is controversial but can be definitely justified in acute onset neovascular glaucoma and in eyes where conjunctiva is not available for any kind of filtering procedure. This modality has been used as primary procedure in HAITIAN GLAUCOMA PROJECT. The workers found it difficult to draw conclusion due to short follow-up and their recommendations are that if a patient has no access to any kind of glaucoma management and visual acuity less than 20/60, limited cycloablation may be considered.

Cyclocryotherapy

Considering the degree of discomfort and postoperative inflammation associated with Cyclocryotherapy the procedure should be reserved for the eyes that have visual acuity of 6/60 or less and laser options are not available. Equipment used for Cyclocryotherapy is the Cryo unit (Frigitronics) with N_2O as the method of freezing with cryoprobe of 4 mm tip that achieves temperature of -80°C. The procedure is performed under peribulbar anaesthesia. The probe is placed perpendicular to the globe 1mm away from the border of the limbus. Moderate pressure is applied to achieve freezing. It is usual to treat 180 degree circumference at a time (6 spots) but lesser (3 spots) can also be treated in eyes that are at higher risk for developing phthisis bulbi like neovascular [5] and uveitic glaucoma. Once the probe is frozen to the globe eye is rotated away from the lid, ice ball extends on to cornea but freezing should be continued till 60 seconds. Similar procedure is repeated till the desired extent of ciliary body has been treated. Post operatively most of the patients have moderate to severe pain and inflammation so adequate analgesia, strong cycloplegia and steroids are started immediately after the effect of peribulbar anaesthesia starts wearing off. Antiglaucoma medications are continued for at least four weeks and IOP and inflammation is monitored at least weekly for first 4 weeks and dosage is adjusted accordingly. Success of therapy is determined by the technique and the underlying diagnosis. Reported success has ranged from 34% in neovascular glaucoma to 90 % in aphakic and pseudophakic eyes. Retreatment is done with one quadrant overlap and one quadrant must be preserved to maintain the integrity of the eye ball. In high risk cases esp seeing eyes one quadrant is treated in first sitting and depending upon the response further treatment is titrated.

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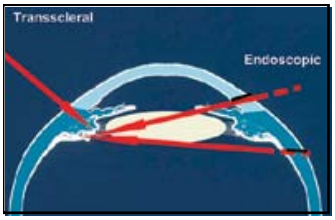
Laser Cycloablation

The advent of lasers in field of cycloablation has given it a new dimension for the seeing eyes of the patients whose glaucoma could not be controlled with prevalent anti glaucoma strategies. Lasers are a more precise and focussed source of energy hence cause less collateral damage and lesser complications than Cyclocryotherapy. They are non-invasive and easy to learn due to short learning curve and ease of application. Many types of lasers have been used over last two decades. This experience has evolved in to relatively safer protocols. The laser procedures can be performed through trans-scleral, trans-pupillary or endoscopic routes on day care basis. Transscleral cycloablation by lasers is done by contact and noncontact methods and both modes are available in Nd:YAG laser but some how this laser did not gain popularity in India.



Transpupillary Cyclophotocoagulation

Ciliary processes are not usually visible through the dilated pupil but can be seen in aphakia, traumatic aniridia or in young patients of neovascular glaucoma due to forward tenting of iris in a widely dilated pupil. Visible ciliary processes are ablated by argon or 532 frequency doubled laser using 3 mirror gonioscopy lens or a specially designed lens with an inbuilt indenter to reduce intraocular pressure. The laser settings used are between 700 to 1000 mw for 0, 2 sec and 100 micron spot size. End point of treatment is taken as a crater in the ciliary process and not the blanching. Entire visible portion of ciliary process is coagulated and about 180 degree treatment is done at a time. This treatment modality was first reported by Lee[6] but has not been found to be very effective by Shield et al. This procedure is used as yet another option in glaucoma management.

Endoscopic Cyclophotocoagulation

Earlier this technique was done in combination with pars plana vitrectomy for control of glaucoma in retinal disorders. Chen and colleagues report the results of 68 eyes treated for 180° to 360° and found that 61 eyes had an IOP of less than 22 mmHg at 12.9 months' mean follow-up.[7] Now new photocoagulation system is available specially for endophotocoagulation of ciliary processes and can be used by anterior segment surgeon. The technique can be combined with phacoemulsification in patients of combined cataract and glaucoma. The advantages of this technique are that it is very precise, controlled and safe without collateral damage. It has been found to be really effective in post penetrating keratoplasty glaucoma and in eyes with visual potential. The disadvantages are the cost of the machine, all the risks of incisional surgery and cyclophotocoaglation.

Noncontact Nd:Yag Laser Transscleral Cyclophotocoagulation-

Noncontact Nd:YAG laser transscleral Cyclophotocoagulation is performed with a slit lamp delivery system in thermal mode with a maximum offset (Offset 9). The suggested settings are 8J, duration 20ms, offset of 9 and total of 32 applications avoiding long posterior ciliary arteries. The reported success has been 45-86 %. Simmons and Shield [8] fashioned a lens for the procedure that was transparent from limbus to 3 mm on to sclera and corneal area was kept opaque to prevent laser damage to other ocular tissues. Markings were made to indicate the target area. This lens was useful in keeping the eye stable and open.

Contact Laser Transscleral Cyclophotocoagulation-

Brancato et al[9] were the first workers to evaluate the success of ciliary body destruction by contact delivery system. Contact **Nd:YAG** laser Cyclophotocoagulation is performed transsclerally with continuous wave Nd:YAG laser that is connected to a fibre optic cable and a synthetic sapphire tipped convex probe with 2.2 mm diameter. The suggested settings are 7 W and 0.7 s and a total of 32 to 40 shots are placed 1 mm behind the limbus.

Diode laser is FDA approved laser for transscleral photocoagulation and is more popular world wide due to low cost, easy availability, portability and no cost maintenance. It's utility in retinal disorders for grid laser and pan retinal photocoagulation and in anterior segment for laser Trabeculoplasty and suturolysis makes it a more desirable laser than Nd:YAG laser. It has been shown in numerous published studies[10,11] that it lowers IOP and allows patients to retain vision in about two-thirds of eyes with intermediate follow-up but requires multiple re-treatments. 810 nm semiconductor diode lasers have very good scleral penetration. Laser machine is light weight and air cooled. It is fitted with fibre optic cable and cycloprobe/ G probe. The laser penetrates through the sclera and is absorbed by the melanin and effect is seen in form of pars plicata damage. Total destruction of ciliary processes, pigment clumping, and loss of vessels is seen in the treated area. However haphazardly regenerated non-pigmented epithelium is seen over damaged pars plicata.

The procedure is performed under peribulbar anaesthesia and is our preferred procedure for cyclodestruction . Just like cyclo Cryopexy lids are separated and the contact probe is firmly applied to sclera 1 mm behind the limbus and G probe has inbuilt measurements. Three quadrants or 270 degree circumference is treated in one sitting sparing 3 and 9 o clock position to prevent damage to long ciliary vessels. There are no fixed laser parameters or dose effect algorithm available yet. The general guideline is to start with 1750

mw for 2 secs and listen for the pop sound. Increase laser settings by 250 increment till the pop is heard and then reduce the settings by 250 from the audible pop sound level and complete the entire procedure. Usual settings in Indian eyes range between 1500 to 2000 mw. Post operative treatment includes analgesia, steroids and cycloplegics.

Antiglaucoma medications are continued as before and are adjusted gradually based on post operative IOP. The patients are subjected to slit lamp examination for assessment of inflammation or other complications and IOP is recorded on each follow up visit. Our follow up protocol includes visits on day 1,7,28 and then patients are followed on monthly basis. Patient requiring retreatment needs to go through the same protocol again but retreatment is not done in less than 6 weeks time.

Complications

Complications can be mild in form of anterior uveitis, conjunctival injection pain, corneal edema and intraocular pressure spikes or moderate to severe in form of hyphema, vit hemorrhage, cataract ,anterior segment ischemia with subsequent phthisis or sympathetic ophthalmia.[12] Choroidal detachment with flat AC[13] and malignant glaucoma[14] have also been reported.

Conclusion

Cyclodestructive surgeries have reincarnated in last 2 decades due to availability of numerous safe lasers. Variable modes of delivery like transscleral route and endoscopic delivery have further enhanced the ease of application and precision. The transscleral CPC has gained wide acceptance due to relative ease and overall effectiveness. The laser CPC has been found to be as effective as Cyclocryotherapy but with lesser complications and post operative pain. [15] 810 nm semiconductor diode laser has been found to as safe and effective as Nd:YAG transscleral CPC laser [16] and is being used more due to it's low cost and other utilities in ophthalmology. The procedures that were reserved for painful blind eyes are now being used on seeing eyes effectively for control of intractable IOP rise. Utility of Cyclophotocoagulation as primary procedure is still debatable and needs a lot more research and reassurance. We do need excellent studies to titrate accurate and best parameters to use to reduce complications, control pressure, and preserve sight.

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Congenital Ptosis Surgery

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Congenital Ptosis is one of the most frequent conditions managed by an oculoplastic surgeon in the country. Management of the condition requires a thorough understanding of the surgical anatomy and a meticulous surgical technique based on a proper evaluation.

It is advisable to wait till 3-4 years of age for surgical correction when the tissues are mature enough to withstand the surgical trauma and a better assessment and postoperative care is possible due to improved patient co-operation. There should be no delay in surgical management in cases of severe ptosis where pupil is obstructed and the possibility of the development of amblyopia is high. In these cases a temporary procedure may be opted for early on, followed by a definitive surgery later.

Surgical Approach

The surgical approach is based on whether the

1. Ptosis is unilateral or bilateral
2. Severity of Ptosis
3. Levator action
4. Presence or absence of abnormal ocular motility, jaw winking phenomen on or blepharophimosis syndrome

The choice of surgical procedure is as is follows:

- Fasanella Servat Operation
  a) Mild ptosis (<2mm or less)
  b) Levator action >10mm
  c) Well defined lid fold-no excess skin
- Levator resection
  a) Mild/moderate/severe ptosis
  b) Levator action ≥ 4mm
- Brow suspension ptosis repair
  a) Severe ptosis
  b) Levator action <4mm
  c) Jaw winking ptosis or blepharophimosis syndrome

Bilateral Ptosis

In cases of bilateral ptosis, sumiltaneous bilateral surgery is preferred to ensure a similar surgical intervention in the two eyes. However in cases where gross asymmetry exists between the two eyes, the eye with a greater ptosis is operated first and the other eye is operated after 6-8 weeks when the final correction of the operated eye can be assessed.

Surgical Techniques

Modified Fasanella Servat surgery

It is the excision of tarsoconjunctiva, Muller's and levator. We use a modified technique that avoids the use of haemostat or a special clamp.

Xylocaine with adrenaline is used for local anaesthesia in adults but general anesthesia is necessary for children.

Surgical steps (*Fig 1a-e*)

Eyelid is everted and the tarsal plate is exposed. Three sutures are passed close to the folded superior margin of the tarsal plate at the junction of middle, lateral and medial one third of the lid. Three corresponding sutures are placed close to the everted lid margin starting from conjunctival aspect near the superior fornix in positions corresponding to the first 3 sutures. Proposed incision is marked on the tarsal plate such that a uniform piece of tarsus, decreasing gradually towards the periphery is excised. This is necessary to avoid a central peaking. A groove is made on the marked line of incision and the incision is completed with a scissors. The first set of sutures help in lifting the tarsal plate for excision. The second set aids suturing by lifting and supporting the conjunctival and tarsal edges during suturing. The tarsal plate not more than 3 mm in width is excised.

5-0 plain catgut is used for continuous suturing and the knot is buried within the wound. Postoperatively the patients are kept on antibiotics and antiinflammatory agents and cornea is observed for any sign of abrasion.

The preoperative and post operative photographs of a

Fig.1(a): Lid everted and sutures passed through the superior margin of the tarsal plate.

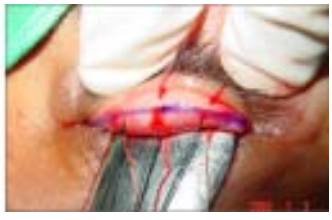

Fig.1(b): Marking along the proposed line of excision. Corresponding sutures passed close to everted lid margin.

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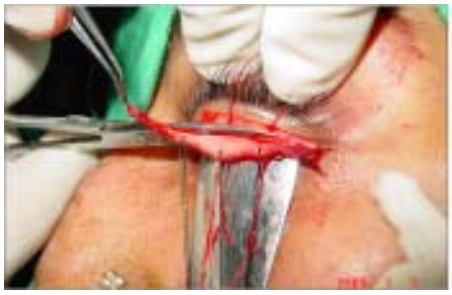


Fig.1(c): A scissors in used to cut along the groove created by a knife

(d): Excision is completed. Conjunctival and tarsal edges are can be raised with the aid of the traction sutures, to assist in suturing.

Fig.1(e): Continuous sutures with 5'0 plain catgut, with knots buried in the wound.

Fig. 2(a)&(b): Preoperative and Postoperative photograph following fasanallea servat primary gaze.

Fig.2(c)&(d): Preoperative and Post operative photograph in upgaze.

Fig.2(e)&(f): Preoperative and post operative photograph in downgaze.

patient taken up for the fasanella servat surgery in the primary, up and down gaze **(***Fig. 2a-2f***)** are shown.

Levator resection

This is the most commonly practiced surgery for ptosis correction. It may be performed by skin or conjunctival route but the former is preferred by most surgeons because of its more universal applicability, a good titration / assessment on the table and creates a good lid fold.

2% Xylocaine with adrenaline is locally infiltrated. The injection is also used in cases being operated in general anaesthesia to achieve haem-ostasis.

Surgical steps

The proposed lid crease is marked to match the normal eye considering the margin crease distance of the normal eye as well as the amount of skin show measured in the primary position. In bilateral cases highest forming crease is used which is usually at the superior border of the tarsus or a standard measurements can be used.

Three 4-0 silk sutures are passed near the lid margin to provide traction. A lid spatula is placed under the lid and incision through the skin and orbicularis is made along the crease marking *(Fig 3a)*. The inferior skin and orbicularis are dissected away from the tarsal plate *(Fig 3b)*. The upper edge is separated from the orbital septum *(Fig 3c)*. The orbital septum is cut completely across exposing the preaponeurotic fat *(Fig.3d)*. Fat is retracted posteriorly exposing the whole of the tendinous aponeurosis *(Fig.3e)*. Three partial thickness traction 4-0 silk suture are passed through the distal end of the aponeurosis. The fibers of the aponeurosis are cut from their insertion in the inferior half of the anterior surface of the tarsus *(Fig.3f)*. The levator is freed from the adjoining structures. The lateral and the medial horn are cut *(Fig.3g)* when ever a large resection is planned. The direction of the cut should be vertical to avoid damage to the lacrimal gland laterally and the pulley of superior oblique muscle medially. Care should be taken that Whitnalls ligament is not damaged which is visualized as a whitish fascial condensation running across the junction of the muscular and aponeurotic part of the levator about 15 mm from the insertion. A double armed 5-0 vicryl is passed through the centre of the tarsal plate by a parsal thickness bite. It is than passed through the levator

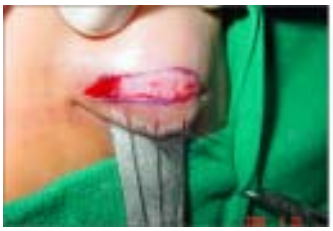


Fig.3(a): Matched lid crease is marked. Skin and orbicularis are incised.

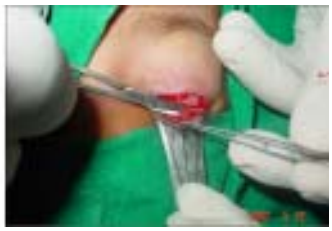


Fig.3(b): Skin and Orbicularis dissected from the tarsal plate.

aponeurosis at height judged by the preoperative evaluation. Intraoperative assessment is made (*Fig. 3h*). Two more double armed vicryl 5 - 0 sutures are passed through the tarsus about 2 mm from the upper border in the center and at the junction of central third with the medial and lateral thirds (*Fig.3i*). These sutures are then placed in the levator and intraoperative assessment made. The lid level and contours are evaluated. The eyelid is left at the position determined preoperatively based on the levator action.

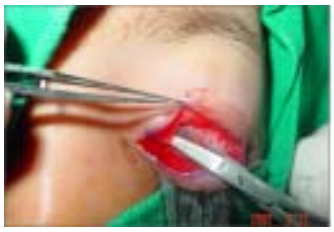


Fig.3 (c): Dissection done superiorly from the orbital septum.

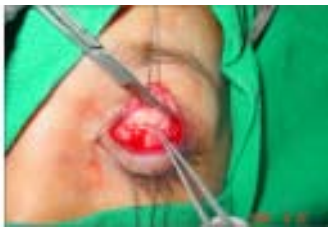


Fig.3(d): Orbital septum cut exposing the preaponeurotic fat.

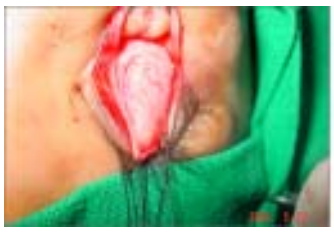


Fig.3(e): Levator is freed from the adjoining structures.

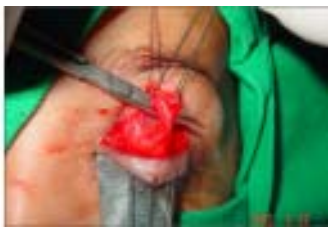


Fig.3(f): Lateral horn being cut.

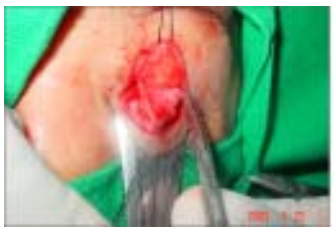


Fig.3(g): Levator fibres of aponeurosis being cut from its insertion in the inferior half of the anterior surface of tarsus.

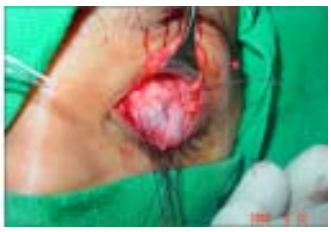


Fig.3(h): Double armed 5'0 vicryl sutures passed through the tarsus and entrap assessment.

Fig.3(i): Three double aimed suture. Suture passed through the levator and tightened.

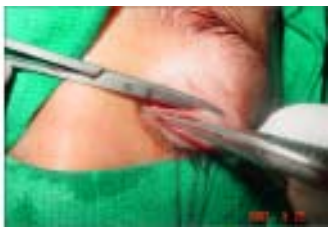


Fig.3(j): Strip of excess skin removed.

Fig.3(k): Skin sutures applied.

Excess levator is excised. If required a strip of skin is removed from above the lid crease (*Fig.3j*). A piece of orbicularis may be excised inferior to the lid crease to debulk the lid. Four to five lid fold forming sutures are placed. The sutures pass through skin edges taking a bite through the cut edge of levator (*Fig.3k*). An inverse frost 6-0 silk suture is passed through the lower lid margin over a bolster.

We use a modification of Berke's criteria based on our postoperative observations.

The position of the lid aimed at during the table assessment should be as follows:

Levator Action	Recommended Placement of lid
2-4mm	1mm above the limbus (when levator resection is chosen to be undertaken)
5-7mm	1mm below the limbus
8mm or more	2mm below the limbus

Patients are prescribed oral antibiotics and antiinflammatory agents.

Preoperative and post-operative Photographs are demonstrated in *Fig . 4a-4f.*

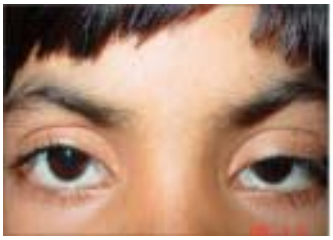


Fig.4(a): Pre operative simple ptosis

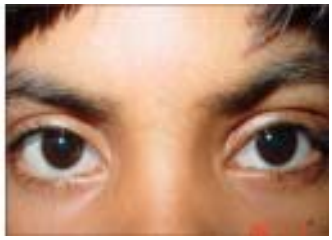
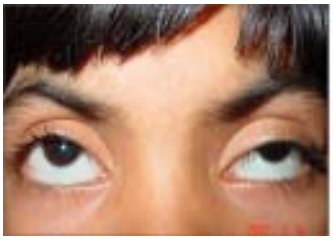


Fig.4(b): Post operative following levator resection. Note the symmetrical lid crease and contour.



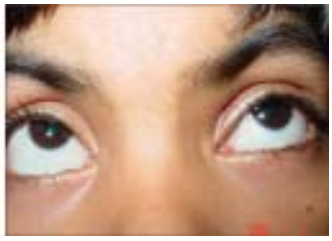


Fig.4(c&d): Preoperative and Post operative: Upward gaze

Fig.4(e&f): preoperative and Post operative: Downward gaze

Brow suspension repair

This surgery is the procedure of choice in simple congenital ptosis with a poor levator action. A number of materials like non absorbable sutures, extended Poly Tetra Flouro Ethylene (ePTFE), muscle strips, banked or fresh fascia lata strips have been used for suspension. We prefer Poly tetra fluoro ethylene (ePTFE) sutures for temporary thread sling procedure and fresh autogenous fascia lata for permanent brow suspension.

Temporary Sling

Thread sling is carried out in very young children with severe ptosis where prevention of amblyopia and uncovering the pupil is the main aim. We use CV 0 ePTFE (Goretex) sutures by modified Crawford technique for brow suspension. The suture sling procedures have a relatively higher recurrence rate of or may show formation of suture granuloma. Definitive surgery may be performed at a later date when a fascia lata sling is carried out.

Fascia Lata Sling

It is considered in children above four years of age having severe congenital simple ptosis with poor levator action We prefer fresh autogenous fascia for suspension. Even in cases of unilateral severe ptosis a bilateral procedure is preferred because a unilateral surgery causes marked asymmetry in down gaze. Results of bilateral surgery are more acceptable.

All cases are done under general anaesthesia. Infiltration with 2% xylocaine and adrenaline is done in the region of the proposed incision in the thigh and the eyelid and the eyebrow region.

Harvesting of fascia lata

A line joining the lateral condyle of femur to the anterior superior iliac spine is marked. A lower thigh incision about 2 inches above the lateral condyle on the marked line in the site of incision. However due to lower thigh scar we often use the upper thigh incision for past 2 yrs. The incision is about one inch in size. The skin incision is deepened through the fat till the glistening fascia is visible. The fascia is then cleared of the overlying tissue for a length of four inches upwards from the incision. A 12 mm incision is given at the lower end of the exposed fascia lata. Dissection is carried out beneath the fascia lata separating it from the underlying vastus lateralis muscle along the whole length of the previous dissection. Two linear incisions are given 12 mm apart on the fascia along the length of dissection using a long scissors. The superior end of the fascia is made free by making horizontal cut using a long bladed scissors while the assistants retract the skin and the subcutaneous tissue. The subcutaneous tissue is closed using 4 - O chromic catgut and the skin is closed using 4 - O silk sutures. We

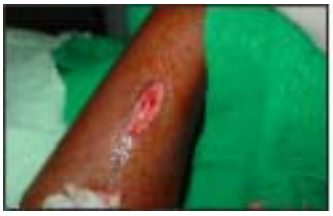


Fig. 5(a): Incision marked. 2 inch above the lateral condyle.

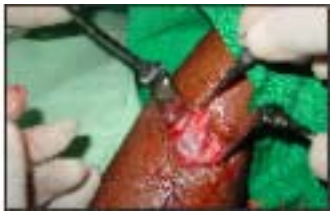


Fig. 5(b): Fascia lata is exposed.

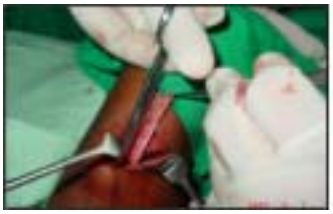


Fig. 5(c): 12 mm fascia lata strip being removed.

Fig. 5(d): Subctaneous tissue closed with 4'0 catgut.

Fig. 5(e): Trimming of the strip, fat removed.

Fig. 5(f): Strip placed on a wooden board and divided into 4 strips of 3mm width each.

now have an autologous fascia lata four inches long and 12mm wide. It is kept in a bowl containing Ringer lactate and 1 cc of gentamycin. (*Fig 5a-d*).

The fat is trimmed from the fascia lata strip (*Fig 5e*).

The fascia lata strip is kept on a wooden board, stretched and fixed. It is divided into four pieces each of about 3 mm width by a scalpel blade. (*Fig.5f*)

Fascia Lata sling suspension

Three traction sutures are passed along the lid border. Four incisions are made on the eyelid 2-4mm above the margin. The placement of these determines the position of lid fold. The two central incisions are on either side of the center of the lid while the other two are at the junction of middle and lateral thirds and middle and medial thirds of the lid. A lid crease incision is also given.

The eyebrow incisions are marked next. They are made at a line perpendicular to the intersection of two lateral eyelid incisions and the two medial eyelid incisions while the eyelid is placed in the desired normal position. A third incision is made in the middle of the first two but 4-6 mm higher than the first two incisions.

The eyelid incisions are made down to the tarsus and the brow incisions are made upto the Frontalis. Blunt dissection is carried out to make pockets for the fascial

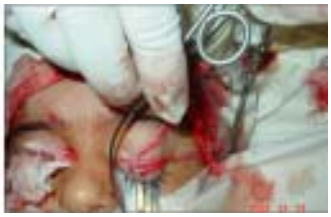



Fig.6(a,b): Fascia lata needle is passed through submuscular plane on medial side and strip pulled.


Fig.6(c): Needle passed from brow incision to medial lid incision

Fig.6(d): Fascial strips pulled from both medial & lateral brow incisions


Fig.6(e): Knot tightened and secured to get proper lid position.

Fig.6(f): One end of each fascial strip passed through central brow incision and tightened.

knots. The two ends of a strip are then passed from the outer eyelid incisions to the outer eyebrow incision using a Wright's fascia lata needle. The needle is passed in the submuscular plain from the lateral brow incision to emerge from the lateral incision in the lid. The fascia is threaded through the eye of the needle and is pulled through. The Wright's needle is again passed from the lateral incision to the second eyelid incision threaded with fascia and drawn up. The procedure is repeated on the medial side (*Fig. 6a,b*). The fascial strips are pulled up and a single tie is made so as to place the eyelid margins as high as possible without lifting the eyelid from the globe (*Fig.6c,d*). After a single tie the position and contour of the eyelid is assessed. Required adjustments are made. Presence of a good lid crease is ensured at this stage. The knots are secured using 5-0 vicryl. A second tie is made and secured (*Fig.6e*).

One end of fascial strip from each brow incision is pulled through the central brow incision. Knots are tied and secured (*Fig.6f*). All the knots are buried in the pockets prepared for them. The excess of skin created by shortening of the posterior lamina is judged and excised by removing a spindle of skin from above the eyelid crease. Eyelid incisions require no closure. The brow incisions are closed with 5-0 silk and the eyelid crease incision by 6-0 silk.

Patients are prescribed oral antibiotics and antiinflammatory agents. An appropriate selection and


Fig.7(a&b): preoperative and Post operative:primary gaze following fascia lata sling surgery


Fig.7(c&d): preoperative and Post operative: upward gaze


Fig.7(e&f): preoperative and Post operative: Downward gaze


Fig.7(g&h): preoperative and Post operative: on lid showing minimal lagophthalmos.

meticulous execution of surgical technique hold the key to obtain excellent functional and cosmetic results in patients with ptosis.

The preoperative and postoperative photographs of patient with severe ptosis corrected by bilateral fascia lata sling are shown in *Fig. 7a-7h*.

Management of Complicated Ptosis

Ptosis with oculomotor abnormalities

It is necessary to correct the ocular motility before correction of ptosis because the restriction of superior rectus and accompanying hypotropia make the assessment of ptosis difficult. Secondly, the hypotropic eye with poor Bell's penomenon is extremely vulnerable to exposure keratopathy due to postoperative lagophthalmos.

Congenital ptosis with Superior rectus weakness

- Superior rectus weakness is a common association as both muscles develop from the same myotome. The

hypotropia is corrected by an inferior rectus recession at times combined with superior rectus resection as the first procedure. Ptosis correction is then carried out using the procedure indicated by evaluation.

Ptosis associated with double elevator palsy

- Knapps procedure may be done for ptosis associated with double elevator palsy. The lateral and medial rectus tendons are transposed to the sides of superior rectus insertion. This does not cause significant limitation of adduction or abduction. Ptosis is corrected 3 months later.

Blepharophimosis syndrome

The blepharophimosis syndrome comprises of ptosis, epicanthus inversus,telecanthus, horizontal shortening of palpebral aperture, flattened supraorbital ridges, arching of the eyebrow and lateral ectropion of the lower eyelid.

- Mustarde's double "Z" plasty or Y-V plasty with transnasal wiring is done as a primary procedure. This gives a good surgical result both in terms of correction of telecanthus as well as deep placement of the medial canthus. The results are long lasting.
- Brow suspension is carried out 6 months after the first procedure for correction of ptosis.

Double Z plasty or Y to V plasty with transnasal wiring

Lateral canthotomy and canthoplasty may be carried out before the skin incision is made.

The markings for double Z plasty are made as shown in Fig 8. The first mark is made just medial to the medial canthus (A). The proposed canthal site (B) is marked such that intermedial canthal distance is half that of interpupillary distance. The two marks are joined. All the other lines drawn are 2 mm smaller than the line AB. Two lines are drawn from a parallel to upper and lower lid margins. From the centre point of AB (C), a line is drawn medially at 60° both above and below (CD). Another line is drawn outwards at an angle of 45° from the point of D (DE).

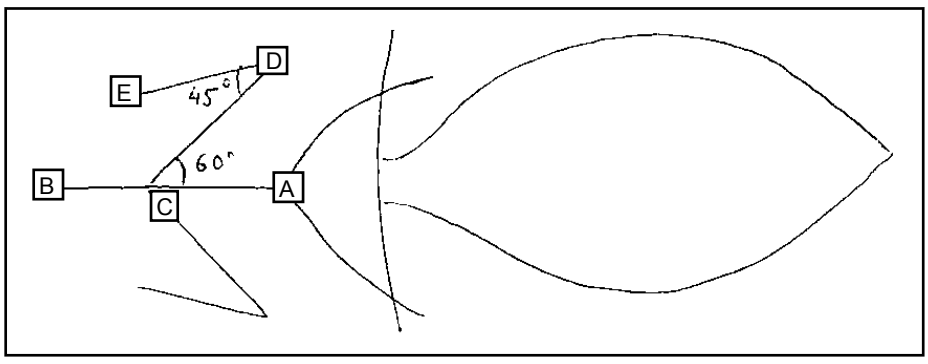


Fig.8: Z-Plasty

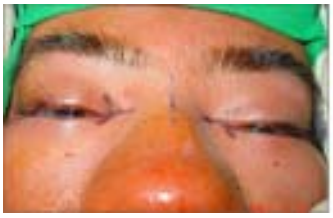


Fig. 9(a): Markings of Y-V plasty

Fig.9(b): Incision given and flap undermined

Fig.10: Transnasal wiring.

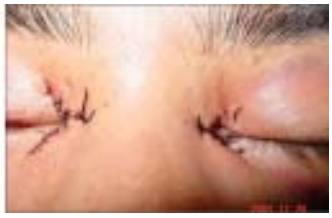


Fig.11: Skin sutured

The markings for Y to V plasty are shown in *Fig. 9a*. The incision are made through the skin down the to the orbicularis. The flaps are undermind. (*Fig.9b*). The site of proposed canthus is cleared of all tissue upto the periosteum and the medial palpebral ligament is exposed. The periosteum is incised medial to the insertion of MPL and is reflected along with the lacrimal sac.

A large bony opening 12-15 mm high and 10-12 mm wide is made as for dacryocystorhinostomy but located more posterior and superior. The edges of the bony opening are smoothened. A similar procedure is performed on the opposite side.

Medial palpebral ligament (MPL) of one side is wired with 24 G stainless steel wire close to its attachment to the tarsus and the two ends of the wire are passed to the opposite side through the bony opening with the aid of an aneurysm needle or a Wright's fascia lata needle. The wire is threaded into the opposite MPL by a similar double bite. The two ends are tightened and a single twist is given to the wires (*Fig.10*). The position of the medial canthus is assessed from the front, above and the sides. Once the desired position is obtained the wire is twisted several times and cut. The ends of the wire are pushed into the bony opening. After achieving the hemostasis the incision is closed in several layers. The skin flaps may need to be trimmed before they are tranposed and sutured with 6-0 silk (*Fig.11*).

Lateral Canthoplasty

The lateral canthus is crushed by a straight haemostat for a few seconds. A lateral canthotomy is performed. The bulbar conjunctiva at the lateral

Fig.12 (a): A patient with Blepharophimosis syndrome

Fig.12 (b): Post-operative photograph following Y-V plasty and transnasal wiring

Fig.12 (c): Post-operative photograph following fascia lata strip surgery

canthus is undermined. The apex of the conjunctiva is sutured to the proposed new position of the canthus which is short of the end of the skin incision. The skin edges distal to the new lateral canthus are apposed with 6-0 silk sutures. A similar procedure is repeated on other side.

The bandage is removed after 24 hrs and sutures are removed between 5-7 days.

Stage II

The second stage is performed after 6 months. A bilateral fascia lata sling is performed.

Fig.12a-c show the pre-operative photograph of a patient with Blepharophimosis syndrome after stage 1 & 2 procedure.

Marcus Gunn ptosis

Ptosis associated with lid retraction on opening the jaw or its movement to the opposite side is classical marcus gunn phenomenon. An inverse Marcus Gun phenomenon is also known.

Management depends on the cosmetic significance of the jaw winking. Where jaw winking is not significant the choice of procedure depends on the amount of ptosis and the levator action is carried out as in any case of congenital simple ptosis. A larger levator resection is necessary and under correction is common. In case with significant jaw winking bilateral levator excision with a fascia lata sling surgery is the procedure of choice. (*Fig. 13 a-d*)

Fig.13(a): A patient with severe ptosis with marcus gunn phenomenon.

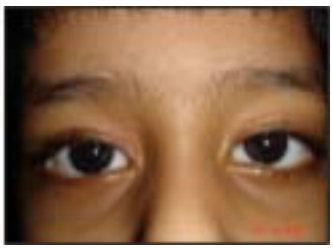


Fig.13(b): Posoperative photo-graph following bilateral levator excision with fascia lata sling surgery.

Fig.13(c): Marcus gunn phenomenon elicited on opening of mouth.

Fig.13(d): Postoperative photograph. Marcus gunn phenomena is eliminated.

Misdirected third nerve ptosis

- In cases of misdirected third nerve ptosis where treatment is indicated levator exersion with bilateral fascia lata sling is the procedure of choice.
- Ptosis associated with third nerve palsy is difficult to manage because of poor Bell's phenomenon. A crutch glass may be prescribed or a conservative sling surgery may be performed.

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Anti-VEGF intravitreal Bevacizumab (Avastin) ® in Ocular Neovascularisation and Macular Edema

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Recent advances in the treatment of age-related macular degeneration (AMD) represent the culmination of over a decade of research into the mechanisms of ocular angiogenesis. Michaelson first suggested that a diffusible "Factor X" from the retina stimulated the retinal and iris neovascularization seen in diabetic retinopathy[1]. However, many years passed before the isolation and identification of such proposed angiogenic factors could be achieved. In 1989, Napoleone Ferrara and colleagues identified a molecule in the conditioned media from bovine pituitary follicular cells that promoted the proliferation of endothelial cells; they called it vascular endothelial growth factor (VEGF)[2]

In 1992, two independent groups demonstrated that hypoxia could upregulate VEGF expression[3,4]. It had long been appreciated that neovascularization of the retina and iris was related to retinal ischemia due to various etiologies, and the property of hypoxia inducibility made VEGF a plausible candidate for the "Factor X," proposed by Michaelson as the mediator of abnormal blood vessel growth in the eye. Evidence from both clinical and animal studies accumulated in the next several years to support the critical role of VEGF in ocular neovascularization.

Measurements of vitreous VEGF levels demonstrated significantly higher VEGF concentrations in patients with active proliferative diabetic retinopathy compared with patients with other retinal disorders not characterized by abnormal blood vessel growth.

The strong supportive evidence from animal studies defined VEGF as an optimal therapeutic target for treatment of ocular diseases in which neovascularization leads to blindness. The need for better treatments for neovascular AMD, leading cause of blindness in individuals over age 55 years, provided the opportunity to develop anti-VEGF agents for clinical use. While regression of retinal neovascularization due to proliferative diabetic retinopathy and ischemic retinal vein occlusion can, in most cases, be successfully achieved with laser photocoagulation, laser treatment for subfoveal CNV due to AMD is suboptimal due to the inevitable destruction of the foveal retina

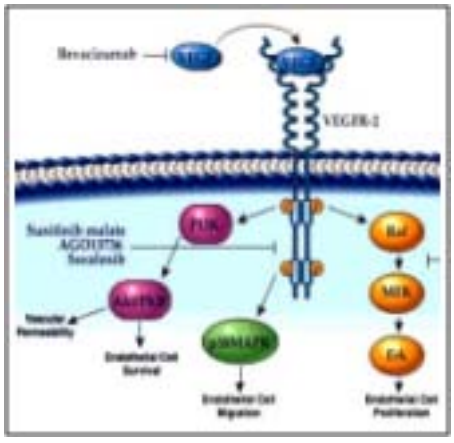

Fig.1: Site of action of Bevacizumab

Pegaptanib [Macugen] emerged as the first antiangiogenic agent with proven efficacy in clinical trials for neovascular AMD. It is a modified 28-base RNA aptamer that selectively binds VEGF165.

The anti VEGF drug which caught the attention of ophthalmologists world over and has opened up new vistas in the management of choroidal neovascular membranes secondary to wet AMD is Bevacizumab. Bevacizumab is a humanized monoclonal antibody that inhibits all active isoforms of vascular endothelial growth factor (VEGF) (Fig. 1) and is approved by the US Food and Drug Administration (FDA) [Feb 2004] for the treatment of metastatic colorectal cancer. It was developed as an intravenous therapy for cancer patients because VEGF is one of the major angiogenic stimuli responsible for neovascularization in tumors.Ranibizumab [Lucentis], a humanized monoclonal antibody fragment against VEGF highly related in structure to Bevacizumab, has also proven efficacious in the treatment of subfoveal CNV due to AMD. A number of studies has shown encouraging results after using Ranibizumab in wet AMD and currently is in the process of review by the US Food and Drug Administration (FDA)

Bevacizumab, which is FDA approved for use in metastatic colorectal cancer came to the attention of ophthalmologists as an anti-VEGF antibody (although not a fragment like Ranibizumab, a VEGF antibody fragment that was specifically developed for intraocular use).

In the open label Systemic Avastin for Neovascular AMD (SANA)[5] trial, patients with progressive visual loss who were ineligible for PDT were given intravenous Bevacizumab. Based on the risks of Bevacizumab therapy, patients were excluded if they had uncontrolled hypertension, a history of thromboembolic events, current anticoagulant therapy,

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proteinuria, or if elective surgery was planned within 3 months. Patients were followed weekly initially and then monthly. Significant improvements in visual acuity and decreased retinal thickness on optical coherence tomography (OCT) were seen. With improvement in visual acuity, OCT and angiographic outcomes, the systemic use of Bevacizumab appeared to be both effective and durable. The only significant adverse event observed was a mild elevation of blood pressure that was easily controlled with antihypertensive medication. There were no thromboembolic events; however, there were far too few patients, and the follow-up was far too short to know the true incidence of adverse events

Fig. 2: 4 ml AVASTIN vial, 0.2ml aliquots and tuberculin syringes for intravitreal injection.

With this background in mind researchers began treating patients with AMD with off-label use of intravitreal Bevacizumab and noted a dramatic therapeutic response. Initially, it was deemed unlikely that this big molecule (it is a humanized monoclonal antibody) would penetrate the full thickness of the retina when given intravitreally. Intravitreal use of Bevacizumab involves both an off-label application of the drug and an alternative route of drug delivery. Positive presentations at retinal meetings[6] and a number of published case reports of visual acuity improvement and decreased retinal thickness have led the retinal community to embrace this new treatment[7,8]. As a result, the use of intravitreal Bevacizumab has increased exponentially in the past few months. The main force driving intravitreal Bevacizumab usage is the high percentage of patients who experience symptomatic relief from active subfoveal CNV.

In many parts of the world, treatment with photodynamic therapy with verteporfin is too expensive and it is not used. On the other hand, Bevacizumab is easily available, relatively inexpensive, and is efficacious. This might include therapy for wet AMD, retinal venous occlusion (CRVO, BRVO) with macular edema, cystoid macular edema from uveitis, proliferative diabetic retinopathy, and causes of macular leakage like Diffuse diabetic macular edema. (DDME).

Preliminary results indicate a response of AMD to Bevacizumab that seems comparable to that of Ranibizumab (Fab fragment of an antibody that binds all isoforms of VEGF). The numbers are small and no randomized clinical trials have been performed.

There are a number of factors surrounding intraocular use of Bevacizumab. These include:

- High efficacy.
- Longer half life thus reducing the number of injections.
- Potential antigenicity of the full antibody.
- Lack of preservatives is a potential benefit with Avastin (Bevacizumab)
- Potential retinal toxicity in higher doses (>3.5mg), we currently use 1.25mg which is extremely safe.

Bevacizumab is a full-sized antibody, unlike the Ranibizumab fragment and has a longer half-life (up to 20 days) and thus longer anti anti-VEGF action. Although an intravitreal injection uses minute fractions of the drug and the systemic absorption is unlikely to be significant, measurement of systemic levels after intravitreal injection has not been done.

Reasons to use Bevacizumab center on this one fact: fast onset of improved retinal morphology and visual acuity. Indeed, the reports show dramatic improvement in OCT appearance and corresponding improvement in visual acuity. Other reasons to use it include its low cost and wide availability, with no experimentally proven toxicity shown to date. For patients who have failed therapy with approved drugs and have not yet evolved to disciform scars, off-label Bevacizumab could be recommended as a salvage therapy. Of all of these reasons, the overwhelming reason to use it is, of course,its high efficacy.

Technique of injecting intravitreal Avastin

We use 100mg vial of Avastin [Fig: 2] which contains 4cc of 25mg/ml concentration of the drug. This is then compounded to divide it into 0.2 cc aliquots. 0.05cc is injected intravitreally to deliver a dose of 1.25mg.under strict aseptic conditions. After giving topical anaesthesia, and betadine application over lids and conjunctiva, 1.25mg (0.05cc) of preservative free Bevacizumab is injected into the eye through pars plana route using a tuberculin syringe with 26 gauge needle. After the injection intraocular pressure and retinal artery perfusion is checked and patient is instructed to administer topical antibiotics for 5 days conditions where Bevacizumab has been tried till date are

1. Wet Neovascular AMD [both occult and classic variety]
2. Macular edema following Vein Occlusions
3. Proliferative diabetic retinopathy and diabetic macular edema (CSME)
4. Iris Rubeosis & Neovascular glaucoma

We have administered intravitreal bevacizumab in

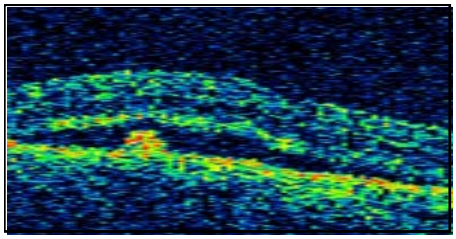


Fig.3(a): Pre Avastin OCT showing CNVM with sub retinal fluid.

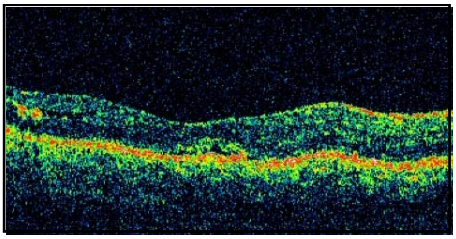
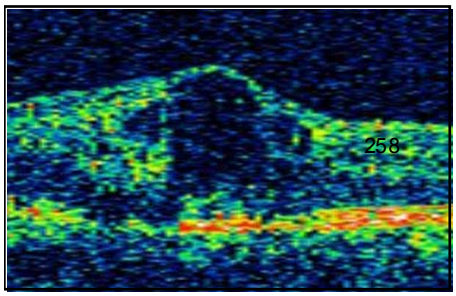


Fig.3(b): Post Avastin resolution of sub retinal fluid and marked improvement in visual acuity.



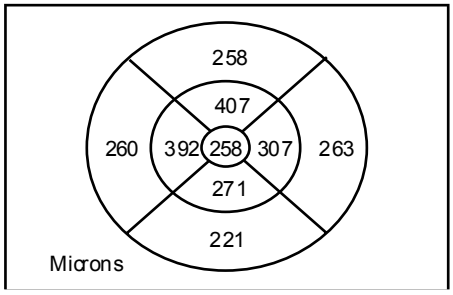
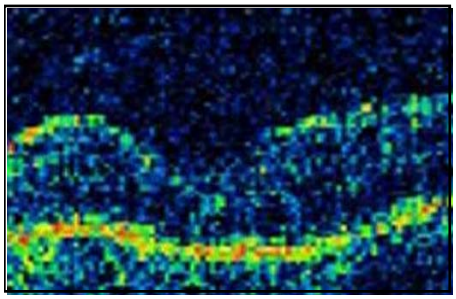


Fig.4(a): OCT showing cystoid macular edema and grossly increased retinal thickness following CRVO.



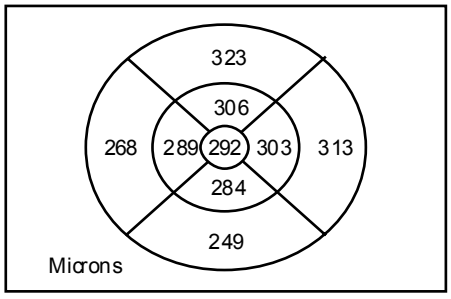


Fig. 4(b): 4 weeks post Avastin shows resolution of macular edema and reduction in macular thickness.

wet AMD (Fig.3a,3b), macular edema due to vascular occlusions like CRVO, BRVO (Fig.4a,4b) proliferative diabetic retinopathy (Fig.5a,5b), diabetic macular edema(Fig.6a,6b) with highly satisfying results.

Very recently, Manzano et al have evaluated the retinal toxicity of varying doses of Bevacizumab when injected intravitreally in rabbits. They concluded that Intravitreal Bevacizumab did not appear toxic to the retina in albino rabbits at a concentration of 2.5 mg[9].Shahar, Jonathan et al have carried out electrophysiologic and retinal penetration studies following intravitreal injection of Bevacizumab (Avastin). Bevacizumab was found to be nontoxic to the retina of rabbits based on electrophysiologic studies. They concluded that full thickness retinal penetration may explain observed clinical effects of intravitreal Bevacizumab[10].

Spaide et al and Bakri S J et al have shown that intravitreal Bevacizumab has resulted in marked regression of neovascularization and rapid resolution of vitreous hemorrhage in proliferative diabetic retinopathy in a limited number of patients[11,12].Iturralde,Diana et al and Jaissle GB et al conducted a retrospective study of patients with macular edema due to CRVO who were treated with at least one intravitreal injection of Bevacizumab 1.25 mg in 0.05 ml. They concluded that Intravitreal Bevacizumab resulted in a significant decrease in macular edema and improvement in visual acuity[13, 14].

Spaide RF et al and Ladewig MS et al have described the short-term anatomical and visual acuity responses after intravitreal injection of Bevacizumab (Avastin, Genentech) in patients with choroidal neovascularization (CNV) secondary to age-related macular degeneration (AMD). Treated eyes had a significant decrease in macular thickness and improvements in visual acuity. [15,16] There are also reports of promising results of Bevacizumab in refractory pseudophakic cystoid macular edema, rubeosis iridis[17]

However long term prospective randomized trials using intravitreal Avastin in varying doses would be required to finally justify its eventual efficacy.

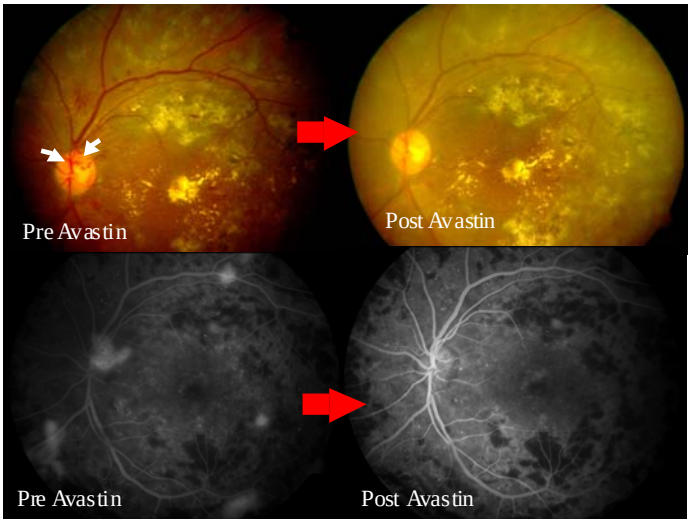


Fig.5: Pre Avastin Colour photograph and FFA showing NVD and multiple NVE, 4 weeks post intravitreal Avastin reveals resolution of neovascularization.

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Optical Coherence Biometry IOL Master

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Ultra-high-resolution immersion A-scan ultrasonography in upright position achieves axial length measurements that are approximately five times more accurate than standard applanation A-scan ultrasonography. The IOL Master employs the principle of Optical Coherence Biometry (OCB) to measure the axial length that matches the accuracy of an immersion A-Scan. Being an optical device, the IOL Master may not be able to measure in the presence of significant axial opacities.

OCB: The Technologic Basis

The use of OCB to measure the axial length of the human eye was first reported in 1986(1). Since then there have been many refinements that have culminated in the introduction of the IOL Master, the first commercial version of OCB for use in general ophthalmology. The IOL Master uses a modified Michelson interferometer to measure axial length with accuracy that is unprecedented. The use of OCB in ophthalmology is based on optical principles laid down more than a century ago by the German-American physicist Albert Michelson (1852-1931). Dr. Michelson's original work in interferometry was so important that in 1907 he received the Nobel Prize in physics.

The Michelson interferometer portion of the IOL Master is used to create a pair of coaxial 780-nm infrared light beams with a coherence length of approximately 130 nm. In the classic Michelson interferometer the eye has to be kept perfectly still but the use of a dual coaxial beam allows the IOL Master to be insensitive to longitudinal movements and makes axial length measurements mostly distance-independent.

One mirror of the interferometer is fixed and the other mirror is moved at a constant speed by a small motor. This process takes one of the light beams out of phase with the other by twice the displacement of the moving mirror. Both beams of light then illuminate the eye to be measured and are reflected at the level of the cornea and the retinal pigment epithelium. After passing through a polarizing beam splitter, all light beam components are combined together, producing interference fringes of alternating light and dark bands . The constant speed of the measuring mirror causes a Doppler modulation of the intensity of the interference pattern. An optical encoder is used to sense the position of the moving mirror, which is then translated into an axial length figure (2, 3, 4).

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The nuances

Every aspect of cataract surgery has an impact on the final postoperative refractive result. If keratometry is inaccurate by 0.50 diopters, then the final refractive outcome will be inaccurate by that same amount. If the capsulorhexis is made larger than the optic of the IOL, an anterior shift in position may occur during capsular bag contraction, resulting in more postoperative myopia than anticipated. Optimization of IOL constants and the IOL power calculation formulae are the other parameters that also influence the refractive outcome. Errors in axial length measurement have a profound effect on the refractive outcome, especially for high axial hyperopes.

Cataract Surgery - A Refractive Procedure

The last few years have witnessed amazing advances in the refractive component of cataract surgery. Increasingly sophisticated IOL designs, IOLs in 0.25-diopter steps, aspheric designs for powers as high as +40.0 diopters, and the limited correction of higher-order aberrations are now available options. However, without the ability to consistently calculate the accurate IOL power, an appreciation of these advancements is lost. Modern cataract surgery is not only a rehabilitative procedure, but a refractive procedure as well. IOL power calculations are a chain of interconnected factors and if one factor is incorrect, the final outcome will be sub-optimal.

Ideal Axial Length Measurement Technique

It has been reported that the most common reason for incorrect IOL power calculations is an error in the measurement of axial length. Conventional 10-MHz A-scan ultrasonography is the measurement technique most commonly available and has limitation of resolution of approximately 0.10 mm. This translates to about ±0.25 diopters under optimal conditions when the axial length is measured using an immersion technique (5). The accuracy of A-scan ultrasonography is less when carried out by the applanation technique that inevitably produces a falsely short axial length and sometimes widely variable results due to varying degrees of corneal compression. The ideal axial length measurement technique should be carried out in an upright position, without corneal contact or

compression, and with a level of accuracy high enough for outcomes consistently within 0.25 diopter of the target refraction. The ideal measurement technique should also be accurate enough to satisfy future requirements, such as the more precise IOL power calculations necessary to correct for any number of higher-order aberrations.

Fig.1

Fig.2

The IOL Master

The Zeiss IOL Master was approved for use in Europe in 1999 and in the United States in 2000. By employing a partially coherent light source rather than ultrasound, the IOL Master has fulfilled nearly all the important objectives for measuring the axial length of the human eye with a level of accuracy unimaginable just a few years ago.

During testing, the patient is seated upright and there is no corneal contact. Because OCB uses an infrared light source rather than a 10- MHz sound beam, measurement accuracy is increased from 0.10 mm to 0.02 mm-0.01 mm, an improvement of approximately five times (6). With the introduction of OCB as part of the preoperative evaluation, axial length measurement errors are no longer a limiting factor.

IOL Master vs. Ultrasound

Because OCB measures length from the corneal vertex to the retinal pigment epithelium, and A-scan ultrasound measures from the corneal vertex to the vitreo-retinal interface, some method of calibration is necessary to avoid a measurement error of 0.20 mm, or the approximate thickness of the retina at the center of the macula.

Haigis and others calibrated axial length readings from the IOL Master against immersion technique measurements with a Grieshaber Biometric System (GBS). The GBS is an ultra-high-resolution ultrasound biometer that employs four 40-Mhz counters and is capable of an accuracy of 20 μm (2).

Based on a comparison of the measurements obtained with the GBS, an internal algorithm for the IOL Master was developed such that the axial length displayed by the IOL Master equals that of the GBS. In essence, the IOL Master is the equivalent of an upright, non-contact, ultrahigh-resolution immersion A-scan, consistently accurate to within 0.01 mm.

Technique

Axial length measurements with the IOL Master are easy and quick. Although mostly operator-independent, some degree of interpretation is still necessary for optimal refractive outcomes. The patient is seated comfortably and positioned in a chin rest similar to a slit lamp (Fig. 1). The overview mode is used for course alignment; the patient looks at a small, yellow fixation light. Once the video image of the eye is centered, the operator switches to the axial length measurement (ALM) mode. The patient then views a small red light and the image of the eye is enlarged, with the iris filling most of the video screen (Fig. 2). It is best if nothing has touched the corneal surface prior to axial length measurements (e.g., an applanation tonometer or contact lenses).

Measuring axial length with the IOL Master allows a high degree of flexibility. Rather than simply positioning a small, in-focus image in the middle of a set of video screen cross hairs, the operator can instead maneuver the focusing spot anywhere within the measurement reticule, and even focus in or focus out. In this way it is possible to sample different areas around the visual axis until the best axial length display is obtained. Then, once that best area is discovered, all subsequent measurements are taken from that location. This technique is especially useful for eyes with small corneal scars, anterior cortical spokes, posterior sub-capsular plaques, or other localized media opacities.

If the signal-to-noise ratio (SNR) of the eye is borderline (2.0-1.6), focusing in or focusing out in such a way that the focusing spot enlarges to about the same size as the measurement reticule often improves the quality of the axial length display. This is possible with the IOL Master because axial length measurements by OCB are distance-independent.

It is useful to take all 20 measurements. At least four of these measurements should be within 0.02 mm of one another and should exhibit the characteristics of an ideal display. In terms of accuracy, an ideal axial length display is far more important than a high SNR (Fig. 3).

During axial length measurements it is important for the patient to look directly at the small red fixation light. In this way, axial length measurements will be made to the center of the macula, giving the refractive axial length rather than the anatomic axial length. For eyes with high

Fig.3: Showing the measuring reticule, SNR and tall primary maxima (Red)

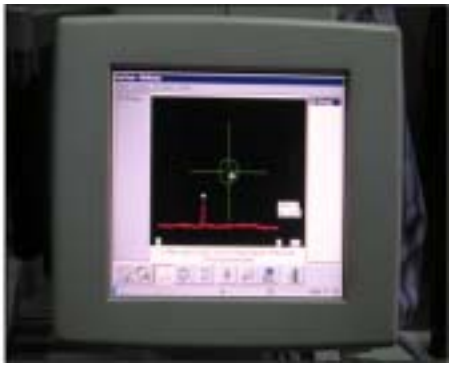

Fig.4

to extreme myopia and a posterior staphyloma, ability to measure to the fovea is an enormous advantage over conventional A-scan ultrasonography.

One other helpful feature of the IOL Master is if there is a high refractive error (more than ±4.00 D), measurement can also be taken with the patient's glasses in place to ensure adequate fixation (3). Measurements with and without glasses are usually identical. The axial length can be determined in most eyes with a high degree of precision, including extreme axial hyperopes and myopes, aphakes, pseudophakes, and even for eyes filled with silicone oil.

The characteristics of an ideal axial length display by OCB are the following: SNR ratio greater than 2.0; tall, narrow primary maxima, with a thin, well-centered termination; and at least one set of secondary maxima. However, if the ocular medium is poor, secondary maxima may be lost within a noisy baseline and not displayed. At least 4 of the 20 measurements taken should be within 0.02 mm of one another and show the characteristics of a good axial length display.

Limitations

Because the IOL Master is an optical device, any significant axial opacity has the potential to be a problem. Clinical situations such as a mature or darkly brunescent lens, central posterior sub-capsular plaques, anterior cortical spokes, corneal scars that pass through the visual axis, and vitreous hemorrhages may interfere with the partially coherent light beams and decrease the SNR to the point that may preclude a meaningful measurement. On the other hand, in eyes with posterior staphylomata or eyes with silicone oil, the IOL Master is the answer. In the ophthalmology practice, approximately 90% of patients can be measured successfully using the IOL Master. The remaining 10% of patients must be measured by A-scan ultrasonography for the reasons outlined above.

Other Features

The IOL Master is an "all-in-one" IOL power calculation device. It not only measures axial length with great precision, but also measures the central corneal power by automated keratometry. The instrument takes five keratometry measurements within 0.5 seconds and averages them. The latest software revision (version 3.01) has an improved keratometry algorithm and will alert the operator if a keratometry measurement is questionable.

The IOL Master will also measure the anterior chamber depth (the distance between the optical section of the cornea and the anterior surface of the crystalline lens) using a lateral slit illumination at approximately 30 degrees to the optical axis. This measurement is helpful for IOL power calculation formulas, such as Haigis and Holladay 2, which require a measured anterior chamber depth.

Included in the standard IOL Master software package are five popular IOL power calculation formulas (Holladay, SRK/T, Haigis, SRK II, and Hoffer Q) (Fig. 4). IOL power calculations can be carried for four IOLs at a time and to a precision of either 0.50 or 0.25 diopters. The IOL Master software will accommodate as many as 20 surgeons, each with up to 20 preferred IOLs and corresponding personalized lens constants.

New IOL Constants

IOL constants for the IOL Master will be closer to those normally seen for the immersion technique. They are typically higher than what would normally be used for the applanation technique, which is based on a falsely short axial length due to corneal compression. In making the transition from applanation IOL constants to IOL Master IOL constants, a good rule would be to increase already optimized applanation IOL constants by 0.50 for the SRK/T formula and by 0.29 for the Holladay and Hoffer Q formulas. Failure to make this initial adjustment may result in approximately 0.50 diopters of initial postoperative hyperopia. The IOL Master software comes with an IOL constant optimization feature that can subsequently be used to refine postoperative outcomes.

Saves time and effort

One additional advantage of the IOL Master is a significant increase in efficiency. With manual keratometry and applanation A-scan ultrasonography, the average time taken to do axial length measurements, corneal power determination, and IOL calculations is nearly 20 minutes. Use of the IOL Master has reduced this time to approximately 4 minutes. For any busy surgical practice, reducing the time needed to complete a common measurement by 80% is of enormous benefit.

Summary

The IOL Master is the equivalent of an ultra-high resolution immersion A-scan ultrasonography, giving the refractive axial length rather than the anatomic axial length. Because the IOL Master is an optical device, measurements may not be possible in the presence of significant axial opacities, such as a central corneal scar, mature cataract, vitreous hemorrhage, or dense PSC plaque. IOL constants for the IOL Master are often slightly higher than the manufacturer's suggested numbers for A-scans carried out by the applanation technique, but they are very close to those for the immersion technique. It is suggested that IOL Master-specific IOL constants be used with the various standard IOL power calculation formulas.

With the introduction of the IOL Master, the era of high-resolution IOL power calculations has begun. Ophthalmology now has a tool that will become indispensable in not-too-distant future when IOL-based higher-order aberration correction will become the norm.

"I have reffered to the user manual and an article by Dr. Warren Hill in preparation of this text."

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A Friendly Computer Program for Calculating Surgically Induced Astigmatism - The SIA Calculator 1.0

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There are many methods of calculating Surgically Induced Astigmatism (SIA), but some of these have been proven to be scientifically unsound. The difficulty arises because astigmatic values are composite in nature, having both magnitude and direction. Any method of evaluation that does not fully consider this vector nature of astigmatism is essentially incorrect. Many such methods were in use in the past, including simple subtraction, algebraic subtraction etc, but with refinement in analytical techniques, these have become obsolete. This is not just an increase in the degree of accuracy, but rather a fundamental shift from the incorrect to the correct. As modern cataract surgeons strive for postoperative emmetropia, precise analysis of astigmatism becomes more than just a theoretical exercise.

The techniques of vector analysis, Cartesian coordinate analysis, and Holladay's method of dealing with aggregate astigmatic data are all modifications on the same theme. As Naeser has proved using Popperian falsification, these methods will all lead to the one correct solution. But the problem lies elsewhere. All these methods are mathematical in nature, and the task of understanding this level of mathematics is daunting to many of us.

Fortunately, the very fact that this analysis is so stringently mathematical is a blessing in disguise. This mathematical nature makes the whole process amenable to computer programming. This was done using the Visual BasicTM platform and the product was the SIA Calculator 1.0, a user friendly computer program that calculates the exact surgically induced astigmatism for a given set of pre and post-operative keratometric data. The methodology used is that recommended by Holladay et al and is internationally accepted.

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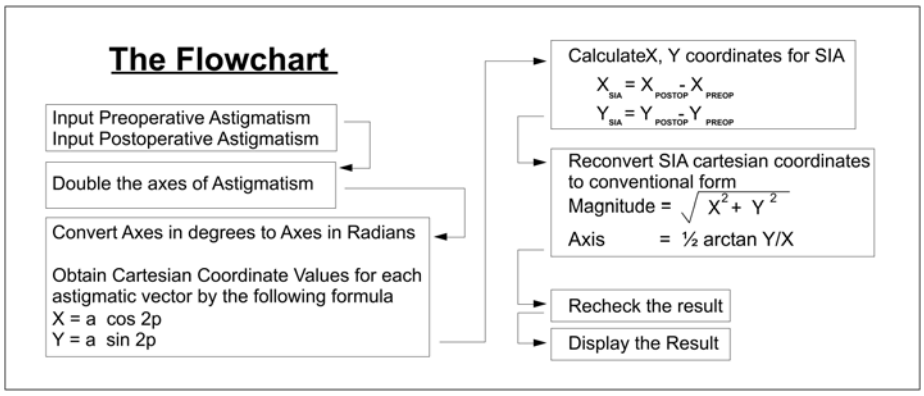
In brief, Holladay recommends that all vectors be analysed using trigonometric functions. This involves a 360 degree system, while astigmatic entities are 180 degree based. To circumvent this, astigmatic axes are first doubled. Trigonometric functions are used to derive 'x' and 'y' values as follows.

x = a cos2p, y = a sin2p, where a is amount of astigmatism and p is the axis.

Next, similar x, y values are calculated for postoperative astigmatism. To calculate induced astigmatism, preoperative x and y values are subtracted from the respective postoperative values, and then reconverted to astigmatic values (amount and axis) using mathematical operatives (please see the flowchart for exact methodology).

The following flowchart was used while developing the software.



The actual program is a windows based application that utilizes only about 44KB hard disk space. Apart from the small file-size, the software is very user friendly and straight forward. It is available on the DOS website for free use, and can be obtained from the author via email.

On double-clicking the program icon, a display appears on the desktop. Make sure that all the boxes are clear. If some key has been pressed inadvertently, just press CLEAR.

The values that need to be filled in are preoperative and postoperative astigmatism. For example, if the preoperative keratometry is

44.0 D x 90 deg and 45.5 D x 180 deg

then the preoperative astigmatism would be

1.5 D x 180 deg (dioptric difference x steeper axis)

Similarly, postoperative keratometry (43.25 D x 60 deg, 46.00 D x 150 deg) would mean a post operative astigmatism of 2.75 D x 150 deg. Fill in these figures in the appropriate places.

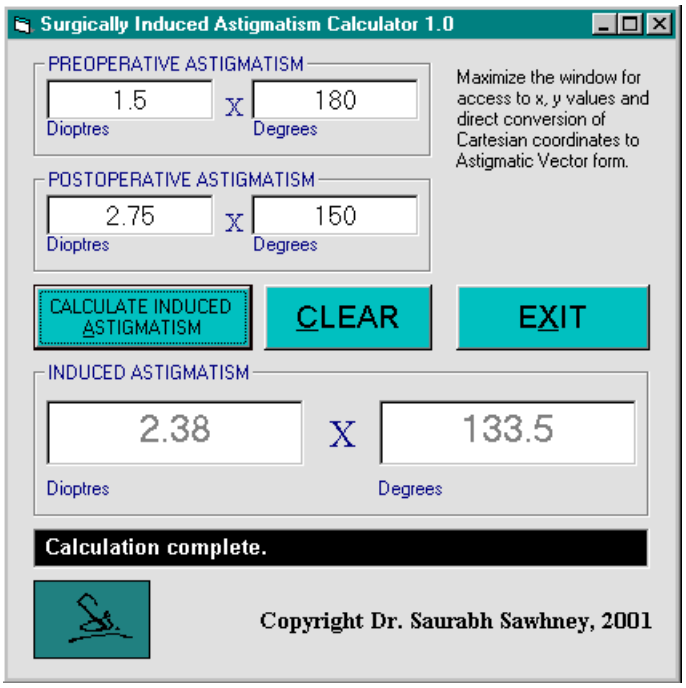
Once you have entered these values, just press the button and the surgically induced astigmatism is displayed. This is what the display looks like now.

The SIA Calculator reduces the arduous task of drawing vectors or looking up sines, cosines and their relatives to a straightforward button pressing job. There is no need to delve into mathematics; the job is accurately and speedily done.

Maximising the calculator window gives ready access to the x,y values that have been calculated by the computer. These are utilized for statistical analysis of aggregate astigmatic data. Since the calculator employs an internationally ratified method, the figures obtained can be used for publication. This is much simpler than using vector analysis for aggregate data analysis.

The calculator can be used for effectively analyzing personal data and identifying the patterns in one's own surgical practice. It comes with a readme text file that describes in further detail the use of the calculator, as well as a troubleshooter.

The authors hope that the Calculator will find widespread acceptance as the tool of choice for accurate calculation of surgically induced astigmatism.



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