The Ocular Communique

The Journal of the Maharashtra Ophthalmological Society

Editor : Dr. B. K. Nayak

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Contents

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Instructions to Authors

The Journal of Maharashtra Ophthalmological Society publishes three issues in a year. It accepts original articles, rare case reports and short reviews. All the articles are subject to editorial revision.

1. All the papers should be accompanied by a statement that, they have not been published in any other journal or presented in any conference and that, if accepted they will not be offered to any other publisher / conference without the consent of the Editorial Board. All the authors should sign in this statement.

   For the articles already published earlier elsewhere, written permission of the relevant authority should be accompanying the article.

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3. The matter must be typewritten in double space on bond paper with adequate margin.

4. The title along with the author's name, address of the institution, and reprint request must be typed in a separate page.

5. The manuscript in the case of scientific papers must be in the following format:

   Title, Abstract & Keywords, Introduction, Material and Methods, Results, Discussions and Reference. The abstract must be short, conveying the aim, method of study, result and conclusions.

6. Table should be typed on separate pages and numbered, titled and with suitable column headings.

7. Photographs should be submitted in quarter size (3 ¼ " x 4 ½ ") on glossy paper. Indication of top and figure number should be shown on the back of the photograph. No author's name or institution must be mentioned in the article or on the photograph. Title of the article may be written at the back of the photograph. Legends must be submitted on a separate paper.

8. Colour photographs shall be published at author's cost. (Film Scanning Charges, extra printing charges etc.) The amount should be paid in advance.

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Examples are given hereunder.

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For books: Mandel Wanger etal,Atlas of corneal diseases, W.B. Sanders, First edition, 1989, 80-2.

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Editorial

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Original Article

Severe Anterior Capsular Phimosis in a High Myope

Dr. Minu Ramakrishnan, Dr. Vishal Raval, Dr. Riddhi Shah, Dr. Sophia Ansari

Anterior capsular phimosis (ACP) or capsule contraction syndrome is a known complication following continuous curvilinear capsulorrhexis(CCC). The degree of ACP is believed to be related to some types of ocular entities like pseudoexfoliation syndrome, diabetic retinopathy, retinitis pigmentosa and myotonic dystrophy. This anterior capsular contraction syndrome occurs when residual lens epithelial cells, located within the capsular bag following cataract extraction, proliferate into myofibroblasts. These myofibroblasts opacify the anterior capsule and produce contractile forces. Patients with weakened lens zonules are unable to counteract these contractile forces, causing the opacified anterior capsule to cinch down and eventually obscure the visual axis.

Case Report

A 45 year old high myope presented with gradual DOV RE for distance and near.He was operated for cataract extraction with PCIOL implantation RE 4 months back with good postop vision.(no zonular laxity, uveitis or pseudoexfoliation noted preop, 5.5 mm rrhexis, SICS with one piece PMMA Rayner IOL implantation, no capsular polishing done).Patient has h/o using high powered glasses since childhood. He also gives h/o cryopexy done in both eyes for peripheral retinal degeneration 3 months prior to cataract surgery.

O/E: Visual Acuity RE - FC 2 metres, BCVA – 6/36. LE- BCVA 6/24 with -8.0d sph.

Post dilation RE: S/L- tilted, crumpled PCIOL, haptics overlapping optic, severe ant. capsule phimosis and opacification. stretched zonules seen(Fig 1). Retinoscopy showing irregular astigmatism with slight hyperopic shift.

Fundus- myopic crescent with peripheral cryo marks.

LE- NS 2 cataract with well dilating pupil and no pseudoexfoliation.

ND YAG capsulotomy was deferred in view of small 1.5 mm central aperture and thick capsular rim to avoid damage to IOL.Hence patient was taken up for surgical capsulectomy. Multiple radial snip capsulotomies were done using capsulotomy scissors, all around the pupillary area, all tags were also cut.(Fig 2,3)The capsular bag was opened up with viscoelastic ,with repositioning of the IOL.

Patient recovered good vision postop(6/12)with improvement in the refractive status.

Other eye surgery was planned with large capsulorrhexis and anterior capsular + posterior capsular polishing,with rigid one piece PMMA lens, which is doing well 6 months post surgery.

Discussion

This entity- 1st described by Hansen, Davison in 1993, who coined the term`capsule contraction syndrome'. Risk factors documented include- pseudoexfoliation with weak zonules[1,2],advanced age, uveitis, pars planitis[1], myopia, retinitis pigmentosa[3], diabetic retinopathy[4], myotonic muscular dystrophy[1]. Also associated with silicone plate haptic IOLs[5](least with acrylic) and presence of a small original capsulorrhexis[1].

Anterior capsular phimosis is caused by proliferation of residual lens epithelial cells followed by fibrous metaplasia. In these cells, alpha-smooth muscle actin leads to anterior capsular contraction. ACP depends on contraction of a fibrous membrane that can be present either beneath the anterior capsule or on the outer surface of the anterior capsule. Although the pathogenic mechanisms responsible for excessive capsule fibrosis and contracture are not well understood, several histopathologic studies have identified the cell types associated with pseudophakic fibrosis. Some studies suggest that the IOL optic material may influence the development of anterior capsular fibrosis. IOL shape, IOL biomaterial, and more specifically, the hydrophilicity of the biomaterial have been associated with a high degree of postoperative inflammation.

It has been suggested that surgical invasion and contact with the IOL stimulate residual lens epithelial cells (LECs) to produce cytokines. These cytokines may induce collagen production and fibrous proliferation. Some of the cytokines induce transformation of the LECs into myofibroblasts, that contain contractile filaments. In parallel with these processes, aqueous prostaglandin E2 concentration is elevated, leading to blood aqueous barrier breakdown and an increased aqueous protein concentration. Previous studies have demonstrated disruption of both the blood-retinal and the blood-aqueous barriers in eyes of patients with retinitis pigmentosa. An increase in blood-derived cytokines in the aqueous humor of these eyes after cataract surgery, results in increased activation of LECs with fibrosis and contracture of the anterior capsule.

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Sequelae: include complete occlusion of anterior capsular opening[6],capsular bag distension syndrome, in-the-bag IOL decentration / dislocation[7], and in severe cases-retinal detachment [8]or choroidal effusions[9] can be caused by traction on zonules.

Prevention strategies include : large capsulorrhexis [1]with / without relaxing incisions (surgical / laser) in high risk cases, use of capsular tension ring[10](not always protective), proper removal of LECs from anterior capsule- using polisher, irrigation cannula or vacuum aspiration curette[11], use of 3 piece IOL or making radial relaxing incisions on the anterior capsule[12].

In limited forms of capsular shrinkage without invasion of the optical zone, Nd YAG laser capsulotomy[13] is considered the first choice. In severe cases with dense fibrous plaques, laser capsulotomy will result in incompletely resolved capsular debris, enhancing the risk for inflammation and recurrence. For this reason, and to avoid the risk of further rise in IOP due to these debris, and with the aim to align the lens properly in the capsular bag, we decided to perform the surgical approach to remove the fibrotic tissue. Other techniques described in literature include the use of intraocular diathermy to remove fibrotic anterior capsules and the excision of the central part of the fibrotic anterior capsule with microscissors in cases with complete occlusion of the capsular opening, or use of vitrector for the same purpose.

Conclusion

Anterior capsular phimosis / capsular contraction syndrome is a known entity. Unless severe, there is no need for intervention. Also, most cases recover good vision with anterior capsulotomy alone (laser / surgical). Role of cryopexy in accentuating capsular contraction is not known. In presence of risk factors, preventive strategies can be undertaken. Also, once it occurs in one eye, other eye needs definitive prevention.

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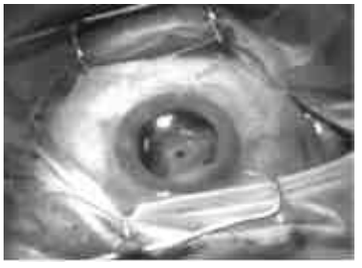
Photos

Fig 1 Slit lamp view- diffuse illumination

Fig 2 Preoperative view- under microscope.

Fig 3 Intraoperative view.



Original Article

Disability, Health and the CBR Matrix: A Paradigm Shift

Dr. G V S Murthy

Background

The World Health Organization estimates that 650 million people live with some type of disability (physical, mental, visual, hearing, learning, speech and intellectual) globally, 80% of whom live in low income countries [1]. Considering this huge burden of disability, the World Health Assembly passed a resolution (WHA58.23) in 2005 that member States of the WHO should be helped to develop policies on disability and rehabilitation and that early detection and treatment of those with disabilities was a WHO priority for action [1].

Till recently disability has been treated purely as a 'disease phenomenon' and therefore it was felt that efforts should be directed to identify and treat the physiology and pathology of the disabling impairment. This traditional approach has recently been replaced by a more vibrant and positive strategy to tackle disability using the 'social model' wherein the individual's functioning is given more importance than the impairment. The WHO has recommended the use of the International Classification of Functioning, Disability and Health (ICF) as the framework for measuring health and disability both at the individual and community level. The ICF domains are classified from body, individual and societal perspectives. The 'social model' of disability has brought about awareness that environmental barriers to participation and social life, are major causes of disability. ICF has enabled the understanding that disability is not only an issue of body structure and function but that 'activities' and 'participation' from an affected individual and a social perspective are essential to tackle problems faced by people with disability (PWD). The paradigm shift now focuses on 'inclusive communities'. An inclusive community emphasises that communities should adapt their structures and procedures to facilitate the inclusion of PWD. It places the focus on all citizens and their entitlement to equal treatment, again reinforcing the fact that the rights of all people, including those with disabilities, must be respected. The community looks at itself and considers how policies, laws, and common practices affect all community members.

United Nations Convention on the Rights of Persons with Disabilities (UNCRPD)

The UNCRPD specifically mentioned that PWD have a Right-to-Health. It recognized that PWD had the right to the enjoyment of the highest attainable standard of health without discrimination [2]. What is significant about the UNCRPD declaration is that it categorically emphasises that the right to health is not only about the access to health services but also about the access to the underlying determinants of health, like safe drinking water, adequate sanitation and housing conditions[2]. The health-related entitlements under UNCRPD include[3]:

- Right to a system of health protection.
- Right to prevention, treatment and control of diseases.
- Access to essential medicines, and
- Participation in health-related decision making.

Barriers to Health Care Services for PWD

Evidence shows that PWD have poorer health status compared to the other members of the community[4]. Data from many countries also supports the universal perception that health care utilization and health expenditure is significantly higher among PWD[5-7].

The barriers to health-care services that people with disabilities and their family members may face include[8]:

- absent or inappropriate policies and legislation – where policy and legislation do exist, they may not be implemented or enforced and can be discriminatory and/or obstructive regarding the provision of health services to people with disabilities;
- economic barriers – health interventions such as assessments, treatments and medications often require out-of-pocket payments, presenting difficulties for PWD and their families who are likely to have limited income for health care
- physical and geographical barriers – lack of

Dr. G V S Murthy

accessible transport and inaccessible hospital buildings and medical equipment are examples of common barriers, as well as the limited health-care resources of rural areas (where many people with disabilities live) and the long distances to reach services in big cities;

- communication and information barriers – communicating with health workers may be difficult, e.g. a person who is deaf might find it difficult to communicate his/her symptoms to a doctor and health information is often not available in accessible formats, such as picture formats for people with intellectual impairment;
- poor attitudes and knowledge of health workers about PWD – health personnel may have inappropriate attitudes, be prejudiced or insensitive and lack awareness and the knowledge, understanding and skills to manage health issues for PWD;
- poor knowledge and attitudes of people with disabilities about general health care and services PWD may be reluctant to use health services; many also have limited knowledge about their rights and health issues and about what health services are available.

Some people with disabilities may be more vulnerable to discrimination and exclusion than others. They may suffer multiple disadvantages because of their disability and other factors like their age, gender etc. and so find it more difficult to access health-care services. It has been shown that women with disabilities are significantly more likely to have experienced intimate partner violence and also report a significantly higher unmet health need compared with those without disabilities[9].

Health concerns of the disabled can be effectively addressed by using a two-pronged strategy:

- Realize that PWD need general health services like the rest of the population.
- Realize that many PWD will have specific needs because of their impairment and would require care and support for the same.

Inclusive Health

Just like inclusive education, recently people are talking about 'inclusive health'. Inclusive health means that all individuals can access health care irrespective of impairment, gender, age, colour, race, religion and socioeconomic status. To make inclusive health work, it is important that adequate time should be invested to develop partnerships by sufficient consultation and collaboration, include and empower PWD and their organizations and to view capacity building as a two-way process[10]. In effect it means that improving lives of PWD is best achieved through processes that are inclusive, owned and driven by local communities[10].

Community Based Rehabilitation

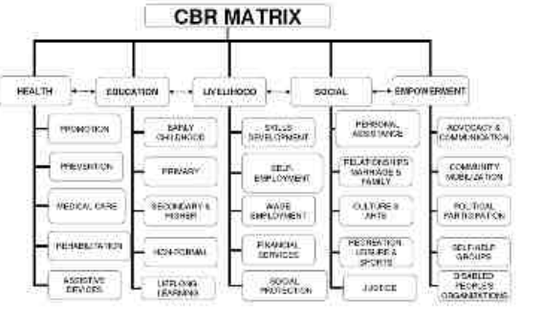
Community-based rehabilitation (CBR) was initiated by the World Health Organization (WHO) following the Declaration of Alma-Ata in 1978. It was promoted as a strategy to improve access to rehabilitation services for people with disabilities in low-income and middle-income countries, by making optimum use of local resources[11]. CBR is currently implemented in more than 90 countries. Recently Guidelines to implement CBR activities was released in October 2010[11].

CBR Matrix

To capture the benefits of a broad development strategy, a CBR Matrix has been developed. This provides a common framework for CBR programmes (Figure 1). The CBR matrix consists of five key components:

- Health
- Education
- Livelihood
- Social Participation
- Empowerment

Within each of the five key components, there are five essential elements:, four of which relate to the development sector and one to the empowerment of people with disabilities, their families and communities. The CBR Matrix and Guidelines are intended to improve the quality of life of PWD[11]. It may not be possible to implement every component and element of the CBR matrix. Therefore the matrix has been designed in such a manner that it allows specific programmes to select the options which best meet local needs, priorities and resources[11].



Source: [11]

To illustrate the use of the CBR Matrix, one could take the example of a young blind woman who became blind due to corneal scarring from vitamin A deficiency in her childhood. She will not currently benefit from health promotion or prevention activities or with assistive devices in the health domain. However she would have reproductive health needs and would require medical care for common ailments as would other women in the community. She would benefit from rehabilitation elements of the Health component in activities such as daily living and life skills. In relation to the education component, she will be too old to benefit from most of the educational elements. However life skills can still be imparted as these skills need a lifelong learning. If she were to be supported to set up a petty shop selling groceries and given basic accountancy skills to run the shop, the livelihood component would be addressed. This would include elements of skill development, self-employment and financial services. In addition, if she were to access schemes like disability pension, it would cover the element of social protection. Because she now has a viable source of income, she decides to get married and have children then the component of social participation is addressed. She may decide to learn classical singing and then she would be participating in culture and arts elements. All these would also impact on the component of empowerment as social participation has been facilitated as has been participation in a disabled peoples' organization (DPO). She could also join a self help group set up by the DPO.

The above example clearly demonstrates that a lot can be achieved through adaptation of the CBR matrix, even if all the components and elements were not to be addressed. What can be done in a particular situation is dependant on the local situation, services and resources. The CBR matrix therefore has facilitated a paradigm shift in addressing disability concerns in the developing world.

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Original Article

Visual Fields in Glaucoma – An Overview

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Dr. Roshni shetty , Dr. Vijay Jadhav

The Normal Visual Field

The field of vision is defined as that volume of space which is simultaneously perceived by a steadily fixating eye.

- 60°superior field,
- 75°into the inferior field,
- 110°temporally
- 60°nasally.

Effects of Normal Aging:

- changes in the ocular media,
- linear reduction in pupil diameter ,
- decrease in the absorbance efficiency of photopigments, and
- neural losses in both the retina and the retino-geniculostriate pathway

Correlation Between Onh and Vf Defects

- disc changes precede detectable field loss
- presence or absence of glaucomatous field defects can be predicted from the appearance of the ONH
- nerve fiber loss occur before reproducible field defects

Correlation Between Onh and Vf Defects

- extent of axonal loss may be much greater
- 25% to 35% of the RGC may be lost in an eye with a normal field by the time field defects are found
- 20% loss of large RGC, in the central 30 degrees of the retina, correlated with a 5-dB sensitivity loss and a 40% loss corresponded with a 10-dB decrease

Modes For Visual Field Assessment

- Amslers grid
- Confrontation tests
- Goldmann perimetry, Friedmann perimetry and Bjerrum screens, Humphrey Field Analyser, Octopus perimeter, Dicon perimeter and Henson perimeter

Bjerrum Screen - Terminologies

- Isopter: contour lines on a map which enclose an area within which a target of given size is visible
- Absolute scotoma: area of total visual loss
- Relative scotoma: partial visual loss
- Decibels: 0.1 log unit of attenuation of maximal available stimulus

Differential light sensitivity: degree by which the luminance of a target exceeds the background luminance in order to be perceives by the eye

Visible threshold: luminance of a given stimulus which when presented is perceived 50% of the times

Basic Principles of Visual Field Testing

Stimuli: Spots of light of various diameter and intensity are projected. The absolute light intensity is measured in units of luminance and is expressed in logarithmic units referred to as decibels

- Stimulus Size-spatial summation
- Exposure time-temporal summation
- Background illumination

Kinetic Perimetry

In kinetic perimetry, a stimulus is moved from a non-seeing area of the visual field to a seeing area along a set meridian. The procedure is repeated with the use of the same stimulus along other meridians, usually spaced every 15°.

In kinetic perimetry, one attempts to find locations in the visual field of equal retinal sensitivity. By joining these areas of equal sensitivity, an isopter is defined. The luminance and the size of the target is changed to plot other isopters.

Static Perimetry

In static perimetry, the size and location of the test target remain constant. The retinal sensitivity at a specific location is determined by varying the brightness of the test target. The shape of the island is defined by repeating the threshold measurement at various locations in the field of vision.

Manual Perimetry: the Goldmann Visual Field

calibrated bowl: 31.5 asb

The stimuli used to plot an isopter are identified by a Roman numeral(I-V), a number(1-4), and a letter(a-e)

Goldmann Visual Field

Humphrey automated threshold perimetry

- the perimetric unit and the control unit
- provides interaction between the operator and computer
- Stimuli are presented in a random, unpredictable fashion

The Differential Light Threshold

Perimetry measures retinal sensitivity (the ability of the eye to detect a difference in contrast between a test target and the background luminance).

The differential light threshold is designated as the dimmest target seen 50% of the time.

The differential light threshold

Threshold at a specific retinal location can be measured directly from a frequency-of-seeing curve.

Stimuli of varying intensities are presented multiple times at one retinal location

Frequency of seeing curve Bracketing strategy

- Impractical to perform FOC. 4-2 algorithm is employed.

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- Octopus perimeter: average of the last seen and unseen intensities.
- HFA uses the intensity of the last seen stimulus as threshold.

What do we achieve?

- Threshold

What do we compare this to?

- A set of values stored in the software

Which are normal Strategies Supra-threshold Static Perimetry?

- present a stimulus brighter than the anticipated normal value for the corresponding retinal location
- detect an abnormality to characterize the topographical nature of the abnormality and to quantify the boundary of the field

Screening Programs

1. Age related screenings: 8 db more than mean age corrected normal sensitivity at each test point
2. Threshold related: target exceeds expected threshold the same across the visual field.
3. Single intensity: by default 10 decibels (III,4e), —is presented at all points

Screening strategies

1. Two zone mode: there is no further retesting, points missed are marker black
2. Three zone:If the ST target is missed twice, the spot is retested later with a maximum intensity target of 0 dB. The 3 zones of results are: normal, relative defect, absolute
3. Quantify deect mode

Full Threshold Perimetry

Measures the retinal threshold at 76 points within the central 30 degrees,

Follows the staircase strategy with 2 crossovers,if thresholds are more than 4 db from expected values, the location is retested

FASTPAC

Thresholding from a single threshold crossing in 3-dB increments, threshold is crossed only once, provides time reduction at some expense of accuracy and reliability

Swedish Interactive Threshold Algorithm (SITA)

Significantly minimizes test time without significant reduction of data quality. SITA uses new concepts, such as visual field modelling, that utilizes frequency-of-seeing curves for glaucoma and normal patients

- threshold determinations for the four primary points in the quadrants
- Maximum posterior estimate
- Measurement error for that value
- incorporating data from neighbouring points in modelling responses

Time Short-Wavelength Automated Perimetry

Blue stimulus (440 nm) is presented on a background of yellow illumination.

The yellow background desensitizes the green and red cones, whereas the blue stimulus activates the blue cones.

SWAPSWAP-controversy

Identifying preferential damage to the blue/yellow cone system, or whether the testing of only a subset of the visual system enables earlier detection even if the damage is not selective.

Frequency Doubling Technology

- tests the magnocellular pathway.
- large diameter fibers –3 -5% of RGC
- 17 regions -tested within central 20°
- black and white bands flicker at 25 Hz
- 97% sensitivity and specificity

Commonly used programs for glaucoma.

Octopus program 32 and the Humphrey program 30-2.

These programs are tests of the central 30°with 6°of separation between locations.

Humphrey program 24-2 eliminates the most peripheral ring of test locations from program 30-2, except in the nasal step region, and tests only the central 24°.

Macular programme: central 5°with 2°separation

Data Interpretation

- Name
- Fixation

Fixation losses

Fixation losses: indicates steadiness of gaze detected by presenting stimuli in the blind spot. A FL of more than 20%needs repetition of the test. A high FL may also occur if the instrument has incorrectly spotted the blind spot

False positives: they are detected when a stimulus is accompanied by a sound. A high FP score more than 33%suggests a trigger happy patient and is the most sensitive indicator of an unreliable field

False negatives: are detected by presenting a stimulus much brighter than threshold at a location where sensitivity has already been recorded. Indicates inattention or due to short term fluctuation. A FN more than 33%needs repetition

Total deviation

Pattern deviation

Mean Deviation

Indicates any overall depression (or elevation) of the patient's hill of vision

Pattern Standard Deviation

Shows how different the numbers, in the total deviation plot, are from each other, highlights any irregularity in the visual field, irrespective of any overall depression in the hill of vision

Short-term Fluctuation

Is the intra-test variation in threshold. It is essentially

the error in threshold determination, its an indicator of reliability; but it could also be an indicator of pathology, diseased points have a greater variability.

Corrected Pattern Standard Deviation

Irregularities in the visual field irrespective of any overall depression due to media opacities as well as after adjusting for errors of threshold determination (SF).

(CSPD)2= (PSD)2+ k(SF)2: k=1.28(30-2). 1.14(24-2)

Glaucoma Hemifield test

Outside N limits

Borderline<3%

Within N limits

Confirmation of glaucoma field defect: Andersons criterion

- 3 or more non-edge points that are depressed as found in less than 5% of the population; one of those points should be depressed to an extent found expected in 1% of the population
- The Corrected Pattern Standard Deviation (CPSD) (or the PSD) should be depressed to an extent found in less than 5% of the population
- Is the Glaucoma hemifield test outside normal limits?

Andersons criterion

If the clinical features strongly indicate glaucoma, even one criterion is good enough to make a diagnosis.

If the clinical features are not suspicious all three criteria must be positive before one even begins to consider a diagnosis.

Progression of Fields

- Overview
- Glaucoma change probability
- Change analysis

Overview Print out

Sequential series of fields of same patient printed on a single piece paper that contains all the data that a single field analysis provides,Up to 16 fields can be printed on a single piece of paper, it determines whether progression is due to cataract or glaucoma.

Overview Print outGlaucoma change probability

Provides necessary statistical help.Two fields as the baseline fields done after the learning curve of the patient. The baseline may need to be changed if the patient has undergone intervention (such as a trabeculectomy),

GCPAGCPA Change analysis

- Analyses 16 test results
- Presents change in the form of a box plot
- Summary of the 4 indices
- Linear regression analysis of MD

Glaucomatous visual field defects

The superior and inferior poles of the optic nerve head are most vulnerable to glaucomatous damage. These areas may be watershed areas at the junction of the vascular supply from adjacent ciliary vessels. The lamina cribrosa shows that the pores in the ST and IT areas are larger. These larger pores may make these regions more vulnerable to compression.

Visual field defects include

- Paracentral defects
- Arcuate scotomas
- Nasal step
- Temporal wedge

Gaze tracker

- Records every movement of the eye
- off fixation as an upward spike on the tracing
- blink is recorded as a downward spike

Octopus printout

Octopus global indices

- MS-mean sensitivity-average of all measured values
- MD -mean defect , average corrected or age
- LV loss variance (PSD)
- SF
- CLV: corrected LV (CSPD)
- Reliability factor

Octopus criterion for a field defect

- MD> 2 db
- LV> 6 db
- At least 7 points with sensitivity decreased by > 5db, three of them being contiguous

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Test parameters-octopus vs HFA

Parameter	Octopus 300	HFA700
Bowl type	Direct projection	Aspheric bowl
Background luminance	31.4 asb	31.6 asb
Stimulus size Duration Luminance or0 db	Goldmann 3 and 5 100ms 4800asb	Goldmann I –V 200ms 10000 asb
Measuring range	0-40 db	0-50 db
Test strategies	4-2-1 db bracketing strategy Dynamic TOP	4-2-1 db bracketing strategy SITA standard SITA fast

Accommodation and Anomalies

Dr. Nayana Potdar, Dr. Chhaya Shinde, Dr. Neha Gadaria, Dr. Vishnukant Ghonsikar

Definition :In an emmetropic eye parallel rays of light coming from infinity are brought to focus on the retina .Our eyes have been provided with a mechanism by which we can even focus the diverging rays coming from a near object on the retina.

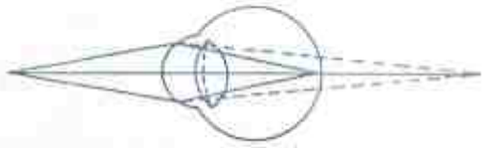


Fig. 4.1. Effect of accommodation on divergent rays entering the eye

- Punctum proximum
- Punctum remotum
- Range of accommodation
- Amplitude of accommodation

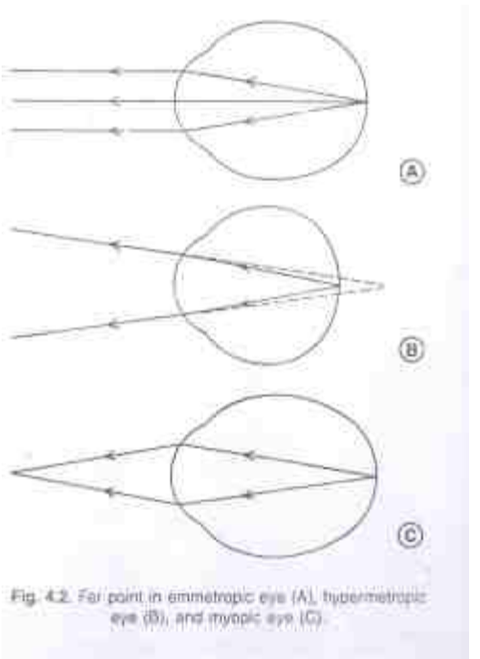


Fig. 4.2. Far point in emmetropic eye (A), hypermetropic eye (B), and myopic eye (C).

v In an emmetropic eye far point is at infinity near point varies With age being about 7 cm at the age of 10 years,25 cm at 40 years,and 33cms at 45 years

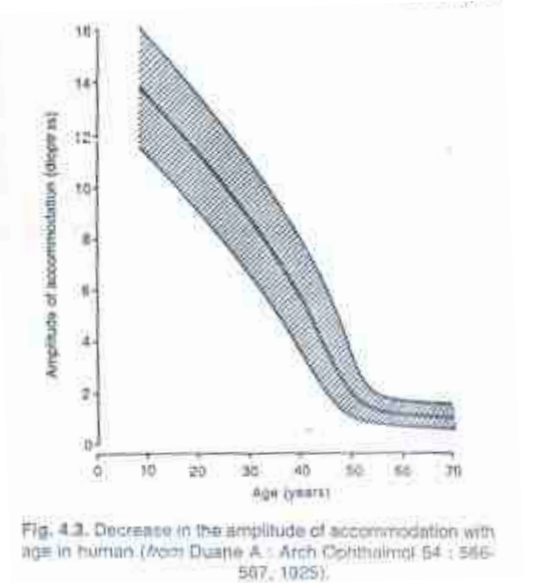
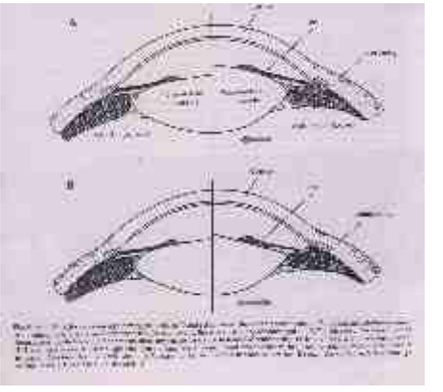


Fig. 4.3. Decrease in the amplitude of accommodation with age in human (From Duane A : Arch Ophthalmol 54 : 566-587, 1925).

Theories of accommodation

v Helmholtz's theory
v Schachar's theory
v Tscherning theory



Role of the lens capsule

- Helmholtz suggested that the lens capsule has elastic Properties which account for the change in the shape Of the lens when it is free from the tension of the zonules. The lens surfaces are not perfectly spherical in contour; The anterior surface is more convex centrally during accommodation.
- Fincham suggested that variations in the thickness of the lens capsule account for the local variations in curvature. During accommodation the thicker ring of anterior capsule Surrounding the central region contracts under the lessened Zonular traction while

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the thinner central capsule bulges forward in a more pronounced fashion. The physiological anterior lenticonus thus formed has a short radius of curvature and high refraction.

- Stimulus for accommodation
  Image blur
  Apparent size and distance of the object
  Chromatic aberrations
  Scanning movements of the eye
- Reaction Time
  Average reaction time for far to near accommodation is 0.64s
  Average reaction time for near to far is 0.56s
  Reaction time is larger than that for the contraction of pupil to light[0.26to 0.30]
  Reaction time of convergence response is 0.20s

Ocular changes in accommodation

- Slackening of zonules
- Changes in the curvature of lens surface
- Anterior pole
- Axial thickness
- Changes in the tension of lens capsule
- Lens sinks down
- Changes within the lens substance
- Pupillary constricton and convergence of eyes
- The choroid
- The ora serrata
- Diminished or deficient accommodation
- Physiological [presbyopia]
- Pharmacological [Cycloplegia]
- Pathological
  Ill sustained accommodation
  Inertia of accommodation
  Paralysis of accommodation
- Increased accommodation
- Excessive accommodation
- Spasm of accommodation

Definition

Presbyopia [Eyesight of old age] ; The condition of failing near vision due to age related decrease in the amplitude of accom Modation or increase in the punctum proximum

Pathophysiology

Decrease in the accommodative power of the crystalline lens with increasing age is due to

- Decrease in the elasticity and plasticity of the crystalline lens [due to age related sclerosis]
- Age related decrease in the power of the ciliary muscles.

Causes of premature presbyopia

- Uncorrected hypermetropia
- Premature sclerosis of the crystalline lens
- General debility causing presenile weakness of ciliary muscle
- Chronic simpl eglaucoma

Symptoms

Symptoms of presbyopia develop when the amount of accommodation needed to focus at near exceeds more than half of the total amplitude of the eye

An uncorrected hypermetrope and a chronically undercorrected myope develop earlier

- Difficulty in near vision
- Asthenopic symptoms
- Intermittent diplopia

Testing

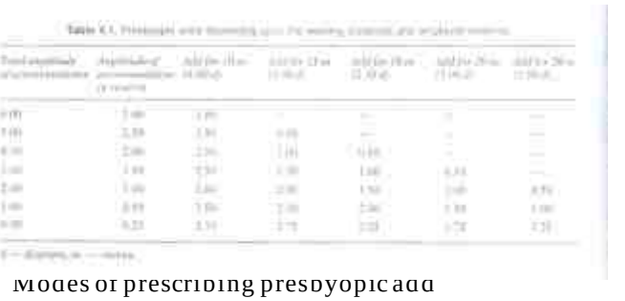
Monoocularly as well as binocularly

Treatment

The difference between the distance correction and the strength needed for near vision is called add.

45years 1to1.25D
50years 1.50 to 1.75 D
55 years 2 TO 2.25 D
60 years 2.5 to 3.00D

Adjustment for working distance



Modes of prescribing presbyopic add

- Single vision reading glasses
- Bifocal glasses
- Trifocal glasses
- Multifocal OR varifocal glasses

Basic principles for presbyopic correction

1] Always find out refractive error for distance and first correct
2] The presbyopic add should leave 50 percent of accommodation in reserve
3] Near point determined considering the profession of the patient
4] Weakest convex lens at which the pt can see clearly at near point
5] Test each eye seperately and add it to the distance correction
6] An additional correction for the intermediate distance may be necessary

Management of Iridodialysis With Traumatic Cataract in Single Sitting

Dr, Pooja Jain, Dr. Asif Virani, Dr. Smita Patil , Dr. Sufiyan Shaikh, Dr. Nayana Potdar ,Dr. Chhaya Shinde

A 6 year old female child presented to the OPD of sion hospital ,Mumbai with history of firecracker injury to left eye followed by diminution of vision and white reflex in LE since 2 months .On examination RE vision 6/6 LE pl pr in all quadrants On slit lamp left eye showed traumatic cataract with d shaped pupil with posterior synechiae & iridodialysis in inferolateral quadrant.(ITQ). Fundus left eye no glow visualized .UBM Left eye showed iridodialysis from 2 to 6 o clock and zonular tear from 3 to 6 o clock . USG Bscan showed normal posterior segment and no evidence of retinal detachment.

Patient was posted for ECCE through STCV and IOL implantation and iridodialysis repair under general anaesthesia.3mm scleral tunnel at 11 o'cl &2mm scleral shelf was made at 5 o'cl. Synechiolysis in ITQ was done After cortical aspiration, heparin coated foldable IOL was implanted in the bag . PCO was noted in ITQ. Iridodialysis repair was done by passing one straight needle of double ended 10 -0 prolene suture through main tunnel, then through the iris stroma of one torn iris leaflet at 3 o'cl further taking out the needle thru scleral shelf at 5 o'cl,then another straight needle passed through the main tunnel, other end of iris leaflet at 6 o'cl and sclera shelf,both ends of suture tied together & buried in the shelf.

FIG 1 shows the 2 straight needles of 10-prolene passing thru main tunnel at 11o'cl traversing torn iris to emerge at scleral shelf at 5o'cl

FIG 2 shows the correction achieved after pulling the 2 ends of the sutFIGFG

Fig 3 shows straight needle of 10 – prolene traversing the torn end of iris & scleral shelf .Fig 4 shows needle pulled out thru shelf with suture holding the iris

Fig5 shows corrected almost circular slightly peaked pupil with good cosmesis PCIOL in place,PCO in the inferotemporal quadrant.Wound & side port is closed

FIG 6 PODay1- left eye vision 6/18 ,AC well formed ,pupil was circular, PCIOL in place,with normal IOP.Patient was started on local antibiotic steroids & homatropine -4wk.1 month FU-LE quiet, vision-6/12 with good cosmetic appearance

Discussion

Iridodialysis frequently occurs as a complication of blunt trauma to the globe & leads to separation of iris root from ciliary body. Small iridodialysis with minimal symptoms require no treatment. Large iridodialysis with double pupil effect,monocular diplopia, glare,poor cosmesis & photophobia require surgical intervention. Surgical repair is usually done by 10-0 prolene suture taking the base of iris avulsion and suturing it to the scleral spur and ciliary body junction,by open / closed chamber technique.

Surgical Planning

Preoperatively, you must determine whether there is sufficient iris tissue remaining to achieve the desired goals. It is often difficult to assess how much tissue is present because the iris stroma may be contracted or rolled over. Careful examination and review of prior operative notes are helpful in determining whether tissue has been removed. Typically, there is more iris present than you might think based on slit-lamp examination. Iris tissue is usually very stretchable and can cover larger areas than you might initially anticipate. Usually, if the patient retains two-thirds or more of normal iris tissue, surgical repair can produce a good functional and anatomic result. For cases in which large amounts of iris tissue is absent, artificial iris diaphragms, overlapping rings or sectoral implants may be a more appropriate option to augment the remaining native iris tissue

Principles of Iris Repair

1. Instillation of a miotic agent, such as acetylcholine or carbachol, puts the iris stroma on maximal stretch, increasing the surface area.

2.Intracameral manipulations should be performed under viscoelastic agents to prevent chamber volatility, iris stretching and corneal

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endothelial damage. When choosing your viscoelastic agent, remember that you may be removing it manually through a small incision. Highly retentive agents may be difficult to remove without automated irrigation and aspiration, while retained bits of overly viscous materials can cause a significant postoperative intraocular pressure rise.

3.The very soft and friable consistency of the iris demands an atraumatic technique. Often, posterior or peripheral anterior synechiae prevent proper mobilization of the iris leaflets. Therefore, gentle blunt or sharp synechiolysis may be the first step in repair. When the sphincter is involved in the injury or damage, reapposing the severed pupil margin establishes a central pupil and creates a more taut iris diaphragm, facilitating further steps.

4.Because patients may develop glare symptoms when the optic margin of an implant lens is exposed, the repaired iris leaflets should cover all IOL edges. When an implant placement or exchange is performed coincident with iris repair, a larger optic implant may facilitate this task.

Conclusion

Iridodialysis repair techniques are classified as open chamber & closed chamber . Open chamber techniques access the iridodialysis site thru a limbal self-sealing incision or a scleral tunnel . Although access to the AC is attained with a needle in closed chamber techniques, the knot of the suture is left subconjunctivally, buried to the sclera or put under a scleral flap as in open chamber techniques .Similar to subconjunctival knots, sclera buried knots cause erosion ,discomfort & infection. To avoid this scleral flap technique like flaps in SFIOL implantation is used to bury the knots.However, scleral flap preparation is time consuming and hard to perfect, & can cause erosion and infection

Advantages of Our Surgical Technique

The prolene suture is resistant to hydrolysis in the anterior chamber hence a better choice than nylon.Less irritation, less exposure,As knot is burried .

Single sitting

Safe ,cheap

Atraumatic with minimal instrumentation

Good cosmesis

FIG A

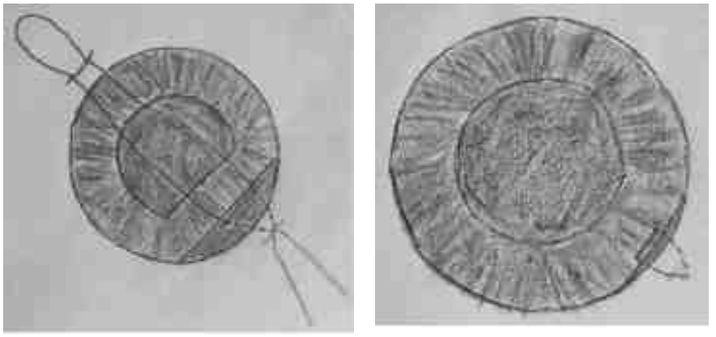


FIG 1&2

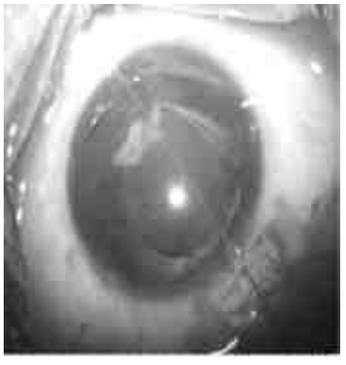


FIG3

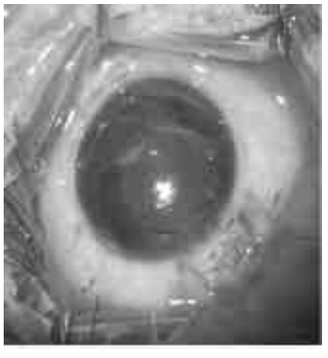


FIG 5

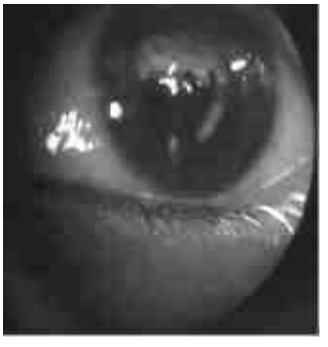


FIG 6

Original Article

Dry Eye Syndrome – an Overview

Dr. Vishnukant Ghonsikar, Dr. Nayana Potdar, Dr. Chhaya Shinde, Dr. Sufian shaikh

Dry eye syndrome (DES) is a break down of the tear film and is one of the most common conditions affecting the eyes. Most people don't realize how extremely important tears are in providing comfortable eyes, clear vision, and protection from infections.

DRY EYE is a result of:

1. not enough tears being produced because of tear gland (lacrimal gland) dysfunction , and / or
2. poor composition of any, or all of the 3 layers that make up tears.

Both conditions result in the tear film breaking down. This break down causes dry areas on the cornea and results in dry eye symptoms

Symptoms

The symptoms can vary greatly and range from mild to severe.

Symptoms include:

- General irritation
- Burning
- Foreign body sensation
- Itching
- Excess tearing
- Eye pain or soreness
- Fluctuating vision
- Mucous discharge
- Redness
- Contact lens discomfort

These symptoms are often amplified or made worse by smoking, wind, heat, low humidity, or prolonged use of the eyes (e.g. computer use or reading).

Causes

There are many conditions and factors which can contribute to causing dry eye. Like most eye conditions, dry eye syndrome is often related to other health conditions in the rest of the body. These systemic health problems include digestive imbalances, and autoimmune conditions such as rheumatoid arthritis, Sjogren's syndrome and lupus erythematosus.

Dry eyes are very common problem for women and seem to be a result of fluctuations in hormone levels. Pregnant women, women who use birth control pills, and post-menopausal women on hormone replacement therapy often suffer from dry eyes.

Contact lens wear is probably the most common cause of dry eye. A contact lens can potentially disrupt the natural tear film. If your eyes do not react well with the contact lens material or they do not produce enough tears, you may not acquire comfortable and clear vision and your wearing time may be reduced significantly. Additionally, long term contact lens wear may cause a reduction in corneal sensitivity. The sensitivity of the cornea determines how many tears your eyes produce. Less sensitivity means less tears.

The next most common cause is the natural aging process. As you age, your tear production decreases. By the age of 65, tear production is reduced to about 60% from age 18. Obviously, this is quite a significant reduction that results in increased discomfort.

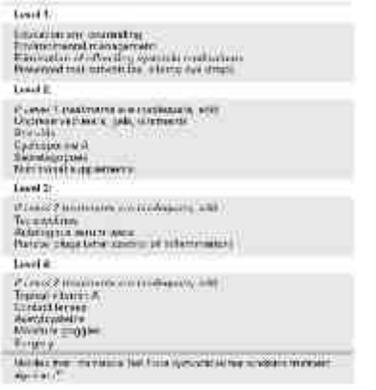
Several medications can lead to dry eyes: birth control pills, antihistamines, decongestants, codeine, morphine, heart medications, and even eye drops like Visine.

Diagnosis

There are some basic tests that may be performed like tear film break-up time, Schirmer test and fluorescein staining. Most other tests are usually performed as part of dry eye research studies. Herein we will list all tests and the cutoff value at which they are considered abnormal and indicative of dry eyes, and will discuss in details the tests that are likely to be performed in a clinical setting.

Test	Abnormal cutoff value for dry eye
Schirmer's I	less than 5 mm wetting over 5 minutes
Tear Breakup time	less than or equal to 10 seconds
Tear Meniscus height	less than or equal to 0.2 mm
Fluorescein staining	more than 3 out of 15
Rose Bengal staining	more than 3 out of 18
Tear film osmolarity	more than 316 mOsm / L
Impression cytology	more than 1
Brush cytology	more than 1
Tear lactoferrin	less than or equal to 0.9 ug / mL

ic approaches to include:



Avoidance of exacerbating factors such as low humidity, wind or drafts, dust or smoke, prolonged visual tasks, exacerbating medications.

Eyelid hygiene (particularly in patients with MGD).

Tear supplementation—for example, artificial tears, autologous serum tears.

Tear retention—for example, punctal plugs, moisture spectacles / goggles, therapeutic contact lenses, tarsorrhaphy.

Tear stimulation—for example, oral cholinergic agents such as pilocarpine or cevimeline (used off-label for aqueous-deficient DED).

Anti-inflammatory agents—for example, topical corticosteroids, oral tetracyclines, topical cyclosporine.

Other therapies—for example, nutritional supplements

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(essential fatty acids); mucolytics (topical acetylcysteine, used off-label in DED with filamentary keratitis); and topical vitamin A (off-label and controversial, but possibly useful in severe DED with squamous metaplasia or ocular surface keratinization).

Artificial tears are the mainstay of DED therapy. Most tear supplements act as lubricants; other actions may include replacement of deficient tear constituents, dilution of proinflammatory substances, reduction of tear osmolarity, and protection against osmotic stress. A wide variety of OTC artificial tear products are available, which differ with respect to a number of variables that include:

Electrolyte composition. Potassium and bicarbonate appear to be the most important.

Osmolarity / osmolality. Some studies suggest that artificial tears should ideally mimic the osmolarity of normal tears; however, others suggest that hypo-osmolar artificial tears are optimal.

Viscosity. Higher viscosity increases tear retention time and may help protect the ocular surface, but is more likely to cause visual blurring. Viscosity agents used in artificial tears include CMC, HP-guar, and lipids such as those that make up castor oil or mineral oil. Lipid-containing products are intended to decrease tear evaporation by restoring the lipid layer of the tear film. HP-guar is believed to form a bioadhesive gel, mimicking the mucous layer of the tear film.

Preservatives. There are 2 main types of preservatives: detergent (eg, benzalkonium chloride) and oxidative (eg, stabilized oxychloro complex). Detergents can irritate or damage the ocular surface with frequent use; oxidative preservatives are less likely to do so. Preserved tears are usually well tolerated in mild DED when used no more than 4 to 6 times daily. If more frequent application is required, unpreserved tears should be used.

Compatible solutes. These are small nonionic molecules (eg, glycerin) that are taken up by ocular surface epithelial cells. Because they increase intracellular osmolarity without disrupting cellular metabolism, they may protect against osmotic stress.

Although artificial tears can improve DED symptoms and objective findings, there is no evidence that they can resolve the inflammation that accompanies DED. Therefore, anti-inflammatory therapy may be indicated, including:

Topical corticosteroids. Although effective, these agents are generally recommended only for short-term use because prolonged use may result in AEs including ocular infection, glaucoma, and cataracts.

Oral tetracyclines. Based on limited evidence, oral tetracyclines have been used off-label to treat DED, primarily DED associated with ocular rosacea.

Topical cyclosporine. Topical cyclosporine is currently the only pharmacologic treatment that is FDA approved specifically for DED. Although its onset of action is relatively slow, it is safe for long-term use and appears to be disease-modifying rather than merely palliative. The most common AE is transient burning or stinging. Because blood levels are negligible even after long-term use, the risk of systemic toxicity is minimal.

Topical NSAIDs have been used off-label in DED; however, their use is controversial because they can promote corneal melting in patients with a compromised ocular surface. Some experts feel that they have no role in DED therapy.

Punctal Plugs - In more serious cases of DES where discomfort is unbearable and / or contact lens wearing time is reduced to the point of being impractical, Punctal Plugs are recommended. A punctal plug is a small collagen or silicone cylinder that is inserted into the punctum (tear drainage hole - see diagram below) to reduce the drainage of tears from the front surface of the eyes. With each blink, your eyelids coral the tears from the lateral surface of your eye towards your punctum, which is the start of your eye's drainage system. If you reduce the amount of tears that are draining, then more tears remain on the surface of your eyes and do their job to coat, comfort, and protect.

Dry eyes are very common problem for women and seem to be a result of fluctuations in hormone levels. Pregnant women, women who use birth control pills, and post-menopausal women on hormone replacement therapy often suffer from dry eyes.

Contact lens wear is probably the most common cause of DES. A contact lens is an unnatural piece of soft plastic that is placed on the cornea, that can potentially disrupt the natural tear film on the front surface of the eye. If your eyes do not react well with the contact lens material or they do not produce enough tears, you may not acquire comfortable and clear vision and your wearing time may be reduced significantly. Additionally, long term contact lens wear may cause a reduction in corneal sensitivity. The sensitivity of the cornea determines how many tears your eyes produce. Less sensitivity means less tears.

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Original Article

The Need for a Public Health Approach to Childhood Disability in India

Dr. G V S Murthy

Introduction

The World Health Organization estimates that 650 million people live with some type of disability (physical, mental, visual, hearing, learning, speech and intellectual) globally, 80% of whom live in low income countries [1]. Considering this huge burden of disability, the World Health Assembly passed a resolution (WHA58.23) in 2005 that member States of the WHO should be helped to develop policies on disability and rehabilitation and that early detection and treatment of those with disabilities was a WHO priority for action [1].

Childhood disability is a major public health problem in low income countries including India. A disabled child is more prone to abuse, higher morbidity and mortality and has reduced educational opportunities compared to other children of the same age group living in the same community. The disability-life-years (Number of years of disability x magnitude) is much more for children than disability that starts later in life due to the magnitude and the remaining life span of these children.

in South Asia and sub Saharan Africa 200 million children < 5 years of age fail to reach their cognitive potential because of poverty, poor health, nutrition and sub optimal home environments [2]. These children consequently have poor incomes in later life, higher fertility and a difficulty in meeting the needs of their own children which leads to intergenerational transmission of poverty and compromised developmental potential [3]. Available evidence shows that early detection and intervention in the first year of life, and preferably first few months of life are of paramount importance in preventing the development of permanent disability [4].

There is a strong correlation between disability and poverty [5]. Poverty leads to increased disability, and disability in turn leads to increased poverty. Thus, a majority of people with disabilities live

in poverty. Studies show that they have higher rates of unemployment compared to non-disabled people even in industrialised countries. In developing countries, where the majority of people

with disabilities live, their rates of unemployment and underemployment are undoubtedly higher [6]. Lack of access to health care and rehabilitation, education, skills training, and employment contributes to the vicious cycle of poverty and disability.

Impact of Disability on Affected Children and their families

As has already been stated, children with disabilities are less likely to attend school than their non-disabled peers.

Living with a disabled child has profound effects on the entire family [7]. For parents, it may increase stress affecting their physical and mental health. It affects their decision about work, education and having additional children. It may be associated with guilt, blame or reduced self esteem. A disabled child impacts on parenting practices, expectations of contributions from other siblings and the siblings' health and development [7]. Parents of disabled children have lower rates of social participation and tend to have smaller families [8].

Unfortunately there is a paucity of accurate data on the magnitude of childhood disability. Though substantial progress has been made in reduction of childhood mortality, there has been very little attention paid to research and progress in relation to childhood disability, especially in the low income countries where there is a higher prevalence of avoidable childhood disability [9].

Data on childhood disability is available from a few sources in India. However, the data is strictly not comparable as the definitions used to define disability vary greatly. The Census of India (2001) showed that the prevalence of disability in India was 2.2% translating into 21.9 million affected individuals [10]. The data also showed that 14.9% of the disabled were children aged <=10 years of age [10] i.e. 3.3 million. It has also been observed that a significant proportion of children suffer from multiple impairments (e.g. vision and hearing etc.) which often interact with each other, further compounding their disability. Since half the population in India is < 20 years old, the proportion of all disabled is significantly higher amongst children and young adolescents.

The Indian Council for Medical Research (ICMR) coordinated a survey of disability among children at three centres in India in 2005 and it was observed that among children aged 0-6 years, the prevalence of disability was 8.8 / 1000 at Delhi, 6.5 / 1000 at Jaipur and 12.6 / 1000 at Lucknow [11]. Using a two stage process

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of first level screening by anganwadi workers followed by a clinical examination, a study in eastern Uttar Pradesh found a prevalence of 7.6% among children aged < 6 years of age [12]. These limited data show that there is an urgent need to harness scientifically valid evidence on disability and the affect of disability on the affected child and their families.

Access to health and rehabilitation services

A recent study in Cambodia observed that the main barriers to use of services were costs of health services and medications, costs of transportation, costs associated with missing work, lack of knowledge about relevant services and how to access them and the distance to a health or rehabilitation facility [13].

There is very limited data on the use of health services by children with disabilities in India. The World Bank reports that access to health services by people with disabilities (PWD) is lower than the non PWB peers [14]. The Report also highlighted that PWD in urban areas and those staying with their parents had better access to health services. The Report also showed that 16% of the PWD did not access health services because of negative attitudes of the providers. The National Sample Survey data (58th round) revealed that only 20% of PWD were ever advised about aids / appliances and that <16% actually acquired aids / appliances [14].

Studies in South India have shown that the mean expenditure on health care of a disabled child is significantly higher compared to what was spent on a non disabled child [15].

Models of Disability Care

Traditionally people viewed disability from a pure 'charity' perspective wherein the disabled person was 'beneficiary' of somebody's sympathy. This was followed by a 'medical' model of care wherein it was presumed that a disabled person had some part of his / her body which was the root cause and this had to be 'fixed' using a medical / surgical intervention. So searching for persons with specific curable 'impairments' and 'correcting' the impairment was the order of the day. However it is now realized that it is not the impairment which causes disability but how the external environment including the 'able-bodied' peers perceive and react to a person who is 'differently-abled'. This leads to stigma, exclusion and isolation which become barriers to the inclusion of a person with a disability in the day-to-day life of the community. This social model brings about a rethinking on how to tackle issues faced by a person with disability. It logically culminated in the rights and inclusive model which realized that every individual has a right to services and a right to lead a life as per his / her own choice. It is no longer a matter of a 'beneficiary' approach but a right to equity and a level playing field for persons with disabilities.

Why a public health approach?

Public health is a multi-faceted discipline which ensures that individuals, families and communities have the best possible quality of life. It looks at the magnitude of a health condition, helps in undertaking a situational analysis which also considers the perspective of the client and not just the health system, the causes of compromised health, risk factors, where there is a higher risk or probability of occurrence of a health condition, the determinants of health (including social determinants), interventions that are likely to succeed at a population level, the cost-effectiveness of the available interventions, utilization and access to services including care and support. Persons with disabilities (PWD) are a high-risk group in a society as they have suffer a higher rate of morbidity and mortality and at the same time have poorer access to health, care and support services. A number of successful programmes are available from many low and middle income countries (LMIC). However many of these programmes cater to the needs of a small segment of population with PWD. If these programmes are to be scaled up to cover larger population groups or whole countries, it is necessary to harness evidence on each component of the programme. The two main disciplines of public health are epidemiology and management principles. Planning any programme needs a thorough situational analysis, determination of specific objectives, implementation of a scientifically valid set of activities, regular monitoring of the progress of the programme and the evolution of the programme as well as evaluation at periodic intervals. These components of the planning process are ably supported by application of epidemiologic principles. Public health therefore has the potential to provide the necessary fillip to interventions targeting PWD as well as the environment in which PWD habitually reside. Scientifically sound evidence is also required for advocacy with the political system as well as programme planners and policy makers. These aspects have helped countries and communities to successfully mitigate major health problems over the past century. This does not mean that disability should be viewed only from a health perspective but that the principles of public health could be effectively used to address all aspects of disability. Some institutions in countries like India have recently provided institutional support to utilize public health skills to address concerns of PWD.

South Asia Centre for Disability Inclusive Development & Research (SACDIR)

The Public Health Foundation of India (PHFI) is a response to redress the limited institutional capacity in India for strengthening training, research and policy development in the area of Public Health. It is a public-private partnership that has collaboratively evolved through consultation with multiple stake holders, both national and international.

SACDIR has been set up as a Centre of Excellence by PHFI in collaboration with the London School of Hygiene and Tropical Medicine, UK. The mission for the Centre is 'Inclusive Millennium: Evidence for Empowering Persons with Disabilities'.

The Objectives of SACDIR are:

- Develop the evidence base for understanding the magnitude of disabilities within the South Asia context;
- Train and reorient health care personnel to concerns of persons with disabilities;
- Organize modules on application of the International Classification of Functioning (ICF) recommended by WHO;
- Run short course training modules on disabling conditions & inclusive development;
- Develop a Masters Course in Disability Management & Research
- Conduct high quality need-based epidemiological, operations, sociological and outcomes-based research to improve the quality of life of persons with disabilities;
- Evaluation of existing programs for persons with disabilities in India and other South Asian countries;
- Develop innovative modalities for identifying persons with disabilities and providing appropriate care;
- Advocate at appropriate congregations and forum for disability inclusive development;
- Assist and influence policy development initiatives to foster disability inclusive development in the country and the region.

There is a need for more public health institutions and professionals to engage with PWD and develop need-based programmes which are cost-effective in reaching a significant proportion of the disabled persons and to bring about an attitudinal change in communities so that inclusiveness could be fostered as a principle in guiding policy and action in LMIC.

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Original Article

Retinopathy of Prematurity - The new challenge

Dr. Parag K. Shah, Dr. Saurabh Arora, Dr. V. Narendran, Dr. N. Kalpana

Introduction

Retinopathy of Prematurity (ROP) is a fibrovascular proliferative disorder which affects the developing peripheral retinal vasculature of premature infants. It is a preventable cause of blindness in children. ROP was a major cause of blindness in children in Europe and North America during the late 1940s and 1950s after the introduction of intensive neonatal care, with unmonitored supplemental oxygen being the major risk factor[1-4]. This was called the "first epidemic". Since then, increased awareness of the importance of monitoring blood oxygen levels has resulted in a lower incidence of potentially blinding ROP in larger, more mature babies, but extremely low birth weight (BW) babies (i.e. less than 1000 g at birth) are still at risk. This has been termed the "second epidemic"[5]. Developing countries are now facing what is termed the "third epidemic" which is a mixture of the first two epidemics[6]. This "third epidemic" is characterized by severe ROP in babies who have a much wider range of BWs and gestational ages (GA) than is now the case in industrialized countries and also in extremely premature babies.

The initial signs of ROP are detectable by a few weeks after birth, and the condition progresses rapidly thereafter. This means that screening has to be timely, and there is only a very narrow window of opportunity for treating those babies where the condition progresses to high risk prethreshold or threshold stages. If not treated the condition progresses rapidly to Stage 4 or 5 in approximately 50% of babies. The visual prognosis for babies with Stage 5 disease (total retinal detachment) is very poor, even in babies having complex vitreoretinal detachment surgery.

The primary goal of screening is to detect all babies with treatable disease in time for treatment to be effective.

Screening

When to screen? When should a pediatrician refer to ophthalmologist for ROP screening?

Ideally babies are to be screened at 31 weeks post conceptional age (gestational age + post natal age) or 4 weeks after birth, whichever is later.[7] However, an easier way to remember is that first retinal examination should be done by first month of life.

Whom to screen?

Screening all premature babies will be a waste of time as we know that all do not develop ROP. One cannot follow the western screening guidelines in developing countries as bigger babies getting severe ROP are still seen here[8-9]. So, for the developing countries all babies having gestational age (GA) 34 weeks or having birth weight (BW) 1850 gms should be screened for ROP[9]. However it is best to have individualized criteria for each neonatal intensive care unit (NICU), as even between NICU's from the same city the age of presentation maybe different. Babies, who are on unmonitored 100% oxygen, even for a few days, should be screened irrespective of BW or GA as they are at the highest risk of getting oxygen induced ROP[10].

How to screen?

Screening is done by a retina specialist or a pediatric ophthalmologist. It is done with the help of indirect ophthalmoscope and 28 D lens. Recently, a new digital camera, Retcam is available for screening but is a very expensive tool[11]. It is the duty of the pediatrician to call these trained ophthalmologists to their NICU or they should refer the children to them at end of first month. ROP screening should be included as a part of neonatal care.

Classification of ROP

An international classification of retinopathy of prematurity was published in 1984 and updated in 1987 and 2005.[12-14] The components of classification is as follows.

1. Location of disease: Each eye is divided in three zones to define the exact location.
   a. Zone I - circle, the radius of which extends from the disc to twice the distance from the disc to the fovea (center of macula).
   b. Zone II – extends from the edge of zone I peripherally to ora serrata nasal and equivalent area near the temporal equator
   c. Zone III – residual crescent of retina anterior to zone II temporally.
2. Extent of disease: In the form of number of clock hours involvement (Figure 1).
3. Staging of disease: Is done according to degree of vascular changes. Each stage is defined by its location in zone & extent in clock hours for documentation.
   a. Normal fundus (Figure 2)
   b. Stage 1 – Demarcation Line: This line is a thin but definite structure that separates the avascular retina anteriorly from the vascularized retina posteriorly. The demarcation line is relatively flat, white, and lies within the plane of the retina (Figure 3).
   c. Stage 2 – Ridge: The ridge is the hallmark of stage 2 ROP. It arises in the region of the demarcation line, has height and width, and extends above the plane of the retina (Figure 4).
   d. Stage 3 – Extraretinal fibrovascular proliferation

Dr. Parag K. Shah

(EPF): EPF or neovascularization extends from the ridge into the vitreous. It is continuous with the posterior aspect of the ridge (Figure 5). It is further subdivided into mild, moderate or severe depending on the extent of EPF infiltrating the vitreous.

e. Stage 4 – Partial retinal detachment: Stage 4 is divided into partial retinal detachment not involving fovea, stage 4A (figure 6) and involving fovea, stage 4B (Figure 7). Visual prognosis of stage 4B is poorer than 4A.

f. Stage 5: Total retinal detachment: These retinal detachments are generally tractional and may occasionally be exudative. Visual prognosis is the worst for stage 5 ROP (Figure 8).

g. Aggressive posterior ROP (AP-ROP): An uncommon, rapidly progressing, severe form of ROP is designated AP-ROP. If untreated, it usually progresses to stage 5 ROP. The characteristic features of AP-ROP are its posterior location, prominence of plus disease, and the ill-defined nature of the retinopathy (Figure 9). It is observed most commonly in zone I, but may also occur in posterior zone II.

4. Plus disease: It is an additional sign indicating the severity of active ROP. This includes increased venous dilatation and arteriolar tortuosity of the posterior retinal vessels (Figure 10) and may later increase in severity to include iris vascular engorgement, poor pupillary dilatation (rigid pupil), and vitreous haze.

5. Pre-plus disease: It is defined as vascular abnormalities of the posterior pole that is insufficient for the diagnosis of plus disease but d emonstrates more arterial tortuosity and more venous dilatation than normal (Figure 11).

Management of ROP

When to treat?

1. Threshold disease: It was defined by the CRYO-ROP study,[15] as Stage 3 in zone I or II involving > 5 contiguous or 8 cumulative clock hours with plus disease. This was the previous “cut off” stage for treatment.

2. Prethreshold Disease: Early Treatment ROP (ETROP) study[16] has revised the treatment guidelines. This study proved that earlier treatment (Pre Threshold stage) has a better outcome. They divide prethreshold ROP into

a. High Risk Prethreshold or Type 1 ROP: This is the new “cut off” for treatment. It should be treated immediately. It is defined as

i. Zone 1 any stage with plus disease or
ii. Zone 1 stage 3 without plus disease or
iii. Zone 2 stage 2 or 3 with plus disease.

b. Low Risk Prethreshold Disease or Type 2 ROP: These eyes should be considered for treatment only if they progress to type 1 or threshold ROP. It is defined as

i. Zone 1 stage 1 or 2 without plus disease or
ii. Zone 2 stage 3 without plus disease.

How to treat?

Principle is ablation of the ischemic peripheral retina stops release of angiogenic factors. Two options are available:

1. Cryotherapy: Involves placing a very cool probe on the sclera and freezing, until an ice ball is formed on the retina inside. Multiple applications are made to treat entire avascular retina anterior to the ridge (Figure 12). However cryotherapy has a lot of disadvantages. It requires general anesthesia, has more local complications like severe lid edema and for zone I cases, the cryo probe cannot reach posteriorly because of the restriction caused by the conjunctival fornix.

2. Laser Photocoagulation: It is a practical alternative after the advent of indirect laser delivery system. Direct treatment of retina from inside, so less local inflammation. The main advantages are that it can be performed under topical anesthesia, systemic and local complications are much less compared to cryotherapy, and it can be done as out patient procedure and posterior retina in zone I cases can be treated easily (Figure 13).

Laser or cryotherapy can only be done till stage 3 ROP. Management of stages 4 and 5 is surgical and final outcome is very poor for most of these stages.

3. Surgical treatment: Surgery is advocated if laser or cryotherapy is unsuccessful in preventing progression to stage 4 or 5. Surgical options available are

a. Scleral buckling
b. Lens sparing vitrectomy
c. Lensectomy + vitrectomy
d. Open sky vitrectomy

In our series[17], anatomical success was 90% (9/10) for stage 4A, 44.4% (4/9) for stage 4B, and 14.3% (2/14) for stage 5 ROP. Visual outcome was worst for stage 4B and 5. ROP management doesn't end with laser or surgery. Once treated, lifelong followup (yearly) is mandatory. All other premature infants irrespective of having ROP yearly followup till the age of 5 years is advisable to rule out refractive errors (most common), squint and amblyopia (lazy eye).

4. Role of anti vascular endothelial growth factor (VEGF) injections in ROP:

It is very controversial as VEGF is also needed for normal vascularization of the retina to be completed. Thus anti VEGF injections may stop growth of not only abnormal new vessels but also of the normal ones. Systemic absorption of these drugs may cause vascular development delay in other developing organs. Thus currently anti-VEGF injections are used in ROP only when the standard treatment (which is laser) fails and the disease progresses. It is not recommended as the first line of treatment

Conclusion

The epidemic of ROP we are facing in developing countries is a repeat of the first epidemic, as shown in our previous study[18] and also in other studies from Turkey[8-9]. There are several possible reasons for this. First, preterm birth is increasing in some sectors of the population due to in vitro fertilization. Second, neonatal provision is expanding and survival rates among premature babies are improving. Third, supplemental oxygen is often given unnecessarily to infants who are stable even when they would be adequately oxygenated without it. Fourth, not all babies receiving supplemental oxygen are being adequately monitored, and for those who are being monitored, target saturations may be set at 100%. Lastly, screening and treatment programs are not in place in all neonatal units in many cities.

A two pronged approach is needed to tackle the emerging epidemic of blindness due to ROP in developing countries, and this will require close collaboration between pediatricians and ophthalmologists. Pediatricians can play a pivotal role in preventing ROP by reducing supplemental oxygen except to those babies who really need it. The use of 100% oxygen saturation should be avoided. All babies receiving oxygen should also be closely and continuously monitored, avoiding fluctuations which are known to be harmful. There is also an increasing body of evidence that lower target oxygen saturations are protective, even in very low birth weight infants, and monitoring should aim to keep oxygen levels between 83-93% and not higher.[19-20] Ophthalmologists can play a key role in preventing blindness from ROP, by undertaking regular, ongoing screening programmes to detect and treat type 1 prethreshold ROP. They need to develop evidence based screening criteria relevant to the population of babies they are responsible for to ensure that all babies at risk of ROP needing treatment are examined. In many settings, this will mean that larger, more mature babies should also be examined than indicated by screening guidelines developed for use in the UK, USA, Canada etc.

We feel that it may be impossible to have uniform national screening guidelines in a developing country till oxygenation policies are uniform. Until these criteria are well established, it is advisable to err on the side of caution and use wide screening criteria and not to rely on criteria published by developed countries. These criteria must be evaluated and revised periodically based on the evolving knowledge and advancements in neonatal care in the particular country.

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Legends:

Figure 1: Schematic diagram of right eye (RE) and left eye (LE) showing zones to describe location of disease and clock hours to describe extent of ROP.

Figure 2: Normal fundus of RE.

Figure 3: Fundus picture of RE showing stage 1 demarcation line (black arrows).

Figure 4: Fundus picture of LE showing stage 2 elevated ridge (black arrows).

Figure 5: Fundus picture of LE showing stage 3, ridge (black arrows) and extra retinal fibrovascular proliferation (white arrows).

Figure 6: Fundus picture of RE showing stage 4A partial retinal detachment (black arrows) nasal to optic disc and vitreous hemorrhage (white arrow) over macula.

Figure 7: Fundus picture of LE showing stage 4B partial retinal detachment involving the macula (black arrows).

Figure 8: Fundus picture of RE showing total retinal detachment.

Figure 9: Fundus picture of RE showing AP-ROP.

Figure 10: Fundus picture of RE showing dilatation and tortuosity of posterior pole vessels signifying plus disease.

Figure 11: Fundus picture of RE showing pre plus disease

Figure 12: Schematic diagram of fundus showing multiple white cryo burns (black arrows) in avascular retina anterior to ridge (white arrow).

Figure 13: Fundus picture of RE showing laser scars (black arrows).

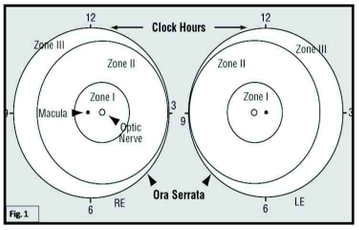
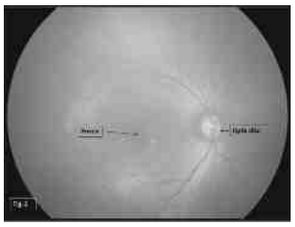


Fig 1



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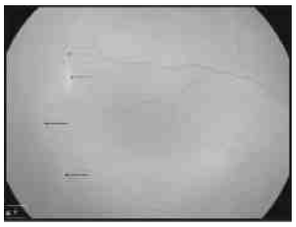

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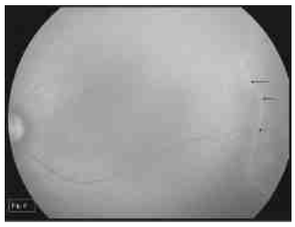

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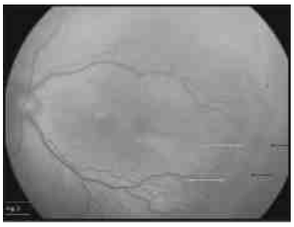
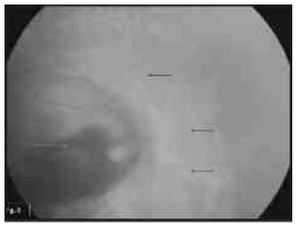


Fig 5



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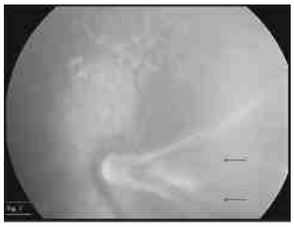


Fig 7

Fig 8

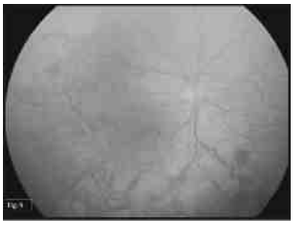


Fig 9

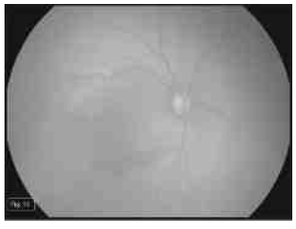


Fig 10

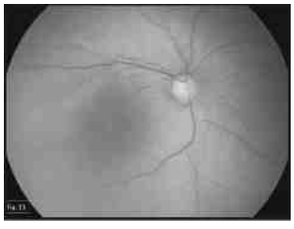


Fig 11

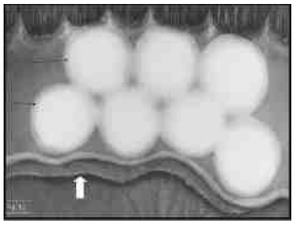


Fig 12

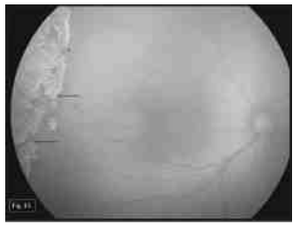


Fig 13

Original Article

Approach to a Patient With Glaucoma

Dr. Amandeep Kaur, Dr. Swati Zawar

Definition: Glaucoma is a multifactorial optic neuropathy, characterized by classic optic disc cupping and /or visual field defects with raised intraocular pressure being the only known modifiable factor.

Glaucoma is one of the leading causes of blindness in most parts of the world despite new medical and surgical strategies to control intraocular pressure (IOP), blindness caused by glaucoma continues to increase, and glaucoma remains the third most common cause of blindness (12%) in the India.

Classification: Broadly glaucoma can be classified into:Open angle glaucoma (OAG) Angle closure glaucoma (ACG) glaucoma Combined mechanism glaucomaDevelopmental glaucoma

Each of these classes has subclasses depending on the mechanism of IOP rise and glaucomatous damage.

Prevalence: According to a recent study, there are about 11.2 million people above 40 years of age in India with glaucoma; out of them 6.48 million have POAG and 2.54 million PACG.

This underlines the importance of evaluating our patients thoroughly; especially those who have not yet developed advanced and obvious disease.

History Taking and Risk Factors: A thorough history taking is must in all glaucoma patients and should include:

Age : The incidence of glaucoma increases with age. The risk of POAG in those above the age of 70 years is five times that of the 40-49 age group. Increasing age is a risk factor for PACG too,with the highest prevalence in 6^{th}-7^{th} decade.

Refractive error : Incidence of POAG is higher in myopes (esp. >8 D) .Recent studies show that hyperopia does not increase the risk for PACG, though the incidence is higher in small eyes with smaller anterior segment.

Heredity : POAG has 22 % incidence in first degree relatives . PACG is essentially sporadic.

Diabetes : Higher incidence of POAG is found in diabetics.

Hypertension : B.P lowering drugs esp. when taken at bed time increase nocturnal hypotension and contribute to optic disc hypo perfusion and may have a role in POAG.

Asthma : History of steroid intake may play role in steroid responders and non selective beta blockers may exacerbate asthma.

Urogenital : History of kidney stones limits intake of carbonic anhydrase inhibitors.

Allergy : Steroid intake / application may be causative in steroid responders. Drug allergy to sulphonamides must be ruled out esp before prescribing carbonic anhydrase inhibitors.

Ocular history : History of intermittent pain, blurring of vision, redness, coloured haloes is suggestive of angle closure attacks. History of any previous long term steroid application, trauma, inflammation, surgery, may be causative factors for secondary glaucoma.

Ocular Evaluation: Detailed slit lamp evaluation of anterior segment should be done .

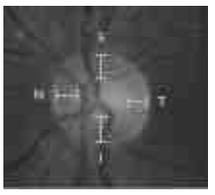
CONJUNCTIVA	CORNEA	ANTERIOR CHAMBER	IRIS	PUPIL	LENS
Pre-existing bleb	Pigment on Endothelium	Depth	Atrophic Patches	Mid dilated non reactive	Intumesence
Episcleral vessels	Keratic precipitates	Cells/ flare	Heterochromia	Ectropion uveae	Glaucomflecken
Ocular surface	Haab's straie		PXF material/ transillumination defects	Posterior Synechiae	PXF Material
	Dryness		New vessels		Pigment on ant. capsule
			Iris nodules		Phacodonesis

Disc Evaluation: All patients should have a dilated fundus examination for stereoscopic disc evaluation with contact / non contact fundus lenses.

Optic disc is vertically oval and cup is horizontally oval and as the fovea is lower than the optic disc, width of neuroretinal rim is as follows :

Inferior>Superior>Nasal>Temporal This ISNT rule is followed in majority of non glaucomatous discs. The deviation from this rule is suggestive of glaucomatous change.

Disc should be evaluated for :

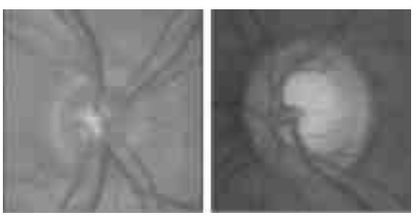


Size: Larger discs have larger cups, more number of axons larger and more no of lamellar pores , whereas smaller discs have smaller cups. A larger cup in a small disc should raise suspicion.

Neuroretinal rim : The width of tissue between the inner margin of cup (identified by bending of vessels) and the margin of disc(identified by defining the sclera ring) defines the neuroretinal rim .

To detect glaucomatous changes attention should be paid to :

- Deviation from ISNT rule.
- Asymmetry in the thickness of NRR between two eyes in absence of disc asymmetry.
- The inner margin of NRR for any localised thinning / notch.



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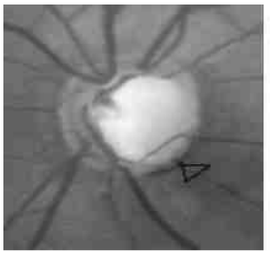
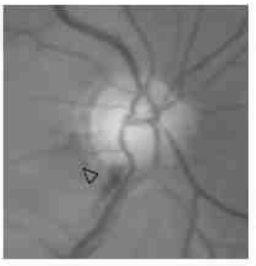
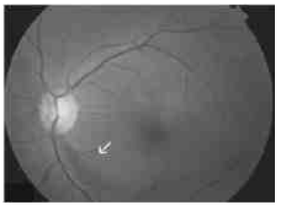
Peripapillary atrophy : The inner beta zone is identified by loss of retinal pigment epithelium and chorio capillaries and the visibility of larger choiroidal vessels and is more common in glaucomatous eyes. The outer alpha zone is identified by hypo and hyper pigmented area and is found in both glaucomatous and non glaucomatous eyes.



In glaucomatous eyes width of beta zone is inversely proportional to NRR width and its enlargement follows glaucoma progression.



Splinter haemorrhage : Any disc with haemorrhage within 1 disc diameter of optic disc should be carefully examined for glaucomatous changes. Splinter haemorrhages can be seen in normal eyes too but are present more commonly in glaucomatous eyes and their presence is suggestive of ongoing glaucoma damage.



Cup disc ratio : Larger discs have larger cups and smaller discs have smaller cups. A difference of more than 0.2 CDR between the two eyes in absence of disc size asymmetry is suggestive of glaucoma.

Retinal nerve fibre layer : RFNL can be identified as fine bright striations starting from the margins of the disc and identifiable up to 2-3 disc diameter area around the disc esp. in polar areas,
and more clearly visible in young adults with clear media .

Glaucomatous change in RFNL can be appreciated as :

- Wedge defects
- Diffuse loss (sharp visibility of small peripapillary blood vessels)

All these disc changes should be documented by taking disc photographs(preferably stereoscopic), which sometimes highlight subtle changes like resorbing haemorrage and rnfl loss, also they are helpful to follow up progression.

Intraocular Pressure (iop): IOP is currently the only known modifiable factor in glaucoma, and often-raised IOP the first finding in many patients.

Baseline IOP is recorded for each patient after measuring at least 3 readings at 3 different visits and a target IOP is determined after assessing the severity of damage, life expectancy, family history of glaucomatous vision loss and baseline IOP.

IOP is measured at every follow up visit and treatment is modified to achieve the target IOP. So there is a need for an accurate and reproducible method for IOP measurement, as all our effort of stopping the progression of damage, is based on this single modifiable factor.

Commonly used methods for measuring IOP are as follows:

Applanation tonometer : Goldmann applanation tonometer is the gold standard against which all other tonometers are compared, and is the most accurate method for IOP measurement. GAT is dependent on corneal thickness and so, pachymetry must be done and the corrective factor applied to give corrected IOP readings.

Tonopen is reliable for scarred and irregular corneas, but for normal corneas it's comparable with GAT in normal range of IOP only, and inaccurate for higher range.

Non contact tonometer : As for tonopen ,it's accurate for normal range of IOP but not for higher range of IOP.

Pascal dynamic contour tonometer : Its is an accurate method of IOP measurement in regular surfaced cornea and is independent of corneal properties, so is free from corneal thickness factor.

Shiotz tonometer: Can be used only for screening purpose, and gives only a
probable range of IOP. It's far from accurate and should not be used as a tool for glaucoma assessment.

24 Hr. Diurnal IOP variation test is time consuming and unfeasible for both the doctor and the patient in today's scenario, instead modified diurnal variation test in which 2 hourly pressure measurements are done during office hrs can be applied in selected cases.

Gonioscopy: The omission of goinioscopy is a common cause of misdiagnosis in glaucoma. It should be done for all suspects and glaucoma patients, initially and repeated periodically in angle closure cases.

The choice of gonioscopy lenses can be any of

- 1,2,3 mirror Goldmann lenses,
- Zeiss, posner or sussman 4 mirror goniolenses.
- Koeppes direct lenses for angle evaluation in paediatric patients

The four mirror lenses in addition allow compression gonioscopy and allow the differentiation of appositional from synechial closure.

Gonioscopy should be done in a dark room, using small and dim slit beam, and high magnification.

Gonioscopy evaluation should include the study of:· Configuration of iris (flat / convex / concave).

- Insertion of iris(at / in front of / behind sclera spur).
- Identification of angle structures and angle grading.
- Presence of PAS, PXF material, precipitates, excessive pigment, new vessels, blood in schlems canal.

Identification of angle structures: From the root of iris towards cornea the angle structures are as follows:

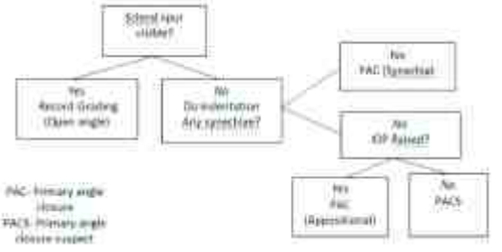
Ciliary body band (CBB): Seen as deeply pigmented band, posterior to sclera spur, to which the iris inserts. It's

easily visible in aphakic and high myopic eyes.

Sclera spur (SS): Seen as gray white band of varying width, posterior to the trabecular meshwork which may be obscured partly by iris processes. It's an important landmark in identifying angle structures.

Trabecular meshwork(TM): It is a finely granular structure that extends from sclera spur to schwalbe's line, with posterior pigmented and anterior non pigmented parts. Even in the absence of pigments it is not as white as sclera background and is translucent white, as opposed to solid white sclera. When the TM is non pigmented CBB is the only pigmented structure in angle.

Schwalbe's line(SL) : This posterior most termination of descemet's membrane can be seen as a white / translucent ledge in the anterior most part of angle where the corneal parallelepiped of slit lamp comes together. It's an important landmark for identifying angle structures. When pigmented, it can be confused for trabecular meshwork.



Grading of Angles:

Grade	0	I	II	III	IV
Shaffer's	Closed	10°	20°	30°	40°
Modified Shaffer's	Shwalbe's line not visible	Shwalbe's line visible	Ant. TM visible	Sclera spur visible	Ciliary body band visible

Perimetry: Automated perimetry is the current gold standard for diagnosis and management of glaucoma.

Visual field assessment should be done in all suspect cases to find out the extent and depth of field defects caused by glaucoma and to follow up progression.

Imaging Devices: Modern technologies to quantitatively and objectively measure glaucomatous structural change in optic nerve head and retina:

Optical coherence tomogram (OCT)

Heidelberg retina tomography (HRT)

Scanning laser polarimetry (GDx VCC, GDx ECC)

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Original Article

How uni-ocular diminution of vision affects binocular single vision in Indian adults.

Dr. Sanjiv Singh, Dr.Parikshit Gogate

Introduction

Humans have a remarkable visual sense that allows perception of depth. This depends on the difference in points of view of two eyes and gives humans what has been termed 3D vision, binocular vision and stereopsis. Uniocular vision loss is not considerd significant in public health realm, as the definition of blindness and visual impairment is based on vision of the better eye. [1] But decrease in vision in one eye has implications for binocular single vision. [2,3,4] Various tasks of daily living like threading a needle and sewing, reaching out to shake someone's hand, pouring into a container, catching or hitting a ball, driving and parking a car, stepping off a curb or climbing steps all require stereopsis. Certain occupations like cricketer, racquet sportsmen, waitress, driver, architect, surgeon and dentist depend heavily on stereo vision. Uni-ocular vision loss thus affects a person's activities of daily living. However there are few studies which demonstrate how much does less vision in one eye affect stereopsis. [5] The present study determined the variation of stereo acuity with binocular visual acuity when one eye had poor vision.

Materials and methods

The participants were briefed about the purpose and nature of the study and a written consent taken. The examination was non-invasive. Each participant was emmetropic with uncorrected 6/6 visual acuity in each eye with no history of using glasses. None of the subjects had history of ocular disease or systemic disease likely to affect visual functions. Each participant underwent a detailed ophthalmic evaluation including visual acuity for distance (6 meter) and near (0.33m) without correction; dry and cycloplegic refraction to rule out any significant refractive error, dilated fundus examination and orthoptic evaluation to rule out any misalignment for near and distance. Subject who had phoria >6 prism diopter or any tropia were excluded. All subjects had all three grades of binocular vision as tested on Randot charts. Participants with any ocular pathology, history of ocular diseases or any systemic diseases likely to affect visual functions were excluded from the study.

Stereoacuity testing was done using Randot test circles which provided a range of 28-526 seconds of arc. The response was considered positive if the subject could identify the circle depth cues with respect of other circles. Testing was done under standard conditions in all subjects. Stereoacuity was measured at 30 cm from root of the nose. All subjects were tested in similar light conditions. Lighting was provided by ceiling fluorescent light (40 W) and an incandescent lamp (100 W) over the subject's right shoulder and at a distance of one meter from the target. All subjects were made to understand the test completely and none of them had difficulty with test compliance. All stereo acuity testing was done by same observer.

The subjects was asked to read the distance visual acuity and then randomly one eye was occluded to keep that eye as normal eye and a +6.0D lens was added to the best correction for the distance to blur the vision. The power of additional convex lens was reduced in 0.25 D steps and lenses required for each standard line of distance visual acuity were noted.

One eye selected randomly was corrected 6/60 for the distance, while the other eye remain 6/6. The Randot response was noted at each combination of binocular visual acuity. Following this the visual acuity at 6 m in the randomly selected eye was improved one line at a time while other eye visual acuity remained 6/6 for the distance at each step. Randot scores were recorded for each combination of binocular visual acuity from 6/60 to 6/6 so that a total of 7 Randot scores were recorded for each subject.

Results

Twenty healthy adult subjects with an average age of 25 years (range 15 - 35years) were studied. Twelve (60%) subjects were males. The 140 unique Randot scores thus obtained (7 in each of the subjects) were used to calculate mean and standard deviation of Randot stereo acuity at each binocular combination of visual acuity as shown in Table 1. The mean stereo-acuity is within 62 seconds of arc even when one eye's vision is 6/18 and the other eye has 6/6 visual acuity. The stereopsis declines when the vision of the poorer eyes falls below 6/24 and declines precipitously when visual acuity of the poorer eye is 6/60 or less (Figure 1). The standard deviation of the stereopsis is also larger; the lesser is the vision of the poorer eye (Figure 2). So at visual acuity levels of 6/36 and less, we cannot be sure about the gross stereopsis that a person has.

Discussion

This study demonstrated that stereo acuity improves with improvement of binocular acuity in Indian patients as also reported by earlier researcher.[5] However, this study demonstrates a linear relationship between binocular acuity $\geq$6/60 and stereo acuity. This is because if one eye is having poor visual acuity, it cannot coordinate effectively with the other eye howsoever good the visual acuity of that eye may be. In fact, the good eye visual acuity increases the image disparity between the two eyes, which is a known hindrance to effective binocular combination. But when worse eye visual acuity improves, the stereo acuity improves.

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If amblyopic and uniocular refractive error patients are treated and if they achieve 6/12 or better visual acuity, then their stereo acuity shall also improve dramatically. So conditions like unilateral refractive error, unilateral cataract, corneal opacity, astigmatism, pterygium and unilateral ocular trauma when treated shall not just improve the vision of that eye, but also improve the person's stereoacuity and allow him/her to function better. Even though their better eye's vision may remain the same, their binocular single vision would have significantly improved.

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Table 1: Randot scores (means and standard deviation) at various Snellen's visual acuity levels, while one eye visual acuity is normal (6/6):

VISUAL ACUITY OF WORSE EYE								
Subject	6/60	6/36	6/24	6/18	6/12	6/9	6/6	STEREO ACUITY
A	526	227	128	66	41	28	28	
B	227	128	66	41	41	28	28	
C	526	227	128	66	41	28	28	
D	526	227	128	66	41	28	28	
E	526	227	128	66	41	28	28	
F	526	227	227	66	41	28	28	
G	227	128	66	41	28	28	28	
H	526	227	128	66	41	28	28	
I	526	227	128	66	41	28	28	
J	227	128	66	41	41	28	28	
K	526	227	128	66	41	28	28	
L	526	227	128	66	41	28	28	
M	526	227	128	66	41	28	28	
N	526	227	128	66	41	28	28	
O	526	227	128	66	41	28	28	
P	526	228	128	66	41	28	28	
Q	526	228	128	66	41	28	28	
R	526	227	128	66	41	28	28	
S	526	227	128	66	41	28	28	
T	526	227	128	66	41	28	28	

Mean of Stereo-acuity	481.2	212.25	123.65	62.25	40.35	28	28
SD of Stereo-acuity	109.54	36.31	33.21	9.16	2.91	0.00	0.00
Lower Confidence Limit	429.89	195.26	108.11	57.96	38.99	28.00	28.00
Upper Confidence Limit	532.42	229.25	139.19	66.54	41.71	28.00	28.00

Figure 1: Graph showing that stereoacuity (SA) is better when Snellen's visual acuity (VA) in both eyes is better.

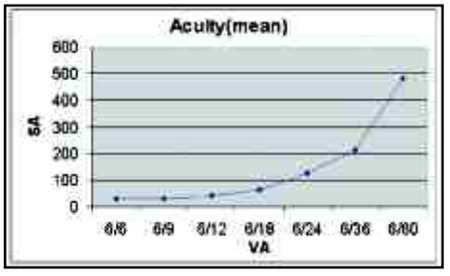
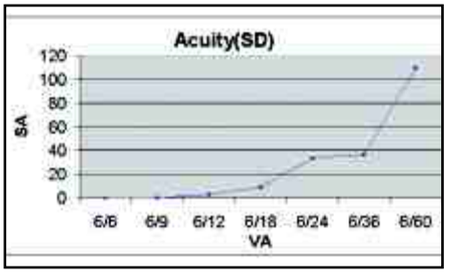


Figure 2: Graph showing standard deviation is (SD) less if binocular Snellen's visual acuity is better, and SD is more when Snellen's visual acuity is poor



Original Article

Pediatric Cataract

Dr. Rupal H. Trivedi, Dr. M. Edward Wilson

Cataract remains one of the most important causes of treatable blindness in children. Overall, it accounts for 3-39% of childhood blindness.[1] Just over 200,000 children are blind from lens disorders, primarily unoperated cataract, but also from dense amblyopia following delayed surgery, surgical complications or from associated ocular abnormalities.[1] In industrial countries, approximately 30 out of every 100,000 infants will be diagnosed with cataract by their first birthday, with a further 10 children being diagnosed by the age of 15 years.[2] The rate of cataract development is likely to be higher in developing countries as a result of consanguinity and congenital rubella syndrome.

Etiology: For many years, it has been commonly believed that approximately one-third of childhood cataracts are inherited, one-third are associated with other diseases or syndromes, and the remaining one third are idiopathic. The causes of pediatric cataract include intrauterine infections, metabolic disorders, and genetically transmitted syndromes. Johar and colleagues[3] reported that 86% of unilateral and 68% of bilateral cataract had no discernible cause. Out of 172 children, 88.4% had non-traumatic cataract and 11.6% had traumatic cataract. In cases with non-traumatic cataract, 73.0% were undetermined, 15.1% were secondary (uveitis, persistent hyperplastic primary vitreous, aniridia, Down, Marfan and Lowe syndromes), 7.2% were hereditary, and 4.6% were due to congenital rubella syndrome. Inherited isolated (non syndromic) cataract represents a significant proportion of congenital cataract cases and many causative genetic mutations have been identified. Inherited cataract is known to be clinically and genetically heterogeneous. Genetic mutation is likely to be the common cause, particularly for bilateral cataract, but the proportion of cases with a genetic basis is still unclear. On the basis of family history, Rahi and Dezateux[4] found that 27% of children with bilateral isolated congenital cataract had a genetic basis compared with 2% of unilateral cases. It is likely that some children with congenital cataracts who don't have a family history of cataract still have a genetic cause; they may represent new autosomal dominant mutations or have autosomal recessive or X-linked forms of cataract. All at-risk family members should be subjected to slit lamp examinations as some affected individuals may have minor lens opacities, which do not cause a significant reduction in visual acuity. Incomplete penetration is uncommon, as is unilateral cataract in affected families. Cataract may be inherited as either autosomal dominant, autosomal recessive, or X-linked recessive traits. Twelve loci and 15 specific genes associated with inherited isolated cataract have been identified to date; it is likely that there are other genes still to be discovered.[5]

Role of the Pediatrician in Early Detection of Childhood Cataract: In children, early detection of cataract is important to avoid lifelong visual impairment. Pediatricians play a key role in early detection of infantile cataract. Red reflex testing is generally used for early detection of cataract. The American Academy of Pediatrics recommends red reflex assessment as a component of the eye evaluation in the neonatal period and during all subsequent routine health supervision visits.[6] The test should be performed in a darkened room to maximize pupil dilation. The direct ophthalmoscope should be focused on each pupil individually approximately 12 to 18 inches away from the eye, and then both eyes are viewed simultaneously at a distance of approximately 3 feet. Eyes which show diminished red reflex should be referred to an ophthalmologist. In the case of infants, the referral should be arranged within a reasonably short time. Direct communication with the ophthalmologist or confirmation of an appointment is optimal since families may not understand the importance of timely interventions or be able to navigate the appointment systems. Infants or children in high-risk categories, including relatives of patients with cataract, should not only have red reflex testing performed in the nursery but also be referred to an ophthalmologist who is experienced in carrying out a complete eye examination regardless of the findings of the red reflex testing by the pediatrician.

History, Examination and Investigation: Patients may present with parents / caregivers complaining of a white spot in the pupil, visual inattentiveness, nystagmus, strabismus, asymmetry of one eye relative to the other (e.g. microphthalmos), photophobia, ocular injury, or simply because of a referral from other physicians who have identified possible lens opacity. At times, the evaluation is scheduled because of a family history of childhood cataract or because the child has one of a growing number of systemic conditions or syndromes that can be associated with cataract. Specific information should be gathered on evidence of maternal infection (especially the TORCH

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infections), rash or febrile illness during pregnancy (may be suggestive of intrauterine infection), any other prenatal and perinatal history that may be pertinent (e.g., alcohol, tobacco, drug use, ionizing radiation during pregnancy), history of ocular trauma (unless cataract appears to be purely non-traumatic), age at onset of visual symptoms, ocular status on previous eye examinations (can be helpful in assessing visual prognosis after treatment), history of corticosteroid therapy (especially in posterior subcapsular cataract). Posing a series of simple questions can help in determining the surgical need, the timing or urgency of surgery, and the visual prognosis after cataract removal (e.g. Does your child appears to see well? Do your child's eyes focus straight or do they seem to cross or drift or seem lazy? How long have you noticed a change in your child's visual function?). In cases of congenital cataract, abnormalities such as poor visual behavior, nystagmus or strabismus (in unilateral cases) are a late sign, and deprivation amblyopia is already present.

Developmental pediatricians and clinical geneticists play an important role in selective investigation based on characteristics of the child. As compared to unilateral cataract, laboratory investigations of bilateral cases are more rewarding. Based on past history of the eye and observations during the examination, customized laboratory investigations can be advised. Since cataract can be the presenting sign of diabetes, children with acquired cataract of unknown etiology should be questioned about symptoms of diabetes and evaluation for hyperglycemia should be performed. Children with Lowe syndrome have hypotonia, mental retardation, aminoaciduria and an abnormal facial appearance with frontal bossing and chubby cheeks. If Lowe syndrome is suspected, the urine should be screened for amino acids. If there is a history of maternal rash, fever, flu-like symptoms, or neonatal physical signs of intrauterine infection, then acute and convalescent TORCH titers should be obtained.

Cataract Surgery: The aim of pediatric cataract surgery is to provide and maintain a clear visual axis and a focused retinal image. Cataract surgery in children is but one-step in the long road to visual rehabilitation, not the end of the journey. Achieving better visual outcome is a team approach involving the patient, the ophthalmologist, parents and other caregivers, while considering visual, economical, psychological, and social issues. It is not just the surgical procedure that determines a successful visual outcome; it also depends on the surgeon's ability to maintain adequate aphakic correction and follow through with amblyopia therapy. The following are indications for cataract surgery: cataract obstructing the examiner's view during fundus examination in non-dilated pupils or a blackened retinoscopic reflex preventing refraction of the eye. Deciding when to remove a partial cataract can be difficult and challenging. The loss of accommodation after cataract removal may negatively affect visual functioning far more than the actual presence of partial cataract did.

Deciding on the appropriate time at which to perform surgery is very critical during early infancy. In the case of unilateral dense cataract diagnosed at birth, it is advisable to wait until the patient is 4-6 weeks of age so as to decrease the possibility of occurrence of anesthesia-related complications and facilitate the surgical procedure. Waiting beyond this time, however, adversely affects the visual outcome. In the case of a bilateral cataract diagnosed at birth, a good visual outcome can be achieved if the child is operated before 10 weeks of age. Surgery in the first eye can be performed when the infant is 4-6 weeks of age; surgery in the second eye can be performed after another 1-2 weeks. It is important to minimize the time interval between the surgeries in the two eyes. For older children, the timing of the surgery is not as critical as it is in the case of infants. In children beyond the amblyopic age, surgery can often be decided based on convenience and other logistic issues.

Pediatric cataract surgery remains a complex and challenging proposition. Pediatric cataract-IOL surgery is quite different from cataract surgery in elderly patients. A propensity for increased postoperative inflammation and capsular opacification, a refractive state that is constantly changing due to the growth of the eye, difficulty in documenting anatomic and refractive changes due to poor compliance, and a tendency to develop amblyopia are among the factors that make pediatric cataract surgery different from that in adults. In addition, the lack of a hard nucleus, vastly reduced scleral and corneal rigidity, and enhanced posterior vitreous pressure in pediatric eyes demand a surgical approach that differs in many ways from the adult procedure. Ocular growth makes selection of an intraocular lens (IOL) power difficult. Normal childhood behavior can make compliance with postoperative instructions difficult, and postoperative eye examinations are also often challenging. The long expected life span after surgery for children also deserves consideration when surgical decisions are made. These special patients are uniquely challenging. There is general consensus that IOL implantation is appropriate for most older children undergoing cataract surgery.[7-8] In contrast, the advisability of IOL implantation during the first year of life is still questionable. Intraocular lens implantation in children has the benefit of reducing dependency on

external optical devices (aphakic glasses and contact lenses) and providing at least partial optical correction. These are important advantages in the visual rehabilitation of amblyopia-prone eyes. However, concerns about primary IOL implantation are the technical difficulties involved in implanting an IOL in pediatric eyes, selecting an appropriate IOL power, and the risk of visual axis opacification (VAO) after implantation. The rate of VAO is higher in pseudophakic infantile eyes as compared with aphakic infantile eyes. On the other hand, although it is possible for an eye with a unilateral infantile cataract to achieve good visual outcome following contact lens correction, it has continued to be the exception rather than the rule. Secondary IOL implantation is far more common in children who have undergone early cataract surgery and who are contact lens or aphakic spectacle wearers.

IOL Power Selection: It is well known that the majority of the eye's axial growth occurs during the first two years of life. This rapid eye growth makes selection of an IOL power for an infant difficult. Selecting the best IOL power to implant in a growing child presents unique challenges. While Gordon and Donzis[9] have documented the axial growth pattern of normal eyes in children, the axial growth of cataractous eyes is different. In the eyes of normal phakic children, there are hardly any changes in refraction (0.9 diopters from birth through adulthood on an average) because the power of the natural lenses decreases dramatically as the eyes grow axially. However, an IOL placed in a child's eye cannot change in power to match the growth of the eye. An IOL chosen for emmetropia in early childhood is likely to leave the patient highly myopic in adulthood. When operating on children, many surgeons prefer to select an IOL power that will leave the eye hyperopic, so that hyperopia will decrease with increasing age. Other authors have advocated aiming for emmetropia especially when operating on children beyond 2 years of age. This approach avoids potentially amblyogenic residual hyperopia but is likely to lead to the development of significant myopia later in life.

Multi-focal and Accommodating IOL Implantation: Multifocal and accommodative IOLs have recently gained popularity in cases of adult cataract surgery. There is an increased use of multifocal and accommodative IOLs during cataract surgeries performed on patients in their teenage years with predictable outcomes. However, we would caution surgeons that the use of these lenses may not be advantageous in growing or amblyopic eyes. With residual refractive error, especially the myopia that develops after eye growth, multifocal IOLs may (ironically) result in a higher level of spectacle dependence compared to the use of monofocal IOLs with residual myopia.

Parental Commitment: A child operated for cataract requires regular scheduled care during the first decade of life, and then every 1-2 years throughout life. To achieve the best visual outcome for the child, a long-term commitment from the parents is required. The changing refraction will require frequent follow-up examinations. Glaucoma is known to develop even years after cataract surgery. Parents need to understand that their children may initially need serial examinations under anesthesia till they are cooperative enough to be examined in the office. Parents of children with lens implants are also made aware that spectacles may still be needed postoperatively even when an IOL is implanted. In addition, changing refraction necessitates changing the power of the spectacles frequently after surgery. Taking the extra time before surgery to help parents understand the implications of the diagnosis of cataract in their children and the treatment options will promote better compliance with medication, glasses, contact lenses, and occlusion therapy. Informed parents usually are willing participants in the treatment of their children. The more they understand and accept the necessary steps, the better partners they will become in the battle for good visual functioning of their children's eyes.

Summary: The pediatrician plays an important role not only in early detection and referral for cataract patients, but also in supporting the family through the process of visual rehabilitation after the cataract is diagnosed and treatment initiated. Pediatric cataract surgery is a complex issue best left to surgeons that are familiar with its long-term complications and lengthy follow-up. Cataract surgery in children is the first stepping-stone in the long road to visual rehabilitation, not the end of the journey. An uneventful surgery is simply the first step toward achieving the main goal. Maintaining a clear visual axis while correcting the changing residual refractive error requires careful observation, sound judgment, and diligent follow-up. Complications may develop in the early postoperative period, or after many years, making it crucial to examine these children regularly on a long-term basis after pediatric cataract surgery. Management of residual refractive error, amblyopia, and strabismus must be customized to each child based on measurements that can be a challenge to obtain, and which can change over time. Despite these uncertainties, diligent teamwork involving the physician and the parents can result in a gratifying visual outcome throughout the long life of the child.