Posterior Capsule Opacification: An Overview

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Posterior capsule opacification (PCO) and posterior chamber intraocular lens (PC IOL) decentration still remain two major complications of successful extracapsular cataract surgery (ECCE) or phacoemulsification.¹⁻⁶ Ridley, who performed the first intraocular lens implantation in 1949, himself noted these complications in his earliest patients.⁷ In his initial publications, he described lens decentration, and remarking that apparently, the most difficult problem was to retain the lens in position. He also recognized the problem of PCO and designated it as " the principal complication" that is not easy to treat, and which requires division of posterior capsule i.e surgical posterior capsulotomy.^{8,9} Control of decentration and PCO is becoming more necessary now that IOL implantation is emerging as a refractive procedure that mandates almost a perfect optical rehabilitation - as opposed to the former goal of simply removing the opaque lens material and achieving safe but less than optimal visual rehabilitation.²⁻ ⁵ As the cataract operation continues to be perfected, major goal is to eliminate these complications.

Clinical studies have reported an incidence varying between 10% - 50% of posterior capsule opacification following ECCE or phacoemulsification with PC IOL implantation.^{1,2,3,12-15} Schaumberg et al conducted an important metanalysis of published articles on PCO and generated pooled estimate of eyes developing PCO over three postoperative points: 1,3 and 5 years. They noted that even today the rate of PCO remains unexpectedly and unacceptably high- still over 25% during the 5-year postoperative period.¹ Furthermore, adverse clinical sequelae may be associated with Nd:YAG laser posterior capsulotomy. Last but not the least, there are very significant and compelling financial reasons to eliminate the necessity to do Nd:YAG laser capsulotomy. Nd:YAG laser posterior capsulotomy now ranks as the second most expensive surgical cost to the US health care system, second only to the cost of the original cataract operation¹³.

The reported Nd:YAG laser posterior capsulotomy rate ranged from 30% to 50% in the 1980s.^{2,3,11} More recent reports document an additional decrease in PCO and Nd: YAG laser capsulotomy rates.^{5, 11,14,15,16,17} With the use of

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modern surgical techniques and IOLs, posterior capsule opacification and Nd: YAG laser posterior capsulotomy rate is decreasing to less than 10%.¹⁸⁻²³ In a recent study by Apple et al¹⁷ comparing foldable versus rigid designs, the foldable IOLs were associated with a much lower Nd:YAG laser posterior capsulotomy rate (14.1% vs. 31.1%). Surgical tools and IOLs are now available to bring these rates down to single digits. Careful application and use of these tools by surgeons can genuinely lead in the direction of virtual eradication of secondary cataract, the second most common cause of visual loss worldwide.

Pathogenesis of posterior capsule opacification

Most secondary cataracts are caused by proliferation of equatorial lens epithelial cells, forming the pearl form of posterior capsule opacification.²⁶ Posterior capsule plaques or fibrous plaque detected in patients after ECCE are not uncommon in the developing countries²⁷ and such plaques are rarely seen in the industrialized world.

The epithelium of the lens consists of anterior epithelial cells known as A-cells which is single continuous cell line. These cells are continuous with the cells of the equatorial lens bow. The cells of equatorial lens bow are the E-cells, which comprise the germinal cells undergoing mitosis as they peel off from the equator. They continuously form peripheral cortical fibers. A- cells tends to remain in place and not migrate and are prone to change toward fibrous tissue (fibrous tissue metaplasia) when disturbed. In contrast E-cells of equatorial lens bow tends to migrate along the posterior capsule and form pearls form of posterior capsule opacification. These equatorial cells are the primary source of classical secondary cataract especially the pearl form of posterior capsule opacification.²⁶

(Fig 1-5). Fibrous form of posterior capsule opacification occurs as result of either posterior proliferation of A-cells or may result from a fibrous metaplasia of posteriorly migrating E cells.

Clinical appearance of PCO may also be caused by a postoperative localized endophthalmitis, a condition which has been recognized as a cause of persistent, usually low grade uveitis. Meisler and associates²⁸ were first to recognize the role of Propionibacterium acnes as an offending organism. Piest and associates²⁹ and Apple and associates³⁰ were the first to emphasize the concept of a post-ECCE localized infectious process caused by sequestrated organism within the capsular bag. Clinically,

it is important to be aware of the fact that clinical picture of PCO may be produced by localized endophthalmitis. The use of Nd: YAG laser capsulotomy to treat the posterior capsule thickening in this condition may lead to precipitation of severe inflammation.

Evaluation techniques for posterior capsule opacification

Precise methods of evaluation are important to measure the progress of posterior capsule opacification. Most of the studies evaluate posterior capsule opacification in patients after ECCE/phacoemulsification after full dilatation of pupil using slit lamp biomicroscopy. PCO is defined as opacification of the posterior capsule in the visual axis that is observed on slit lamp biomicroscopy which includes Soemmering's ring (PCO peripheral to the IOL optic), Elschnig's pearls and fibrous opacification behind the IOL optic. The degree of opacification is assessed using distant direct ophthalmoscopy, direct visualization by slit lamp biomicroscopy, and decrease in best corrected visual acuity after surgery. Visually significant posterior capsular opacification is defined as a decrease in the best corrected post-operative vision by two Snellen lines. Tetz described a photographic image analysis system that can morphologically score posterior capsule opacification without dependence on visual acuity testing.31 Standardized slitlamp retroillumination photographs are analyzed. Posterior capsule opacification score is calculated by multiplying the density of opacification and graded from 1-4 by



Fig 1: Visually significant PCO in an eye implanted with single piece PMMA IOL 18 months after cataract surgery



Fig.2: Thick PCO in eye implanted with hydrophilic acrylic multifocal IOL, 2 years after phaco surgery



Fig.3: PCO after sulcus fixation of square edge Pharmacia 911A IOL into sulcus. Square edge effect to prevent PCO is achieved only after implantation of these IOLs in the capsular bag.

Miyake-Apple posterior photographic technique for analyzing commonly used IOL model in eyes obtained post-mortem to evaluate PCO and whether or not an eye had an Nd: YAG laser capsulotomy.¹⁷

Management of posterior capsule opacification:

In the past, invasive surgical posterior capsulotomy was the primary treatment of posterior capsule opacification and it is still performed where Nd: YAG laser facility is not available or in cases with very dense or fibrotic membrane particularly in children.³³ The treatment of choice for clinically significant posterior capsule opacification is Nd: YAG laser posterior capsulotomy.34-36 It is an effective modality in the management of posterior capsule opacification. (Figure 6)

There are several disadvantages of Nd: YAG laser capsulotomy:

a. There are several visionthreatening complications such as damage to IOL optic, postoperative intraocular pressure elevation, cystoid macular oedema, retinal detachment, IOL subluxation or dislocation and exacerbation of localized endophthalmitis. Nd:YAG laser posterior capsulotomy significantly increases the overall cost of cataract surgery beside being a burden on the health care.

Keeping in view several visionthreatening complication of Nd: YAG laser capsulotomy or surgical capsulotomy, peeling or removal of epithelial cells from the posterior capsule in eyes with pearl type of PCO with automated irrigation mode or capsule vacuuming

the fraction of capsule area behind the IOL optic that is opacified. This technique shows good inter- and intraobserver reliability. Pande et al reported a more sophisticated system of retro-illumination imaging of the posterior capsule using a computerized high resolution digital system that can produce excellent images for objective documentation and quantitative measurement of posterior capsule opacification.³² Apple et al utilized mode or using two-ways Simcoe cannula is recommended particularly in patients with high myopia where incidence of retinal detachment increases several fold after Nd: YAG laser or surgical posterior capsulotomy.

Prevention of posterior capsule opacification

Although all the steps of cataract surgery are important in reducing this entity, six factors are



Fig.4: Visually significant PCO 5years after capsular bag fixation of hydrophobic acrylic Alcon Acrysof IOL



Fig.5: PCO in an eye implanted with hydrophilic acrylic rollable IOL. Although this IOL can be implanted with very small incision but this advantage if offset by high rate of PCO.



Fig.6: Treatment of PCO is usually achieved with Nd: Yag laser posterior cap sulotomy. This eye had clear visual axis after Nd: Yag laser capsulotomy.

particularly important in relation to eliminating or at least delaying posterior capsule opacification.

First, very essential step in reducing PCO is the reduction of formation of postoperative

Soemmering's ring, which is a precursor of PCO. This can be reduced not only by excellent hydrodissection enhanced cortical clean up but also by use of a highly biocompatible IOLs that reduce stimulation of cellular proliferation.^{2,3,5,13,26} The six factors influencing PCO formation are described



Fig.7: Tecnis foldable IOL, 1 year after implantation into capsular bag following phacoemulsification with clear visual axis. Lens epithelial cells stopped at the square edge optic.



Fig.8: Capsular bag fixated Alcon AcrySof MA60AC foldable IOL, 18months after implantation showing clear visual axis. Note anterior capsule opacification.



Fig.9: Clear visual axis in an eye implanted with Pharmacia 911A silicone IOL 8 months after phacoemulsification.

below:

1. Hydrodissection-enhanced cortical clean-up: First formal publication on this procedure was by Faust³⁷ in 1984 and later on in 1992, Howard Fine ³⁸ perfected the technique of subcapsular fluid injection and coined the term cortical cleavage hydrodissection. Cortical clean-up hydrodissection is used by many surgeons to facilitate lens substance removal and enhance the safety of surgery. The goal of hydrodissection is to remove equatorial cells and cortex, as opposed to removal of the single layer of anterior epithelium that does not migrate.13,26

2. In-the bag fixation of IOL: The obvious advantage of in-the-bag fixation is accomplishment of good centration and more important advantage that is not often appreciated is reduction in incidence of PCO.^{2,3,5,13,26,39,40} The hydrodissection enhanced cortical clean up and in-thebag fixation of IOL are two most important surgical factors in reducing PCO (Fig 7-10). In-the-bag fixation of IOL functions primarily enhances the IOL-optic barrier effect. When the IOL optic is fully in the capsular bag, it's contact is maximum with the posterior capsule and the barrier effect is functional. When one or both of the haptics are out-of-the-bag, a potential space exists that allows ingrowth of cells towards the visual axis.41,42

3. Capsulorhexis edge on the IOL surface: A significant factor which helps in reducing PCO is creation of a capsulorhexis with a diameter slightly smaller than that of IOL optic, so that the anterior capsulorhexis edge rests on the IOL optic. This helps to provide a tight fit (analogous to a " shrink-wrap") of the capsule around the optic.^{26,43,44,45}

4. Biocompatibility of IOL: In general, the amount of PCO depends in part on the biocompatibility of the IOL. The less the cell proliferation, the less the chance of posterior capsule thickening. The amount of PCO depends on many factors such as the quality of surgery, duration of



Fig.10: Clear visual axis following implantation of hydrophobic acrylic Sensor optiedge foldable IOL 6 months after surgery



Fig.11: Square edge barrier effect of acrylic hydrophobic IOL helps to prevent migration of lens epithelial cells beyond the edge of IOL optic, thus preventing formation of PCO

implant in the eye and biocompatibility of IOL material. It

- has been reported that acrylic IOLs display the lowest amount of cell proliferation, and hence are the most biocompatible.⁴⁶⁻⁴⁹
- 5. Maximum IOL optic posterior capsule contact: In-thebag fixation of IOL helps to maintain a tight contact between the IOL optic and posterior capsule and helps to inhibit the migration of cells across the visual axis.^{10,14,47,50-54} Posterior angulation of IOL haptics and a posterior convexity of IOL optic also contribute significantly in maintaining this maximum posterior capsule contact. Still another factor which appears to contribute is related to stickiness of IOL biomaterial which in turn might create an adhesion of the capsule and IOL optic.
- 6. Barrier effect of IOL optic: The IOL optic barrier effect comes into play as a second line of defence against PCO.⁵⁵⁻⁶⁰ Implanting IOL in the capsular bag enhances the barrier effect. It has been shown that optic with round edges might have negative influence by allowing some of the cells to migrate under the tapered edge of the optic onto the posterior capsule. (Figure 11). A truncated optic edge appears to create an abrupt and effective block to cells growing onto the posterior capsule. Examples of square edge optic IOLs are Alcon Acrysof®, Pharmacia Cee On 911 etc. (Figure 7-10)

Pharmacological techniques and immunological inhibitors of PCO:

Pharmacological techniques which could accomplish the reduction or destruction of lens epithelial cells would perhaps be effective in reducing PCO.^{2,61-67} The various pharmacological studied till date are caffeic acid phenethyl ester in a rabbit model, hypo-osmolar drugs (sterile water), and antimetabolites. Antimetabolites that have been studied are daunomycin, methotrexate, 5-fluoro-uracil and colchicine. The rationale for use of these agents is to inhibit lens epithelial cell mitosis while avoiding toxic effects to non-mitotic cells. Some investigators are studying immunological agents such as monoclonal antibodies targeted to lens epithelial cells.

A new entity: Interlenticular opacification (ILO) or opacification of piggyback IOL:

The use of piggyback IOL i.e use of paired IOLs in one eye is becoming more and more common for correcting residual refractive error after IOL surgery or as primary procedure in high refractive error.⁶⁸⁻⁷⁴ Opacification between two-implanted IOL has been termed as " Interlenticular

opacification" or "interpseudophakos Elschnig pearls". In contrast to PCO, this entity occurs as a result of pearls formation or opacification between the two IOLs, undoubtedly due to ingrowth of cells from the equatorial lens bow. Werner et al have suggested implanting the posterior IOL in the capsular bag and anterior IOL in the sulcus to reduce this complication besides all the factors listed for preventing PCO.

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Nystagmus: Pearls and Paradigms of Management

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Involuntary rhythmic oscillations of the eyes are referred to as nystagmus. The clinical features of the nystagmus often give the clue to the diagnostic significance of the eye movements. Usually, movements associated with nystagmus have a slow component and a fast component. It is important to remember that the slow component is the pathologic element in the nystagmus, but the jerk nystagmus is characterized by the corrective fast component. In other words, nystagmus is conventionally described by the direction of the fast phase i.e. if the fast phase of the nystagmus is to the left, it is called a left beating nystagmus. The other terms used frequently while describing nystagmus is the frequency and the amplitude of the nystagmus. Frequency is the to and fro movement of the nystagmus in a second while amplitude is the extent of the excursion of the nystagmus. The intensity of the nystagmus is the amplitude multiplied by the frequency.

The severity of the nystagmus can be graded if its direction is constant (i.e. if the fast phase does not reverse). For example, in a right beating nystagmus the amplitude will increase on right gaze and decrease on left gaze. The severity of the nystagmus can then be graded as mentioned below:

- 1. *Grade 1*: Nystagmus is present on right gaze.
- 2. *Grade 2*: Nystagmus is present on right gaze and in the primary position but is absent on left gaze
- 3. *Grade 3*: Nystagmus is present even on left gaze

Nystagmus has been the waterloo of many a strabismologists for a very long time and its management has interested many ophthalmic surgeons for many years.

Classification of Nystagmus

Nystagmus can be physiological or pathological. Most examples of true physiological nystagmus are produced only under laboratory conditions. Some of these conditions are the end-point nystagmus and the nystagmus induced by caloric testing.

Pathological nystagmus can be classified in different ways. One of the ways in which it is classified is dependent on the waveforms of the movements seen in the nystagmus. The waveforms may be pendular or jerky.

Pendular Nystagmus

In pendular nystagmus, the to and fro movements of the eyes are of relatively equal rate and amplitude. The oscillations are usually horizontal, and remain the same on gaze upwards and gaze downwards. On lateral gaze to either side, the oscillations frequently convert to jerk type nystagmus, with the rapid component towards the direction of gaze.

Pendular nystagmus is commonly referred to as sensory or "ocular" nystagmus, implying an underlying ocular or vision defect. It usually occurs in patients with bilateral central vision defects of congenital origin like congenital cataracts or optic nerve lesions, when the central fixation does not develop appropriately.

Jerk Nystagmus

In the jerk type nystagmus, the to and fro movements of the eyes are unequal; there is a slow component in one direction followed by a rapid component in the other direction. In congenital jerk type nystagmus, the oscillations remain horizontal in all fields of gaze, though the amplitude and the rate may vary with the direction of gaze. In many of these cases, there is a null point, a point of gaze in which the oscillation of the eyes diminishes or abates. In such cases the patient usually exhibits compensatory head posturing, keeping the eyes in the preferred gaze where the nystagmus is least and vision is best, and turning the face in the opposite direction.

When there is no obvious underlying sensory or ocular abnormality, visual acuity in patients with nystagmus is usually subnormal usually because of the decreased foveation time. There may also be associated foveal or optic nerve head hypoplasia.

Theoretical basis of the origin of the nystagmus

Failure of the step part of the pulse-step mechanism controlling saccadic movements has been argued to be the principle cause of nystagmus. The basis of this theory is that any change in the extraocular muscle position is accompanied by a change in the extraocular muscle tone in order to maintain the new position of gaze. Maintenance of the eye position is the function of this step. If, for example, the eyes move into left gaze, the tone in the agonist left lateral rectus muscle and right medial rectus muscle is increased and that in the antagonist left medial rectus and right lateral rectus muscle is correspondingly decreased. If the muscle tone cannot be maintained, the eyes are pulled

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 Table 1: (Face turn present to the right; nomogram for shift of eyes from the null point in the left) – the Kestenbaum's Anderson's nomogram

Face Turn	Left Eye	Right Eye			
(Degrees)	LR Recession	MR Recession	MR Recession	LR Recession	
< 20 (Classical Kestenbaum's Procedure)	7 mm	6 mm	5 mm	8 mm	
30 (Augmented KA Procedure)	9 mm	8 mm	6.5 mm	10 mm	
45 (Augmented KA Procedure)	10 mm	8.5 mm	7 mm	11 mm	
>45 (Augmented KA Procedure)	11 mm	9.5 mm	8 mm	12.5 mm	



Fig. 1: Patient with left beating Congenital Idiopathic Nystagmus with esotropia. Note the right head turn of the patient *(Photo Courtesy: Dr Hanumantha Reddy)*

Fig. 2: Patient with upbeating nystagmus. Note the chin down position. (Photo Courtesy: Dr Hanumantha Reddy)

back towards the mid-line by the natural drag of the orbital tissues until the drift (the slow phase of the nystagmus) is corrected by a saccade (the fast phase of the nystagmus). The saccade is initiated by the pulse part of the pulse-step mechanism. A defect in the step will allow repeated drift and the cyclical pattern of the nystagmus becomes established. The step defect may be due to failure to supply and maintain sufficient innervational drive to the agonist muscles, as in gaze evoked nystagmus, or it may be due to an imbalance in the tonic drive to the agonist muscles, as in unilateral labyrinthine damage.

Neuronal Integrator

The neural integrator is a theoretical concept of a center in the brainstem, which adjusts the input from the pursuit and vestibulo-ocular systems to ensure maintenance of the correct innervational drive to the extra-ocular muscles. The pursuit system maintains foveal fixation and the vestibulo-ocular system maintains the position of the eyes in space, counteracting the effect of head rotation. As both the systems bring about an appropriate change in the muscle tone, their input must be co-ordinated to maintain a steady eye position. It is possible that a number of such centers exist which contribute to the formation of the neuronal integrator. There are different theories as to how stimulating the neuronal integrator or reducing the drive to it can produce different types of nystagmus in different circumstances. The details of these are outside the preview of this article.

Management of Nystagmus

It is important to distinguish the acquired and the congenital varieties of nystagmus. The main types of acquired nystagmus are:

1. Labyrinthine nystagmus: Labyrinthine nystagmus is caused by diseases affecting one labyrinth, which causes horizontal jerk nystagmus. Vertical nystagmus does not occur because each of the two vertical semi-circular canals is paired with the corresponding canals on the opposite side, giving bilateral control over vertical eye movement. Torsional oscillations may however be seen. Characteristics of a labyrinthine nystagmus are that this is a

nystagmus of small amplitude and high frequency, with the fast phase being away from the site of the lesion (localizing). The amplitude of the nystagmus also increases to the side away from the lesion. This nystagmus can be suppressed by the pursuit system and is thus best demonstrated when the foveal fixation is inhibited. The pursuit system compensates for the imbalance after the few weeks and hence this nystagmus is transient.

2. *Nystagmus due to central vestibular disease:* Damage to the central vestibular apparatus may produce a nystagmus that can have any combination of horizontal, vertical and torsional movements, not dampened by fixation.

3. *Nystagmus due to cerebellar disease:* This is usually a nystagmus of a large amplitude and low frequency (coarse nystagmus), not dampened or compensated by pursuits. The nystagmus can be localizing with the fast phase being directed to the side of the lesion and the amplitude of the nystagmus increasing when the eyes are directed to the side of the lesion.

4. Nystagmus due to the cerebellopontine angle tumour (Brun's nystagmus): This type of nystagmus as may be seen in patients with acoustic neuromas, demonstrate both types of nystagmus, the labyrinthine type and the cerebellar type. This pattern of the nystagmus is localizing and this particular type of nystagmus thus has a name (Brun's nystagmus)

5. *Gaze Evoked Nystagmus*: This common form of nystagmus represents the inability of the eyes to maintain an eccentric gaze because of the weakness of the muscle tone in the agonist muscles. This weakness may be neurogenic or myogenic. The nystagmus has a slow phase of decreasing velocity followed by a refixation saccade in the direction of the defective gaze.

6. Convergence Retraction Nystagmus (Parinaud's Syndrome): This is a specific type of nystagmus syndrome, which is localizing for dorsal midbrain lesions. There is progressive upgaze palsy, with a rhythmical retraction and convergence movement on attempted upgaze. An associated light-near dissociation may be present.

7. *Spasmus nutans*: This condition comprises of a combination of nystagmus, which classically consists of bilateral asymmetrical oscillations; involuntary head movements and head nodding, with an abnormal head posture. The onset of this disorder is between 3 to 18 months of age and usually resolves spontaneously by 3 years of age. Sometimes, it may be the manifestation of an optic nerve glioma, especially an optic chiasmal or

 Table 2: (Face turn present to the right; nomogram for shift of eyes from the null point in the left) – Other nomograms

Procedure	Left Eye LR Recession	e MR Recession	Right Eye MR Recession	LR Recession
Johnson's procedure)	10 mm	10 mm	10 mm	10 mm
> 20 (Spielmann's Procedure)	KA nomogram with posterior fixation	KA nomogram	KA nomogram with posterior fixation	KA nomogram
Modified Anderson's procedure (Von Noorden)	12 mm	_	10 mm	_

parachiasmal glioma. The nystagmus is jerky, of small amplitude and frequency and the movements may be horizontal, vertical or rotary. The nystagmus varies in different positions of gaze and is rarely compensated by the head movements or the head posture because the two movements are of different frequencies. The head movement is usually in the direction opposite to the eye movements. The vision is usually good, bordering between 20 / 30 to 20 / 40.

8. *Seesaw nystagmus*: This is a rare nystagmus, where one eye elevates and intorts while the other eye depresses and extorts. Subsequently, the sequence is repeated in the opposite direction. This disorder is commonly seen in patients with bitemporal hemianopia, parasellar

neoplasms and vascular disorders affecting the midbrain area.

9. *Dissociated nystagmus*: This term is used to describe cases where the oscillations of the eyes are of different amplitude. A common cause is INO, where there is marked coarse nystagmus in the abducting eye but little or no nystagmus in the adducting eye.

10. *Periodic Alternating nystagmus (PAN):* This is an uncommon nystagmus that is characterized by spontaneous oscillations that alter directions every few minutes (usually with the fast component going to one side for 60 to 90 seconds, followed by 3 to 5 seconds of little or no nystagmus, with the fast component shooting over to the other side again for about 60 to 90 seconds



(Fig. 3A): A 6 year old albino child with CIN, and optic nerve head pallor, demonstrating a left divergent squint with a right head turn (Fig. 3B): Note how closely the child holds the object of regard while doing near work. This should not be discouraged. (*Photo Courtesy: Dr Zia Chaudhuri*)

and so on). It is seen commonly in patients with multiple sclerosis, Arnold Chiari malformation, drug toxicity and disorders of the craniocervical junction. These patients respond well to the drug Baclofen and Carbamazepine.

11. Ocular flutter: This condition is not a true nystagmus but a series of to and fro saccades. When the movements are limited to the horizontal plane, the disorder is known as ocular flutter. These movements usually imply an underlying cerebellar disorder. When the oscillations occur in all directions, it is called opsoclonus or saccadomania. These movements are also sometimes referred to as "ataxic" conjugate movements of the eyes. This can be a presenting symptom of bronchial carcinoma, encephalitis, myoclonic seizures and neuroblastomas.

12. Ocular Dysmetria: This particular disorder denotes a lack of precision in performing movements of re-fixation. On changing the gaze from one point to another, there is an overshoot of the eyes. This phenomenon is usually associated with diseases of the cerebellar pathways.

13. Ocular Bobbing: In this condition, the eyes dip downwards

intermittently and then return back to the mid-position. This movement is associated with pontine lesions and has been described with pontine infarction, hemorrhage and tumours of the pons.

The management of acquired nystagmus requires an extensive neurological work-up as on most occasions, they denote an inherent cerebellar or vestibular lesion, which often gets detected because of the presence of the nystagmus. The ophthalmologist should be aware of these conditions with an aim to be able to appropriately refer these patients.

The different types of congenital nystagmus are:

1. Congenital Nystagmus due to low vision: This term implies that the nystagmus is present from birth, possibly due to a congenital anomaly of the motor systems or due to a congenital disorder of vision. These can manifest shortly after birth, or can develop at a later stage. Examples of later development include nystagmus secondary to low vision or manifest latent nystagmus. Nystagmus secondary to low vision is characterized by slow rhythmic

l IR Recession + SR Resection (Parks and l, Roberts et al, Taylor and Jesse) ssion + SO Recession (Pierse) ction + IO advancement (Schlossman) ession + IR resection (Parks) ession + IO Myectomy (Taylor and Jesse)
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cement of the anterior part of the riate oblique muscles (Conrad and de nd infraplacement of the horizontal rectus s (de Decker) g the insertions of the 4 recti muscles ann) ral Anterior tenectomy of the SO with lateral IO Myectomy and Harada-Ito on the SO (Pratt-Johnson) osition of the vertical recti muscles (von n)

dampening the intensity of the nystagmus and increasing foveation time

Bietti et al	•	Retroequatorial recession of all 4 horizontal muscles
Von Noorden et al Helveston et al	•	10-12 mm recession of all 4 horizontal muscles MR placed 11.5 mm behind the nasal limbus and the LR placed 13 mm behind the temporal limbus in both eyes.

oscillations of the eyes with similar movements in both directions. However, the waveform in low vision nystagmus can be variable and in some cases, the jerk pattern is prominent. The common causes of such low vision resulting in nystagmus include bilateral corneal scarring, infantile glaucoma, dense cataracts, ocular albinism, optic nerve hypoplasia, optic atrophy, macular hypoplasia and heridomacular degenerations

2. Congenital Idiopathic Nystagmus (Jerk): This is by far, the most common type of nystagmus seen. It is characterized by an early onset with slow wide movements (Triangular movements)- the phase 1; evolving into a pendular type nystagmus (Phase 2); and if it persists, it progresses into a jerk nystagmus with null zone with acceleration in the slow phase, the direction of the jerk being on the side of the fixation (Phase 3). The vision typically continues to improve upto 20/80 by the age of 9 years, improving by 1 line for every 10 years. The accommodation however deteriorates early, sometimes as



(Fig. 4A): Pre-operative photograph of a patient with CIN with a null towards the right, face turn towards the left and a left divergent strabismus. The patient had good visual acuity in both eyes of 6/9 in both eyes. (Fig. 4B): The patient had good vision for near work. The head posture was equally prominent during near work as it was while doing distance work. (*Photo Courtesy: Dr Zia Chaudhuri*) Fig.4C: Post-operative photograph of the patient demonstrating good ocular alignment in primary gaze with marked amelioration of the head posture. Fig.4D: Post-operative photograph of the patient doing near work. Note the marked improvement in the head posture with respect to the posture present pre-operatively. (*Photo Courtesy: Dr Zia Chaudhuri*)

early as 30 years. The patient may require regular correction of the hyperopia and near vision as the only treatment. Clinical features of this type of nystagmus are:

- a. The nystagmus is bilateral, associated and of equal amplitude in both eyes
- Many patients adopt a head posture, so as to place the eyes within the null zone and have better visual acuity.
- e. The vision usually improves on convergence, therefore near vision may be better than the distance vision. The intensity of the nystagmus however usually increases on attempting fixation.
- b. The ocular structures are usually normal in such cases. Though the constant oscillations reduce vision to an
- f. A superimposed latent component may be present,



extent, when the visual acuity is significantly reduced, an occult sensory cause for the nystagmus should be suspected, such as optic atrophy, optic nerve hypoplasia or retinal dystrophy.

- c. The nystagmus is predominantly uniplanar, usually in the horizontal plane. The movements remain horizontal on lateroversions, upgaze and downgaze.
- d. Once the nystagmus has gone to Phase 3, there is the presence of a null zone, usually sited eccentrically, where the nystagmus is minimum and the vision maximum. Visual acuity is relatively good when the null zone is used.

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Fig.5: ENG Recordings of different eye movements-the smooth pursuit movements in this case (Photo Courtesy: Dr. Hanumantha Reddy)

which may then cause the uniocular visual acuity to be worse than the binocular visual acuity.

- g. The nystagmus is abolished in sleep but may persist in darkness with both eyes open. The nystagmus is reduced by eyelid closure. There may be dampening of the optokinetic response.
- h. Compensatory mechanisms adopted to minimize nystagmus and improve vision in such cases are:
 - i. A face turn to centralize the null zone
 - ii. Voluntary Convergence
 - iii. Head bobbing / shaking (Especially if the patient has oscillopsia, which is extremely rare but may be present in an isolated case)

3. Latent and Manifest Latent Nystagmus: Latent nystagmus is a condition in which a horizontal jerk nystagmus is present when the light stimulus to either eye is reduced, as what may occur when one eye is covered, but there is no significant nystagmus binocularly. This may be present with congenital nystagmus. The characteristics of the latent nystagmus and the manifest latent nystagmus are the same with the differences between then being only quantitative. By definition of the condition, binocular vision is usually present. Aims of treating manifest latent nystagmus comprise of obtaining binocular single vision and manage any other cause of decreased vision that may be causing the nystagmus to get manifested like the presence of congenital cataracts, amblyopia and strabismus. The characteristics of latent nystagmus are:

- a. The nystagmus is bilateral and jerky, with the fast phase invariably to the side of the fixing or uncovered eye.
- b. The amplitude increases on occlusion of either eye. A face turn to the side of the fixating eye is commonly

seen, particularly while testing the visual acuity.

c. The slow phase of the waveform shows an exponential slowing, as opposed to the exponential increase seen in congenital nystagmus. The intensity of the nystagmus increases on adduction with the nystagmus frequently manifesting when both the eyes are open.

The management of congenital nystagmus can be a difficult proposition and has to be approached with clarity of thought and aims. With appropriate clinical judgement, proper diagnosis and treatment of these children, not to mention the immense role of parent counseling, predicting the performance of these children is possible. Appropriate consideration has to be given to the limitations of education, vocation and even genetic problems that these patients may have. Help can often be sought from the neuro-physiologists and neuro-anatomists to aid the understanding of the subject.

The primary aims of the management are:

- a. To diagnose the type of nystagmus and its diagnostic and prognostic significance
- b. To elucidate the cause of the nystagmus, whenever possible
- c. To assess the effect of the nystagmus on the patient's vision and binocularity
- d. To alleviate the abnormal head posture, whenever possible. This is especially true of patients with congenital nystagmus.
- e. To assess the effect of a co-incident strabismus

Management of a patient with Congenital Idiopathic Nystagmus (CIN)

Conservative treatment: This is indicated when the visual acuity and binocularity is good in the presence of an insignificant head posture. Patients with CIN usually have good vision for near and also for distance, if there is a null point. Patients should not be stopped from taking up their compensatory head posture. The parents and the teachers should be reassured and made to understand that the habit of holding the book very close to the face or adopting the compensatory head posture should not be discouraged. Deviating prisms and converging prisms can be used to alleviate vision. These can also be used routinely in the pre-operative assessment of the child as they help in assessing the response to the planned surgery. Other protocols that need to be adopted are the management of amblyopia, if present, correction of refractive errors, and prescribing low vision aids, which though may not be universally accepted, are of immense help in some cases. Contact lenses have been used to improve vision in patients with CIN. The mechanism of the beneficial effect of contact lenses is however, not well elucidated.

Botulinum Toxin has been used in many cases of acquired nystagmus. The role in patients with CIN is not well described. The retrobulbar route of injection is believed to be more effective than the direct rectus muscle injection. The advantages to an ophthalmologist as far as a retrobulbar injection is concerned are that only one injection needs to be given, and an EMG monitoring, which may not be accessible to many, is not required.

b. Surgical Management

i. *Patients with binocular single vision*: Surgery is only indicated if there is a cosmetically poor or extremely uncomfortable head posture (> 30⁰ in any axis as measured with the goniometer or a simple protractor). The aims of the surgery in this condition are to reduce the abnormal head posture without disturbing the ocular balance. This is achieved by moving the eyes in the direction of the face turn. Both eyes are rotated an equal amount in order to maintain parallelism of the visual axis. There are various methods adopted for different types of nystagmus depending upon the clinical picture of the patient.

ii. *Patients with strabismus*: In patients with associated strabismus, the protocols of management undergoes a change. Most patients with esotropia or exotropia with the torticollis fixate with the adducting eye. Depending upon the surgeon choice and the patient's clinical profile, the following modalities can be adopted:

- a. Perform staged surgeries; first correcting the torticollis and then the strabismus
- b. Perform both the procedures simultaneously with best guess dosage. There are no clear-cut nomograms related to this. Some of the situations that may arise are cases of esotropia with face turn and exotropia with face turn. This, we will discuss in some details as elucidated below:
- 1. Esotropia with face turn
- a. Recess the MR and LR of the fixing eye (adducting eye). This should correct both the face turn and the esotropia
- b. Alternatively, recess the MR muscle and resect the LR muscle on the abducting (non fixing) eye to correct the squint and then recess the LR on the adducting eye to change the face turn
- 2. Exotropia with face turn
- a. Recess the LR and resect the MR of the fixing (adducting) eye corrects both the esotropia and the face turn
- b. Along with the above, recession of the LR in the non fixing abducting eye can neutralize the over correction and correct the face turn
- c. Perform surgery on both eyes simultaneously, but use one or more adjustable sutures to provide flexibility in correcting the associated strabismus.

It is important to explain to patients undergoing

surgery for nystagmus that:

- 1. Any of the above procedures may induce a new deviation and a new head posture. The chances are much less with the Kestenbaum-Anderson's procedure and rather high with the 4 muscle recession surgeries. Any subsequent strabismus that may occur may require subsequent surgery. The modified Anderson's procedure has been noted to produce the least amount of post-operative change in head posture.
- 2. Any correction of the head posture is usually associated with undercorrection, with the head posture being within the accepted limit as the end point. Sometimes there may an overcorrection. Undercorrection is more acceptable to the patient than overcorrection.

While describing the association of nystagmus with strabismus, two other conditions need special mention, which is as described below:

1. *Nystagmus Blockade Syndrome*: This is a condition with an early onset, with manifest nystagmus in primary position. The esotropia is variable, often unilateral, with a head turn towards the fixing eye. There have been reports of an etiological association between this syndrome and infantile esotropia with an inverse relationship between the angle of the esotropia and the intensity of the nystagmus. Both hydrocephalus during infancy and an increased intraocular pressure are associations of the condition. The vision is usually good, of the range of 20/40. Bilateral MR recessions, a unilateral recess-resect procedure or recessions with posterior fixation are all procedures that may be performed to alleviate this condition. The results are usually sub-optimal because of the variability of the deviation. Pre-operative prism adaptation may be of great help in determining the extent of the surgery to be performed.

2. Albinism: The origin of nystagmus in albinos remains unknown. Both sensorineural and motor factors may be contributory in its pathogenesis. Sensory factors that may be contributing are high refractive errors, high astigmatism, increased intraocular scatter, optic nerve and foveal aplasia etc. Congenital idiopathic nystagmus (CIN) and Periodic Alternating Nystagmus (PAN) are commonly associated with albinism. Patients with albinism also have a high incidence of concurrent strabismus, which is often sensory in origin. The visual acuity in these patients is poor, of the order of 20 / 200. Both the abnormal sensorial apparatus with secondary sensory nystagmus and the presence of CIN or PAN could be contributing to the low vision. It is important to correct the refractive errors, where present, provide appropriate amblyopia therapy and give anti-glare treatment to help palliate the symptoms of the patient. Surgical modalities similar what has been described above for the management of CIN and PAN can be adopted.

Electronystagmography

The physician gains useful information about the patient who complains of disturbed equilibrium by observing the patient's eve movements during visual and vestibular stimulation. However, as already mentioned, visual fixation may have a strong suppressive effect on certain types of nystagmus making such observations redundant. Electro-oculography (EOG) is an important modality of assessing such eye movements and keeping a permanent and repeatable record of the same, in all kinds of external conditions (light or dark), with the eyes closed or open. An adaptation of the EOG used in the study of eye movements in nystagmus is called electronystagmography (ENG). ENG owes its existence to the fact that the eye behaves like a battery, with the cornea acting like a positive pole and the retina acting like a negative pole. The potential difference between the two poles is at least 1 mv. This electrical potential, called the corneoretinal potential, creates an electrical field in front of the eyeball when it rotates. These electrical changes are recorded and amplified by the electrodes placed on the skin and a tracing

of the eye movements taken. The ENG cannot detect torsional movements, as these movements produce no change in the orientation of the corneoretinal potential. This is one of the limitations of the ENG vis-à-vis the clinical method of examining the patient and taking a video record of the same. The ENG can be used to perform the gaze test (the eye movement recordings with the patient looking in different gazes); the saccade test; the tracking test, the optokinetic test, the positional test and the caloric test.

These tests extensively help in the diagnosis of otoneurological disorders. However, the results of these tests are often subjective and must be interpreted in the light of the patient's history and clinical findings.

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CRVO : Current Practice

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Introduction and etiology: Central retinal vein occlusion (CRVO) is the third most common vascular retinal disorder after diabetic retinopathy and branch retinal vein occlusion. It may be caused either from thrombus formation within the central retinal vein, or vascular compression.¹²

CRVOs are complex multi factorial disorders, occurring as a result of both systemic³⁻¹⁰ and local factors.¹¹ In critical numbers and/or intensity, these precipitating and predisposing factors result in the final catastrophic event.

Pathogenesis: The initiating event for a CRVO is thought to be thrombus formation in the central retinal vein, histologically localized at the level of the lamina cribrosa. ^{12, 13} Here, the central retinal vein and artery share a common adventitial sheath. It occurs that artherosclerotic changes in the central retinal artery transforms it into a rigid structure that presses upon the pliant central retinal vein. This results in hemodynamic alterations, increased turbulence of the blood flow, resultant endothelial damage, and thrombus formation. The central retinal vein has no room to move or expand because of the restricting adventitial sheath.

Clinico-pathological correlation: Once the central retinal vein is occluded, drainage of blood through the venous system is resisted. This increased resistance causes stagnation of blood and resultant ischemic damage. It has been shown that ischemic damage to the retina in eyes with CRVO stimulates increased production of vascular endothelial growth factor (VEGF).¹⁴VEGF has been shown to stimulate neovascularization within the eye, of both the posterior and anterior segment as well as leading to capillary leakage and causing macular edema and resultant vision loss.

CRVO is classified into two distinct entities—ischemic and non-ischemic—each with differing clinical presentation, prognosis, complications and management options. On initial presentation, it is often difficult to predict the course, since CRVO may change with time. An intermediate form also exists, but 80 percent of these intermediate cases progress to the ischemic variety over time. Non ischemic CRVO is considered to occur due to obstruction of the central retinal vein and ischemic CRVO due to obstruction to both the vein and central retinal artery.

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In accordance with the pathogenesis, patients with CRVO typically present with sudden loss of central vision. Upon early examination, fundus examination reveals generalized venous dilation and tortuosity, superficial and deep intraretinal hemorrhages, and/or cotton wool spots. Accompanying macular and disc edema may be seen. In order to differentiate between ischemic and non-ischemic CRVO, ophthalmoscopy alone is inadequate. Clinically, visual acuity <20/200, the presence of an afferent papillary defect, and extensive retinal hemorrhages are consistent with ischemic CRVO. Abnormal ancillary test results such as an altered electroretinogram (reduced scotopic and photopic b-wave amplitude), widespread non-perfusion (>10 disc areas) on fluorescein angiography, and peripheral visual field defects (as assessed by Goldmann perimetry) indicate ischemic CRVO. In some cases it may be difficult to assess the perfusion status by FA until the retinal hemorrhages resolve. Other angiographic signs of ischemia include severe large-vessel leakage, marked macular edema and an arteriovenous transit time of greater than 20 seconds.

Natural history of CRVO: 15

Neovascularization: Non-ischemic CRVO eyes do not develop neovascularization unless associated with another disease process such as diabetes mellitus or ocular ischemia.¹⁶ In ischemic CRVO the risk of developing anterior segment NV exists mainly during the first 7-8 months of the disease, the maximum risk of developing NVG is about 45%, and 1/3 of eyes with iris NV and ¹/₄ of eyes with iris and angle NV do not progress to develop NVG.

Cumulative chances of conversion of non-ischemic CRVO to ischemic CRVO during follow-up: It has been found that in a series of 500 eyes with non-ischemic CRVO, from the time of onset conversion happened within 6 months in 9.4% and reached almost its maximum within 18 months in 12.6%.¹⁷

Resolution of retinopathy: With a great degree of variation, both types of CRVO are *self-limiting diseases*, with the retinopathy spontaneous resolution of retinopathy. It may occur faster in younger than older people and during the period of active disease various complications may be noted.

Visual outcome:^{18,19,20,21} In a natural history study by Hayreh¹⁸ on non ischemic CRVO, following visual results were obtained:

Hayreh et al found that a visual acuity of better than

Final visual acuity after resolution of retinopathy					
Visual acuity	Non-ischemic CRVO				
20/15 to 20/40	65%				
20/50 to 20/80	9%				
20/100 to 20/200	11%				
20/400	8%				
CF or worse*	7%				
Total eyes	144				
* = Main cause of poor vision other than retinopathy, e.g., cataract, macular degeneration, glaucoma, etc. CF = Counting fingers					

20/200 (6/60) was seen in 58% of the non-ischemic, as compared to only 1.7% in the ischemic type of CRVO. A visual acuity of better than 20/400 (6/120) was seen in 81% of the patients of the non-ischemic, as compared to about 7% of the ischemic type of CRVO only. A visual acuity of 20/400 or worse was seen in only 19% of the non-ischemic, whereas it was seen in 93% of the ischemic type of CRVO. Thus, if an eye with CRVO has 20/400 or worse vision, then there is about a 90% chance that that eye has ischemic CRVO.

In more than 90% of patients with ischemic CRVO, final visual acuity may be 20/200 or worse.

According to the central vein occlusion study,¹⁹ visual acuity outcome was largely dependent on initial acuity. Sixty-five percent of patients with initially good visual acuity (20/40 or better) maintained visual acuity in the same range at the end of the study. Patients with intermediate initial acuity (20/50-20/200) showed a variable outcome: 19% improved to better than 20/50, 44% stayed in the intermediate group, and 37% had final visual acuity worse than 20/200. Patients who had poor visual acuity at the first visit (< 20/200) had an 80% chance of having a visual acuity less than 20/200 at final visit, whether perfused or nonperfused initially. In the first 4 months of follow-up, 81 (15%) of the 547 eyes with perfusion converted to ischemia. During the next 32 months of follow-up, an additional 19% of eyes were found to have converted to ischemia for a total of 34% after 3 years. The development of nonperfusion or ischemia was most rapid in the first 4 months and progressed continuously throughout the entire duration of follow-up. Iris neovascularization (INV) of at least 2-clock hours, and/ or angle neovascularization (ANV) developed in 117 (16%) of the 714 eyes. Sixty-one of the 117 eyes that had INV/ ANV were initially categorized as nonperfused or indeterminate; 56 of the 117 eyes were initially categorized as perfused. When INV/ANV occurred, it was treated promptly with panretinal photocoagulation. The strongest

predictors of INV/ANV were visual acuity (P < .001) and the amount of nonperfusion seen by fluorescein angiogram (P < .001). For eyes initially categorized as nonperfused or indeterminate, 35% (61/176) developed INV/ANV, compared with 10% (56/538) for eyes initially categorized as perfused. Other risk factors were venous tortuosity (P = .02), extensive retinal hemorrhage (P = .07), and duration less than 1 month (P = .08). Neovascular glaucoma that was unsuccessfully managed with medical treatment developed in only 10 eyes. No eye was enucleated. Thus, visual acuity at baseline is a strong predictor of visual acuity at 3 years for eyes with good vision and eyes with poor vision, but a poor predictor for intermediate acuities. Visual acuity is also a strong predictor for the development of INV/ANV, as is nonperfusion. During the course of follow-up, one third of the eyes with perfusion converted to eyes with ischemia.

Fluorescein Fundus Angiography: In fluorescein fundus angiography, typically the retinal capillary non-perfusion or obliteration is considered the diagnostic criterion of ischemic CRVO. In addition, fluorescein angiography may reveal delayed arteriovenous transit, staining along the retinal veins, microaneurysms, arteriovenous collaterals, NVD, NVE, and dilated optic nerve head capillaries. Resolved CRVO may be completely normal.

Macular edema may be detected as leakage from perifoveal capillaries, leakage from microaneurysms, or diffuse leakage on fluorescein angiography. If extensive edema is present, fluorescein angiography may show pooling of dye in large cystoid spaces. In addition, capillary nonperfusion around the fovea may indicate macular ischemia.

However, often the quality of angiograms in unreliable due to media opacities due to cataracts, small pupil, etc. In addition, there are multiple limitations in the evaluation of retinal capillary non-perfusion by fluorescein fundus angiography in CRVO, especially in fresh cases. These limitations include:

- During the very early stages, in spite of retinal ischemia, retinal capillary non-perfusion may not be seen, because it takes time for the retinal capillaries to obliterate completely.
- If there are extensive retinal hemorrhages, it is usually almost impossible to evaluate capillary non-perfusion accurately, because of the masking effect by the blood.

A recent multicenter study ²²on CRVO show ed that eyes with less than 30 disc diameters of retinal capillary nonperfusion and no other risk factor are at low risk for developing iris/angle NV, "whereas eyes with 75 disc diameters or more are at highest risk". Thus "10 disc area of retinal capillary obliteration" is a poor and unreliable parameter in differentiating ischemic from non-ischemic CRVO as well as predicting ocular NV." Optical coherence tomography: Optical coherence tomography (OCT) scanning is a noninvasive, noncontact, transpupillary imaging technology that can image retinal structures in vivo with a resolution of 10-17 μ m. OCT can detect subtle macular edema in the presence of significant hemorrhages, which is not evident by fluorescein angiography because of blockage from hemorrhage. It is useful in quantitatively monitoring the development of macular edema and its resolution.

Treatment

It is clear from the Central Vein Occlusion Study that when left to follow its known course, the vision in patients with CRVO will most likely worsen or remain unchanged and that those patients with poor vision initially have little hope of significant spontaneous recovery. Current treatment is based on the central vein occlusion study^{22,23} recommendations. All patients should undergo close follow up (every three to four weeks) including undilated slit lamp examination of the iris and gonioscopy. Panretinal photocoagulation (PRP) is recommended if and when iris or angle neovascularization is detected (the CVOS used two clock hours of NVI). Prophylactic panretinal photocoagulation for ischemic CRVO is recommended only if close follow-up is not possible. Eyes treated with prophylactic laser were less likely to develop neovascularization of the iris or angle, but this result was not statistically significant. In addition, prompt regression of anterior segment neovascularization was more likely to occur in eyes that had not received prophylactic PRP (56%) than those who received prophylactic PRP (22%). According to the CVOS, grid laser photocoagulation for macular edema results only in angiographic improvement but not visual improvement and therefore is not recommended.

Laser induced anastomosis²⁴ between a retinal vein and the choroid is a procedure that seeks to bypass the central retinal vein outflow. At a site at least three disk diameters away from the optic nerve and usually along the inferotemporal vein, a green argon laser using a power of 2.5 to 3.5 W creates a small spot to rupture Bruch's membrane and a second spot to rupture an adjacent venule. The anastomosis bypasses the postulated obstruction at the lamina cribrosa and creates another outflow for the venous blood.

Limitations of this technique include a low success rate (30 to 50 percent) and complications such as vitreous hemorrhage, choroidal neovascularization, preretinal fibrosis and traction retinal detachment.

Surgical treatment: Thrombolytic agents such as t-PA (tissue plasminogen activator) have been used via the systemic, intravitreous,²⁵ and retinal venous route.²⁶ Systemic administration is associated with the risk of

systemic and intraocular bleeding. Intravitreous administration of t-PA shows outcomes that are similar to the natural history of CRVO. Cannulation and injection of t-PA into a peripapillary branch retinal vein specifically targets the thrombus at the lamina cribrosa, but it is not clear if the reported beneficial effects are from lysis of the thrombus or from mechanical dilation of the central retinal vein. This technique (retinal venous route) may work via several different mechanisms. Vitrectomy with peeling of the posterior hyaloid face may help reduce macular edema. t-PA itself may lyse the clot, and the bolus of fluid may "flush" the thrombus downstream. It is more logical to use it in acute phase of the disease before the thrombus organizes as in the treatment of stroke where most of the success of t-PA lies within first few hours of the thrombotic event. Soon after forming, the thrombus becomes organized and in a long-standing occlusion, it may be difficult to dissolve.

Other evidence suggests that the inner retina may suffer irreparable damage after several weeks and that even if a clot is dislodged, the retina may be too damaged to recover. Also, if collateral vessels have time to form, t-PA may be routed away from the thrombus and may not reach it in sufficient amount to dislodge the clot.

Radial optic neurotomy^{27, 28,29} (figure 3) is a procedure that attempts to decompress the central retinal vein at the lamina cribrosa to release the 'napkin-ring / bottle-neck' constriction. A radial incision of the lamina and sclera is made at the nasal edge of the optic disc. The proposed mechanisms for its functional results include relief of mechanical pressure exerted on the central retinal vein by the distended optic nerve, the creation of chorioretinal anastomosis (42- 46% cases) and the enhancement in the exchange between retina and vitreous cavity secondary to posterior hyaloid peeling thereby reducing oedema.. The complications associated are retinal detachment, vitreous haemorrhage, globe perforation, laceration of central retinal vein & artery, optic nerve damage.

We published encouraging results with this procedure.³⁰ 83.33% of our 24 eyes with CRVO and vision <6/60 showed increase in vision (of average 3 lines). Decline in macular edema decreased or resolved intra retinal haemorrhages resolution of venous dilatation and disc edema could be appreciated in all cases. Average Pre- and post-operative macular thickness on OCT was 834.17 and 556.17 microns. Both visual and OCT findings were statistically significant.

Intravitreous triamcinolone acetonide (IVTA)(figures1,2): In patients with macular edema, injection of triamcinolone (0.1 mL/ 4 mg) into the vitreous cavity through pars plana has been shown to be effective not only in resolving the edema but also in corresponding improvement in vision. It has been used to treat macular edema in both ischemic

and non-ischemic CRVO.³¹⁻³⁴ Reduction in macular thickness with improved visual acuity can last three to six months; however, improvement is unlikely in eyes with chronic macular edema and secondary macular changes. Furthermore, especially in the ischemic group, visual acuity may not improve despite resolution of macular edema

The role of corticosteroids in the treatment of macular oedema is based primarily on the inhibition of biosynthetic pathways of leukotrienes and prostaglandins, the inflammatory mediators implicated in the pathogenesis of macular oedema. It inhibits the expression of inflammatory adhesion molecules thus contributing to the stabilization of blood-retinal barrier thereby reducing cellular permeability. It has also been shown to downregulate the induction of VEGF by platelet-derived growth factor and platelet-activating factor in time and dose dependent manner.³⁵ This leads to a reduction in capillary permeability and macular edema.

The main drawback of an injection of triamcinolone remains the post treatment recurrence of macular edema, requiring repeat triamcinolone injections, often every 3-6 months. Researchers estimate the risk of non-infectious endophthalmitis after IVTA injection to be as high as 2 percent, and the risk of infectious endophthalmitis to be between 0.16 and 0.6 percent.^{36,37} In reports of outcomes following intravitreal triamcinolone injection for the treatment of macular edema, up to a third of patients may develop an IOP increase.³⁸

Role of anti-VEGF agents(figure 4): ^{39,40,41}The role of VEGF is already clear in the pathogenesis of macular oedema.³⁹ It is also known that VEGF is essential and sufficient to produce pathological neovascularisation and blocking VEGF inhibits abnormal vessel growth while sparing normal vessels. With this background lot of research is on to evaluate the efficacy of anti-VEGF agents in CRVO. A correlation has been found between aqueous VEGF concentrations and the onset, persistence, and regression of NVI; extent of retinal capillary nonperfusion; and vascular permeability. NVI occurred when aqueous VEGF concentrations were 849 to 1569 pg/mL and regressed fully when they fell below 550 pg/mL. The close temporal correlation between aqueous VEGF levels and the course of neovascularization and permeability in human ischemic central retinal vein occlusion indicates that increased aqueous VEGF level may predict the need for treatment, and that anti-VEGF therapy at an early stage of ischemic central retinal vein occlusion may be therapeutically beneficial, as shown by Iturralde, et al.

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Understanding Oblique and Vertical Muscle Dysfunction Physiology of Extra Ocular Muscles and Eye Movements

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6 extra ocular muscles (Four recti and two obliques) ensure precise movement of each eye ball in the orbit. Eye movement and position are both influenced by a number of factors. The shape of the orbit, the points of origin and insertion of the muscles, the course or the path of the muscle in the orbit, and connective tissue of the orbit the EOM pulleys are some of them. The inter muscular septa and the check ligaments prevent the side slip of the extra ocular muscles on the globe. The vertical slip of the horizontal recti on the globe, when the eye moves up or down and the horizontal slip of the vertical recti when the eye moves sideways are very minimal.

The retinal sensorial input is of paramount importance in deciding the extent of action of a given extra ocular muscle and also appropriate muscle combinations. For example temporal disparity of images on the retina, stimulates a convergence eye movement leading to precise contraction of both the medial rectus muscles, till the image is positioned on the foveae again and a nasal disparity encourages a divergence eye movement by a precise contraction of the two lateral rectus muscles. Temporal disparity in one eye and nasal disparity in the other eye stimulates a version or saccadic eye motion depending on the velocity of moving object.

The precise eye movement due to contraction of the extra ocular muscles is in turn due to retinomotor value of the retinal receptor. The fovea has zero retinomotor value which means that the eye will not move ones the image is positioned on the fovea. The farther away the retinal receptor is from the fovea, the greater is its retinomotor value for obvious reasons. To bring the image on the far periphery of the retina on to fovea, the eye will have to make a greater excursion, than say an image nearer the fovea.

Anatomical variations in origin and insertion of agonist and its antagonist muscle can cause functional abnormalities even if the extra ocular muscles and innervations are perfectly normal. The action of any given extra ocular muscle is further dependent on the angle between the muscle plane and visual axis of the eye. This angle can vary according to the position of the globe in the orbit. For example the lateral recti act as pure abductors in

Kamala Nethralaya, 81, 7th Cross, Koramangala, Bangalore primary position and as the eye moves up or down the lateral recti also contribute to elevation and depression.

There are four extra ocular muscles involved in vertical and torsional movements of each eye. A precise understanding of their structure and function is important to learn their dysfunction. The insertion of an EOM is not a point insertion but a line. The line of insertion in case of the horizontal recti is concentric with the limbus and in vertical recti is oblique and does not correspond to the limbus. In case of obliques, the insertion is quite tortuous. This is the reason for the multiple actions of the oblique and vertical muscles.

A vertical deviation is a vertical misalignment of visual axes named after the vertically deviating non-fixating eye. Vertical deviation can be paralytic or restrictive. Some of the common vertical/oblique muscle dysfunctions seen in clinical practice are follows:

The diagnosis, differential diagnosis and management of these entities are discussed.

Superior Oblique Palsy

Superior Oblique Palsy is mostly congenital. Acquired SO palsy is commonly traumatic in origin, though there are many other causes. Severe closed head injury associated with loss of consciousness usually gives rise to bilateral SO palsy.

Superior oblique palsy patient presents with vertical diplopia more pronounced on down gaze (such as while descending stairs and while reading) and with characteristic head posture. Head is tilted to the opposite side & face turned towards uninvolved side and chin down position. Cover test has to be performed in all the cardinal gaze positions or else subtle palsy may be missed. Facial asymmetry is quite marked in congenital cases. There is hypoplasia of the face on the side of head tilt.





Right superior oblique palsy

Left superior oblique palsy



In left gaze the up shoot is

With head posture the eyes are straight







Bielchowsky's head tilt test is positive to involved side. Here the left eye is involved and notice that the left eye shoots up when the head is tilted to the left shoulder. But the eye position remains more or less normal when the head is tilted to the right shoulder. To attain binocularity the patient would tilt the head to a position where the eyes would be straighter. In this case the patient had a head tilt to the right shoulder.

While planning treatment, the actions of the involved SO, the antagonist IO, yoke muscle IR & it's antagonist SR have to be carefully checked. Indications for treatment are abnormal head posture and diplopia.

One scheme of treatment of SO palsy proposed by Knapp is as follows.

Note where is the maximum diplopia in the 9 positions of gaze. Depending on that the options could be Ipsilateral inferior oblique recession/ superior oblique tucking/Harada Ito's procedure and contralateral inferior rectus recession or superior rectus resection as well.

Inferior Oblique Over action

Inferior Oblique Over action may be primary or secondary to SO palsy. Etiology of PIOOA (primary inferior oblique over action) is not well understood. It could be due to mechanical or innervational factors.



Bilateral primary Inferior Oblique Over Action

Note that the adducting eye elevates on examining versions.

Treatment is by maximal weakening of Inferior Oblique.

Dissociated Vertical Deviation

DVD is a common innervational disorder usually associated with congenital esotropia. It is characterized by elevation & extorsion of either eye when the other eye is occluded or during periods of visual inattention. It doesn't obey Hering's law in that the other eye does not move down when hyper tropic eye fixates. DVD may be spontaneous or latent.





Dissociated Vertical Deviation

Treatment of DVD is by antero-position of inferior oblique (if there is an associated IO over action as well) and SR recession and Fadenisation.

Brown's Syndrome

Congenital or acquired shortening of SO tendon sheath can cause limitation of elevation in adduction and can be confused with IO palsy. Diagnosis is by forced duction test.

Treatment: Tenotomy of the posterior two thirds of the Superior oblique tendon if found to be tight.

Blow out fracture of Orbital floor

An orbital blow out fracture can cause herniation of orbital contents into maxillary antrum and also entrapment of IR or IO muscle. The patient presents with history of trauma, vertical diplopia, limitation of elevation, depression with enophthalmos and hypesthesia. FDT and radiological studies clinch diagnosis.

Treatment involves repair of the orbital floor combined with Inferior rectus recession at a later date.

Double Elevator Palsy

It is very rare for both IO and SR to be involved. Most cases of double elevator palsy are in fact SR paresis with contracture of IR and spread of comitance. The double elevator palsy may be associated with true and pseudo ptosis. Treatment involves a two stage procedure of muscle transplantation and ptosis correction followed by inferior



Treatment is indicated if DVD is spontaneous, frequent or is cosmetically significant.



Brown's Syndrome Right Eye



Brown's Syndrome Left Eye



Clinical photograph

rectus recession after 6 months to a year. The artery sparing Inferior rectus recession is a better alternative to prevent anterior segment ischaemia.

Inferior rectus fibrosis

This can result from endocrine myopathy, primary fibrosis of EOM, as well as from accidental xylocaine injection into the muscle prior to cataract surgery. FDT is positive.

Patient presents with vertical diplopia and limitation of elevation. IR recession can relieve diplopia.

A & V patterns

20% to 50% of patients with horizontally comitant strabismus have A or V pattern. In the proper management of horizontal strabismus, recognition and treatment of these patterns are important.

A pattern is significant when the difference in deviation between upward gaze and downward gaze is 10 prism dioptre or more and V pattern when the difference is 15 prism dioptre or more.

Pattern strabismus may be associated with head posture such as chin elevation or chin depression. Increase in deviation in down gaze is functionally more significant.

Aetiology of AV pattern strabismus

Various etiological factors are proposed such as



Clinical photograph



Inferior Rectus fibrosis following cataract surgery



V Exotropia



V Esotropia

horizontal, vertical, oblique muscle dysfunction as well as facial characteristics and abnormal muscle insertions. Clinically the most important etiological factor is oblique dysfunction. Most cases are now thought to be due to oblique dysfunction. How ever the contrary over action of obliques like for example the inferior oblique over action in A and superior oblique in V tropias makes it difficult to substantiate this theory in all cases.

Abnormal path of the extra ocular muscles particularly the recti, due to heterotopic pulleys as an important cause of A-V pattern strabismus is gaining acceptance. This can be demonstrated with MRI. A connective tissue sheath at mid orbital level connects the extra ocular muscle to the orbit. This connective tissue has smooth muscle fibers, which are influenced by sympathetic and parasympathetic fibers. Contraction of these smooth muscle fibers of the pulleys of EOM, would alter the direction of pull and therefore the force of the EOM. This can explain the AV phenomenon. In cases of V tropias the Medial recti are relatively shifted up and lateral recti are shifted down and the opposite in cases of A tropias. Hence the medial recti have to be shifted down in treating V tropias and Lateral recti have to be shifted up and opposite in cases of A tropias.

Treatment

The surgical goals of treatment are the same viz: to maintain BSV, to eliminate head posture or cosmetic. Conventional unilateral or bilateral surgical procedures for horizontal strabismus can be combined with slanting recessions or muscle displacements in the absence of demonstrable oblique over action. Strengthening or weakening of appropriate oblique muscles, when indicated, has proved effective in patients with A & V patterns.

Bilateral superior oblique recession or posterior

tenotomy is more effective in A tropias than are bilateral inferior oblique recessions are in alleviating V tropias.

Nearly 40 prisms dioptres of A deviation can be corrected by the superior oblique surgery alone. Bilateral inferior oblique recession corrects up to 25 prism dioptres of V.

While treating AV pattern strabismus its important to treat the associated over action of the obliques at the same time as treating the horizontal muscles.

It is important not to tackle the obliques in cases of pattern strabismus, where it is not indicated.

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Cultivated Limbal Stem Cell Transplantation: Promise and Practice

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The ocular surface comprised of by the epithelia of the palpebral and bulbar conjunctiva and the corneal epithelium, is in a state of dynamic equilibrium. More than two decades ago, Thoft and Friend described the X, Y and Z hypothesis which explained how corneal epithelial cells are continuously shed from the surface and regenerated by cells from the periphery.¹ It was previously thought that conjunctival transdifferentiation was responsible for replenishing the corneal epithelium following surface damage.²³ However, the limbal stem cells residing in the palisades of Vogt are now believed to be the never-ending source of corneal epithelial cells.⁴

The conjunctival epithelium and underlying stroma with lymphoid tissue protects the underlying sclera and provides nutrition to the sclera and cornea through its blood vessels. Goblet cells present in the conjunctiva secrete mucin which forms an important constituent of the tears and renders the hydrophobic corneal surface into a hydrophilic one to enable the spread of tears.

The corneal epithelium provides a water-tight barrier which protects the underlying stroma and endothelium and maintains the transparent refractive surface.

Ocular surface failure may be of two types depending on the epithelial phenotype as identified by impression cytology.⁵ Type 1 failure is characterised by squamous metaplasia where the non-keratinised corneal epithelium is converted to a keratinised epithelium. Type 2 failure is characterised by limbal stem cell deficiency where the normal corneal epithelium is replaced by conjunctival epithelium.

Promise of Stem Cells for Incurable Eye Disease

The use of stem cells to regenerate and repair tissues and organs has been the subject of intense scientific and media interest. There are practical and ethical issues to the use of embryonic stem cells and the alternative of using somatic or adult stem cells has major advantage in terms of immediate clinical application. The adult human eye harbors stem cells in the limbal region, in the conjunctiva, the pars plana & plicata of the retinal ciliary margin and adult human retina. The successful use of *ex-vivo* cultivated limbal epithelial stem cells to treat limbal stem cell deficiency provides a model for treatment of other incurable eye diseases in future.

Practice of Limbal Stem Cell Transplantation for Ocular Surface Reconstruction

Limbal stem cell deficiency (LSCD) (Figure 1) characterised by conjunctivalisation of the corneal surface with neovascularisation and chronic inflammation⁶⁻⁸ is managed by limbal stem cell transplantation which provides the cells required to replenish the corneal epithelium and restore the ocular surface integrity. Management depends on whether the condition is unilateral or bilateral and whether some or all of the stem cells are lost. Partial stem cell deficiency (Figure 2) may only require observation if the patient is asymptomatic,⁹ conjunctival debridement,⁹ or amniotic membrane transplantation.¹⁰⁻¹³

Successful transplantation of 8 clock hours of conjunctival limbal autograft from the healthy fellow eye for a large series of cases with unilateral LSCD was first reported by Kenyon and Tseng in 1989.¹⁴ Bilateral cases require allografts either from living-related donors or cadaveric tissue with lifelong immunosuppression to prevent the risk of rejection. The results of all these



Fig.1: Slit lamp photo showing causes of limbal stem cell deficiency (LSCD), **(A)** Corneal pannus due to Vernal Keratoconjunctivitis (VKC). **(B)** Dysfunction of limbal stem cell in a patient with VKC. **(C)** Total stem cell deficiency with conjunctivalisation and multiple symblephara in a patient with chemical injury. **(D)** Peripheral corneal destruction due to Mooren's ulcer which leads to limbal stem cell deficiency over a period of time.

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techniques have been summarised in a recent review.¹⁵ The long-term outcome of limbal stem cell transplantation is awaited.

Limbal Stem Cells: Pathophysiology

Evidence regarding the existence of limbal stem cells at the limbus is suggested by indirect experimental and clinical evidence, the description of which is beyond the scope of this review but has been compiled earlier.¹⁶ These cells function as a barrier preventing the growth of conjunctiva onto the cornea and replenish the corneal epithelial cells. The cells have a long life with a slow cell cycle and the capability of error-free replication. Upon replication, one daughter cell remains at the limbus thereby ensuring that the stem cell pool remains unaltered while the other



Fig. 2: Slit lamp photo of total (a) and partial (b) limbal stem cell deficiency due to chemical injury.

by demonstrating the presence of goblet cells on the cornea by impression cytology and histopathology or by showing the breakdown of the epithelial barrier by fluorophotometry.

Management of LSCD: Algorithm

Prior to limbal stem cell transplantation, concomitant lid pathology, dry eye and associated systemic disorders have to be treated as these may adversely affect the outcome of surgery. Figure 4 shows the algorithm for management of LSCD. Unilateral cases may have partial or total LSCD, the management of the former has been described earlier in this review. Total LSCD requires an autograft which may be harvested from the healthy fellow eye, and either transplanted directly or following

cultivation of stem cells ex vivo. An allograft may have to be used in case the recipient is one-eyed. In bilateral cases, the donor tissue may be obtained either from a cadaveric or a living related donor and either transplanted directly or after culturing the stem cells. To ensure the viability of the transplanted stem cells and prevent rejection, immunosuppression is necessary for all allografts.

Surgical Management

Role of Amniotic Membrane (AM)

Since LSCD manifests with recurrent epithelial breakdown, neovascularisation and chronic inflammation, amniotic membrane transplantation prior to stem cell transplantation may have a therapeutic benefit as amniotic



Fig.3: Schematic diagram explaining the steps in multiplication of limbal stem cell and their differentiation. **SC:** Stem cells, **TAC:** Transient amplifying cells, **PMC:** Post mitotic cells, **TDC:** Terminally differentiated cells.

forms the transient amplifying cell (Figure 3).

Limbal stem cell deficiency may be primary, without identifiable external factors and inadequate stromal microenvironment to support stem cells or secondary, due to destruction of stem cells by external factors. Table 1 shows the conditions with primary and secondary LSCD. The signs of LSCD include conjunctivalisation of the cornea with chronic inflammation, neovascularisation and recurrent epithelial breakdown.¹⁷ This may be confirmed

Table 1: Causes of Limbal Stem Cell Deficiency (LSCD)					
Primary LSCD (Insufficient microenvironment)	Secondary LSCD (LSC destruction by external factors)				
Aniridia	Chemical or thermal injuries				
Erythrokeratoderma	Ultraviolet and ionising radiation				
Multiple endocrine deficiency	Stevens-Johnson syndrome(SJS)				
Neurotrophic keratopathy	Ocular cicatricial pemphigoid(OCP)				
	Contact lens wear				
	Servere microbial infection				
	Multiple ocular surgeries				



membrane promotes epithelialisation,¹⁸ and reduces angiogenesis and inflammation.^{19,20}

Amniotic membrane also preserves and maintains the epithelial progenitor cells and has been used successfully instead of limbal transplantation, in the management of partial limbal stem cell deficiency ranging from 40-330°.^{10-13,21}

Live-Related Vs Cadaveric Limbal Transplantation (LT)

In unilateral and bilateral cases of LSCD, fresh donor tissue may be harvested either from the healthy fellow eye in the former, or from a live-related donor or corneo-scleral rim or enucleated eye of a cadaver in case of the latter.

It is important to exclude limbal stem cell deficiency in the fellow eye especially since some conditions like chemical injury may be bilateral, and pseudopterygium has been described in the apparently healthy donor eye.²² Upto 8 clock hours of tissue may be excised from the healthy eye of the donor though we recommend upto 6 clock hours.¹⁵ We also recommend harvesting tissue from one eye each (3 clock hours superiorly and inferiorly) of 2 relatives, thereby ensuring 360° coverage of the limbus of the recipient.

It is essential to screen the donor thoroughly prior to surgery for evidence of limbal stem deficiency. Close monitoring of the donor is advised after surgery, as it is yet unknown how these eyes will respond to an epithelial insult in the future and there may be stem cell attrition due to

inflammation and sub-clinical donor disease.23,24

The other alternative in bilateral cases is cadaveric limbal allo-transplantation. The following recommendations for harvesting limbal tissue from a cadaveric donor have been made: ²⁵

- 1. Maximum age limit of the donor should be 50 years
- 2. The surgery should be performed within 72 hours since the cells are expected to be more viable.
- 3. The scleral rim should be 4-5 mm wide, conjunctival tissue adjacent to the limbus should be preserved and damage to the epithelium should be avoided.

The advantages and disadvantages of kerato-limbal allograft and conjunctival limbal allograft are shown in Table 2.

Table 2: Advantages and disadvantages of conjunctival limbal allograft and kerato-limbal allograft								
Conjunctival	l limbal allograft	Kerato-limbal allograft						
Advantages	Disadvantages	Advantages	Disadvantages					
Fresh tissue provides more viable stem cells	Risk of iatrogenic LSCD	Readily available tissue	Fresh tissue, immediate surgery preferred					
Reduced antigenic load since minimal stroma harvested	Transplantation of vascularised tissue potentially increases risk of rejection	360° limbal coverage possible	Doubtful viability of stem cells					
HLA matching reduces risk of rejection		Simultaneous PKP with central cornea	Increased risk of rejection					
Technically easier		Normal healthy eyes spared	Technically difficult					
LSCD: Limbal stem cell deficiency; HLA: Human leukocyte antigen; PKP: Penetrating keratoplasty								

The growth potential of fresh tissue harvested from a living related donor was shown to be two times better than that from a cadaver, however once growth had commenced, there was no difference between the two groups.²⁶

Cultivated Vs Direct Limbal Transplantation

The potential problems with direct limbal transplantation include harvesting a relatively large amount of limbal tissue (upto 6 clock hours) from healthy donor eyes with the risk of inducing iatrogenic limbal stem cell deficiency. It is unknown how these eyes will respond to epithelial trauma in the future. There may be stem cell attrition with time resulting in epithelial breakdown.

Pellegrini et al²⁷ were the first to describe culturing of limbal stem cells ex vivo thereby spear-heading ophthalmology into the field of regenerative medicine.

The basic principle of this procedure involves harvesting a small population of the limbal epithelium which presumably contains LSC and cultivating these cells under controlled conditions in a laboratory.

A limbal biopsy (approximately 2x1mm²) is taken from the donor eye thereby minimising the chance of LSCD in the donor. In addition a lesser amount of Langerhan's cells and blood vessels are transplanted to the recipient theoretically reducing the risk of allograft rejection in bilateral cases.

It may be possible to store the donor tissue in a stem cell bank obviating the need for repeated surgeries in the future.

Technically, the only disadvantages of the procedure are

- 1. It takes 10 days to 2 weeks for the cells to grow and hence there is a gap between harvesting and transplanting the cultivated tissue.
- 2. A dedicated stem cell laboratory is required for culturing the cells and hence the facility may be available at tertiary eye care centres.

Autologous Vs Allogeneic LT

Unilateral cases of LSCD require an autograft while bilateral cases need an allograft from a live related donor or a cadaver. Indefinite immunosuppression is required to prolong graft survival though it may not prevent allograft rejection.²⁸ Immunosuppression may have potential for systemic side effects and the cost may be prohibitive.

Cultivated Limbal Epithelium Transplantation

Case Selection / Indications

The cases in which cultivated limbal epithelial transplantation is indicated would depend on the etiology and severity of LSCD.

Limbal stem cell transplantation may not be effective in all cases of limbal stem cell deficiency. In primary LSCD in which the stromal microenvironment is unable to support stem cells, limbal transplantation may not be beneficial. However in secondary LSCD due to chemical injury, Stevens-Johnson (SJS), Ocular cicatricial pemphigoid (OCP), ionising radiation or iatrogenic causes, LT may stabilise the damaged ocular surface. The outcome of LT in these conditions is variable and better for chemical injury.²⁹ In SJS or OCP, underlying ocular surface inflammation, dry eye and associated adnexal problems may adversely affect the outcome.

Limbal Biopsy

The donor eye is closely examined for any evidence suggestive of limbal stem cell deficiency by taking a detailed history and assessing for loss of the palisades of Vogt, epithelial breakdown, chronic inflammation or corneal neovascularisation.

The site of biopsy depends on the severity and laterality of the condition and may be taken from the ipsilateral eye in unilateral partial LSCD or the contralateral eye in unilateral total LSCD. In one-eyed patients with LSCD in one eye and phthisis bulbi with intact palisades of Vogt in the fellow eye, a limbal biopsy can be taken from the phthisical eye. In bilateral cases with asymmetric involvement and partial LSCD in one eye, the biopsy may be taken from the eye with less severe damage. It is difficult to predict the outcome in cases wherein the tissue is harvested from the ipsilateral eye. In bilateral cases with total LSCD, the only option is an allograft harvested from a live-related donor or a cadaver.

Informed consent is taken from the patient or the guardian. Under strict aseptic precautions under topical, peribulbar or general anesthesia (depending on the age and compliance of the patient), with a conjunctival spring scissors, a conjunctival peritomy is made 2-3 mm from the limbus and dissection of the conjunctiva is carried forwards 1mm beyond the limbal arcade. The harvested tissue should exclude the Tenon's capsule and should include the palisades of Vogt. A 1x2mm² piece of tissue is excised and placed in Human Corneal Epithelial Cell Medium (HCEC medium). It is then transported to the stem cell laboratory.

Post operatively topical antibiotic is used four times day for two weeks. Topical steroids are used in tapering doses for 4 to 6 weeks or until complete healing of donor site.

Growth On Amniotic Membrane by Explant Tissue Culture Technique (Figure 5)

Placed in the constituents of the culture medium, the limbal epithelial cells would multiply and form a sheet.

However in order to transplant this sheet onto the ocular surface, a medium or substrate is required. In early experimental studies, silicone rubber film was used to sandwich the cultured epithelial cells and transplant them as orthotopic xenografts in mice.³⁰ Subsequently, petrolatum gauze and a hydrophilic bandage soft contact lens were used as carriers for expansion of the limbal stem cells in two patients with LSCD.²⁷ Other carriers like type 1 collagen, collagen shields and corneal stroma were also used.

Widespread use of amniotic membrane in promoting epithelial healing both as an onlay and an inlay material^{18,31-34} led to its inevitable use in limbal stem cell transplantation. In a rabbit experiment, heterologous limbal epithelial sheets were separated from the corneo-scleral rim by dispase. The sheets were then placed on an amniotic membrane sheet anchored to the recipient bed of rabbits with total LSCD, and then covered by another sheet of amniotic membrane. Complete epithelisation

was noted a week after this procedure. This finding was not seen in the controls, which had only AM transplanted.³⁵

Tsai et al³⁶ were the first to culture autologous limbal epithelium on AM and transplant both onto the damaged ocular surface of 6 patients with complete reepithelialisation in a week, maintained throughout follow up. A rabbit experiment produced similar results.³⁷

Several groups of workers thereafter have examined the anatomy, ultrastructure and function of the human limbal epithelial cells cultured on AM, including the role of AM in promoting the growth of these cells. The features of the limbal epithelial stem cells, which differentiate them from TAC, are the lack of cornea specific differentiation keratins (K3/K12) (Figure 6), connexin 43-mediated gap junction intercellular communication, cell cycle duration, BrdU retention.

AM mimics the stromal niche

The limbal stroma influences epithelial differentiation, proliferation and less apoptosis while the corneal stroma promotes differentiation and apoptosis³⁸ indicating that there is a unique environment at the limbus which nurtures the limbal SC. It was suggested that the limbal basal epithelial cells do not communicate and lack gap junction intercellular communications, and this is a feature



Fig.5: Schematic diagram showing the steps in cultivation of limbal epithelial stem cells. **(A)** Technique of limbal biopsy (See text for details) **(B)** Processing of tissue in the laboratory and making the explant culture. **(C)** Petridish, glass slide (white) with de-epithelialized human amniotic membrane with explants (red dots) with growing cells around it (blue dots). **(D)** Monolayer of cells (10–14 days old) under phase contrast microscope.

of the microenvironment in which the LSC lie.³⁹⁻⁴¹ Limbal epithelial cells grown with the 3T3 fibroblast feeder layer system do express connexin 43 ⁴⁰ while with AM cultures having devitalised amniotic epithelium, the limbal epithelial cells are devoid of connexin 43 expression.⁴¹ It is believed that AM with devitalised epithelium promotes transient amplifying (TAC) differentiation providing another valuable clue towards the uniqueness of the stromal niche in which the limbal stem cells lie. ⁴²

AM preserves and expands the LSC in culture

BrdU labelling demonstrated that the slow cycling limbal epithelium is maintained.^{41,43} There is a higher outgrowth rate of limbal epithelial cells cultured on AM as compared to peripheral and central corneal epithelial cells and the cells have all the phenotypic characteristics of in vivo limbal epithelium thereby proving that the AM maintains and expands the human limbal stem cells.^{41,43}

AM and NGF signaling for ex vivo expansion of LSC

Nerve growth factor (NGF) plays an important role in promoting epithelisation mediated via a high affinity receptor tyrosine kinase inducing receptor A (Trk A).⁴⁴ The corneal basal epithelium expresses TrkA *in vivo*⁴⁵ indicating that NGF directly affects the corneal epithelium. Experiments have shown that LSC express TrkA, AM contains significant amounts of NGF and the growth of epithelial cells in culture was stopped by specific TrkA inhibitor thereby indicating that AM per se does affect the growth of LSC.⁴⁶



Fig.6: Photomicrograph of cultivated limbal epithelial cells stained for keratin 3 and 12 **(A)** showing the corneal epithelial phenotype and basement membrane (green) secreted by the growing cells **(B)**.

AM and Growth Factors

Transforming growth factor (TGF b2) inhibits the proliferation of corneal and limbal cells in culture⁴⁶ however this is down regulated for 96 hours in the presence of AM signifying a beneficial effect.⁴⁸ Tissue inhibitor of metalloproteinase 1 (TIMP1) expression is increased during limbal epithelial cell (LEC) culture, increases expression of epidermal growth factor receptor and its effect.⁴⁸

AM and keratocyte culture

Bioengineering the corneal stroma is a distinct possibility as culturing of keratocytes on AM stroma (and not on plastic) yielded keratocytes with dendritic morphology and intercellular networks which expressed keratocan.⁴⁹ However further studies are needed to determine how corneal stromal transparency is influenced.

AM and Corneal Neovascularisation

One of the anti-angiogenic factors expressed by AM is pigment epithelium-derived factor (PEDF) which would have a beneficial effect in reducing corneal neovascularisation seen in LSCD.⁵⁰

AM and Air lifting

Air lifting of corneal epithelial cells on AM promotes more well-formed desmosomal junctions, with less intercellular spaces in between the cells.⁵¹

AM & Cell-suspension vs explant

Since it was suggested that limbal epithelial cells may not migrate readily from the explants, separation of these cells enzymatically has been recommended. Treatment of the limbal explant with dispase forms a suspension of the corneal limbal epithelium (including the stem cells) which is seeded on AM. The growth from a single cell suspension was faster⁵² and a greater number of hemidesmosomal attachments and smaller intercellular spaces were noted with the cell suspension as compared to the explant technique.⁵³

AM and Corneal Endothelium

Experiments with rabbit corneas receiving human corneal endothelial cells cultured on AM demonstrated the possibility of using AM to culture endothelial cells with preservation of their morphology and more importantly their function.⁵⁴

AM preparation for Cultivation

AM is prepared and preserved by our eyebank (Ramayamma International Eye Bank) using a previously described technique.55 Prior to use, the AM is thawed at 37°C for 30 minutes. Under sterile conditions, a 3 x 4mm² piece of AM is de-epithelized by addition of 0.25% trypsin and EDTA solution for 15 minutes and examined under a microscope for complete removal of amniotic epithelial cells. The de-epithelized AM (basement membrane side up) is spread on a glass slide with the edges of the AM tucked under the glass slide and placed in a petri dish containing culture medium.

Growth Medium

The preparation of the culture medium has been described earlier.⁵⁶ The constituents of the culture medium promote epithelial cell proliferation, provide nutrition to the multiplying cells and contain antibacterial and antifungal agents which will prevent contamination.^{57,58}

For culturing the limbal epithelial cells, human corneal epithelial cell medium (HCEC) with 10% fetal calf serum is used. Currently we have switched over to autologous serum instead of fetal bovine serum. HCEC medium is prepared using 9.7g/l modified Eagle medium with the addition of 16.2 g/l Ham F12 serum, 0.01mg/l epidermal growth factor, 0.25mg/l insulin and hydrocortisone. We have discontinued the use of cholera toxin, a mitogen which promotes epithelial migration, due to difficulties in its procurement and our explant outgrowths are unaffected by its absence (unpublished data). The medium is filtered with 0.22 mm membrane filters using a vacuum pump. The fetal calf serum or autologous serum is added to this sterile medium at the time of use.

Processing of Limbal biopsy tissue

The limbal explants obtained from the limbal biopsy are shredded into pieces under a laminar flow hood in the stem cell laboratory and distributed in the centre of the deepithelialised AM. The culture medium is then added and the culture system is incubated at 37°C with 95% air and 5% carbon dioxide. Under a phase contrast microscope, the growth is monitored daily, and the medium is changed every alternate day.⁵⁶

Duration of culture

Within 10-14 days, the growth from the explants becomes confluent and a monolayer of cells is observed. The incubation is terminated at this point of time.

The whole mount preparation, stained with hematoxylin and eosin appears as a monolayer of polygonal cells with an epithelial appearance.⁵⁶

Surgical Technique

The surgery is performed 10-14 days after the limbal biopsy. Following strict aseptic precautions, under local or general anesthesia, a drop of 1:1000 epinephrine is instilled into the conjunctival cul de sac to ensure



Fig. 7: Slit lamp photograph of a patient with total limbal stem cell deficiency (A) with multiple symblephara following acid injury. One year after autologus cultivated (B) limbal stem cell transplantation shows clear central cornea and no recurrence of symblephara and a stable ocular surface.

hemostasis. Dissection of the fibrovascular pannus is started 2-3mm beyond the limbus using a conjunctival spring scissors until the limbus and beyond. A plane of dissection is usually noted in patients who have undergone previous ocular surface reconstruction with amniotic membrane, facilitating the excision.

Symblepharon release is carried out at the same time and amniotic membrane is used to reconstruct the ocular surface. Fornix reconstruction is also done where needed.

The amniotic membrane with its monolayer of cultured limbal epithelial cells is then transferred to the ocular surface and anchored in place at the limbus with 10-0 monofilament nylon sutures. The knots are trimmed and buried. The peripheral skirt of the AM is anchored to the conjunctiva with 8-0 vicryl sutures. Sub conjunctival dexamethasone is given at the end of surgery. Some authors recommend mitomycin C subconjunctivally to prevent recurrence of symblepharon⁵⁹ and a bandage contact lens is inserted.

Since difficulties may be encountered in suturing the donor corneal tissue to the thin recipient bed in patients with chemical injury and corneal scarring who had undergone prior cultivated limbal stem cell transplantation, cultured LSC transplantation may be combined with either a lamellar keratoplasty or deep anterior lamellar keratoplasty. Preoperative anterior segment interferometry or intraoperative pachymetry after pannus resection may indicate the residual stromal thickness and aid the decision for lamellar keratoplasty (LK) or deep anterior lamellar keratoplasty (DALK).

In cases with anterior to mid stromal scarring and thinning, a lamellar keratoplasty may be done using a crescent blade or an automated keratome. The lamellar donor corneal tissue is sutured to the recipient bed with interrupted 10-0 monofilament nylon sutures and the AM with cultivated limbal epithelium is placed on the whole de-epithelized surface.

Alternatively, deep anterior lamellar keratoplasty can be done to provide a smooth interface.

Penetrating Keratoplasty (PK) after Limbal Stem Cell Transplantation (LSCT)

A penetrating keratoplasty may be done at least 3 months post operatively once the ocular surface has stabilised for visual loss related to corneal scarring in the visual axis. The recipient cornea is trephined in the centre with a disposable trephine and a donor tissue 0.5mm larger is anchored to the recipient bed with interrupted 10-0 monofilament sutures. A lensectomy, vitrectomy,

synechiolysis and or intraocular lens implantation may be carried out if deemed necessary. In the immediate postoperative period, it is imperative to watch for signs of epithelial breakdown and allograft rejection.

The early outcome of PK following cultivated limbal epithelial transplantation is very promising with 87% of grafts remaining clear at mean follow up of 8.3 months post PK and 25% developing corneal allograft rejection.⁶⁰

Outcome of LSCT (Figure 7)

Though for almost a decade there were several reports describing the growth and characteristics of corneal epithelial cells in culture, the first report of transplantation of cultivated limbal epithelial cells was in 1997.²⁷

In two patients who underwent cultivated autologous corneal epithelial transplantation, the ocular surface was stable without recurrence of conjunctivalisation for more than two years. Corneal biopsies done 1.5 years after limbal stem cell transplantation showed the presence of cornea specific keratin K3.²⁷

The outcome of cultivated limbal epithelial transplantation in ocular surface disorders is summarised in table 3.

The factors affecting the outcome have been evaluated in a rabbit model.⁶⁵ Eighty-seven percentage (9/12) success was noted 6 months post operatively in eyes with amniotic membrane used as a biological bandage contact lens over the cultured cells. Success was less than 30% when a concomitant tarsorrhaphy and or sub conjunctival triamcinolone acetonide was administered. In addition, severe lid deformities and the appearance of an epithelial defect in the early post operative period were associated with failure.

Long Term Outcome

An interesting observation made by Daya et al⁶⁶ was that DNA analysis of 7 cases, who had undergone cultivated allo-limbal stem cell transplantation with follow up

Table 3: Sum	mary	of outcome	of cultiva	ated limb	al epithe	lial cel	ls for o	cular surfac	e reconstructi	ion.	
Autho	No of eyes	Cause LSC	Partial Total	Auto allograf		BCV		Duration FU month (range	Outcom No	PK/L (no)	Addition procedure
					Improve	Sam	Wors		Epithelialisat	i	
Pellegrini et ⁷	2	Burn	Total	Auto	1	1		24	6	Y(1)	
Tsai et ³⁶	6	Burn Pterygium Other	Partia (4)	Auto	5	1		15	6		
Schwab et ⁶¹	14	Burn Pterygium Other	Partia (2)	Auto Allo	6	8		13 (6-	10	Y(6)	
Koizumi et [®]	3	SJS	Total	Allo	3			6	3		
Koizumi eť	13	SJS Bum OCP	Total	Allo	10	3		11.2+/-	13	Y(5)	
Shimazaki et ³	13	SJS OCP Bums	Total	Allo	8	5		NA	6(46.2%		Repeat (5)
Sangwan et ⁶⁴	3	VKC	Total	Auto Allo	3			26 (24-	3		
Sangwan et ²⁹	88	Burns Others	Total (60)	Auto	24	NA	NA	18.3	51(73%	Y	

LSCD: Limbal stem cell deficiency; BCVA: best corrected visual acuity; FU: follow up; SJS: Stevens Johnson syndrome; OCP: ocular cicatricial pemphigoid; NA: not available; LT: Limbal transplant

greater than 16 months, showed only host cell DNA beyond 9 months. The authors suggested that immunosuppression may not be required beyond this period however further studies are required to substantiate this discovery.

Since there are not any studies with long term outcome, its is difficult to draw substantial conclusions regarding this. It is unknown whether the limbal stem cells actually home in to the limbus once transplanted onto the cornea and continue to function as they normally do. Studies have demonstrated that the epithelium is of the corneal phenotype but what is yet unknown is whether there are undifferentiated slow cycling cells replenishing the transient amplifying cells. In some cases where there is focal limbal exhaustion after LSC transplantation, partial limbal stem cell deficiency develops and this may be managed by regrafting either from a donor cornea or cryopreserved tissue harvested during the primary surgery.

Regrafting of cultivated corneal epithelium was done in 3 eyes of a cohort of patients who had undergone a similar surgery between 3-12 months earlier. The tissue was harvested and cultured from a donor cornea. After a follow up of more than a year the ocular surface in all 3 patients was stable.⁶⁷

Cultivated corneal epithelial cells cryopreserved in 90%fetal bovine serum with 10% dimethylsulfoxide were successfully transplanted in a patient who had undergone a primary cultivated corneal epithelium 5 months earlier. Co-expression of keratin 14 (present in basal epithelial cells signifying normal behaviour of these cells) and keratin 19 (indicates presence of progenitor cells) with low levels of keratin 3 (indicating less differentiated cells) suggests that the basal epithelium has progenitor cells with basal cell activity.⁶⁸

In patients with extensive ocular surface damage with symblepharon and LSCD, transplantation of cultured limbal and conjunctival epithelium may be beneficial.⁶⁹ Autologous limbal tissue was harvested from the eye with partial LSCD, shredded and the explants placed in the centre of de-epithelized AM. Bits of conjunctiva were placed circumferentially around this at a distance of 1cm from the limbal explants.

Future Direction / Unresolved Questions

Cultivated limbal epithelial transplantation is currently the most exciting and promising technique for ocular surface reconstruction. The risk of iatrogenic LSCD to the donor eye is minimised. Cryopreservation of these cells can ensure long-term supply of cultivated epithelial cells for supplementing focal areas of LSCD that may occur in the future and is a step towards establishing a limbal stem cell bank.

In bilateral OSD, the potential adverse effects and cost of immunosuppression are unavoidable. Some groups have speculated that the oral mucosa contains stem cells which can be cultivated on AM and its phenotype resembes corneal epithelium.⁷⁰ Successful ocular surface reconstruction using cultivated autologous oral mucosal epithelium, was achieved in 10 of 15 eyes (67%) followed up between 3-34 months⁷⁰ however all eyes manifested some grade of neovascularisation which the authors claimed did not interfere with vision. In an attempt towards providing an autologous source of cells thereby avoiding immunosuppression in bilateral cases, research should be directed towards identifying progenitor cells of hematopoietic origin which can transdifferentiate and provide an inexhaustible source of autologous tissue.71

Efforts are being made towards replacing the conjunctiva in severe OSD by cultivating conjunctival epithelium.⁷² The function of cultured oral mucosa to replace the conjunctiva is an aspect that needs further study.

Stromal⁴⁹ and endothelial cells⁵⁴ have been cultured on AM. Bioengineering the whole cornea is an exciting prospect though immunosuppression would be inevitable.

Summary

Since limbal transplantation is the only modality for management of total LSCD and while the early results of cultivated limbal stem cell transplantation are promising, long-term results with larger series are awaited. The outcome of cultivated LSC transplantation is comparable to that of cadaveric kerato-limbal or conjunctival limbal allografts or conjunctival autografts and hence is definitely a viable option for minimising damage to the donor eve. What we are experiencing with regards to cultivated limbal stem cell transplantation is merely the tip of the iceberg and holds tremendous possibilities in the field of regenerative medicine and towards a completely bioengineered ocular surface.

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Balloon Dacryoplasty

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Congenital Nasolacrimal Duct Block

Congenital nasolacrimal duct obstruction, or dacryostenosis, is a relatively common disorder encountered in infants. Some studies show that only 37% of full-term infants have patent nasolacrimal drainage systems at birth.¹Normally, most blockages resolve spontaneously by six to eight months of age, but approximately 5-6% remain blocked, resulting in chronic symptoms of epiphora, mucoid or mucopurulent discharge, and perhaps concurrent conjunctivitis and/or dacryocystitis¹.

Persistent nasolacrimal blockage can be due to many congenital abnormalities, including failure of the puncta or canaliculi to develop properly, the presence of punctal membranes, or the existence of congenital tumours or mucoceles within the lacrimal drainage system. The most common cause of such blockage, however, results from a failure of the nasolacrimal duct to open spontaneously. Either the duct itself fails to canalise, or a persistent layer of lacrimal or nasal epithelial cells fails to open at the valve of Hasner.

Management

The treatment of congenital dacryostenosis may vary between practitioners, but a conservative approach with nasolacrimal massage and topical and/or systemic antibiotics is initially recommended. With conservative

treatment alone, approximately 54% of nasolacrimal obstructions resolve by six months of age, and another 17% resolve by approximately one year of age¹. Any persistence of symptoms beyond the age of 12 months usually warrants some form of surgical intervention.

Subsequent approaches may include surgical procedures like probing and irrigation, silicone stent intubation, or dacryocystorhinostomy (DCR) surgery. Studies vary as to their reported success rates but, in general, if the child is 12 months of

Sankara Nethralaya, Medical and Vision Research Foundation, 18, College Road, Chennai age or less, probing and irrigation alone can provide a success rate of around 92%¹. If the child is older than 18 months, rates drop to around 50%². Because probing exhibits a significant decrease in its effectiveness with increasing age, many surgeons will actually skip probing and proceed directly to silicone stent intubation in children older than one year, even though it is traditionally the second line of defence after conservative treatment and probing have failed⁶. When used before or after probing, silicone stent intubation can be successful 90-97% of the time^{7,8.} Dacryocystorhinostomy (DCR) has a 93-95% reported success rate¹¹

Despite these more common approaches, balloon dacryoplasty (DCP) with or without fluoroscopic guidance, can be considered as an adjunctive or alternative procedure in congenital dacryostenosis especially in children older than 13 months who may be resistant to other treatment modalities especially before considering DCR. In fact, when used as an alternative procedure in patients who had failed prior probings or silicone intubations, balloon dacryoplasty was shown to be up to 94% successful³.

In addition, it has been shown to be up to 96% effective as a primary procedure in patients over 12 months of age³

Nasolacrimal duct obstruction in adults

Balloon dacryoplasty (DCP) is also recommended for treatment of acquired incomplete nasolacrimal duct

obstructions. In adults, it is indicated in cases with incomplete obstruction especially when their general condition does not permit a DCR operation. It provides complete relief in 56% of patients or substantial improvement in an additional 34% of patients.¹⁴

Balloon dacryoplasty technique

Balloon dacryoplasty combines traditional probing and irrigation with an added ability to expand the nasolacrimal duct with an inflatable balloon.

The nasal cavity is packed with gauze containing vasoconstrictor agent. Local anaesthesia of nasal mucosa, eyes and infratrochlear nerve block/ general anaesthesia is given.

After dilation of the puncta, a Bowman probe is passed

Fig.1: Trolley setting showing flourescein

stained saline, topical anaesthetic, antibiotic

ointment, syringing cannulae, Bowman's

probes, punctum dilator.



Fig.2: Balloon in inflated state

through the system until it reaches and passes through the valve of Hasner.

Passage of the probe into the nasal cavity is confirmed by inserting a Bowman probe of larger diameter through the nostril to establish metal to metal contact or by direct visualization using an endoscope.

The Bowman probe is then withdrawn and the balloon catheter is inserted through the upper punctum. In children below 33 months of age balloon with outer diameter of 2mm is preferred and in older children 3mm diameter is recommended (Figure 2). The balloon length is 15mm and the overall length of the catheter is 24 cm. The catheter is advanced until a marking 10mm above the edge of the working segment of the balloon lies at the threshold of the upper punctum (Figure 4,5).

In this position, the balloon sits primarily within the nasolacrimal duct (Figure 7). It can be visualized directly in the inferior meatus using an endoscope (Figure 6).

Using a standard cardiac balloon inflation system (consisting of a syringe with volume 10 cc and a pressure gauge ranging from 0-15 atm (Figure 3), the balloon is inflated to a pressure of eight atmospheres for 90 seconds and then deflated for five to 10 seconds. A repeat inflation of eight atmospheres is then performed for 60 seconds .

After both inflations, the balloon is deflated and then retracted approximately 5mm. At this level, the balloon rests at the junction of the lacrimal sac and the nasolacrimal duct (Figure 8).

Two more inflations are performed in the same manner as described above. The catheter is then fully deflated and dialed out through the punctum in a clockwise fashion.



Fig.3: Balloon inflation system with syringe and pressure gauge



Fig.4: Balloon within the nasolacrimal duct system. The markings are seen.



Fig.5: A dacryoplasty procedure in progress



Fig.7: Schematic representation of Balloon within the nasolacrimal duct system past the valve of Hasner



Fig.6: Endoscopic view of the inflated balloon in the inferior meatus



Fig.8: Schematic representation of Balloon at the junction of the lacrimal sac and the nasolacrimal duct

In order to determine if patency has been achieved, the system is irrigated with a sterile saline solution mixed with fluorescein dye. Any solution that passes through the system is aspirated by a suction catheter inserted into the nose. The presence of fluorescein dye in the aspirated fluid confirms that an open pathway has been established by the procedure. In cases done under fluoroscopic guidance, a dacrycystogram(DCG) is done after the procedure to confirm the patency.

Contraindications

Active dacryocystitis, dacryolithiasis, anatomic malformation in the canal, bony canal and post traumatic

lesions are contraindications to the procedure.

Postoperative care

Post-operatively, the patient is placed on nasal spray decongestant, and a combination of steroids and antibiotics that are administered both systemically and topically. This combination of agents helps to ensure proper healing and to prevent secondary scarring from any trauma induced by the surgery itself¹².

Results

Balloon dacryoplasty not only provides comparable success rates to probing, silicone intubation, and DCR, but also has particular advantages that make it a more desirable procedure than its alternatives. One of those advantages is the radial dilation of the nasolacrimal duct provided by inflation of the balloon. Probing alone simply aims to establish an epithelial channel, in most cases, through a minor membrane at the lower end of the nasolacrimal duct¹³. It has been observed that obstructions proximal to the valve of Hasner, such as focal areas of fibrosis or diffuse narrowing of the nasolacrimal duct, respond poorly to probing and irrigation alone³. The additional dilation at the level of both the nasolacrimal duct and lacrimal sac performed during balloon DCP may be particularly useful in patients with such forms of obstruction. In fact, older children who show success with balloon DCP after failed probings may have avoided repeated general anesthesia, if balloon DCP had been performed as a primary procedure.³ Other advantages of balloon DCP are its low risk of complications and its relative ease of performance.

Complications

As the procedure is very similar to a probing and irrigation, the risks and complications are no more numerous than those encountered with probing alone. Such complications include nosebleeds, canalicular damage and the creation of false passages. In comparison, silicone intubation involves a greater number of possible complications, including dislocation of the stents by the child, corneal abrasions, and a risk of further damage to the nasolacrimal system when the stent is removed two to six months later^{9,10}. Moreover, this second procedure usually requires additional general anaesthesia, which can involve even further risks to the child. When compared to dacryocystorhinostomy, balloon DCP is simply much easier to perform and is far less traumatic to the patient; furthermore, it leaves no visible scar behind.

Conclusion

When a case of congenital dacryostenosis has proven resistant to conservative forms of treatment, some form of surgical intervention is usually necessary to correct the

problem. As an alternative or adjunct procedure to probing, silicone intubation, and DCR, balloon DCP is an excellent choice. While it is not indicated for use in all cases of congenital dacryostenosis, balloon DCP has been shown to be highly effective as a primary or secondary procedure, especially in children over 12 months of age due to its effectiveness with resistant cases^{3,12} Although it remains to be seen whether or not balloon DCP is consistently effective in the treatment of patients under 12 months of age, it is a relatively safe, effective and simple approach to consider for the primary treatment of congenital dacryostenosis in that age group. It could potentially save a child repeat trips to the operating room. In adults with incomplete obstructions and poor general condition, it is a safe and effective alternative to DCR. Meticulous application of the cannulation devices and balloons is required for optimal results. Nasal videoendoscopy adds significantly to the understanding of this procedure and its potential for success.

Our experience at Sankara Nethralaya

The procedure of Balloon Dacryoplasty including Endoscopy Assisted Balloon Dacryoplasty (EADCP) was started at Sankara Nethralaya in early 2006. The procedure has been used in two children aged 8 years and 4 years respectively who presented with previous failed probing and history of troubling symptoms of congenital nasolacrimal duct block and dacryocystitis. The procedure of Balloon Dacryoplasty was performed in these two patients using the 3 mm lacricath balloon with complete relief of symptoms following the procedure. Lacrimal intubation was not performed in either case.

Balloon Dacryoplasty procedure has also been used in two adult patients with partial nasolacrimal duct block and evolving nasolacrimal duct block. Both the patients were experiencing troubling symptoms and one of them had a mucocele which was relieved on lacrimal sac massage. Endoscopy Assisted Balloon Dacryoplasty was considered since one of the patients was unwilling for surgery and the patient with the intermittent mucocele had chronic renal failure for which he was on hemodialysis thrice weekly rendering him a poor risk for dacryocystorhinostomy. Both patients experienced partial to good relief of symptoms but without complete resolution of the symptoms. Intubation was not performed in either case.

The cost, at about Rupees 25,000 per disposable balloon DCP set, is a limiting factor. However, the minimally invasive nature of the procedure, and the potential benefits as a result of this, does make the procedure good alternative to more invasive procedures and with careful patient selection, it is an important addition to the armamentarium of the lacrimal and oculoplastic surgeon.

Balloon catheter advantages

- Minimally invasive
- Less trauma to patient and less post-operative discomfort
- Radial dilation of nasolacrimal duct with no punctures
- Procedure similar to Bowman probing (easy to perform)
- 94%+success rate
- No external surgical scars
- Less blood loss
- Quick recovery time
- No tubes no second procedure necessary
- Dilation of narrow passages possible

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Deep Anterior Lamellar Keratoplasty

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Lamellar keratoplasty (LKP) is mostly recognized as therapeutic keratoplasty, as restoration of visual acuity postoperatively is often poor with this procedure. However, deep anterior lamellar keratoplasty (DALK) in which, pathological corneal stroma is completely excised up to descemet membrane, leads to a marked improvement in post operative visual acuity, and the postoperative results are comparable with penetrating keratoplsty.

History of Lamellar corneal surgery

Von Walther first suggested lamellar keratoplasty in 1830. In the late 1940, Panflaque further developed surgical techniques. In 1950 Hallermann tried to achieve deep preparation approaching descemet's membrane (DM). He first used full thickness grafts with aim of improving the optical outcome of LKP. Malbran believed that interface granulation would be reduced by non-instrumental dissection of host cornea. He therefore, dissected only in periphery, peeling the tissue in the central, thinnest part in keratoconus.

Anwar¹ described deep dissection in a potential cleavage plane between stroma & DM in 1974. He also used full thickness donor corneal stroma, but in contrast to Hallermann he removed both endothelium and DM from donor button to avoid causing an inflammatory reaction and probably scarring & wrinkling at interface. Archila again described dissection down to DM. He was the first to use intrastromal air injection and spatula dissection to facilitate access to DM without perforating it. Price and Rostron used a similar technique.

Sugita² used hydro delamination and spatula delamination. A divide and conquer technique of dividing the corneal stroma into four quadrants in two successive layer to reach DM, was described by Tsubata.

A special semi sharp spatula was used by Melles³ to create deep lamellar dissection in a closed fashion. He also used intracameral air injection to visualize the posterior corneal surface and injection of viscoelastic into the cornea to dissect the DM from stroma. The most recent development in the field of lamellar surgery is the "big bubble" technique based on a particular way of injection air to facilitate separation of DM from the corneal stroma before excising the stroma.

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Advantages of DALK

1. *Extra ocular surgery*: DALK is an extraocular surgery like the conventional lamellar keratoplasty. There are less chances of postoperative inflammation as well as secondary glaucoma.

2. *No risk of endothelial graft rejection:* All lamellar surgeries are devoid of any risk of endothelial graft rejection.

3. *No need for long term steroid prophylaxis:* The need for postoperative topical corticosteroids is very less due to the absence of inflammation and a faster healing.

4. *Rapid functional recovery of vision:* DALK is associated with a shorter recovery time as compared to a conventional penetrating keratoplasty.

5. *No interface haze:* DALK involves the complete removal of corneal stroma to the level of descemet's membrane. Therefore here is no or minimal interface haze associated.

6. *Very good best spectacle corrected visual acuity (BSCVA)* is achieved due to a rapid visual recovery and very low astigmatism.

7. *No significant endothelial cell loss:* The endothelial cell loss described in DALK is very less as compared to the endothelial cell loss after a penetrating keratoplasty.

8. *Lesser postoperative glaucoma:* DALK being an extra ocular surgery does not cause postoperative intraocular pressure rise.

9. *Less astigmatism then penetrating keratoplasty:* The amount of astigmatism induced is low after a lamellar keratoplasty. Early suture removal helps in a faster recovery.

10. *Penetrating Keratoplasty can be done* if recurrences occur or descemet's membrane perforation occurs intra operatively

Indications of DALK

DALK is indicated for eyes in which there is stromal opacification with no epithelial or stromal edema and where it is though that endothelial cell function is preserved. Main indications are

- 1. Keratoconus
- 2. Mucopolysaccharidoses
- 3. Stromal corneal dystrophies
- 4. Descemetoceles
- 5. Trachomatos keratopathy
- 6. Healed keratitis
- 7. Gelatinous drop like dystrophy





Figure 1



Figure 3

Figure 4

Preoperative Evaluation

- *Visual acuity*: Uncorrected visual acuity and best corrected visual acuity measured after refraction is important in all the cases.
- *Tear film status*: The evaluation of ocular surface with special attention to the tear film status is mandatory in all cases of corneal transplantation.
- *Intraocular pressure*: Any cause of raised intraocular pressure has to be treated preoperatively or concomitantly.
- *Slit lamp examination*: All cases for DALK need a thorough clinical examination in order to confirm the area of thinning and any signs of descemet's scarring.
- *Fundus evaluation*: Cases of keratoconus may be associated with retinal abnormalities and therefore need evaluation preoperatively.
- *Keratometery*: Keratometry can be performed manually but sometimes is difficult.
- *Corneal pachymetery*: Ultrasonic pachymetry is one of the most important preoperative investigations before DALK so as to know the area of maximal thinning as well the position of any cone if present.
- *Videokeartography and Orbscan*: Preoperatively VKG and Orbscan can help in obtaining more information regarding the corneal topography. Orbscan is helpful in measuring corneal thickness as well as the point of maximal corneal thinning. Sometimes in cases of very high astigmatism e.g. keratoconus, these investigations are not possible.

• *Specular microscopy*: Non contact specular microscopy is useful in diagnosing any preoperative low endothelial cell count e.g. in cases of macular corneal dystrophy. It is also useful in measuring the postoperative loss of endothelial cells.

Surgical Technique (Anwar's "Big Bubble" technique)

Between 60 to 80 % of stromal thickness is cut by means of a suction trephine. A 27 or 30 gauge needle is attached to an air filled syringe. The needle is bent about 5 mm from its tip in such a manner that the terminal segment angles up by about 60 degree and bevel of the needle faces down. The tip is inserted bevel down into corneal stromal deep in the trephination groove where corneal opacities are present clearest area of cornea is chosen. The needle is

advance in a direction half way between a tangential and radial one until the tip of needle reaches a paracentral position (Figure 1). The bevel of the needle facing down is important to decrease the risk of puncturing the descemet's membrane and to facilitate posterior spread of the air. The oblique direction of advancement avoids the visual axis and the thinnest area of the cornea this is particularly important in cases of keratoconus. The plunger of air filled syringe is depressed most commonly after infiltrating part of central corneal disc. The air suddenly forms of large bubble with a closed a circular outline between descemet's membrane and deep stroma (Figure 2). This is the desired result and is indicated by the sudden easing of resistance of the plunger of the syringe as well as the explosive appearance a whitish semi opaque disc, edges of which usually coincide with trephination groove. A paracentesis is performed at a site peripheral to the edge of bubble. A partial thickness anterior keratectomy is carried out leaving a layer of corneal stroma in place anterior to the bubble (Figure 3). Using a sharp tipped blade that is held almost tangentially to the cornea a small nick made just of center through the remaining stromal layers. This is followed by escape of air from bubble. From this opening viscoelastic is injected to push descemet's membrane back. The remaining host stroma is divided into four quadrants and removed manually by blunt tipped scissors (Fig. 4).

Donor Tissue Preparation

The donor endothelium is stained by trypan blue (0.06%). Stromal rim is firmly held with a forceps and a dry wick cell sponge is used to peel of stained endothelium

and descemet's membrane.

Suturing of donor button (Fig. 7,8)

Standard suturing techniques are used. Interrupted, running, or combined interrupted running sutures with 10-0 monofilament nylon can be used for suturing the donor corneal button to the recipient bed. A single or double running suture has the advantage of permitting adjustment in cases of high astigmatism. If interrupted sutures are used, selective suture removal is an option for reducing astigmatism. Sutures are usually removed about six month postoperatively.

Problems with DALK

However there are problems associated with DALK procedure which include risk of intraoperative perforation of descemet's membrane

while dissecting host stroma, microperforation during suturing, double chamber formation during postoperative period, also the procedure is technically challenging and requires expertise from part of surgeon.

Postoperative course

Usually patients have an uneventful course. The graft is clear on day1. Patient is put on topical prednisolone acetate 1% QID, topical moxifloxacin hydrochloride 0.5% TDS; and preservative free artificial tears four hourly. The steroids are tapered from 4-5 month postoperatively and patient kept on artificial tears.

Follow up (Figure 5,6)

Patients are reviewed on 1 postoperative day, 1 week, 1 month, 3 month, and 6 months follow-up period.

Our Results (RPC experience)

- No of eyes 21
- Follow up range 1 month to 16month
- Mean age –26.09 years
- Male-11, Female-10

Indications

Keratoconus- 14



Figure 5



Fig.7: Macular Corneal Dystrophy



Figure 6

- Macular dystrophy- 4
 Hurlers disease- 2
- Descemetocele-1
- Trachomatous keratopathy- 1

Follow up: 16 months

- ➢ 6/12 or better 90.47% (19eyes)
- ➢ 6/6 or better − 14.28% (3 eyes)

Conclusions

The success rate of penetrating keratoplasty is rising every year. However endothelial cell continue to reduce at a considerable speed even 5 years after surgery and a longterm reduction over 10-15 years often result in a relapse of opacification due to decompensation of endothelial cells.

DALK should be used more frequently because it offers very good visual outcomes and stability for a long period of time.

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Hang-Back Muscle Recession Technique and its Modifications

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A successful strabismus surgery should balance muscle forces to restore central or peripheral fusion when possible or acceptable cosmesis when fusion cannot be expected^{1,4-6}. Weakening procedure on the recti constitute an important surgical modality for correcting various forms of strabismus.

Although many surgical techniques are available for weakening of recti muscle (Table 1), the need for a safe, easy and effective surgical procedure led surgeons to experiment with synthetic absorbable sutures. These high tensile strength absorbable sutures form the basis of suspension recession techniques.

Table 1: Weakening procedures on Recti muscles

- Conventional Recession
- Marginal Myotomy/ Myectomy
- Tenotomy/ Tenectomy
- Disinsertion
- Loop Insertion Technique
- Reteroequatorial myopexy
- Suspension Recession Techniques

As early as 1800's, surgeons performed weakening techniques like myotomies by cutting the extraocular muscles posterior to their insertion on the globe. In order to improve the predictability of the weakening procedures and to ensure a good surgical result, Jameson¹ in 1922 introduced the technique on which all conventional recessions are based. The conventional recession technique involved direct suturing of the muscle to the sclera posterior to their insertion at the site of recession.

However, scleral suturing alone neither guaranteed precise muscle reattachment nor perfect ocular alignment and non-muscular variables such as variations in wound healing, mechanical factors from the connective tissues of the orbit and dynamic sensory factors affected the patient's postoperative outcome².

In an effort to overcome the non-muscular variables responsible for the post-operative alignment and to prevent

¹Guru Nanak Eye Center, Maulana Azad Medical College, New Delhi ²Department of Ophthalmology, University of Louisville, Kentucky, USA various problems encountered with conventional muscle recession techniques, suspension recession i.e. hang-back muscle recession was introduced (Table 2). Emphasizing the safety advantages of hang-back muscle recession, this procedure was advocated as a safe, easy and effective alternative to conventional muscle recession technique³.

Table 2: Problems of Conventional muscle recessionsurgery

- Inadvertent perforation of sclera resulting in retinal detachment, endophthalmitis or phthisis bulbi
- No provision for customization
- Problematic in large recession, in high myopes, in infants
- More chances of tenons capsule snaring in large recessions.

Hang-back muscle recession also formed an integral part of adjustable suturing technique, a procedure that was introduced by Jampolsky⁴ in 1970's. Adjustable suture modification to traditional surgery was introduced to improve surgical outcome and reduce frequency of reoperations by eliminating undesirable early post-operative under or over-corrections⁷⁻¹¹. Although this technique has been widely accepted as an effective procedure to customize strabismus repair, it is unsuitable for less cooperative pediatric and adolescent patients. In order to circumvent this problem, non-adjustable hang-back muscle recession technique was introduced by Mills and co-workers¹².

Although non-adjustable hang-back muscle recession surgery undoubtedly proved to be safe and easy to perform, it carried certain disadvantages such as central posterior muscle bowing. In order to tackle these disadvantages, Macleod and co-workers¹³ described a modified suspension recession technique, an anchored hang-back muscle recession technique. Hang back muscle recession technique and its various modifications are now being extensively used because of the multiple advantages that they offer along with increased safety and a lower complication rate (Fig.1).

Review of Literature

In the early 1800's, German ophthalmologists such as Dieffenbach and Deffendorf performed myotomies by



Fig.1: This figure compares the muscle position in (i) conventional recession, (ii) Hang-back muscle recession and (iii) Anchored hang-back muscle recession. *(Courtesy: Macleod et.el)*

cutting the extraocular muscles posterior to their insertions on the globe. By the mid 1800's general anesthesia had been introduced and many surgeons were attempting to improve the predictability of the weakening techniques by using partial myotomies an tenotomies. These efforts were generally unsuccessful and surgeons were frustrated by the random retraction and reattachment of the cut muscles to the globe.

In 1922¹, Jameson introduced the technique on which all conventional recessions are based, which involved direct suturing of the muscle to the sclera posterior to their insertions at the site of recession. This procedure allowed accurate grading of the surgery permitting a definite understanding of the location of reattachment of the recessed muscle. Though it turned out to be a gold standard procedure, scleral suturing alone neither guaranteed precise muscle reattachment nor perfect ocular alignment. In practice, nonmuscular variables such as variations in wound healing, mechanical forces from the connective tissue of the orbit and dynamic sensory factors affect the patient's postoperative course.

In order to circumvent various problems encountered with conventional scleral suturing suspension recession surgeries were introduced. This technique has been extensively used in the surgical management of simple horizontal and/or vertical strabismus, dissociated vertical deviations and in cases of iatrogenic mechanical incomitance produced by retinal reattachment surgery, glaucoma surgeries, functional endoscopic sinus surgery etc.

Suspension Recession Surgery (Hang-back muscle Recession): Historical Aspects

The term, loop recession was first used in the literature by Gobin¹⁴ in the 1960's. He proposed using two 5-0 silk

sutures attached to the ends of the muscle when recessing the medial rectus muscle more than 5 mm. In this procedure, the detached muscle was sutured to the sclera at two points separated by a distance equal to the width of the original insertion and located 5 mm behind it. The loose ends of the suture ends were tied over a 2 mm diameter probe and the muscle was allowed to recess another 5 mm for a total recession of 10 mm. Gobin postulated that the gap produced between the tendon and the insertion becomes bridged by connective tissue so that the tendon acquires a sort of extension thereby attaching 5 mm behind the original insertion. Thus on one hand it spares the arc of contact maintaining the contraction of the muscle and on the other hand results in a maximal recession of 10 mm.

This technique of loop recession underwent many modifications and in the early 1970's with the advent of high tensile strength synthetic absorbable sutures, the suspension recession techniques were introduced. These materials gave surgeons the confidence to experiment with suspension recession techniques. Suspension recession would seem to represent a departure from the security gained by attaching the muscle to a fixed point on the globe. There success depends upon attachment of the cut muscle to the sclera before complete hydrolysis of the suture material occurs. Synthetic absorbable sutures such as vicryl are not completely absorbed until 90 days after intramuscular implantation. Firm muscle scleral union occurs within 1-2 months after surgery.

In 1970's adjustable modifications to traditional surgery were introduced thereby allowing the surgeons to customize strabismus repair thus improving the rate of surgical success. Adjustable procedures did not differ in the methods of repositioning of muscles, it merely added a satisfactory monitoring of the total end result by extending surgery into the immediate postoperative period when the patient is alert. Suspension recession surgery constituted an essential step in adjustable sutures thereby adding a new armamentarium to the existing techniques. Over the years adjustable sutures have undoubtedly proved most useful in complicated strabismus surgeries such as mechanical incomitant strabismus, paralytic strabismus, strabismus associated with myopathies, reoperations, iatrogenic strabismus etc. Some surgeons started using adjustable sutures in all routine strabismus surgeries in cooperative patients. Thus adjustable suture techniques allowed surgeons to customize strabismus repair thereby reducing the rate of repeat operations. It also offered a convenient site for scleral suturing thereby reducing the incidence of complications and problems encountered in the previous conventional surgical technique.

However adjustable techniques are not suitable for every patient. This is certainly true in the less co-operative pediatric and adolescent age group where conventional recession has been gold standard. Adjustable suture technique also cannot be used in patients who are prone to cardiac arrhythmias due to manipulation of the extraocular muscles.

Medical and surgical alternatives to adjustable and conventional muscle recession techniques have been developed in the last decade. These include intramuscular botulinum toxin injections, non-adjustable hang-back, hemi-hang back, anchored hang-back, and modified anchored hang-back techniques.

Table 3: Hang-back muscle recession and its modifications

Hang-back Muscle Recession

Adjustable hang-back

- Standard hang-back
- Modified Anchored hang-back

Non-adjustable hang-back

- Standard hang-back
- Hemihang-back
- Anchored hang-back

Mills and coworkers¹² conducted the first study of nonadjustable hang-back recession on patients with either horizontal or vertical strabismus. Although not controlled, the study found that 55 of 62 (89%) patients had a successful outcome, which was defined as any alignment that did not require reoperation.

In 1987, Repka and Guyton³ proposed hang-back medial rectus recession as an alternative to conventional recession in esotropia. They studied a comparison of hangback medial rectus recession with conventional recession in cases of both congenital and acquired esotropia. After a one year follow up, 86% of the hang-back group and 67% of the conventional group was aligned within 10 prism diopters of orthophoria. In this study over-correction occurred more frequently with the suspension recession technique than with conventional medial rectus recession. There were, conversely, fewer under-corrections with the suspension recession technique. This suggested that a greater surgical effect was obtained with hang-back surgery as compared to conventional recession surgery. One of the complications as described by the authors was possibility of a sideslip of the sutures up or down over the surface of the globe with vertical movement of the eye. This might produce temporary upshoots or downshoots of the eye during the healing period. Also, the extraocular muscle might attach to the globe above or below the horizontal meridian, thereby producing an A or V pattern strabismus or possibly permanent upshoots or downshoots. Despite the described complications the

authors emphasized the safety advantages of hang-back surgery describing it as a safe, effective and easy alternative to conventional recession.

Potter and Nelson², compared the surgical results of hang-back muscle recession technique with conventional muscle recession technique in cases of large angle esodeviations (mean 60 prism diopters). They reported a motor success defined as within 10 prism diopter of orthophoria in 86% of the patients in the hang-back group as compared to 75% in conventional muscle recession group. According to these authors suspension recession techniques have enabled the surgeons to perform surgery more quickly and easily than conventional muscle recession techniques thereby reducing the general anesthesia time for the patient.

Capo and coworkers¹⁵ studied hang-back lateral rectus recession and reported a 78% success rate at 6 weeks follow up compared to a 75% success rate with conventional recession. However significant late over corrections were found with the hang-back surgery which led the authors to reduce the amount of recession by 0.5-1mm for deviations between 15-35 diopters. They emphasized the safety of anterior suture placement which avoids the thinner sclera between the insertion and the equator which is most prone to perforations

Some workers felt that unpredictably large deviations may occur as a result of suspending the muscle and not attaching it with scleral sutures to the globe¹⁶⁻¹⁸. Lost muscle and late over-corrections may occur if muscle reattachment lags behind suture hydrolysis and the muscle slips posteriorly. Under corrections may develop if the recessed muscle migrates forward on the globe. Two kinds of attachments develop with suspension recession surgery, namely direct muscle-scleral adhesion and pseudo tendon formation, both of which are capable of maintaining an arc of contact between the muscle and the globe. In a primate study, Repka¹⁹ reported that direct muscle attachment occurred in 75% cases while pseudo tendon formation occurred in 37% cases.

In 1992, Potter and co-workers² described the fornix incision in primary cases of strabismus using the nonadjustable hang-back muscle recession and hemi hangback muscle recession techniques. They advocated the use of hemi hang-back muscle recession in cases of large recessions greater than 7 mm. The hemi hang-back muscle recession technique is derived from three procedures mentioned in the literature: conventional scleral suturing, hang-back muscle recession and loop suspension recession technique. In the hemi-hang-back muscle recession technique, the muscle is reattached and suspended from the sclera posterior to the original insertion. This procedure is illustrated in Fig.2. After disinserting the muscle, it is suspended, with the help of sutures, from a point midway



between the original insertion and desired muscle insertion site Fig.2 (A-G). This technique is recommended for management of difficult strabismus cases as in paralytic strabismus; large deviations or following maximal recessions with residual large deviations and in cases with good vision in one eye and the deviating eye has already undergone maximal surgery.

In 1996, Macleod and co-workers¹³ described a modified suspension recession technique, an anchored hang-back muscle recession in order to overcome the problems associated with hang-back recession. As shown in the figure1, the central posterior bowing noted with hangback recession led to enhanced recession making the results less predictable. In addition, the muscle cannot be displaced accurately upwards or downwards as may be required for correction of A and V patterns or vertical deviations. The tendency of the muscle to bunch centrally leads to lack of control over the final vertical position of the muscle thus hindering the correction of coexisting vertical deviation of A and V pattern. . Macleod has recommended an anchoring scleral bite which localizes the muscle precisely and maintains its normal width thereby overcoming the problems noticed with hang-back recession. This technique does still require the passing of a needle into thin sclera as in conventional surgery, although only a thin superficial bite is required. Thus anchored hang-back muscle recession is designed to overcome the problems of hang-back muscle recession while maintaining the ease and safety of the latter procedure. This technique is illustrated step by step in Fig.3.The muscle is exposed using a paralimbal fornix based incision and carefully hooked (Fig3 A-C). Locking bites are

taken to secure the muscle with 6-0 vicryl suture (Fig3 D-G). The muscle is then severed from the original insertion and markings taken for the site of desired insertion site (Fig3 H-J). Then a superficial anchoring bite is taken at the desired site of muscle recession and the muscle is suspended from the original muscle insertion using the same sutures (Fig3 K-O). The conjuctival flap is then sutured back (Fig3 P).

In 1998 Kanwar et al²⁰ compared the results of strabismus surgery using adjustable and non-adjustable hang-back muscle recession in 38 patients having comitant exotropia. At 6 months follow up, 18 of the 19 (95%) patients in the adjustable hang-back group and 17 of the 19 (90%) in the non-adjustable hang-back group had ocular alignment within 10 PD of orthophoria. In a follow up of 3 years, 92% in the adjustable group and 86% in the nonadjustable group had ocular alignment within 10 PD of orthophoria. A post-surgical undercorrection of 5-14% in the non-adjustable group and 5-8% in the adjustable group was noted which was attributed to the anterior migration of the muscle fibres due to contraction of the pseudotendon. They concluded that non-adjustable hang-back muscle recession should be used as a routine surgical procedure in cases of concomitant exotropia.

Kamlesh et al compared the Anchored hang-back muscle recession with conventional recession in 60 consecutive patients of concomitant exotropia. At 2 years of follow up, they observed that 80% of patients with anchored hang-back and 86.67% patients with conventional recession had ocular alignment within 10 PD of orthophoria. No significant post-operative drift was



noted in the anchored hang-back group compared to the conventional group. They also noted that anchored hangback muscle recession caused less manipulation of the eye, less inflammation and was safer and easier than conventional recession.

Although anchored hang-back muscle recession appears to overcome most of the disadvantages of the hangback muscle recession, it lost the major advantage of customization in terms of post or intraoperative adjustment offered by the hang-back recession. In order to integrate the advantage of both Kamlesh et al²¹ in 2002 described a new procedure Modified Anchored Hang-back muscle recession technique. In this technique, after disinserting the muscle, a superficial scleral anchoring bite is placed midway between the intended recession site and the original insertion (Fig.4: A-I). This procedure on one hand maintains the width of the muscle preventing central bowing and on the other hand allows for intra and postoperative customization.

In 2005 Chung et al²² compared modified hang-back recession with conventional recession in patients with esotropia. The overall success rate was found to be similar between the two groups however modified hang-back recession was found to have significantly lower rate of consecutive exotropia at 6 months.

New Developments

Over the last decade hang- back muscle recession has undoubtedly proven to be a safe, easy and effective surgery in both routine as well as complicated strabismus procedures for both horizontal and vertical recti muscles. Modified Anchored hang-back surgery has added a new armamentarium to the existing surgical techniques as a procedure overcoming the disadvantages of hang-back



surgery and at the same time offering an opportunity for intra as well as postoperative customization.

In another break through development, hang back recession was carried out on the inferior oblique muscle for the first time in the history of strabismus surgery by Kamlesh and co-workers. They evaluated 30 patients of V pattern strabismus with inferior oblique overaction and performed 10mm suspension recession of the inferior oblique muscle. In this study a mean V pattern correction of 20.2 prism diopters was obtained. In 85.37% patients the V pattern was found to be fully corrected. The mean inferior oblique overaction correction was found to be 17.04 prism diopter. No significant complications were encountered. This is the only study, which has evaluated the outcome of suspension recession technique on the inferior oblique muscle.

Hang-back muscle recession offers an exciting and challenging alternative to conventional recession for the correction of strabismus. Various modifications have been studied and evaluated as outlined above with encouraging results. While hang-back muscle recession was limited to the rectus muscles for more than a decade after it was first described, the pioneering work of Kamlesh et al has introduced this masterful surgery to the inferior oblique muscle as well. While dedicated research is pursued, we hope that the human mind will continue to evolve and put forth better ideas and solutions to unravel the mystery of strabismology.

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Management of Posterior Capsular Tear during Phaco

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No Surgery is complication free, neither can any surgeon claim to never have had any complication. It is the skill in management of a complication, which actually decides between a good and a moderate surgeon. Cataract surgery is the most commonly performed ophthalmic surgery and almost all cataract surgeons would have experienced a PC tear some time in their life. In the era of ECCE, the nucleus was already out, thus managing a PCtear was not that dreaded in an open wound. In Phacoemulsification PCT is even more dreaded because a PCT during phaco can lead to a nucleus drop, which can then take the case out of the hands of an anterior segment surgeon. In the present day of a closed chamber technique surgeon also has to keep up to various promises like that of sutureless surgery and implanting a foldable lens etc. Also vitreous loss can lead to complications like uveitis, secondary glaucoma, retinal detachment, CME etc. So preventing any complication is essential and we will be highlighting how one can recognize and manage such a situation. The golden rule in the early days of phacoemulsification was to convert, but now the aim is to safely complete the surgery, without any complication inspite of an underlying rent.

PCT can occur at any stage of surgery

(i) During Hydrodissection

Some cataracts such as posterior polar cataract or very hard cataracts with sticky fibers are more prone for a PC rupture during hydrodissection. If the lens is hard and the fluid gets entrapped below the nucleus, the PC can give way. To prevent this a good pre-operative assessment is very important. Hydrodissection should be avoided in cases like the posterior polar cataracts as there is a likelihood of posterior capsule weakness. It is better to just do a hydrodelineation. Some of the signs of a PC rupture during hydrodissection are – a sudden deepening of the chamber, an abnormal tilt in the nucleus and an inability to rotate the nucleus.

(ii) During Nucleotomy

The posterior capsule can get damaged during trenching in very soft cataracts if one is not careful to use

Department of Ophthalmology Sir Ganga Ram Hospital, New Delhi appropriate power. This damage can also occur if the surgeon does not follow the contour of the posterior capsule, as the lens is hardest in the center, achieving almost a conical shape posteriorly. The movement of the phaco probe must follow this curvature, if adequate depth is to be achieved avoiding capsular injury.

In cases of very hard cataracts with leathery bridging fibers complete splitting is difficult and a strong forceful attempt to divide such a nucleus may end in damaging the posterior capsule. The force used to split these bridging fibers should be moderate to avoid damage to the posterior capsule.

(iii) Capsular injury during chopping

Use of a long chopper on a soft nucleus or in an unstable AC can lead to a peripheral capsular tear. If a chopper is accidentally placed over the anterior capsule instead of under it, and a chop is initiated, it may lead to a rhexis margin tear or zonulolysis. An unstable chamber due to surge can also be a cause for a PCT. As long as there is some nuclear piece in the capsular fornix, the PC is pushed back and this prevents it from any direct damage with the phaco probe. However once the capsular fornix is empty, the PC can move forward particularly in hypermature cataracts where the epinuclear plate protection is absent. To prevent this one should reduce the vacuum settings and then perform phaco more anteriorly under VES cover, specially while handling the last piece or removing the ENP.

(iv) PCT during cortical aspiration

More than 50% of PCT occur during cortical aspiration, especially in the sub-incisional area. This is because of poor access and decreased visibility, particularly with a coaxial hand piece. Distortion of wound causes the wound to leak which increases the surge, causing the PC to come anteriorly, PCT does not occur if the PC is only caught in the probe; sudden movement after holding with the probe/ cannula is what causes it to tear. When the PC is caught in the suction port a star-shaped tented area appears. Immediately, **without moving the hand piece/cannula**, release the foot pedal to stop suction. In some cases, reflux may be required. Catching does not tear the posterior capsule but pulling does.

(v) PCT during capsular polishing

It should be kept in mind that high myopes are particularly prone to PCT. If the bag is lax and some





Fig.1: PC Tamponade-never withdraw the probe suddenly in case of a PC tear

Fig.2: PC Tamponade with the probe in replace the BSS with viscoelastic from the side port



Fig.3: Withdraw the probe only once the anterior chamber is filled with viscoelastic to push back the vitreous and not let the chamber collapse

wrinkling appears on the capsule during the movement of rounded repositor/polisher, the chances of creating of PCT are high. During polishing, a well-focussed PC under high magnification and a bag filled with VES is a must to prevent PCT. tear can be identified. In case of any doubt, stop and try to look for any of the signs of PCT described above. Keep a swab stick/merocel sponge in the section and see if a vitreous strand is picked up or the pupil/CCC gets peaked. If this happens then it implies that vitreous is in the wound.

Recognition of PCT

PCT without rupture of anterior hyaloid phase

Failure to detect PCT in time may finally result in nucleus, nucleus fragment or IOL drop. Recognition of PCT is of prime importance. If the anterior hyaloid phase is not disturbed, recognition of PCT may be difficult. However on careful examination under high magnification an increase in brightness of red-reflex at that site and the margin of the PCT will clinch the diagnosis. If most of the nucleus and ENP is out, put VES to flatten the bag, or keep it slightly convex but not concave to elicit halo test. Try to look for the ring reflex by applying the pressure in the center of the capsule with the help of rounded repositor. If the PC is intact, a halo will be seen which will vary in size depending on the amount of pressure applied. This ring reflex will be broken at the site of PCT. Note: the ring reflex will not be elicited if the PC is concave forward due to over inflation of the bag by VES.

PCT with rupture of anterior hyaloid phase

Once the hyaloid phase is disturbed, vitreous prolapses into the AC making it deeper. Vitreous being an elastic, fibrillous substance does not flow and fill the AC completely but tends to come to the incision site. Phaco cannot cut the vitreous strands and therefore any nuclear fragement/cortical matter entangled in the vitreous cannot be removed with phaco. Rotation of lenticular matter also is not smooth and it tends to snap back to its original position. Forceful rotation may only result in pushing the fragment further into the vitreous cavity. Lens matter also does not move with the eddy currents in the BSS. It may in fact gradually sink into vitreous cavity. With higher magnification, on careful examination, the margins of the

Management of PCT

After having recognized a rent or on having a suspicion of PC-defect it is most important not to panic. In this crisis situation the reflexes need to be more stable and the mind needs to work faster. Any wrong movement/step can result in a vitreous prolapse or more dreaded complications i.e. nucleus drop.

Once a posterior capsule tear is detected the first step is to prevent the tear from extending, and prevent the nucleus and nuclear fragments from dropping posteriorly. The natural instinct is to quickly withdraw the probe. This leads to sudden decompression of the AC, which can cause further extension of the tear and vitreous prolapse through the defect. The more the vitreous prolapses, the less the support to the remaining posterior capsule and fragments and the greater the chances of the nucleus dropping to the vitreous cavity. Thus, one should try to avoid fluctuations in the AC depth by injecting an OVD through the side port to fill the AC at the same time (Fig. 1,2,3) or before the probe is removed. Forming the chamber with the OVD helps keep the vitreous back and is better than using a balanced salt solution because it does not hydrate the vitreous. In the setting of a PCT a cohesive agent like Healon GV is the visco-material of choice because this being a high viscosity substance gives a good tamponade to the vitreous and flows very smoothly through a 27 gauge cannula. A dispersive viscoelastic can be used in conditions where one plans to emulsify the nucleus fragments with an underlying PCT. eg Viscoat. This high viscosity substance will tamponade the vitreous posterior to the capsule and will not to be aspirated from the eye easily.

In cases with a posterior capsule tear, the decision to continue phacoemulsification or convert to a large-incision surgery depends on factors such as the type of cataract,



Pic.1: Releasing the CCC to prolapse the nucleus out of the bag

the amount of nucleus remaining, the state of the pupil and cornea, and the expertise of the surgeon. Though no cases is the same, these are some basic principles that may be followed in a posterior capsular tear.

(i) PC tear with most of the nucleus remaining

If the posterior-capsular tear occurs early in the surgery with almost the entire nucleus remaining in the bag; it is most important to secure the nucleus by bringing it out of the bag and positioning it above the anterior CCC. In doing so it is important to analyse the size of CCC, if the CCC is small, release it by giving relaxing cuts in the CCC margin (Pic.1). Removal from the bag is best attempted with the use of 2 instruments. The instruments that could be used are the sinskey hook, chopper, rounded repositior or dumbbell dialer. Sinskey hook is particularly good as it gets buried into the nucleus and provides a good grip. Both the instruments can be introduced from the main port or one from the main port and another from side port. One is put below and are above so that the fragment is sandwiched. The fragment is gripped firmly between the two instruments and is now moved onto the intact anterior capsule or into the AC away from the site of tear. Any

nucleus fragment lying in the capsular fornix must be prolapsed out into supra-capsular area.

In case the nucleus in hanging down in the anterior vitreous cavity, never try to fish it out through the anterior route. Such an impending nucleus drop may be best prevented by what we call the 'Chopstick technique'. (Fig.4) In this, one port is made through the pars plana by



Fig.4: *Chopstick technique* of stabilization of nucleus in impending nuclear drop. Note the placement of once instrument above and one below the nucleus through the parsplana



Pic.2&3: The incision is enlarged and the inclues is comfortably removed with a two instrument grip

giving a stab incision with 15 blade or v-lance knife 3.5 to 4 away from the limbus. Through this parsplana incision an instrument eg. Sinskey hook is passed and buried into the nucleus; this helps to support the nucleus and prevents it from dropping into the vitreous. The nucleus is stabilized with the second instrument from above which may pass through the side port or the main port. The nucleus is then brought into the anterior chamber after having stabilized.

The incision is enlarged and the nucleus is comfortably removed with a two instrument grip (Pic.2,3).

(ii) More than half the nucleus remains, which is grade II to III or more

Managing the nucleus in such a situation depends on the extent of capsular tear and the presence or absence of vitreous in the anterior



Fig.5: Injecting viscoelastic through the side port to push back the vitreous and stablize the nucleus





Fig.6: Pulling and stablizing the nucleus with a chopstick grip



Fig.7&8: Mechanically crushing the nucleus fragment and prolapsing it out of the anterior chamber

chamber. It the capsular support is minimal and the tear is large, it is safer to stop emulsification. Do not withdraw the probe suddenly as this can cause a sudden decompression of the AC and a positive pressure from behind may cause vitreous there prolapse causing extension of the tear. Inject viscoelastic from the side-port to fill the anterior-chamber so that the vitreous is pushed back (Fig.5). The VES is injected under the nucleus fragments to hold back the vitreous and at the same time prolapse the nuclear fragments out of the bag. Once the fragments are away from the tear, they can be removed by mechanical crushing (Fig.6,7,8) (Pic. 9,10,11).

In the process of mechanical crushing, care is to be taken that repeat VES injection may be done to keep back the vitreous and provide a mechanical cushion.

The smaller nuclear fragments are removed out of the AC either by sandwiching them between 2 instruments or by visco-expression. One should take care not to depress the posterior lip of the wound too much but try to inject the VES in front of the piece and bring it out (Pic.12).

(iii) Nucleus grade II-III with a small tear and no vitreous

A small rupture in the posterior capsule during emulsification of the nucleus can be managed by altering the surgical technique. If the capsular support is good and there is no vitreous in the anterior chamber the most important step is to secure the nucleus by bringing it away from the site of PC-defect. Viscoat is placed below the



Pic.9&10: Pulling and stablizing the nucleus with a chopstick grip





Pic.11: Prolapsing the nucleus fragment out of the anterior chamber



(iv) Soft nucleus grade II-III with large tear

In case the tear is large and the surgeon does not feel it safe to carry on with phaco, it is better to secure the nucleus using the sandwich technique by holding the nucleus between two instruments and prolapse it into the anteriorchamber. After the nucleus can then be broken into smaller fragments and expressed out through visco-expression. During visco-expression case has to be taken not to depress the posterior lip too much as it may cause the A/c to collapse and positive pressure from behind may behind level to enlargement of tear and vitreous prolapse (Pic.12).



Pic.12: Injecting viscoelastic to viscoexpress the epinuclear plate without pressing the posterior lip of the incision

(v) Epinuclear plate removal

Before any manover a proper assessment should be mode about the presence or absence of vitreous. It is important to clear the chamber of any vitreous by doing a good vitrectomy preferably with an automatic cutter, but if the same is not available even a good manual vitrectomy will do. Having most of the vitreous cleared fill the capsule bag with viscoelastics to open the capsular fornix. The EPN is mobilized using a rounded repositor and taking counterpressure from the anterior capsule. Rotating the epinucleus helps to dislodge it from the fornix and prolapse into the anterior-chamber. It can then be is expressed through viscoexpression.

(vi) Tackling the residual cortex

Cortex should be removed by dry aspiration after ensuring that no vitreous is in the chamber. The chamber

is formed with VES and manual dry aspiration is done using Simcoe cannula or 26G cannula. First aspirate at the site where vitreous is absent. If the need be, another side port can be constructed.

Sometimes a little infusion may be required to release the cortical matter. For this purpose, enter with irrigation tube of Simcoe cannula pinched between thumb and forefinger and as and when required, allow the irrigating fluid through. Continuous irrigation should not be done.

At times it may not be possible to remove the ENP and cortical-matter due to vitreous in the AC. In such a situation automated or manual vitrectomy may have to be performed. Automated vitrectomy through the pars plana route is preferred though it can be done through the side port after enlarging it. The infusion cannula is introduced through a second side port. Using low vacuum settings and high cutting rate, the vitreous, EPN and cortical matter are removed. The cortical matter can also be removed after vitrectomy by manual/dry aspiration. If this facility is not available and the section has been converted to an ECCE is incision, do liberal vitrectomy till the AC is cleared of vitreous any vitreous remaining in the section removed with the help of swab and Vannas.

Some vitreous may remain in the anterior chamber even in the best of the hands. After IOL insertion, clear any vitreous in the section. Use pilocarpine in the anterior chamber to identify peaking of the pupil. Remove this vitreous with cutter or the Vannas to leave a round pupil. Vitreous in the section is much more damaging than vitreous in the AC. Sweep away any vitreous in the section from the side port in such a manner that it lies away from the wound. These patients have a higher chance of inflammation keratitis, cystiod macular edema, and retinal detachment postoperatively. Hence, management should be modified accordingly.

Insertion of IOL in cases with PCT

No IOL

It is better not to insert the IOL if the lenticular matter is not cleared adequately. In 2-3 weeks the cortex becomes fluffy. At this point of time, the vitreous in the AC is more clearly identified and a secondary anterior vitrectomy and IOL insertion is a much safer procedure.

Sulcus Fixated IOL

In case of a large posterior capsular tear with a damaged CCC

If the posterior capsular support is not sufficient enough it is better to place the IOL in the sulcus. Best is to choose a large optic PMMA IOL if the CCC is not intact. Before the IOL insertion a good cortical cleanup with adequate vitrectomy should be ensured.

IOL in the bag

In case of a small capsular tear with a good capsular support

(a) PMMA IOL:- (Pic.17-22)

Before inserting the IOL, inflate the bag with viscoelastic and ensure that it is clear of vitreous. Place the IOL over the anterior capsule then place the haptics into the bag one by one. Avoid dialing the haptics. Alternatively one can place the leading haptic and the optic in the bag and then insert the trailing haptic with a Mc-pherson forceps. Pronate the hand and press the haptic down to place the haptic into the bag without causing any traction on the posterior capsule.

(b) Foldable IOL:-

Fill the anterior chamber and the bag with a cohesive, high viscosity substance like HealonGV, which prevents a



Pic.17: Injecting viscoelstic in the fornix of the capsular bag to inflate the bag and the anterior chamber



Pic.18: Placing the leading haptic and the optic in the capsular bag



Pic.19&20: Pulling the optic inwards and supporting to place the trailing haptic in the capsular bag without dialing. This causes minimum traction on the posterior capsule and does not cause the rent to enlarge



Pic.21: Placing the trailing haptic in the bag

jerky opening of the IOL. Release the IOL in the sulcus and place the haptic one by one with forceps. It is better to avoid dialing in these cases. Hydrophobic acrylic lens is preferable as it opens more gradually as compared to a silicone lens. These can easily be inserted over a PCC or a small rent.

Foldable IOLs in the sulcus (Pic. 13-16)

If the anterior rhexis is intact and a large posterior capsular tear lies underneath the foldable IOL can be placed in the sulcus with a reverse optic capture. After releasing the IOL in the sulcus the optic is gently pressed underneath the anterior rhexis margin by placing the entire optic behind the anterior rhexis with the optics placed in the sulcus

Anterior Chamber (AC) IOL



Pic.22: Well centred IOL placed in the bag

AC IOL. PC IOL in the AC can cause fibrosis of the angle structures leading to glaucoma. The angulation of the PC IOL is unsuitable for the angle. The forward angulation leads to corneal problems and if placed the other way, the backward angulation may lead to papillary block. AC IOL may cause ciliary tenderness, inflammation, CME and corneal complications. It is better to wait and insert a secondary IOL later.

Scleral fixated IOL

Scleral-fixated IOL can be implanted in the same sitting if proper sutures and IOL are available. It may also be implanted later after the inflammation settles.

Secondary IOL

Usually, it is possible to put secondary IOL after 3 to 6 We do not recommended use of PC IOL in AC or use of months, whatever the extend of PCT. A secondary PC IOL



Pic.13: Gently Opening a hydrophobic acrylic IOL in the sulcus for sulcus fixsation and anterior optic capture



Pic.14: Placing the leading haptic over the anterior rhexis



Pic.15: Gripping the trailing haptic with mac-pherson forcep and placing with in the sulcus for sulcus fixsation of the lens

is a better option than AC or Scleral fixated IOL.

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Pic.16: Final vitrectomy with visco clean-up after reverse anterior optic capture of foldable hydro phobic IOL

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Surgical Management of Morbid Obesity

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Morbid obesity is a disease of excess energy stores in the form of fat. This is a direct result of a mismatch between energy intake and energy expenditure. It is a recognized major public health risk throughout the world. The morbidity is related to both the physical bulk of the patient as well as comorbid conditions including cardiac disease hypertension, NIDDM, obstructive sleep apnoea, hypovetilation, dyslipidemia, cholelithiasis, GERD, degenerative arthritis, stress incontinence, infertility and cancers. The most pronounced effect is a direct increase in mortality rate related to increase in body weight.

Recognition of obesity as a disease began in the middle of the last century. It was about this time that attempts were made to find answers for its management through surgical procedures. Substantial progress has since been made to understand the underlying pathophysiology of this chronic disease resulting in a more comprehensive multidisciplinary approach to treat the illness. It has also been proved that bariatric surgery offers the most effective means of prophylaxis against life threatening complications and severe degenerative problems of morbid obesity. What needs to be understood beyond doubt is that bariatric surgery is not a cosmetic surgery. This surgery is aimed only to treat the comorbities consequential to obesity thus increasing the longevity and quality of life in morbidly obese patients.

There are well accepted guidelines and indications for bariatric surgery as recommended by National Institute of Health consensus conference in 1991, these are:

Indications for Surgery

- 1. Individuals with a Body Mass Index (BMI) of greater than 40 kg / m2.
- Individuals with a BMI of > 35 kg/m2 with significant comorbities with unsuccessful attempts at nonoperative (dietary) weight loss.

Surgical Goals

Surgery is medically indicated, as it is the only means of achieving long terms weight control for the morbidly obese. Surgery does not aim at removing fatty tissues of these patients. The aim of surgery is:

- To reduce the gastric reservoir with or without.
- Creating a degree of malabsorption.

Minimal Access and Bariatric Surgery Centre Sir Ganga Ram Hospital, New Delhi • Bariatric surgery in the young is performed with the aim to increase longevity and quality of life whereas in the elderly it is primarily for improving quality of life.

It is necessary to target realistic goals and to set a protocol for the best and most appropriate utilization of time tested operations.

The Professional Team

The Surgeon: should be the head of the professional team and shoulder the overall responsibility for the long term well being of the patient. He / She should be aware of all possible complications related to the bariatric procedures and should be competent enough to diagnose and manage them.

A dietician / nutritionist: To guide the patient through alteration of eating habits and to develop and maintain a good balanced diet and eating behaviors following surgery.

The Clinical Psychologist / Psychiatrist: It needs to be understood that these are special patients who have suffered a lifetime of prejudice and are bound to have psychological and behavioured problems which require assessment and simultaneous management with the surgery.

Infact it is necessary to adopt a multidisciplinary approach for management of these patients. Therefore other professional required include:

A medical specialist(*Endocrinologist / Cardiologist*): To assess the risk of existing comorbidities and their management in the perioperative period.

A Physiotherapist: for developing an exercise programme for the patients and for training them to follow an appropriate exercise schedule

Special Nurses: The morbidly obese have special needs physically and emotionally special trained nurses are required for managing these needs with empathy and firmness.

Patient Selection

Indications for surgery have already been defined. The patients apart from being morbidly obese must be motivated, and well informed regarding the surgical procedure and what to expect postoperatively. There must be willingness for long term follow up. The operative risk must not exceed morbidity and mortality risk due to obesity. All patients with a BMI exceeding 40kg/m² desiring weight loss and patients with a BMI between 35-40 kg/m² with significant obesity related comorbidities posing a high



Biliopancreatic diversion (BPD)

Biliopancreatic Diversion

Laparoscopic Adjustable Gastric Banding

risk to their lives are candidates for bariatric surgery

Preoperative management

- Thorough evaluation (History)
- a) What is patients obesity risk: Obese & Fit / Obese & unfit (require surgery more urgently)
- b) How is obesity affecting the patient's health (obesity related comorbidities)
- c) Why is the patient obese (genetic, environmental, psychosocial, cultural, and hormonal).
- d) What are the patient's goals and expectations? Is the patient motivated to enter a weight management programme (Aim: 10% of current weight over 6 months)
- e) What kind of help does the patient need diet, physical activity, behavioral modifications.
- Chronological history of body weight, weight gain and inciting events.
- Response to previous weight loss attempts
- Understanding of how body weight is regulated
- Perception of how excess BW affects health
- Expectation of weight Management program
- Lifestyle, including diet and physical activity patterns.
- Social history (occupation / travel / home environment)
- Family history
- Psychiatric & psychological history (binging, bulimia, body image disturbances)
- Medication history
- System review
- Motivation and readiness
- Physical Examination
 - Body weight (kg)
 - Height (m)

- Waist circumferences
- BP with appropriate pressure cuff
- Rest as dictated by assessment of risk factors
- Laboratory investigations
 - Lipid profile

Gastric Bypass Roux-EN-Y

- Blood sugar F/PP
- Remainder of lab with diagnostics determined by lap assessment of risk factors and Index of suspicion e.g. cushings, hypothyroidism, sleep apnoea etc. (associated with upper body obesity)

Surgical Techniques:-

The bariatric procedures are grouped into 3 categories:

- 1. Restrictive AGB (Adjustable Gastric Band), VBG (Vertical Banded Gastroplasty)
- 2. Combined Roux-en-Y Gastric Bypass.
- 3. Malabsorptive BPD (Bilio-Pancreatic Diversion), BPD + DS (Duodenal Switch)

1. Restrictive: Adjustable Gastric Band

It is the least invasive procedure possible amongst all bariatric surgeries. It is accomplished by placing a silastic band around the upper part of the stomach to create a tiny stomach pouch. Hence early satiety is attained. The band is adjustable, that is, it can be inflated / deflated with saline via the access port placed subcutaneously and fixed to the rectus sheath. It is preferred in less obese patient.

It has its own advantages in that it can be placed laparoscopically, does not involve division or anastomosis of stomach or bowel, is adjustable and reversible. It allows a slower weight loss, which is more physiological and is associated with lesser nutritional problems.

It has a low morbidity with negligible mortality. Complications reported, occur in less than 2% of patients, which are band erosion or slippage, injury to nearby organs, failure of esophageal function, hardware breakage and reaction to silastic material in band. The reduction in weight, according to some studies, is slower than Gastric bypass.

2. Combined: Roux -en- Y GBP

It involves creation of a gastric pouch of volume 20ml after transecting the stomach. A Roux-en-Y jejunal limb is constructed and attached to the pouch.

The advantages of this procedure over others are that it is a time-tested procedure and affords consistent weight loss with significant improvement in co-morbidities. Like others, it too can be performed laparoscopically. One of the side effects of this procedure is that it is associated with dumping syndrome, which again helps in controlling their food intake, although it is troublesome for the patient.. Unlike AGB, it is not a reversible procedure and is associated with nutritional disturbances and hence requires long-term follow up and mineral supplementation.

3. Malabsorptive: Bilio-Pancreatic Diversion + Duodenal switch

The stomach is divided at D1 so as to preserve the function of pylorus hence allowing normal filling of stomach & maintenance of satiety sensation. A modification of this procedure involves a vertical sleeve gastrectomy. The advantage of this procedure is that it can be performed laparoscopically. It is an excellent option for super obese i.e. BMI > 50. It affords good relief from comorbidities, like diabetes & hypertension.

Again, these patients need to be monitored for nutritional & metabolic abnormalities and thus should receive aggressive supplementation. It has a steep learning curve.

Virtually all bariatric procedures may be performed by minimal access. However sufficient experience is required both in bariatric and advanced laparoscopic surgery. It is wise to begin with the simplest and least morbid procedure (gastric lapband) and then move on to more complex procedures.

It must be emphasized that a well trained operating team familiar with the equipment instrument and techniques of bariatric surgery augments the overall performance and outcome of the procedure.

Post operative management

- Multidisciplinary approach
- Management of complication Early / late
- Maintain permanent loss at least half of the preoperative excess weight (usually reverses most weight loss related comorbidities) in 70% of patients – strong role for a good dietician nutritionist.
- Surgical failures reoperative bariatric surgery
- Regular counseling
- Support groups
- Developing exercise schedule

Dispensing quality care

- Assume that obese individuals know they are overweight
- Listen patiently to patients presenting problem, independent of weight
- Provide unprejudiced care to obese patients medical advise to be same for lean / obese patients
- Do not blame the patient for less than desired outcome

Creating a user friendly office

- The weighing scale should be calibrated for all patients
- Have examination gowns in all sizes
- Use of appropriate sized BP cuff etc.

Summary

Morbid obesity is a chronic disease of multifactorial origin. Approach to treating morbid obesity must be multidisciplinary and realistic. Bariatric surgery is medically indicated for treating this disease.

Nearly all bariatric procedures may be performed laparoscopically. It is prudent to be conversant with the least morbid procedure before progressing to the more complex procedure. The nutritionist has a significant role in pre and post-operative diet management of these patients and may contribute significantly for maintaining long term weight loss. Management of these patients is prolonged (usually lifetime). Awareness of surgical complications and their management is a must for all bariatric surgeon.