

Jyotirmoy Datta
Chandana Chakraborti



With Interactive
DVD-ROM

Sure Success in

OPHTHALMOLOGY

Viva Voce &
Practical Examination

**Sure Success in
OPHTHALMOLOGY**

Viva Voce & Practical Examination

Sure Success in OPHTHALMOLOGY

Viva Voce & Practical Examination

Jyotirmoy Datta MBBS (Cal) MS (Ophtha)

Professor and Head of the Department of Ophthalmology
Calcutta National Medical College and Hospital
32 Gorachand, Road Kolkata
West Bengal, India

Chandana Chakraborti MBBS (Cal) MD (Ophtha) AIIMS

Assistant Professor
Department of Ophthalmology
Calcutta National Medical College and Hospital
32 Gorachand, Road Kolkata
West Bengal, India



JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD

New Delhi • Panama City • London • Dhaka • Kathmandu



Jaypee Brothers Medical Publishers (P) Ltd

Headquarters

Jaypee Brothers Medical Publishers (P) Ltd
4838/24, Ansari Road, Daryaganj
New Delhi 110 002, India
Phone: +91-11-43574357
Fax: +91-11-43574314
Email: jaypee@jaypeebrothers.com

Overseas Offices

J.P. Medical Ltd
83, Victoria Street, London
SW1H 0HW (UK)
Phone: +44-2031708910
Fax: +02-03-0086180
Email: info@jpmedpub.com

Jaypee-Highlights Medical Publishers Inc.
City of Knowledge, Bld. 237, Clayton
Panama City, Panama
Phone: +507-301-0496
Fax: +507-301-0499
Email: cservice@jphmedical.com

Jaypee Brothers Medical Publishers (P) Ltd
17/1-B Babar Road, Block-B, Shaymali
Mohammadpur, Dhaka-1207
Bangladesh
Mobile: +08801912003485
Email: jaypeedhaka@gmail.com
Website: www.jaypeebrothers.com
Website: www.jaypeedigital.com

Jaypee Brothers Medical Publishers (P) Ltd
Shorakhute, Kathmandu
Nepal
Phone: +00977-9841528578
Email: jaypee.nepal@gmail.com

© 2013, Jaypee Brothers Medical Publishers

All rights reserved. No part of this book may be reproduced in any form or by any means without the prior permission of the publisher.

Inquiries for bulk sales may be solicited at: jaypee@jaypeebrothers.com

This book has been published in good faith that the contents provided by the **author(s)** contained herein are original, and is intended for educational purposes only. While every effort is made to ensure accuracy of information, the publisher and the **author(s)** specifically disclaim any damage, liability, or loss incurred, directly or indirectly, from the use or application of any of the contents of this work. If not specifically stated, all figures and tables are courtesy of the **author(s)**. Where appropriate, the readers should consult with a specialist or contact the manufacturer of the drug or device.

Sure Success in Ophthalmology Viva Voce & Practical Examination

First Edition: **2013**

ISBN : 978-93-5025-955-9

Printed at



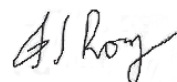
DEDICATION

*To our parents and teachers for their blessings.
To our students and residents who were
our source of inspiration.*

FOREWORD

It is indeed very satisfying to write the foreword for a very different kind of book, one on how to succeed in ophthalmology oral and practical examination. The book is well illustrated for understanding basics of ophthalmology. It almost covers all probable questions along with answers which are usually asked in MBBS examination.

So my hat is off to authors Dr Jyotirmoy and Dr Chandana, for doing such a nice job.



I S Roy

*Ex-Professor and Director
RIO, Kolkata*

PREFACE

We are excited to bring you the First Edition of the 'Sure Success in Ophthalmology Viva Voce & Practical Examination'. We realized the need for this book while conducting Final MBBS Part 1 Examination. The book is primarily meant to help the undergraduate students to face the oral and practical examination of ophthalmology. The book will be helpful also for postgraduates and PG entrance examinee. Optometry students will be benefitted if they go through a few chapters of the book.

Each chapter is devoted to a system of the eye in brief in question answer form, relevant long and short cases and surgery related to the chapter. We have included a list of instruments and relevant viva questions. A few questions have been intentionally repeated to emphasize their importance.

A miscellaneous chapter has been included which consists of some important tables and charts for quick revision.

The book is amply illustrated by photographs, line diagrams, and flow charts.

In spite of our best efforts, the book may have some inaccuracies; feedback from teachers, students will be most welcome to improve the book.

We hope you will enjoy reading the book before going to face the examiner.

Jyotirmoy Datta
Chandana Chakraborti

ACKNOWLEDGMENTS

We are indebted to the help and support of all our colleagues and students during writing the book. Our sincere thanks to our four residents (MS, PGT) Dr Jayanta Das, Dr Kritika Pal Choudhury, Dr Malay Mondal, Dr Dhananjoy Giri for their constant effort during preparation of the manuscript and DVD. We convey our sincere regards to Prof. Mita Khan for her last minute suggestions. Our sincere thanks to Mr Sambhunath Das for helping us to prepare the computer-generated images and illustrations of the book.

We are thankful to Mr Somnath Maitra and Mr Ajay Kumar Mourya (Cine India International) for helping us to prepare the DVD. We are grateful to Dr Dayal Bandhu Mazumder (RMO) for his valuable suggestions during preparing the DVD.

We express our sincere thanks to the publisher, Jaypee Brothers Medical Publishers (P) Ltd and their technical personnels to support us. The enthusiastic cooperation received from Mr S Hazra of Jaypee Brothers is appreciated. Last but not the least, the credit of meticulous publication of this book goes to Jaypee Brothers.

We are indebted to our families for their constant support while preparing the manuscript.

<p align="center">DISTRIBUTION OF MARKS IN OPHTHALMOLOGY (MBBS EXAM)</p>
--

Total Marks : 100
 Pass Marks : 50
 Theory Marks : 40

Module of Questions in theory paper

Pre and Para clinical subject:

1. Question (short answer type) 10 Marks

Operative and Clinical question:

2. Two out of three of which one operative question 10 Marks

3. Problem based questions 10 Marks

4. Short notes (Two out of three) 10 Marks

Oral :

10 Marks

a. Instrument (two) 3 Marks

b. X-ray and other imaging 3 Marks

c. Oral questions 4 Marks

Practical :

30 Marks

One long case 20 Marks

One short case 10 Marks

Internal Assessment:

20 Marks

5th semester and 6th semester

(Continuous Assessment) 10 Marks

Final assessment 10 Marks

REFERENCES

- Sihota R, Tandon R. Parsons' Diseases of the Eye. 20th ed. 2007.
- Nema HV, Nema N. Textbook of Ophthalmology. 6th ed. Jaypee brothers medical publishers (P) Ltd, 2012.
- Mukherjee PK. Clinical examination in ophthalmology, Elsevier, 2007.
- Basak SK. Essentials of Ophthalmology, 4th ed. Current Books International, 2007.
- Shingleton BJ, Hersh PS, Kenyon KR. Eye Trauma, Mosby Year Book.
- Khurana AK. Comprehensive Ophthalmology. 4th ed. New Age International (P) Limited, 2009.
- Sharma YR, Sudan R. Concise textbook of Ophthalmology, Elsevier, 2007.

CONTENTS

1. History Taking and Examination of An Ophthalmic Case	1 – 12
2. Eyelid	13 – 25
3. Conjunctiva and Sclera	26 – 49
4. Cornea	50 – 65
5. Lens	66 – 99
6. Uvea	100 – 117
7. Glaucoma	118 – 139
8. Retina and Vitreous	140 – 149
9. Orbit and Globe	150 – 152
10. Neuro-ophthalmology and Pupil	153 – 159
11. Squint	160 – 164
12. Refraction	165 – 176
13. Lacrimal Apparatus	177 – 187
14. Trauma	188 – 196
15. Community Ophthalmology	197 – 200
16. Ocular Therapeutics	201 – 208
17. Instruments	209 – 244
18. Miscellaneous	245 – 260
Glossary	261 – 263
Index	265 – 274

Chapter

1

History Taking and Examination of An Ophthalmic Case

Name : _____

Age : _____

Sex : _____

Religion : _____

Address : _____

Occupation : _____

Date of admission/Date of examination : _____

Chief complaints:

Dimness of vision - distant/near/both/night vision/day vision (mention the eye involved).

Redness : _____

Itching : _____

Headache/Eyeache : _____

Pain : _____

Watering : _____

Discharge : _____

Foreign body sensation : _____

Opacity over the black of the eye (cornea) : _____

Deviation of the eye : _____

Double vision (diplopia) : _____

Abnormal fleshy mass in the eye : _____

Protrusion of eyeball : _____

Drooping of eyelid : _____

H/O present illness:

Visual disturbance - onset, progress, distance/near, association with pain.



Headache-relation with close work, nausea, vomiting, tinnitus, vertigo.
H/O trauma.

Past history:

Any ocular disease.

Systemic disease-Infective, metabolic, autoimmune diseases.

Treatment history:

Medical treatment : _____

Surgical treatment : _____

History of allergy and drug reaction:

Exogenous : _____

Endogenous : _____

Drugs : _____

Personal history:

Smoking, alcohol intake.

Diabetes, hypertension, hyperthyroidism, bronchial asthma, tuberculosis.

Family history:

History of glaucoma, metabolic disorder, myopia, retinal detachment, night blindness.

Occupational history:

Other relevant history:

Birth history : _____

Antenatal history : _____

EXAMINATION

General examination: Apparent age, anaemia, jaundice, cyanosis, pulse, respiration, temperature, BP.

Ocular examination: Head posture, orbital examination, facial asymmetry.

Visual acuity(VA)-

RE

LE

(Unaided/aided/with pin hole)

Eye ball: Size, shape.



Palpebral aperture: Symmetrical/asymmetrical, shape, width, height.

Ocular movements:

Eye lids: Position, movements, lid margin, cilia, canthi, skin of the lids.

Lacrimal apparatus: Any regurgitation on pressure over sac area, swelling over medial canthal area.

Conjunctiva: Congestion, discharge, subconjunctival haemorrhage, nodule.

Sclera: Colour, shape, nodule, vessel, ectasia.

Cornea: Size, shape, transparency, ulcer, vascularisation, opacity, KPs, sensation.

Anterior chamber: Depth, Contents (cells, flare, hypopyon, hyphaema).

Iris: Colour, pattern, holes, synechiae, vascularisation, nodules, iridodonesis

Pupils: Direct and consensual light reflex, shape and size, position.

Lens: Transparency, position-pseudophakia/aphakia/subluxation/dislocation, deposits on anterior/posterior surface.

Purkinje images: 1st, 2nd, 3rd, 4th (present/absent).

IOP: Digital tonometry.

DETAILS OF OCULAR EXAMINATIONS

Visual Acuity (VA)

Distance vision

- VA is tested in each eye separately (with and without glass).
- The patient is asked to read the Snellen's chart at a distance of 6 meter. VA of 6/6 is taken as normal (**Fig. 1.1**) (Rays coming from a distance of 6 m are considered to be parallel).
- If the VA is < 6/6, a pin hole is used (to test whether vision is improved or not).
- The last line he/she reads is recorded as VA of that eye e.g top letter is 6/60, 2nd line 6/36, 3rd line 6/24, and so on.
- If he can't read the 6/60 line then he is asked to step forward till he is able to read the top letter of the chart and the vision is recorded as 5/60, 4/60 and so on.
- If he fails to read 6/60 line at 1 meter distance then he is asked to count the examiner's finger. The distance at which he counts finger is recorded as VA -FC (FC = finger counting).



Fig. 1.1 : Snellen's test type

- If FC is absent then hand movement (HM) close to face is noted.
- If the patient's vision is less than HM, perception of light (PL) is tested by closing one eye. Ask the patient to close one eye and the light is thrown on uncovered eye and ask him whether he can sense the light. If he fails then vision is recorded as no perception of light (NO PL).
- Projection of rays (PR) is tested in all cases. Patient is asked to look straight, other eye is closed, a small beam of light is thrown from nasal, temporal, superior and inferior direction. The patient is asked to detect from which direction light is coming, and PR is denoted as (+) and absence(-) in particular quadrant. (If there is no PL, there is no need to examine PR).

Near vision

Tested after correcting the distant vision.

Charts used-

- Snellen's near chart
- Jaeger's chart

Procedure

Ask the patient to sit and read the near chart at a distance of 25-33 cm(bright illumination).



Acuity is recorded - e.g. N6/N8/N12

(Normal near acuity is N6).

Q.1. A patient has VA 6/60 in RE, what does it mean?

Ans. It means the patient can read the topmost letter (Snellen's chart) at a distance of 6 meter with the RE ,which a normal person will read from a distance of 60 meter.

Q.2. How will you test visual acuity in an illiterate patient?

Ans. 'E' acuity or Landolt C chart.

Q.3. What do you mean by perception of light?

Ans. It is the ability to perceive light anywhere in the retina (PL once lost is lost forever).

Q.4. What are the causes of loss of PL?

Ans. Optic atrophy

- Central retinal artery obstruction(CRAO)
- Total retinal detachment
- Avulsion of optic nerve

Q.5. What do you mean by projection of rays?

Ans. It denotes gross retinal function at periphery.

Q.6. A patient has VA 6/36, will you test PR?

Ans. Yes. It should be a routine practice to test PR with every level of VA (The patient may have inferior RD with defective PR superiorly).

Q.7. What are the causes of defective PR?

- Ans.**
- Retinal detachment(RD)
 - Advanced glaucoma
 - CRAO

Q.8. Which part of retina you are testing when you are projecting light from temporal side?

Ans. Nasal retina

Q.9. How will you test for colour vision?

- Ans.**
- Ishihara plates(most common)
 - Hardy Rand Rittler plates
 - Farnsworth -Munsell: 100 hue test
 - Edridge Green lantern test



Q.10. Which type of colour vision defect is detected with Ishihara plates?

Ans. Red green defect.

Q.11. What are the conditions where colour vision testing is important?

Ans. Jobs which require identifying coloured signals, like pilots, locomotive drivers, electricians, artists.

Q.12. Which is the commonest colour vision defect?

Ans. Red-green defect.

Q.13. What do you mean by field of vision?

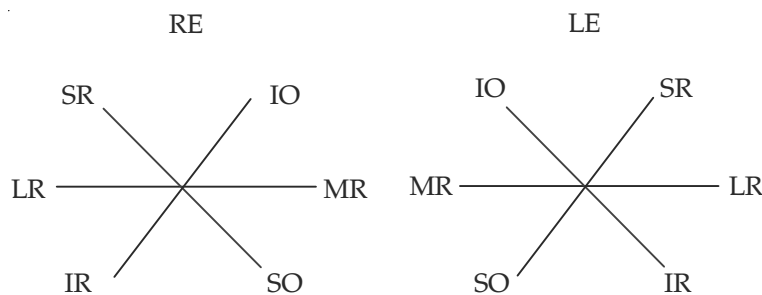
Ans. It is the area that is visible at a given time around a point of fixation (for that eye in that gaze).

Q.14. What are the different methods of testing visual field?

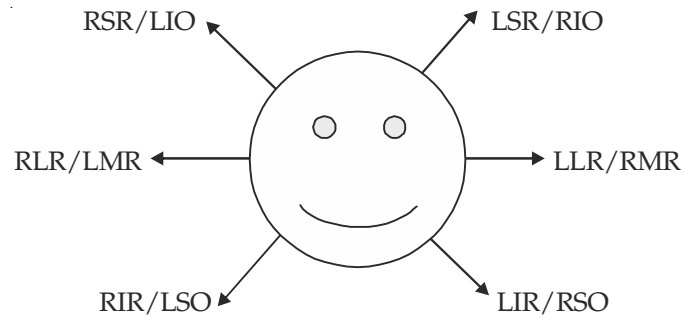
Ans.

- Confrontation test(OPD/bed side procedure)
- Automated perimetry (Humphrey/Octopus perimeter)
- Goldmann perimetry (manual)
- Amsler grid (central 10 degree)

OCULAR MOVEMENTS



Uniocular movements (Duction)



Binocular movements (version)

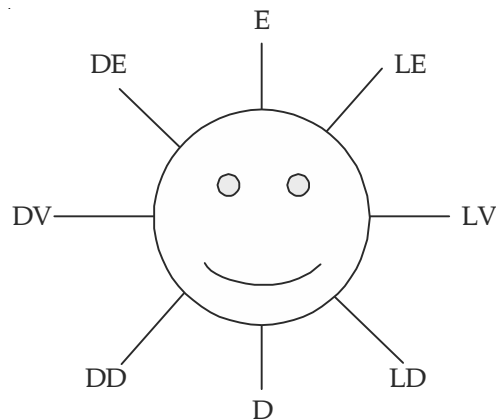
Q.15. What are the 6 cardinal gazes ?

- Ans.**
1. Dextroversion
 2. Levoversion
 3. Dextrolevation
 4. Levoelevation
 5. Dextrodepression
 6. Levodepression

(The movement of one muscle is paired with muscle of the opposite eye ,those are called yoke muscles.)

Q.16. Give one example of yolk muscles.

Ans. Rt lateral rectus and Lt medial rectus(dextroversion).





Q.17. What is the primary position of the eye?

Ans. It is noted by asking the patient to fix an object in a straight ahead position.

Q.18. What are secondary positions of gaze?

Ans. From primary position move the target to right, left, up, down, these are secondary positions of eye.

Q.19. What are tertiary positions?

Ans. a. Dextroelevation - Target is moved up and right.
b. Levoelevation - Target is moved up and left.
c. Dextrodepression - Target is moved down and right.
d. Levodepression - Target is moved down and left.
(**N.B** - every time the light is brought to the primary position).
(**N.B** - there are 6 cardinal gazes and 9 cardinal points).

Examination of Eyelids

Lid margin- Position, any thickening, white scales, erythema, ulcer, pus point, swelling.

Eyelashes- Misdirection, loss of eyelashes, matting of the eyelashes.

Lid skin- Colour, nodule, oedema, swelling.

Examination of conjunctiva-(Examined with Torch light , Loupe and Slit Lamp)

The lower fornix is easily exposed by drawing the lower lid down while patient looks up.

The upper palpebral conjunctiva is exposed by everting the upper lid.

Eversion of the lid : A probe or a thin pencil or index finger is placed along the skin of the upper lid at the level of upper border of the tarsus with the patient looking towards his feet.

Or

The eye lashes are grasped between the index finger and thumb, and the lid is drawn away from the globe, using the probe as a fixed point.

Examination of bulbar and limbal conjunctiva are done by separating the lids while the patient is asked to move his eye ball in different directions.



Look for

- a. F.B
- b. Inflammation (dilated vs.)
- c. Papillae
- d. Follicles
- e. Cysts
- f. Concretions (upper tarsal conj)
- g. Tumor
- h. Tear film abnormalities
- e. Any pigmentation, naevus, fleshy mass, dryness/ wrinkling

Examination of Cornea and Sclera

(Examined with Torch light, Loupe and Slit Lamp).

Cornea

(Transparent small optical part) .

Look for

- Size
- Shape
- Surface
- Sheen
- Transparency
- Opacity if any (with grade)
- Vascularisation
- Degeneration
- Arcus Senilis
- Band shaped keratopathy
- Pigmentation
- Purkinje's image

Sclera (opaque non optical part)

Look for

- Colour
- Any nodule
- Congestion
- Staphyloma



Examination of Anterior Chamber(AC)

Look for

- Depth of AC
- Hyphaema
- Hypopyon
- Turbid aqueous (cells, flare) by SL
- Any foreign body

Examination of Iris

Look for

- Color
- Pattern(crypts, fissures)
- Synaechie
- Nodules
- Hole
- Neovascularisation (NVI)
- Coloboma

Examination of Pupil

- Size
- Shape
- Colour
- Reflexes – Direct and consensual

Method of Testing Pupillary Reflexes

(Dark room)

Sit in front of the patient

Direct Reflex

One eye is covered with patients own palm, bright light is thrown on uncovered eye and constriction is noted.

Indirect/consensual—Pupil of the contralateral eye contracts when the light beam is thrown on ipsilateral eye.

Examination of Lens

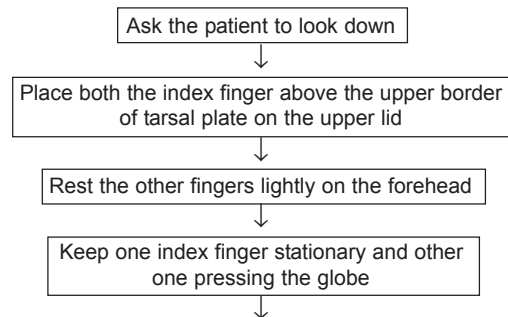
- Color : Transparent/greyish white/pearly white/amber/brown/black.



- Position: Normal/subluxation/dislocation/aphakia/pseudophakia.
- Iris shadow(present/absent).
- Pigmentation over lens surface.
- Purkinje's images.

Digital Tonometry- (rough estimation of IOP)

Method



Try to feel the resistance (like fluctuation test of a cyst)

Q.20. Why you will ask the patient to look down?

Ans. To avoid the resistance of tarsal plate.

Examination of Lacrimal Apparatus

Puncta

- Open/stenosed/inflamed.
- Position- normal(in contact with the globe)/everted/absence.
- Signs of inflammation-discharge/erythema.

Lacrimal Sac

Any swelling, erythema, scar mark, fistula near the medial canthal skin area.

Press over sac (pressing with the thumb just below the medial canthal tendon) and look for any regurgitation .



Examination of the Fundus

Ophthalmoscopy

- Direct
- Indirect

Q.21. What are the differences between direct and indirect ophthalmoscopy?

Ans.

<i>Features</i>	<i>Direct</i>	<i>Indirect</i>
Image	Virtual, erect, magnified 15 X	Real, inverted, magnified 1X to 4X
Stereopsis	Nil	Good
Field	Small 10°	Large 37°
Peripheral view	Not possible	Well seen
Media	Difficult to examine in hazy media	can be examined through hazy media

Short case

1. Name, age, sex, occupation
2. Chief complaints
3. Any relevant history
4. Ocular examination
5. Diagnosis

Chapter

2

Eyelid

Q.1. Describe the structure of eyelids.

Ans. (Figs 2.1a and 2.1b)

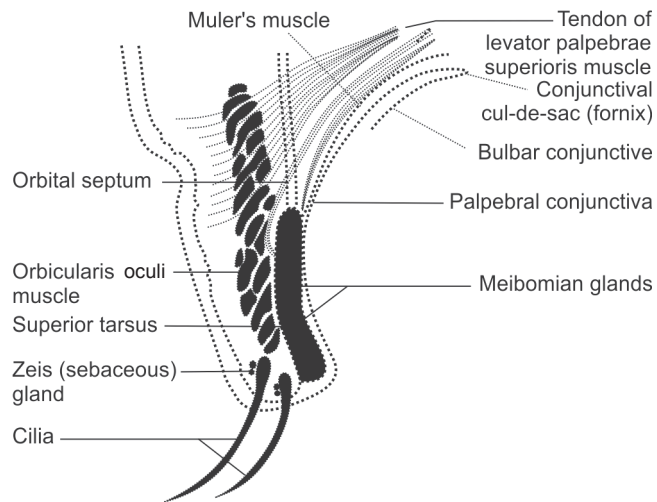


Fig. 2.1a : Anatomy of eyelid

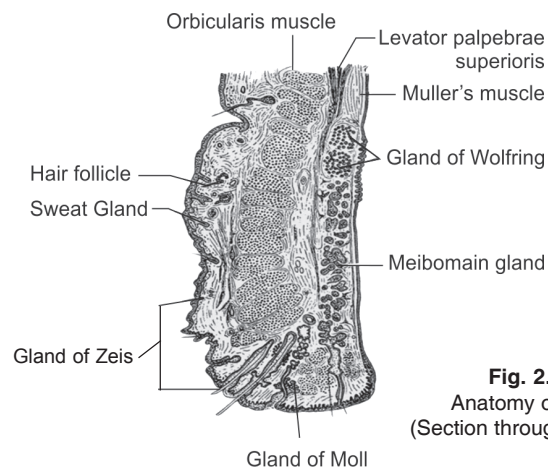


Fig. 2.1b:
Anatomy of eyelid
(Section through upper lid)



Lids are multilayered in structure.

Anterior Lamella

- a. Cutaneous layer
- b. Subcutaneous areolar tissue layer
- c. Muscular layer

Posterior Lamella

- a. Fibrous layer
 - Orbital septum
 - Tarsal plate
- b. Palpebral conjunctival layer

Q.2. Is there any subcutaneous fat in eyelid?

Ans. No

Q.3. Describe the anatomy of the lid margin.

Ans. 2 mm broad

2 parts:

- a. **Lacrimal** part (medial to puncta)- no eyelash or gland
- b. **Ciliary** part (lateral to puncta)-
 - Anterior border- rounded
 - Posterior border- sharp (against the globe)
 - Intermarginal strip- between the two border

Grey line

- Marks junction of skin and conjunctiva
- Divides the intermarginal strip into anterior strip (bearing 2-3 rows of eyelashes) and posterior strip (with opening of meibomian glands)

Q.4. What is the importance of the grey line?

Ans. During operation of eyelids, splitting of the lids is done at the level of grey line.

Q.5. What is so important about tarsal plate?

Ans. Meibomian glands are embedded in it.

Q.6. What are the muscles of eyelid?

Ans. a. Orbicularis oculi
b. Muller's muscle
c. Levator palpabrae superioris (only in upper lid)

Q.7. What are the functions of orbicularis oculi? What is its nerve supply?



Ans. It causes closure of the lids and helps in drainage of tears.
Nerve supply- Zygomatic branch of facial nerve.

Q.8. What will happen if there is paralysis of orbicularis (7th nerve palsy)?

Ans. • Lagophthalmos
• Exposure keratitis

Q.9. Describe the origin, insertion, nerve supply and function of *Levator palpebrae superioris* (LPS).

Ans. Origin: Apex of orbit
Insertion : It is inserted to –
a. Skin of lids
b. Conjunctiva of superior fornix
c. Upper margin of tarsal plate
Nerve supply - 3rd nerve
Function—Raises the upper lid
Paralysis → ptosis

Q.10. What is the origin, nerve supply of *Muller's muscle*? What type of muscle is it?

Ans. In upperlid , it arises from fibres of LPS.
Nerve supply- Cervical sympathetic.
It is a non striated muscle.

Q.11. What is the function of *Muller's muscle*?

Ans. Upper lid - elevates the eye lid.
Lower lid - elevates lower lid.

Q.12. Name one clinical condition where there is ptosis due to paralysis of *Muller's muscle*?

Ans. Horner's syndrome.

Q.13. What are the clinical features of *Horner's syndrome*?

Ans. a. Ptosis
b. Miosis
c. Iris heterochromia
d. Anhydrosis
e. Enophthalmos
f. Loss of ciliospinal reflex

Q.14. Name the *glands of eye lids*.

Ans. a. Meibomian gland
b. Gland of Zeis
c. Gland of Moll
d. Gland of Krause and Wolfring



Q.15. Where are the *meibomian glands* situated?

Ans. They are situated within the substance of tarsal plate.

Q.16. What type of gland is *meibomian gland*?

Ans. Modified sebaceous gland.

Q.17. How many *meibomian glands* are present?

Ans. Upper lid - 30 – 40

Lower lid - 20 – 30

Q.18. What is the function of *meibomian gland*?

Ans. They secrete oily layer of tear film which lubricates the eye and prevents evaporation of tears from the cornea.

Q.19. What is the arrangement of their ducts?

Ans. Vertically arranged ducts.

Q.20. What type of gland is *gland of Zeiss*? Where are they present?

Ans. Modified sebaceous gland.

They lie in the lid margin and open in the follicles of eye lashes.

Q.21. What type of gland is *gland of Moll*?

Ans. Modified sweat gland.

Their ducts open into the duct of gland of Zeiss or into the follicle.

Q.22. What are the *accessory lacrimal gland*?

Ans. Gland of Krause and wolfring.

They are situated on the palpebral conjunctiva.

Q.23. What is *grey line* and what is its importance?

Ans. It is a line in intermarginal strip of lid margin just anterior to the orifice of the duct of meibomian gland.

It is an important landmark for lid surgery in which lid is split.

It indicates the position of loose fibrous tissue between orbicularis oculi and subcutaneous tissue.

Q.24. What are the *functions of eye lids*?

Ans. Protects the eye ball from external injuries, F.B., etc.

Maintain precorneal tear film.

Drainage of tears by lacrimal pump.



Q.25. What is the normal position of upper eye lid?

Ans. Normally upper lid covers the upper 2 mm of cornea.
Lower lid just touches the limbus.

Q.26. What is the normal height of the palpebral aperture?

Ans. 10-11mm

CHALAZION (SHORT CASE)

Q.27. What is your case?

Ans. A case of chalazion on Upper/lower lid of RE/LE. (**Fig-2.2**)

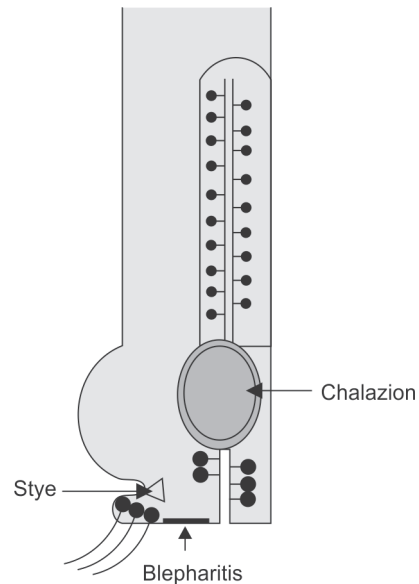


Fig. 2.2 : Inflammation of the lid margin

Q.28. What are the points in favour of your diagnosis?

Ans.

- Painless, non-tender round swelling on lid, away from lid margin.
- Sense of heaviness, no pain.
- It is fixed to tarsal plate, but free from overlying skin.
- On eversion of the lid, the conjunctiva over the chalazion appears bluish.



Q.29. What is chalazion?

Ans. It is a chronic non-specific inflammatory granuloma of the meibomian gland.

Q.30. What is the histological picture of chalazion?

Ans. Centre-cheesy, sebaceous material surrounded by granulation tissue containing – lymphocyte, foreign body giant cell and epithelial cell.

Q.31. What are the predisposing factors of chalazion?

Ans.

- Young adult and children
- Refractive error
- Blepharitis
- Chronic conjunctivitis
- Diabetes mellitus in adults

Q.32. What is the fate of a chalazion?

Ans.

- Spontaneous resolution
- Stationary
- Secondary infection – internal hordeolum
- Large chalazion – mechanical ptosis
- Burst through the palpebral conjunctiva (fungating mass of granulation tissue) or through the skin
- Rarely malignant changes (adult)
- Calcification

Q.33. What are the differential diagnosis?

Ans.

- Stye
- Internal hordeolum
- Meibomian gland carcinoma

Q.34. What is the commonest eyelid malignancy?

Ans. Basal cell carcinoma

Q.35. What are the T/T of chalazion?

Ans.

- Warm compresses
- Steroid-antibiotic ointment with lid massage
- Oral doxycycline 100 mg BD x 5 days
- Anti inflammatory drugs
- Intralesional injection of steroid (Triamcinolone acetate)



Large chalazion

If chalazion fails to resolve after 3-4 weeks
of medical therapy

Patient wishes to have it removed

} Surgical T/T

Q.36. What surgery is done for chalazion?

Ans. Incision and curettage.

Q.37. Describe the steps of chalazion surgery?

Ans.

Local anaesthesia, infiltration of the skin over the
chalazion with 2% lignocaine hydrochloride,
subcutaneous tissue

Topical 4% lignocaine in conjunctival sac

Fixation of chalazion from the conjunctival
side with chalazion forceps/clamp

Vertical stab incision with no. 11
surgical blade over the chalazion site

With chalazion scoop the cyst is
curetted by rotating movement

End point of curettage is a gritty sensation

Removal of clamp

Antibiotic eye drop/ointment

Firm pressure bandage

Oral anti-inflammatory & analgesic for five days

Topical antibiotic steroid eye drop from next day

INTERNAL HORDEOLUM/ HORDEOLUM INTERNUM

(SHORT CASE)

Q.38. What is an internal hordeolum/ hordeolum internum?

Ans. This is a suppurative inflammation of a meibomian gland
and may be due to secondary infection of a chalazion.



Q.39. How will you treat internal hordeolum?

- Ans.**
- Hot compress
 - Systemic analgesics
 - Local antibiotic drops and ointment
 - Systemic antibiotic
 - Once the acute condition subsides → incision and curettage

EXTERNAL HORDEOLUM (SHORT CASE)

Q.40. What is external hordeolum (stye)?

Ans. It is an acute suppurative inflammation of glands of Zeiss and Moll.

Q.41. What type of gland is Zeiss gland?

Ans. Modified sebaceous glands at lid margin.

Q.42. What are the complications of stye?

- Ans.**
- Ulcerative blepharitis
 - Lid abscess, Cellulitis
 - Facial cellulitis

Q.43. What is the treatment of stye?

- Ans.**
- Hot compress
 - Topical antibiotic drops/ointment 4-6 times daily
 - Systemic NSAID
 - Broad spectrum systemic antibiotic 1 week
 - Evacuation of pus by pulling out the affected eyelash/ drainage with small stab incision

Q.44. What is lagophthalmos?

Ans. Inability to close the eyelids
It is seen in Facial palsy, severe proptosis, symblepharon.

Q.45. What is the treatment of lagophthalmos?

- Ans.**
- Artificial tear drops
 - Lubricating eye ointment
 - Tarsorrhaphy

Q.46. What is tarsorrhaphy?

Ans. Closure of the palpebral aperture by suturing the lid margins temporarily or permanently to avoid exposure of the ocular surface.



Q.47. What are the indications of tarsorrhaphy?

- Ans.**
- Neurotrophic keratopathy
 - Lagophthalmos
 - Neuroparalytic keratitis

Q.48. What are the different types of tarsorrhaphy you know?

- Ans.**
- Lateral
 - Medial
 - Paracentral (Fig-2.3)

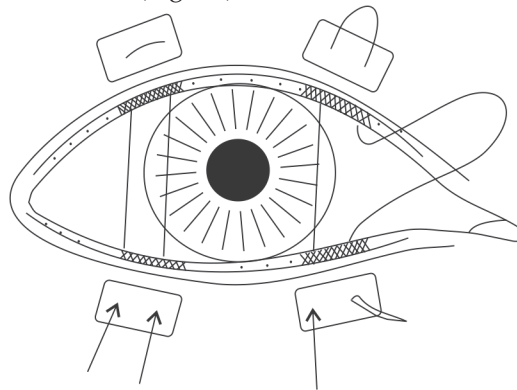


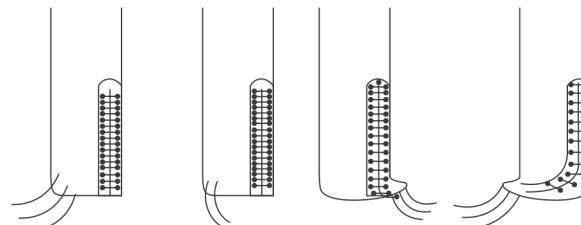
Fig. 2.3 : Tarsorrhaphy for Lagophthalmos

ENTROPION (SHORT CASE)

Q.49. What is *entropion*?

Ans. (Fig-2.4)

Inward turning of eye lid



(A) Normal lid margin, (B) Trichiasis (C) Entropion (D) Ectropion
anterior border rounded lashes in normal position, inner margin sharp.

Fig. 2.4 : Deformity of lid margin



Q.50. What are different types of entropion?

- Ans.**
- Spastic (due to aging)
 - Involutional
 - Cicatricial
 - Congenital

Q.51. What are the symptoms of entropion?

- Ans.**
- F.B sensation
 - Watering
 - Blepharospasm

Q.52. What are the complications of entropion?

- Ans.**
- Corneal erosion
 - Corneal vascularisation
 - Keratitis

Q.53. Name one condition where there will be cicatricial entropion?

- Ans.** Trachoma. Caused by cicatricial contraction of the palpebral conjunctiva.

Q.54. What is the treatment of entropion?

- Ans.**
- Lubricating drops/ointment
 - Surgery (type of surgery varies according to aetiology)

ECTROPION (SHORT CASE)

Q.55. What is ectropion?

- Ans.** Ectropion is defined as eversion of lid margin and eye lashes away from globe.

Q.56. What are the clinical types of ectropion?

- Ans.**
- Congenital
 - Senile
 - Cicatricial
 - Paralytic
 - Mechanical

Q.57. What is the treatment of ectropion?

- Ans.** (according to aetiology)

Surgical

Kuhnt-Szymanowski procedure modified by Smith.
Lateral Tarsorrhaphy.

**PTOSIS (SHORT CASE)**

Q.58. What is *ptosis*?

Ans. Drooping of upper lid.

Q.59. What are the causes of ptosis?

- Ans.**
- a. Congenital
 - b. Acquired:
 - 1. Neurogenic
 - 2. Myogenic
 - 3. Mechanical
 - 4. Aponeurotic

Q.60. What is pseudoptosis?

Ans. Apparent drooping of upper eyelid due to lack of support.

Seen in:

- a. Phthisis bulbi
- b. Anophthalmos
- c. Microphthalmos

Q.61. What is *trichiasis*?

Ans. Distortion of the cilia, so that they are directed backward and rub against the globe.

Q.62. What are the causes of trichiasis?

- Ans.**
- a. Congenital- distichiasis
 - b. Acquired:
 - Blepharitis
 - Conjunctivitis
 - Trachoma
 - Burns
 - SJS
 - Entropion

Q.63. What are the complications of trichiasis?

- Ans.**
- Recurrent corneal erosion
 - Corneal atheromatous ulcer
 - Vascularisation of cornea

Q.64. What is distichiasis?

Ans. Congenital condition
An extra posterior row of cilia is seen



Q.65. What are the treatment options of trichiasis?

- Ans.**
- Epilation of offending lashes
 - Electrolysis of hair follicle
 - Eletro diathermy
 - Cryo surgery

Q.66. What is *madarosis*?

- Ans.** Loss of cilia

Q.67. What is *poliosis*?

- Ans.** Whitening of cilia
Seen in VKH syndrome, ageing

Q.68. What is *ankyloblepharon*?

- Ans.** Narrow palpebral tissue due to adhesion between two lid margin at canthus.

Q.69. What is *blepharophimosis*?

- Ans.** Narrow palpebral fissure

Q.70. What is *xanthalesma*?

- Ans.** Yellow subcutaneous plaque on eye lid
Seen in increased cholesterol level or familial

Q.71. What is *blepharitis*?

- Ans.** Inflammation of lid margin

Q.72. What are the types of blepharitis?

- Ans.**
- Squamous blepharitis
 - Ulcerative blepharitis

Q.73. What are the clinical features of *squamous blepharitis*?

- Ans.** Itching of eyes
Yellowish crusts at lid margin

Q.74. What is the aetiology of squamous blepharitis?

- Ans.** Associated with dandruff in scalp.

Q.75. What are the clinical features of the ulcerative blepharitis?

- Ans.** Crusts at lid margin if removed tiny ulcers cause bleeding.

Q.76. What are the predisposing factors of blepharitis?

- Ans.**
- Staphylococcal infection
 - Refractive error



- Chronic conjunctivitis
- Unhygienic lid condition
- Use of – kajal, surma, mascara etc

Q.77. What are the complications of blepharitis?

- Ans.**
- Trichiasis
 - Madarosis
 - Ectropion
 - Tylosis
 - Blepharoconjunctivitis

Q.78. How will you treat blepharitis?

- Ans.** To maintain hygienic status
- Use of medicated shampoo
 - Correction of refractive errors
 - Treatment of louse infestations
 - Local- 3% sodium-bicarbonate lotion is applied with cotton buds to lid margin to soak the crusts or scales 2-3 times daily or baby shampoo may be used.
 - Antibiotic steroid ointment to be applied by rubbing the lid margin 3 times daily.
 - Stop using oil on scalp.
 - An antibiotic eye drop – if secondary infection is suspected.
 - Systemic—a course of tetracycline or doxycycline orally for 2-3 times is useful in severe ulcerative blepharitis.

Chapter

3

Conjunctiva and Sclera

Q.1. Define conjunctiva.

Ans. It is a mucous membrane covering the inner surface of the eyelids and reflected to cover the anterior part of the eye ball over sclera, upto the equatorial margin.

Q.2. What are the parts of conjunctiva?

Ans. (Fig. 3.1)

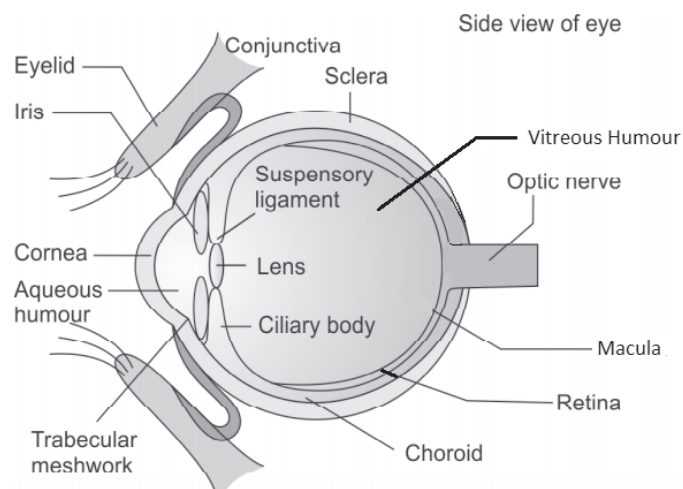


Fig. 3.1 : Conjunctiva with eyeball

- **Palpebral part** - it starts at the grey line of the lid margin. It consists of marginal, tarsal and orbital part. It is firmly adherent to the deeper tissues.
- **Bulbar part** - overlies the sclera, it is freely mobile.
- **Fornices** - junction of palpebral and bulbar conjunctiva (cul-de-sac).
- **Limbal** - conjunctiva around the limbus, it is adherent.



Q.3. Describe the microscopic structure of conjunctiva.

- Ans.**
- **Epithelial layer** - two layers of epithelium over the palpebral conjunctiva and the transitional stratified squamous epithelium at the inter marginal strip .
Epithelium becomes gradually thicker from fornices to the limbus, forming stratified non- keratinized epithelium near the corneal margin.
The rest of the palpebral conjunctiva leaving the lid consists of thinner non keratinized epithelium .
Goblet cells are present throughout the epithelium; more near the fornices.
 - **Subepithelial layer** – Adenoid layer consisting of loose connective tissue containing leucocytes.
 - **Fibrous layer** – Below the adenoid layer a much denser layer, blends with the deeper structures - lid or sclera.

Q.4. What is the blood supply of the conjunctiva?

- Ans.**
- Anterior conjunctival artery from the anterior ciliary artery
 - Posterior conjunctival artery from the lacrimal artery
 - Palpebral branch of the nasal artery

Q.5. What is the nerve supply of the conjunctiva?

- Ans.** Ophthalmic division of trigeminal nerve.

Q.6. What are the functions of conjunctiva?

- Ans.**
- Tear production (mucin by the goblet cells, aqueous by the accessory lacrimal gland)
 - Supply oxygen directly to the cornea when eyes are open
 - Protection of the eye by defence mechanism e.g an intact epithelial barrier and lacrimation
 - Specific immunologic mechanism - outpouring of mast cells , leucocytes, antibody IGA

Q.7. What are the organisms normally present in the conjunctival sac ?

- Ans.**
- Staph. Epidermidis
 - Diphtheroids
 - Propionibacterium acne
 - Corynebacterium xerosis



Q.8. What are the common symptoms of conjunctival disorders?

- Ans.**
- Redness
 - Stickiness
 - F.B sensation
 - Grittiness
 - Lacrimation
 - Photophobia
 - Slight blurring if excess secretion
 - Burning sensation

Q.9. What is papilla?

- Ans.**
- They are hyperplasia of the normal vascular system with glomerulus like branches of capillaries growing into the epithelium in inflammatory conditions.
 - They are raised areas with flat topped velvety appearance and reddish in color.

Q.10. What is follicle?

- Ans.** Localized aggregation of lymphocytes in subepithelial adenoid layer. They appear as yellowish white round elevation, 1–2 mm in diameter.

Q.11. What is chemosis ?

- Ans.** Odema of conjunctiva due to exudation from the abnormally permeable capillaries is chemosis. Conjunctiva becomes swollen and gelatinous in appearance .

Q.12. What are the conditions where you might get chemosis of conjunctiva ?

- Ans.**
- Acute inflammation—
Conjunctivitis
Orbital cellulitis
Panophthalmitis
 - Obstruction to the circulation — dysthyroid ophthalmopathy
 - Blood disorders — anaemia, angioneurotic edema.

Q.13. What is concretion?

- Ans.** It is inspissated mucous secretions seen in the palpebral conjunctiva.

Cause – Idiopathic conjunctival degeneration .



Q.14. What are the causes of conjunctivitis?

- Ans.**
- **Infectious**
 - Bacterial
 - Staph . aureus / albus
 - Haemophilus aegyptus
 - N . gonorrhoea
 - Viral
 - Herpes simplex
 - Herpes zoster
 - Adeno virus
 - Chlamydia- Trachoma
 - Fungal - Aspergillus
 - Parasite
 - **Non infectious**
 - Allergic
 - Toxic
 - Irritants

Q.15. What are the causes of membranous conjunctivitis?

- Ans.**
- Corynebacterium diphtheriae
 - Beta haemolytic streptococcus
 - Strept. Pneumoniae
 - N . gonorrhoea

Q.16. What is pseudomembranous conjunctivitis?

Ans. It is caused by organisms (mentioned above), but in milder form. On everting the lids, palpebral conjunctiva is seen to be covered with a white membrane which peels easily without much bleeding.

Q.17. What is symblepharon?

Ans. If the palpebral and bulbar conjunctiva are stuck together, it is called symblepharon.

Q.18. What are the causes of symblepharon?

- Ans.**
- Chemical injury
 - Burn
 - Stevens-Johnson's syndrome

PTERYGIUM (SHORT CASE)

Q.19. What is your case?

Ans. A case of pterygium(RE/LE).



Q.20. What is pterygium?

- Ans.**
- It is a wing shaped fold of fibrovascular tissue, arising from the interpalpebral conjunctiva and extending onto the cornea.
 - It is caused by degeneration of subconjunctival tissue.
 - It invades the cornea , involving the Bowman's membrane and superficial stroma, the whole thing being covered by conjunctival epithelium.

Q.21. Which is the usual location of pterygium?

- Ans.** Nasal (3 or 9 O'clock position at the limbus).
May be temporal

Q.22. What is the pathogenesis of pterygium?

- Ans.** Elastotic degeneration of deep conjunctival layers. The superficial layers of the stroma and Bowman's membrane is destroyed.

Q.23. What are the aetiologies of pterygium?

- Ans.** Exact cause is unknown. Following factors are responsible.
- Hot, sandy and dusty weather
 - Chronic ultraviolet light exposure (commonly seen among farmers and outdoor workers)
 - Pinguecula is a precursor

Q.24. What are the parts of a pterygium?

- Ans.** (Fig. 3.2)

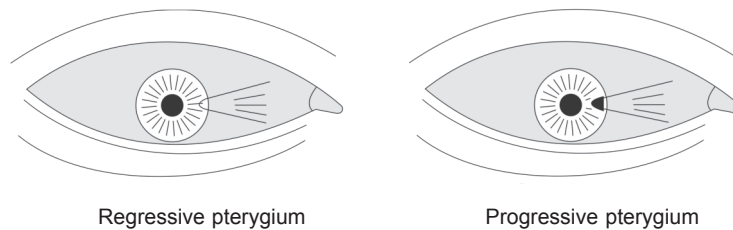


Fig. 3.2 : Types of pterygium



- Apex(apical part of the triangular mass)
- Neck(constricted portion at the limbus)
- Body(the bulky part beneath the conjunctiva)
- Cap – a semilunar white opacity just in front of the apex or head (may or may not be present)

Q.25. What is the literal meaning of pterygium?

Ans. Wing

Q.26. What are the different types of pterygium you know?

Ans.

- Progressive
- Atrophic
- Stationary

Q.27. What are the features of progressive pterygium?

Ans. Thick, Vascular, fleshy, increasing in size and cap is present.

Q.28. What are the features of atrophic pterygium?

Ans. Thin, attenuated, no cap, less vascular, no progression.

Q.29. What is a Pseudopterygium? What are the causes of Pseudopterygium?

Ans.

- Found in any meridian
- Usually Unilateral
- Seen at any age
- Stationary
- History of injury or surgery may be there
- A probe can be passed under the neck of the pseudopterygium but not in true pterygium

Causes:

- Chemical injury
- Marginal corneal ulcer
- Injury or Surgery

Q.30. What are the usual symptoms of pterygium?

Ans. Irritation, dimness of vision.

Q.31. What are the causes of dimness of vision in pterygium?

Ans.

- Corneal astigmatism
- Obstruction of pupillary area



Q.32. When a pterygium should be treated?

- Ans.**
- If it is progressive
 - Corneal astigmatism
 - Cosmetic

Q.33. What are the different treatment options?

- Ans.**
- Excision of pterygium and the bare sclera is covered with **limbal conjunctival autograft**. (Currently the treatment of choice)
 - **Bare sclera technique** - Simple excision of pterygium with conjunctiva, and keeping the limbus and adjacent sclera bare
 - **Transplantation operation** (Mc Reynold)
 - **Subconjunctival dissection** and excision

Q.34. What is the treatment of *recurrent pterygium*?

- Ans.**
- The bare sclera is covered with limbal conjunctival autograft
 - Bare sclera is treated with beta irradiation
 - Thiotepe solution as drops four times daily for six weeks
 - Mitomycin C (0.02%) locally during operation or post operatively as drops
 - Lamellar keratoplasty of the affected area of the cornea
 - Excision with graft by using amniotic membrane

Q.35. If it involves pupillary area, what is the treatment?

- Ans.** Resection with key hole lamellar keratoplasty (LK) or large LK with conjunctival autograft

Q.36. What are the indications of *pterygium operation*?

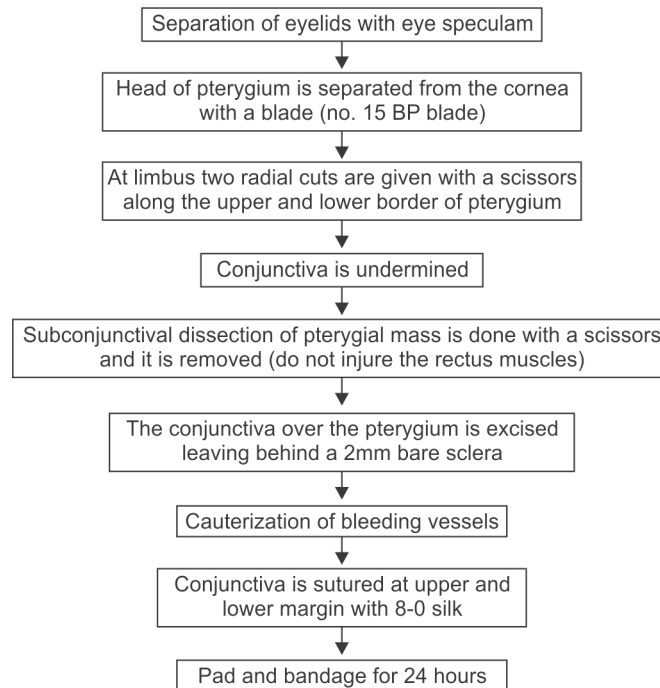
- Ans.**
- Pterygial growth encroaching the visual axis
 - Reduced vision due to astigmatism
 - Severe irritation poorly controlled with medicine
 - Reduced ocular motility
 - Cosmesis

**Q.37. Describe the steps of pterygium surgery.****Ans. Anaesthesia**

- Surface anaesthesia (topical 4% lignocaine hydrochloride/0.5% tetracain)
- Infiltration anaesthesia (2% lignocaine hydrochloride)
- Peribulbar anaesthesia (as in cataract surgery)

Steps of surgery

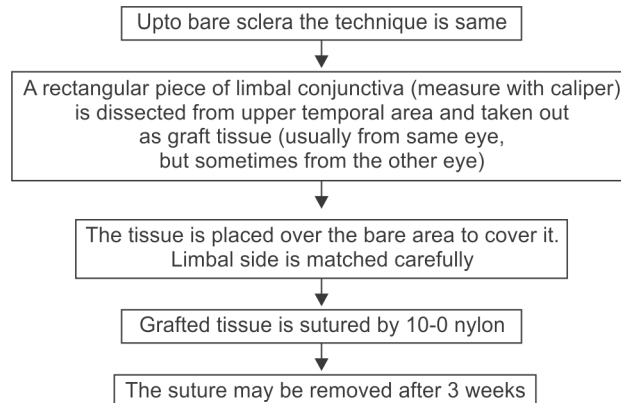
[Bare sclera technique (**D'ombrian's technique**)]





Q.38. Describe the pterygium excision with limbal autograft.

Ans.



Q.39. Which one of the above two surgery is better and why?

Ans. Excision with limbal autograft. It has shown decreased incidence of recurrence by 50%.

Q.40. What are the complications of pterygium surgery?

Ans.

- Recurrence
- Loss of graft
- Excess cautery → thinning of cornea
- Massive bleeding
- Medial rectus injury → divergent squint

CONJUNCTIVITIS (SHORT CASE)

Q.41. Define acute conjunctivitis.

Ans. Conjunctivitis which resolves in 4 weeks is termed as acute conjunctivitis.

Q.42. Define chronic conjunctivitis.

Ans. Conjunctivitis of more than 4 weeks duration is defined as chronic conjunctivitis.

Q.43. What are the clinical features of acute mucopurulent conjunctivitis?

Ans. Symptoms:

- Usually bilateral



- Redness of the eye
- Mucopurulent discharge
- Grittiness or F.B sensation
- Stickiness of the eyelids
- Coloured halos and photophobia

Signs:

- Lid oedema
- Matting of the eyelids
- Crusts at the lid margins
- Conjunctival congestion and chemosis
- Discharge or flakes of mucopus
- Subconjunctival haemorrhage (may be)

Q.44. How will you manage the above condition?

- Ans.**
- Frequent wash with luke warm saline solution
 - Use dark glasses to prevent photophobia
 - Cleaning of discharge
 - Broad spectrum antibiotic eye drop. (4-6 times/day for 2 weeks) (e.g. Ciprofloxacin, Ofloxacin, Moxifloxacin, Chloramphenicol)
 - An antibiotic eye ointment at bed time to prevent gluing of the eyelashes at morning

Q.45. What are the Clinical features of angular conjunctivitis?**Ans. Symptoms:**

- Itching and excoriation of the skin at the outer and inner angle of eyes
- Discomfort, with slight mucopurulent discharge
- Frequent blinking

Signs:

- Congestion of the conjunctiva, limited to inter marginal strip, especially at the outer and inner canthi and neighboring bulbar conjunctiva.
- Excoriation of skin around inner and outer canthus.

Q.46. What are the causative agents of angular conjunctivitis?

- Ans.**
- Typically by the **Moraxella lacunata** (they produce a proteolytic ferment which acts by macerating the epithelium)
 - Gram negative diplo-bacillus
 - Sometimes by Staphylococcus



Q.47. What is the treatment of angular conjunctivitis?

Ans.

- Tetracycline 1% eye ointment 3–4 times daily
- Zinc oxide eye drop 4–6 times daily (it inhibits the proteolytic ferment)

Q.48. What are the common viruses causing viral conjunctivitis?

Ans.

- Adeno virus
- Picorna virus
- Herpes simplex virus
- Herpes Zoster virus

Q.49. Name the conjunctivitis caused by adeno virus

Ans. Epidemic Kerato Conjunctivitis (EKC)

Q.50. Which serotypes of adeno virus causes EKC?

Ans. Sero types 8, 19 (some times 3, 7)

Q.51. What are the clinical features of EKC?

Ans.

- Marked inflammatory symptoms
- Follicular reactions
- Scanty Exudates
- Pseudomembrane (occasionally)
- SPK (7-10 days after infection)
- Pre auricular adenopathy

Q.52. How will you treat EKC?

Ans.

- Non specific
- Anti histaminics
- Lubricants
- Antibiotics to prevent secondary bacterial infection.

(Specific antiviral is not necessary)

Q.53. What is haemorrhagic conjunctivitis?

Ans. Acute haemorrhagic conjunctivitis is characterized by conjunctival hyperemia, multiple conjunctival haemorages and mild follicular hyperplasia caused by picorna virus.

Q.54. What is Inclusion Conjunctivitis?

Ans. It is an acute mucopurulent conjunctivitis caused by Chlamydia (serotype D,K).

Two types:

- **Neonatal type:** Causes ophthalmia neonatorum



- **Adult type:** Adult type occurs secondary to urethritis in male and cervicitis in female through contaminated fingers.

Q.55. Define trachoma?

Ans. It is a chronic keratoconjunctivitis caused by chlamydial infection (*Chlamydia trachomatis* serotype A,B,C). It is characterized by follicles on upper tarsal conjunctiva.

Q.56. What is the WHO classification of trachoma?

Ans.

- TF - Trachoma follicle > 5 on upper tarsal conjunctiva
 - TI - Trachoma inflammation
 - TS -Trachoma scarring
- TT - Trachoma trichiasis
- CO - Corneal opacity (FISTO)

Q.57. What is the treatment of trachoma?

Ans. Erythromycin/Tetracycline/Sulfonamides.
 For trachoma control programme these are given for 5 consecutive days a month for 12 months.
 Oral Tetracycline/Erythromycin -250-500 mg 4 times a day
 Or
 Doxycycline- 100 mg BD -3-6 wks
 Or
 Azithromycin- 1 gm single dose
 Treatment of secondary infection should be done.

General Measures

Avoid kajal/surma.
 Avoid person to person contact.
 Improvement of personal hygiene.

Q.58. What are the sequelae of trachoma if not treated?

Ans.

- Trichiasis
- Entropion
- Tylosis
- Xerosis
- Ptosis



Q.59. What is blanket treatment?

Ans. Mass treatment given in endemic areas of trachoma.

Indication -prevalence of >5% severe and moderate trachoma in children <10 yrs.

Application of 1% tetracycline ointment BD for 5 consecutive days each month or once daily for 10 days each month for 6 consecutive months.

Q.60. What is pannus?

Ans. Infiltration of the cornea beneath the epithelium with neovascularisation and round cells is called pannus.

Q.61. What are the types of pannus?

- Ans.**
- *Progressive:* Cellular infiltration extends beyond the neovascularisation.
 - *Regressive:* Infiltration stops short of the vessels.

Q.62. Name one condition where you will get pannus?

Ans. Trachoma

Q.63. What is ophthalmia neonatorum(neonatal conjunctivitis)?

Ans. It is defined as mucoid, mucopurulent or purulent discharge from one or both eyes in the 1st month of life due to maternal infection acquired at the time of birth.

Q.64. What are the causative agent of Ophthalmia neonatorum?

Ans. N gonorrhoea
Chlamydia oculogenitalis
Strept. pneumoniae
Gram negative coliform
Herpes simplex virus
Chemical causes (silver nitrate)

Q.65. What are the C/F of ophthalmia neonatorum?

- Ans.**
- Purulent/mucopurulent discharge
 - Conj- hyperemic, chemosed
 - Lids- swollen
 - Pseudomembrane

**Q.66. How will you manage ophthalmia neonatorum?****Ans.**

<i>Time of onset after birth</i>	<i>Differential diagnosis</i>	<i>Treatment</i>
Within the first 48 hours	Neisseria gonorrhoeae	Ceftriaxone injection I/M gentamicin drops, bacitracin eye ointment
	Chemical	Wash eyes, erythromycin ointment, observe, usually improves in 24-48 hours.
48-72 hours	Other bacteria	Neomycin-bacitracin eye ointment, gentamicin or tobramycin drops.
5-7 days	Herpes simplex virus (HSV II)	Acyclovir 3% eye ointment, systemic acyclovir for systemic involvement in consultation with a pediatrician.
>1 week	Chlamydia trachomatis	Erythromycin or chlortetracycline eye ointment, oral erythromycin for systemic infection.

Q.67. What is Crede's method?

Ans. Instillation of 1% silver nitrate solution in baby's eye after birth (obsolete).

(Any discharge from a baby's eyes during the 1st wk should be viewed with suspicion, since **tears are not secreted so early in life.**

Q.68. What are the D/D of discharge from a child eyes within the 1st month of life?

Ans.

- Ophthalmia neonatorum
- Congenital NLD block
- Congenital glaucoma
- Keratitis

Q.69. What are the tumours seen in the conjunctiva?

Ans.

- Dermoids
- Dermolipoma



- Papilloma
- Squamous cell carcinoma
- Lymphoma
- Kaposi sarcoma
- Malignant melanoma

Q.70. What is Kaposi sarcoma?

Ans. It is a malignant tumour seen in AIDS patient. It is highly vascular and bluish red in colour; looks like a subconjunctival haemorrhage.

Q.71. What is Xerophthalmia?

Ans. The characteristic ocular manifestations of Vit-A deficiency ranging from night blindness to corneal melting are termed as 'Xerophthalmia' or dry eye.

Q.72. What is the WHO classification of Xerophthalmia?

Ans. XN = Night blindness
X1A = Conjunctival xerosis
X1B = Bitot's spot
X2 = Corneal xerosis
X3A = Corneal ulceration < 1/3 of corneal surface
X3B = corneal ulceration > 1/3 of corneal surface
XS = corneal scar
XF = Xerophthalmic fundus

Q.73. What is Bitot's spot?

Ans. It is a small triangular, white patch on the outer (inner rarely) side of the cornea. It is covered by a material resembling dried foam which is not wet by the tears. This is an early stage of Xerophthalmia (vit-A deficiency).

Q.74. What is the direction of the triangle?

Ans. The base of the triangle is towards the limbus.

Q.75. What is the cause of the foamy appearance of the Bitot's spot?

Ans. Due to gas production by *Corynebacterium xerosis* in the horny epithelium.



Q.76. What is *Nyctalopia*?

Ans. Difficulty in night vision

Q.77. What is *haemarolopia*?

Ans. Day blindness

Q.78. Name one condition where we find day blindness.

Ans. Cone dystrophy

Q.79. What are the clinical features of xerophthalmia?

- Ans.**
- **Conjunctiva**
 - Dry lustureless
 - Muddy discolouration
 - Wrinkling of bulbar conjunctiva
 - Bitot's spot
 - **Cornea**
Dull, lustureless

Q.80. What are the different types of xerosis?

- Ans.**
- a. Xerosis epitheliasis - Due to vitamin A deficiency and associated with protein energy malnutrition.
 - b. Xerosis parenchymatas - Sequale of ocular inflammation
 - Trachoma
 - Chemical injury
 - Diphtheria
 - Ocular pemphigoid

Q.81. What are the causes of conjunctival xerosis?

- Ans.**
- Vit A deficiency
 - Syestemic conditions
 - Stevens johnson'ssyndrome
 - Sjogren syndrome
 - Exposure
 - Lagophthalmos
 - Proptosis
 - Ectropion

Q.82. How will you *treat xerophthalmia*?

- Ans.**
- 2 lakh IU vit. A orally on
1st day
2nd day
Within 1-4 Weeks
(Children < 1 year -1/2 of the above dose).
(If diarrhoea present I/M 1 lakh IU in > 1 yr in same
schedule)



- Eye care
- Broad spectrum antibiotic drops/ointment -4 times a day
- Artificial tears
- Atropine 1% eye ointment
- General health
- Protein rich food
- Treatment of concurrent illness- URI, diarrhea

Q.83. What is rhodopsin?

Ans. It is a photosensitive pigment present in rods and help in night vision.

Q.84. What is the name of the pigment present in cone cells?

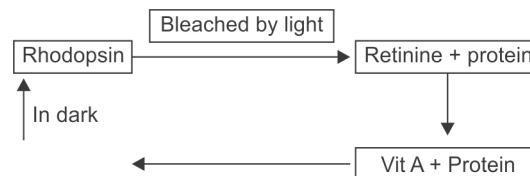
Ans. Iodopsin- Helps in day vision and colour vision.

Q.85. What is the name of the cycle which regenerates rhodopsin?

Ans. Wald's cycle

Q.86. What are the steps of wald's cycle?

Ans.



Q.87. What are the complications of keratomalacia?

Ans. Corneal ulcer → sloughing of ulcer → iris prolapsed → anterior staphyloma → secondary glaucoma → blindness.

Q.88. What is ophthalmia nodosa?

Ans.

- Irritation caused by hair of certain caterpillars produce it.
- Conjunctivitis characterized by formation of nodules.
- Yellowish grey nodules are found in conjunctiva, cornea, iris.

VERNAL CONJUNCTIVITIS (SHORT CASE)

Q.89. What is your case?

Ans. A case of vernal conjunctivitis.(mention age and sex of the patient)



Q.90. What are the points in favour of your diagnosis?

Ans. C/O - (unilateral/bilateral)

- Itching, burning , redness
- Ropy discharge
- Photophobia (may or may not).

H/O - Similar attack in past during spring/summer.

O/E -

- Conjunctiva - Congested.
- Bulbar type-Congestion of bulbar conjunctiva, circumcorneal gelatinous thickening .
- Palpebral type - Papillae in the tarsal conjunctiva.
- Mixed form -Features of both palpebral and bulbar forms.
- Family history of atopy (may be).

Q.91. What is vernal conjunctivitis?

Ans. This is a recurrent bilateral allergic conjunctivitis which occurs in summer or spring.

Q.92. Who are affected usually ?

Ans. Young boys

Q.93. What is the causative agent?

- Ans.**
- Pollen
 - Other atmospheric exogenous allergen

Q.94. What type of reaction it is?

Ans. Type IV hypersensitivity reaction.

Q.95. What are the different types of vernal conjunctivitis?

- Ans.**
- Bulbar
 - Palpebral
 - Mixed

Q.96. What are the features of palpebral form?

Ans. On everting the upper lid flat-topped papillae on the palpebral conjunctiva (papillae resembling cobblestone appearance with milky hue may be seen).

Q.97. What are the features of bulbar form?

- Ans.**
- Congestion of bulbar conjunctiva.
 - Gelatinous hypertrophy of limbal conjunctiva (either discrete or confluent).
 - Discrete white raised dots along the limbus (Horner-trantas spots).



Q.98. What is the differential diagnosis of palpebral variety?

- Ans.**
- Trachoma (non seasonal)
 - Giant papillary conjunctivitis (contact lens users).

Q.99. What is *Horner trantas spots*?

- Ans.** Found in limbal or bulbar form. These are white dots consisting of eosinophils and epithelial debris seen at the limbus.

Q.100. Does it involve the cornea?

- Ans.**
- Yes, in both the types there may be a diffuse superficial punctate keratitis.
 - Corneal ulcer - In severe case known as shield ulcer.
 - Presence of pseudoxerontoxon which looks like arcus senilis (in bulbar form).

Q.101. What is the treatment of vernal conjunctivitis?

- Ans.**
- Purely symptomatic.
 - Cold compress to reduce inflammation.
 - Antihistaminic eye drops (4-6 hourly).
 - Steroid eye drops (tapered gradually).
 - When the acute condition subsides - mast cell stabilizers, e.g. Di sodium cromoglycate 2-4% or Olopatadine 0.1% BD dose (dual action).

Q.102. What are the risk of long term use of steroid?

- Ans.**
- Steroid induced glaucoma (topical use).
 - Corneal infection
 - Cataract (more in systemic use).

Q.103. What is the treatment of cobble stone?

- Ans.**
- Subtarsal injection of Triamcinolone.
 - Cryotherapy.

Q.104. What is the prognosis of vernal conjunctivitis?

- Ans.** Usually self limiting. Symptoms disappear as the child reaches at puberty. But in some cases it can persist .

PHLYCTENULAR CONJUNCTIVITIS (SHORT CASE)

Q.105. What is your case?

- Ans.** A case of phlyctenular conjunctivitis (mention age and sex).



Q.106. What are the points in favour of your diagnosis?

Ans. C/O

- Irritation
- Redness
- Photophobia

C/E

Conjunctiva - (Fig. 3.3)

- One or more small, round ,grey or yellow nodules at limbus
- Congestion of vessels around the nodules
- Rest of conjunctiva is normal
- Multiple phlycten around the limbus (may be present)
- Associated blepharitis (may be)

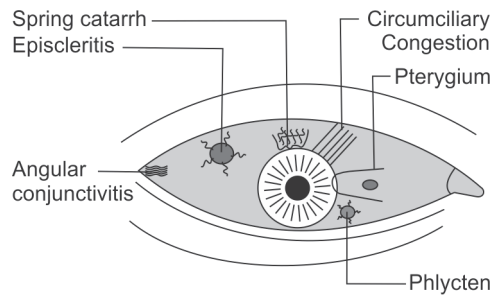


Fig. 3.3 : Various sites of localised conjunctival congestion

Cornea:

Similar nodules may be seen near the limbus.

Q.107. What is phlyctenular keratoconjunctivitis?

Ans. It is an allergic conjunctivitis characterized by formation of phlycten, where both the conjunctiva and adjacent cornea is involved.

Q.108. What is phlycten?



Ans. An endogenous allergic reaction of conjunctiva characterized by bleb formation.(Fig 3.4)

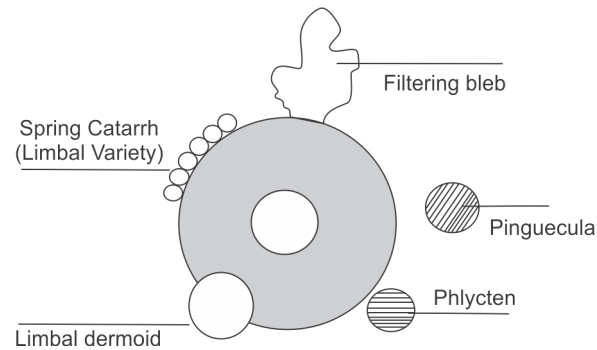


Fig. 3.4 : Nodule at and near the limbus

Q.109. What is the etiology of phlyctenular conjunctivitis?

Ans. Allergic reaction caused by endogenous bacterial proteins, e.g. tubercular, chronic tonsillitis or adenoid infection.

Q.110. Which age group is commonly affected?

Ans. 5-15 years.

Q.111. What is the pathogenesis?

Ans. It is a delayed type of hypersensitivity (type IV) reaction. Nodule formation (infiltration of lymphocytes) → Ulceration of nodules → Granulation tissue formation → Scarring.

Q.112. What are the differential diagnosis (D/D) of phlycten?

Ans.

- Nodular episcleritis/scleritis
- Foreign body granuloma at limbus
- Inflamed pinguecula (elderly)

Q.113. What is the management of phlyctenular conjunctivitis?

Ans. Investigaton:

TC, DC, ESR

X-Ray chest

Mantoux test

Neck glands evaluation (To exclude Tuberculosis)

Local:



- Steroid drops/ointment
- If cornea is involved - antibiotic + cycloplegics drops
- Dark glass

Systemic - treatment of underlying cause.

EPISCLERITIS (SHORT CASE)

Q.114. What is your case?

Ans. A case of episcleritis in Right eye/Left eye.

Q.115. What are the points in favour of your diagnosis?

Ans. C/O

- Irritation, F.B sensation
- Redness of eye
- H/O similar episodes in past(may be)

O/E of Conjunctiva and sclera-

- No ocular swelling
- Reddish pink in colour
- Size like lentil
- Nodule fixed to underlying structures
- Conjunctival congestion over the nodule

Q.116. What is episcleritis?

Ans. Inflammation of deep subconjunctival tissue which somehow involves superficial lamella of sclera.

Q.117. What are the D/D of episcleritis?

Ans. • Phlyctenular conjunctivitis
• Inflamed pinguecula

Q.118. What are the other conditions which can be seen with episcleritis?

Ans. • Uveitis
• Scleritis

Q.119. What is the fate of the above nodule?

Ans. Complete absorption (Sometimes can take a long time).

Q.120. What is scleritis?

Ans. Inflammation of sclera proper



Q.121. What are the systemic conditions which are known to cause episcleritis/scleritis?

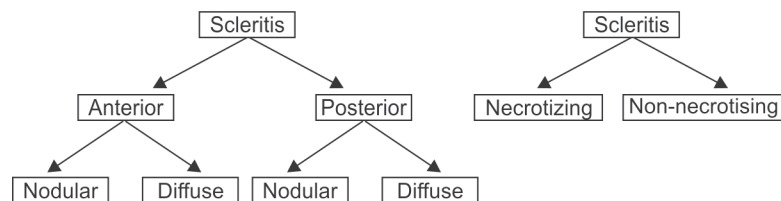
- Ans.**
- Idiopathic
 - Autoimmune collagen disease
 - Rheumatoid arthritis
 - SLE
 - Ankylosing spondylitis
 - Polyarteritis nodosa
 - Metabolic disorder
 - e.g. T.B
 - Leprosy

Q.122. What are the complications of scleritis?

- Ans.**
- Sclerosing Keratitis
 - Uveitis
 - Complicated cataract
 - Scleral necrosis → ciliary staphyloma

Q.123. What are the different types of scleritis?

Ans.



Q.124. What are the investigations needed?

- Ans.** TC, DC, ESR, Hb%
Rh factor
ANA
Mantoux test
Chert X-Ray

Q.125. What is dry eye ?

- Ans.** Deficiency of tears or instability of tear film causes dry eye syndrome which is a leading cause of ocular discomfort.

Q.126. What are the causes of dry eye?

- Ans.**
- Aqueous tear deficiency (keratoconjunctivitis sicca)



- Mucin deficiency (goblet cell damage): e.g. xerophthalmia, SJS, chemical burn, trachoma
- Lipid deficiency (chronic blepharitis)
- Impaired lid function e.g. exposure keratitis, dellen
- Epitheliopathies (irregular corneal surface)

Q.127. What are the C/F of dry eye?

Ans. • Symptoms:

- Ocular discomfort
- FB sensation
- Heaviness of the lids
- Burning sensation

• Signs:

- Lusterless dry ocular surface
- Conjunctival xerosis
- Presence of stringy mucous
- Reduced or absent marginal tear strip
- Severe case: punctate epithelial erosion and filaments

Q.128. What are the diagnostic tests for dry eye?

- Ans. •**
- Schirmer test – done ... > 15 mm
 - Tear film break-up time (BUT) : normal value 15-35 sec , <10 sec is abnormal
 - Rose Bengal staining
done with a 5x35 mm strip of Whatman - 41 filter paper ; normal: >15 mm

Q.129. How will you manage a case of dry eye?

- Ans. •**
- Supplementation with tear substitute e.g. Methyl cellulose
 - Preservation of existing tears
 - Reducing evaporation (decreasing room temperature, protective glass)
 - Punctal occlusion with collagen implant

Q.130. What one systemic disease is associated with dry eye?

Ans. Sjogren's syndrome

Chapter

4

Cornea

Q.1. What are the layers of cornea?

Ans. (From anterior to posterior) (Fig 4.1)

- Epithelium
- Bowman's membrane(BM)
- Corneal stroma
- Descemet's membrane(DM)
- Endothelium

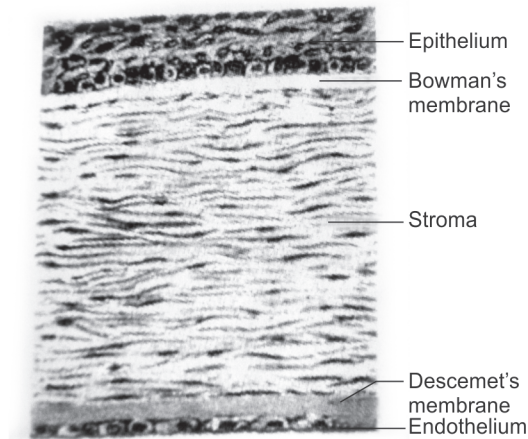


Fig.4.1 : Microscopic structure of Cornea

Q.2. Describe briefly the structures of cornea?

Ans. Epithelium

- Stratified epithelium
- Five to six layers
- Continuation of conjunctiva over cornea
- Basal cells lie on a lamina of substantia propria (BM)

**Bowman's membrane**

Once eroded never regenerate → leads to scarring.

Stroma

- Turns 90% of the total corneal thickness
- Composed of regularly arranged collagen

Descemet's Membrane

- Thin elastic membrane, covered on its posterior surface
- Regenerates after an injury

Endothelium

- Single layered, flattened cells
- Never regenerates after injury/loss

Q.3. What is the nerve supply of the cornea?

Ans. Ophthalmic division of 5th cranial nerve via nasociliary branch.

Q.4. How does cornea get its nutrition?

Ans.

- O₂ from atmosphere via tear film
- Perilimbal plexus of the blood vessels
- Aqueous humour

Q.5. How does corneal transparency is maintained?

Ans.

- Regular arrangement of the stromal collagen fibrils
- Absence of blood vessels
- Absence of myelinated nerve fibers
- Active transport of fluid outwards (maintained by endothelial Na-K-ATPase pump system)
- Optimum IOP to control fluid transport

Q.6. Which layer is most important to maintain corneal transparency?

Ans. Corneal endothelium

Q.7. How can you examine corneal endothelium?

Ans.

- Slit lamp biomicroscopy
- Specular microscopy



Q.8. What is the average endothelial cell count in young adult?

Ans. 2500-2800 cells/mm²

Q.9. What are the conditions where there is decrease endothelial cell count?

Ans.

- With aging
- Injury
- Corneal dystrophy
- Intra ocular surgery

Q.10. What is the normal shape of the cornea?

Ans. Spherical

Q.11. What is keratoconus?

Ans. Inherent weakness of cornea → conical cornea

Q.12. From which primitive tissue does cornea develops?

Ans.

- Corneal epithelium → Surface ectoderm
- Corneal Stroma → Mesoderm
- Endothelium and Keratocytes → Neural crest

Q.13. What is the diameter of the cornea?

Ans.

- Horizontal – 12 mm
- Vertical – 11 mm

Q.14. What is the radius of curvature of cornea?

Ans.

- Anterior surface – 7.8 mm
- Posterior surface – 6.5mm

Q.15. What is the thickness of cornea?

Ans.

- Central -0.52 mm
- Peripheral -0.70 mm

Q.16. What is microcornea?

Ans.

- Horizontal diameter < 10 mm
- Causes-microphthalmias, Phthisis bulbi

Q.17. What is megalocornea?

Ans.

- Horizontal diameter >13 mm
- Causes – Buphthalmos, hereditary

Q.18. What is arcus senilis?

Ans. It is an age related degeneration of cornea, caused by lipid infiltration of corneal periphery, characterized by whitish ring just inside the limbus. (Arcus juvenalis - similar change can occur in young).

**Q.19. What is BSK?**

Ans. Band shaped keratopathy. It is a degenerative condition due to deposition of calcium salts in Bowman's membrane and superficial part of the stroma.

Q.20. What are the causes of BSK?

- Ans.**
- Chronic uveitis
 - Still's disease in children
 - Phthisis bulbi
 - Chronic glaucoma
 - Ocular trauma

Q.21. What is the position of BSK?

Ans. Interpalpebral zone of cornea.

Q.22. What is the treatment of BSK?

- Ans.**
- Chelation with EDTA after removal of epithelium
 - Keratoplasty

Q.23. How will you test for corneal sensation?

- Ans.**
- Ask the patient to look straight
 - Wisp of cotton is brought from side (to avoid blinking response)
 - Touch it 2 mm inside the limbus
 - Look for blink reflex

Q.24. What are the causes of diminished sensation of cornea?

- Ans.**
- Herpes
 - Leprosy
 - Neuroparalytic keratitis
 - Absolute glaucoma

Q.25. What is Kayser-Fleisher ring?

Ans. Deposition of copper at the Descemet's membrane (seen in Wilson's disease and intraocular Copper foreign body).

Q.26. What are the conditions where transparency of cornea is lost?

- Ans.**
- Oedema
 - Opacity
 - Ulceration
 - Dystrophy



Q.27. What are the dyes used for staining cornea and bulbar conjunctiva?

- Ans.**
- Fluorescein 2% solution/strip (FL)-It stains damaged corneal epithelium (epithelial defect) bright green, best seen under cobalt blue and ultraviolet light.
 - Rose Bengal-Stains the devitalized corneal and conjunctival epithelium.

Q.28. In which condition FL dye is used?

- Ans.**
- To delineate corneal abrasions or corneal ulcer
 - Applanation tonometry
 - Siedel's test

Q.29. What is pachymetry?

Ans. Measurement of corneal thickness.

Q.30. What are the different types of pachymetry?

- Ans.**
- Optical
 - Ultrasonic

Q.31. What are the causes of thick cornea?

- Ans.**
- Corneal oedema
 - Corneal leucoma

Q.32. What are the causes of thin cornea?

- Ans.**
- Keratoconus
 - Buphthalmos

Q.33. What are the causes of corneal vascularisation?

- Ans.** (Normal cornea is avascular except peripheral 1 mm)
- Trachoma
 - Phlycten
 - Interstitial keratitis
 - Deficiency of Riboflavin
 - Corneal foreign body
 - Long standing corneal ulcer

CORNEAL ULCER (LONG CASE)

Q.34. What is your case?

Ans. A case of corneal ulcer with or without hypopyon of RE/LE (mention age and sex).



Q.35. What are the points in favour of your diagnosis?

Ans. Symptoms:

- Pain
- Watering
- Intolerance to light
- Dimness of vision

Signs:

- Diminished vision
- Lid edema (may or may not)
- Blepharospasm
- Discharge- purulent/serous
- Congestion- conjunctival/ciliary
- Cornea-hazy, ulcer area surrounded by infiltration
- Satellite lesion (if any)
- AC- normal/hypopyon
- Iris- iritis (may or may not be present)

Q.36. Define corneal ulcer.

Ans. It is a defect in the normal epithelial surface of cornea associated with necrosis of surrounding corneal tissue along with edema and cellular infiltration.

Q.37. What are the predisposing factors for corneal ulcer?

Ans. (*damage of the corneal epithelium is a pre-requisite for most of the organism to produce ulcer*).

- Trauma to corneal epithelium (FB, trichiasis)
- Underlying corneal diseases:
 - Bullous keratopathy
 - Punctuate keratitis
 - Keratomalacia
 - Dacryocystitis
 - Dry eyes
 - Exposure keratopathy
 - Long tem use of topical steroid
 - Contact lens wearer
 - Immunocompromised subject

Q.38. Classify keratitis etiologically.

Ans. Infective-

- Bacterial
- Fungal
- Viral
- Chlamydial
- Protozoal



Non-infective:

- Allergic:
 - Phlyctenular
 - Vernal
 - Atopic
- Trophic keratitis:
 - Central -
 - Exposure keratitis
 - Atheromatous
 - Keratomalacia
 - Peripheral:
 - Marginal
 - Mooren ulcer
 - Phlyctenular
 - Associated with collagen disorder

Q.39. Which bacteria can invade intact corneal epithelium?

- Ans.**
- Neisseria gonorrhea
 - Neisseria meningitidis
 - Corynebacterium diphtheria

Q.40. What are the stages of corneal ulcer?

Ans. Stage of infiltration

- Infiltration of PMNL/Lymphocytes into the epithelium and stroma

Stage of progression

- Necrosis and sloughing of the necrotic material
- Surrounding area is packed with leucocytes. Wall of the ulcer projects due to edema and infiltration of cells
- Zone of infiltration extends beyond and underneath the ulcer margin
- Ciliary congestion
- Involvement of iris and ciliary body (due to absorption of toxin)–causing iritis/cyclitis
- Hypopyon formation

Stage of regression

- Induced by immunodefence mechanism and treatment.
- Line of demarcation develops around the ulcer area.



- Necrotic material is sloughed off and the ulcer bed enlarges.
- As the surrounding infiltration and swelling disappears, the floor and edges become smooth and transparent.
- Superficial vessels grow in from the limbus near ulcer, to restore the loss of substance and supply antibody.

Stage of cicatrization

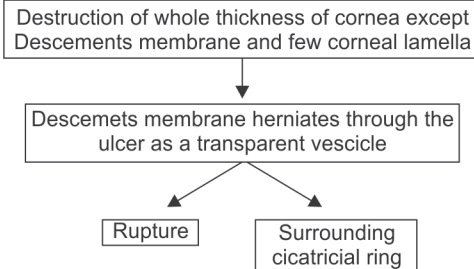
- Vascularisation of ulcer
↓
- Regeneration of collagen and formation of fibrous tissue (causes corneal opacity)

Q.41. What are the complications of corneal ulcer?

- Ans.
- Iridocyclitis
 - Secondary glaucoma
 - Ectatic cicatrix - thinning of cornea → bulging of the scar (IOP may be normal)
 - Descematocele
 - Perforation

Q.42. What is descemetocele?

Ans.



Q.43. What will happen if a corneal ulcer perforates?

Ans. (Fig 4.2)

It varies according to the location and size of the perforation.

- Anterior synechiae
- Iris prolapse → adherent leucoma
- Total iris prolapse → pseudocornea → anterior staphyloma
- Phthisis bulbi



- Perforation → subluxation/dislocation of lens → expulsive haemorrhage
- Endophthalmitis
- Panophthalmitis

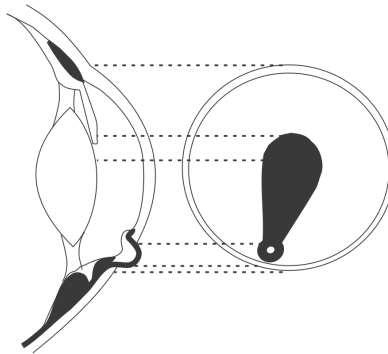


Fig.4.2 : Showing a longitudinal section of a perforated corneal ulcer with iris prolapse.

Q.44. What is hypopyon corneal ulcer?

Ans. An infective purulent corneal ulcer associated with collection of pus in the AC.

Q.45. What are the features of ulcer serpens (hypopyon corneal ulcer)?

Ans. (Fig. 4.3)

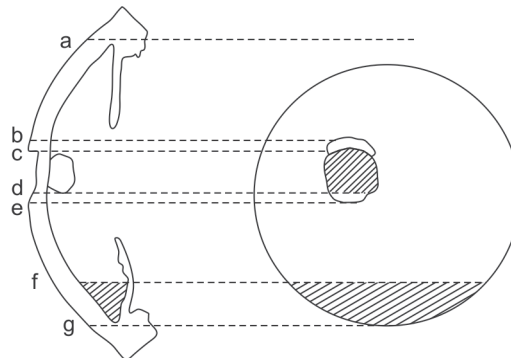


Fig.4.3 : Hypopyon corneal ulcer.

c - e extent of ulcer

b - c actively infiltrated border

c - d leucocytes and fibrin adherent to endothelial surface

f - g hypopyon



- It is caused by pneumococcus
- Characterized by hypopyon ulcer
- The ulcer creeps over the cornea in a serpiginous fashion
- It is associated with violent iridocyclitis

Q.46. What are the causes of corneal ulcer with hypopyon?

- Ans.**
- Pneumococcus (commonest)
 - Pseudomonas pyocyane
 - Gonococcus, staphylococcus, streptococcus
 - Mycotic hypopyon ulcer

Q.47. What are the signs of impending perforation?

- Ans.**
- Descematocele formation
 - Thinning of cornea in progressive stage

Q.48. What are the features of perforation of corneal ulcer?

- Ans.**
- Sudden relief of pain
 - Sudden gush of hot fluid coming out of the eye
 - Shallow AC with iris prolapse

Q.49. What are the advantages of perforation?

- Ans.**
- Decreased IOP
 - Decreased pain
 - Rapid healing of ulcer, specially when iris tissue comes forward and seals the perforation
 - Improve diffusion of fluid and better nutrition
 - More antibody /antibiotic reaches the ulcer site

Q.50. How will you diagnose whether it is a bacterial /viral/ fungal corneal ulcer clinically?

Ans. Clinical features of bacterial ulcer :

- Symptoms are proportionate to the signs
- Mucopurulent/Purulent discharge
- Wet looking ulcer
- Surrounding area is edematous
- Mobile hypopyon
- Rapid progression of ulcer

Clinical features of fungal ulcer :

Symptoms are less than signs.

- History of trauma with vegetative matters
- Dry/leathery appearance
- Raised surface



- Feathery margin of infiltrates
- Satellite lesions
- Fixed hypopyon
- Endothelial plaque
- Slowly progressive ulcer

Clinical features of viral corneal ulcer :

- Symptoms are more than signs
- Serous discharge
- Usually superficial ulcer with less infiltration
- No hypopyon
- Decreased corneal sensation
- Slow progression of ulcer

Q.51. What are the clinical feature of Acanthamoebal corneal ulcer?

- Ans.**
- History of contact lens wear /bathing in swimming pool/ pond
 - Ulcer not responding to conventional antimicrobial treatment
 - Severe pain (due to radial keratoneuritis)
 - Ring shaped lesion
 - Chronic indolent ulcer

Q.52. How will you manage corneal ulcer?

- Ans.**
- **Slit lamp** examination to diagnose clinically the causative agent
 - Lacrimal sac examination-syringing (to rule out chronic dacryocystitis)
 - General examination to rule out any systemic problem.

Management of corneal ulcer :

- S/L exam : Site, size, depth of ulcer
- Blood for TC, DC, sugar
- Scraping
 - Smear
 - Gram stain
 - KOH
 - Culture
 - Bacterial (Blood agar/ Glucose broth)
 - Fungal (Sabouraud's media)



- Suitable topical antibiotic at frequent interval (1 hourly), e.g.- ciprofloxacin, tobramycin, gatifloxacin.
- Topical fortified (concentrated) preparation- cefazoline/ tobramycin etc.
- Subconjunctival injection of antibiotic and atropine.
- Atropine drop 1% 3 times daily- to rest ciliary body and control iritis.
- Scraping of ulcer bed at frequent interval for better antibiotic penetration.
- Carbolic cautery (if not responding to any medication).

CORNEAL OPACITY (LONG/SHORT CASE)

Q.53. What is your case?

Ans. Corneal opacity RE/LE – Nebula/macula/leucoma/adherent leucoma(mention age and sex).

Q.54. What are the different types of corneal opacity?

Ans. (Fig 4.4)

- Nebula
- Macula
- Leucoma
- Adherent leucoma
- Mixed

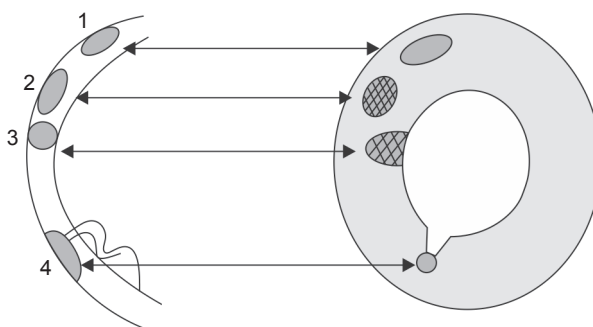


Fig.4.4 : Grades of corneal opacity

1. Nebula - not deeper than Bowman's membrane
2. Macula - depth up to anterior stroma
3. Leucoma - depth up to posterior stroma
4. Adherent leucoma - an opacity in which iris is incarcerated.



Q.55. What are the ocular findings in favour of your diagnosis?

Ans.

- Dimness of vision
- Corneal opacity - type
- Corneal opacity - vascularised/ non-vascularised
- Mention - site/size/shape of opacity/pigmentation on opacity
- Iris adherence
- Corneal sensation - present/diminished
- Lacrimal sac - Regurgitation on pressure over sac - present/absent

Q.56. What is nebula?

Ans. Very faint corneal opacity. Epithelium and Bowman's membrane are involved.

Q.57. What is macula?

Ans. Denser than nebula. Underlying structures are hazily visible through it, corneal stroma is also involved.

Q.58. What is leucoma?

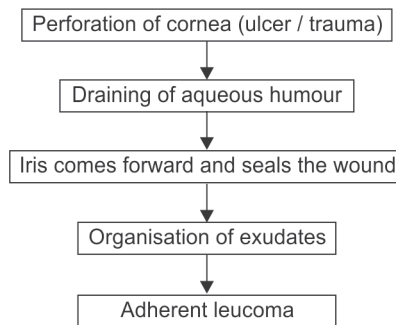
Ans. Most dense opacity. Underlying structures not visible. Deeper layers of stroma involved.

Q.59. What is adherent leucoma?

Ans. Iris tissue is incorporated with corneal scar.

Q.60. How is an adherent leucoma formed?

Ans.



Q.61. What is the complication of adherent leucoma?

Ans. Secondary glaucoma.

**Q.62. What are the causes of corneal opacity?**

Ans. (STUMPED)

S - Surgical scar

T - Traumatic

U - Ulcer

M - Metabolic (Mucopolysaccharidoses)

P - Peter's anomaly

E - Endothelial dystrophy

D - Degenerative

Q.63. Nebula or Leucoma - Which is better for the patient?

Ans. Nebula in pupillary region is more harmful for the patient than a small leucoma. As nebula causes irregular astigmatism, it can only be treated with contact lens. A small leucoma cuts only a few rays going inside the eye so clarity of vision is better.

Q.64. What are the treatment options for corneal opacity?

Ans. a. Nebula - Contact Lens (Irregular astigmatism).

b. Nebula or Leucoma -

i. Peripheral - Refraction only for optical reason, Tattooing for cosmetic reason.

ii. Central - If PL/PR present - Penetrating keratoplasty. Optical Iridectomy - If penetrating keratoplasty facility not available.

No PL - Tattooing (cosmetic)

Q.65. What is tattooing?

Ans. Colouring the cornea, with chemicals like gold, platinum, chloride with hydrazine hydrate.

Q.66. What is keratoplasty?

Ans. It is a surgical procedure in which diseased or scarred cornea is replaced by donor's healthy cornea.

Q.67. What are the different types of keratoplasty?

Ans. • PK – Penetrating keratoplasty (PK) (full thickness) (Fig. 4.5)

• LK – Lamellar keratoplasty (partial thickness corneal transplant)

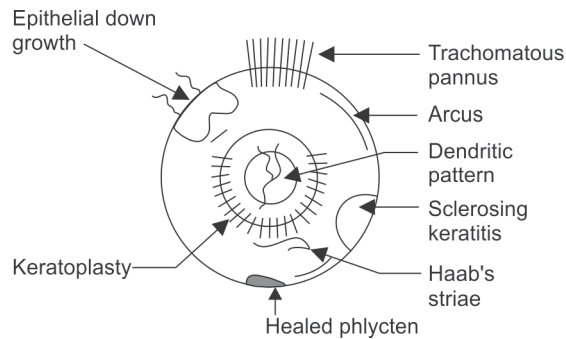


Fig.4.5 : Positions of various kinds of corneal opacities

Q.68. What are the indications of PK?

- Ans.**
- Optical-it is done for visual improvement, e.g.- ABK,PBK
 - Therapeutic-it is done in case of impending perforation due to inflamed/infected cornea
 - Tectonic-for supporting eye in thin or melting cornea
 - Cosmetic-where there is no hope of vision

Q.69. What is the commonest indication of PK ?

Ans. Leucoma/adherent leucoma.

Q.70. What are the common viruses which causes keratitis?

- Ans.**
- Adenovirus
 - Herpes simplex virus
 - Herpes Zoster virus

Q.71. What are the different types of keratitis caused by Herpes simplex virus?

- Ans.**
- Epithelial keratitis
 - Superficial punctuate keratitis(SPK)
 - Dendritic keratitis
 - Geographic ulcer

Q.72. How will you treat epithelial keratitis?

- Ans.**
- Acyclovir eye ointment- 5 times daily x 2 wks
 - Cycloplegic eye drop- atropine/pentol eye drop x 1 wk



Q.73. What are the ocular lesions caused by Herpes Zoster virus ?

Ans. Herpes Zoster Ophthalmicus.

Keratitis, conjunctivitis, iridocyclitis all can occur together.

Q.74. How will you treat the condition?

Ans.

- Same as HSV + Topical corticosteroid.
- Oral acyclovir tab - 800mg 5 times daily 10 days.

Skin lesion : Steroid antibiotic ointment, Lactocalamine lotion.

Stromal keratitis/Disciform keratitis: Atropine 1% eye ointment, acyclovir eye oint, Topical steroid drops/oint.

Q.75. What is exposure keratopathy? What are the causes of exposure keratopathy?

Ans. Non-infective condition of the cornea due to exposure (lagophthalmos). Inferior punctate erosions is the classical lesion.

Etiology

- Facial nerve palsy
- Severe proptosis
- Comatose patient

Q.76. What is the management of exposure keratopathy?

Ans. Treat the underlying cause :

- Short term- Artificial tears
- lubricating ointment at night
- Bandage contact lens
- Lid taping
- Long term- Tarsorrhaphy.

Q.77. What is neurotrophic keratopathy? How to manage the condition?

Ans. Occurs in anaesthetic cornea due to trigeminal nerve palsy. Treatment same as above.

Q.78. What are the causes of non-healing corneal ulcer ?

Ans.

- Poor body resistance
- Inappropriate antibiotic therapy
- Chronic dacryocystitis
- Trichiasis
- Diabetes melitis
- FB
- Raised IOP

Chapter

5

Lens

Q.1. What is the shape of the lens?

Ans. Biconvex, flatter anteriorly than posteriorly.
(At birth - spherical)

Q.2. What is the diameter of the lens?

Ans. 9 mm(adult)

Q.3. What is the thickness of the lens?

Ans. 4-4.5 mm

Q.4. From which primitive tissue does the lens develops?

Ans. Surface ectoderm overlying the optic vesicle

Q.5. Describe the structure of the lens (Fig 5.1).

Ans. *Layers (from without inwards):*

Lens capsule (thinnest at posterior pole)

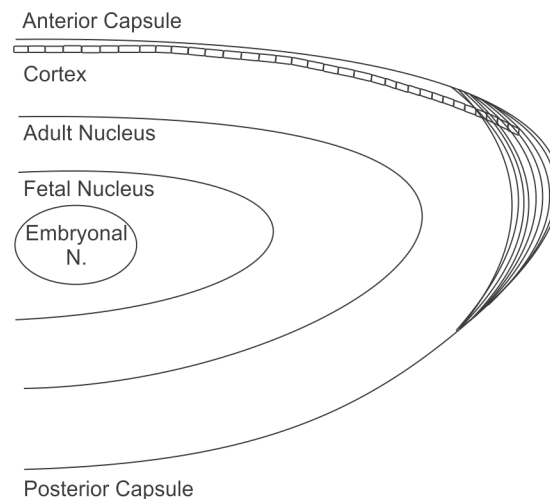


Fig. 5.1 : Showing a section of lens.



Epithelium (missing from posterior capsule, only underneath ant. capsule)

Cortex (Youngest fibres)

Epinuclear Cortex

Nucleus (from without inwards):

- Adult
- Adolescent
- Infantile (Lens at puberty)
- Fetal (Lens at birth, contains anterior and posterior Y-sutures)
- Embryonic (central)

Epithelium divides most actively in the periphery and differentiates into lens fibers.

Q.6. Name the congenital abnormalities of the lens.

- Ans.**
- Ectopia lentis (subluxation/dislocation)
 - Lenticonus
 - Congenital cataract

Q.7. What is the thickness of the lens capsule?

- Ans.**
- Anterior capsule – 14 micron
 - Posterior capsule – 4 micron (posterior pole is thinnest)

Q.8. Which is the thickest part of the lens capsule?

- Ans.** Equator

Q.9. Which is the thickest basement membrane of the body?

- Ans.** Lens capsule

Q.10. What is the chemical composition of lens?

- Ans.**
- 66% water
 - 33% protein
 - Others 1%

Q.11. What is the weight of the lens?

- Ans.** It varies from 135mg (0–9 yrs) to 255mg (40–80 yrs).

Q.12. What are the functions of lens?

- Ans.**
- Refraction of light (+18 D)
 - Accommodation

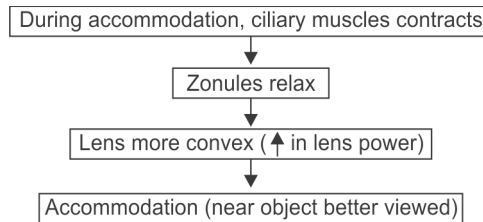
Q.13. What structures hold the lens in position?

- Ans.** Suspensory ligament or zonule of Zinn. They are attached to ciliary body at one end and equator of lens at the other end.



Q.14. What is the function of zonule of Zinn?

Ans. They have role in **accommodation**.



CATARACT (LONG CASE)

Q.15. What is your case?

Ans. It is a case of senile immature/mature cataract of right/left eye in an elderly patient (mention age of the patient).

Q.16. What are the points in favour of your diagnosis?

Ans. H/O Painless progressive dimness of vision.

O/E:

- V/A – Finger counting/hand movement (IMSC)
- PL/PR only (in mature senile cataract)
- Presence of iris shadow (in IMSC)
- Presence of Purkinje images - 4th image absent in MSC/HMSC/Adv IMSC; 1st, 2nd, 3rd present in all cases.

Q.17. Define *cataract*.

Ans. Any opacity of the lens or loss of transparency of the lens that causes diminution or impairment of vision is called cataract.

Q.18. Why do you say it is a mature cataract?

- Ans.**
- Vision is reduced to PL/PR
 - Pearly white appearance of the lens
 - No iris shadow
 - 4th Purkinje's image absent
 - No fundal glow (ophthalmoscopy)

Q.19. What is iris shadow? What does it indicate ?

Ans. It is a shadow formed on the anterior lens surface in IMSC cast by the concave iris border when light is thrown obliquely upon the lens. (Fig. 5.2) It indicates that there is some clear cortex in front of opaque lens fibres.

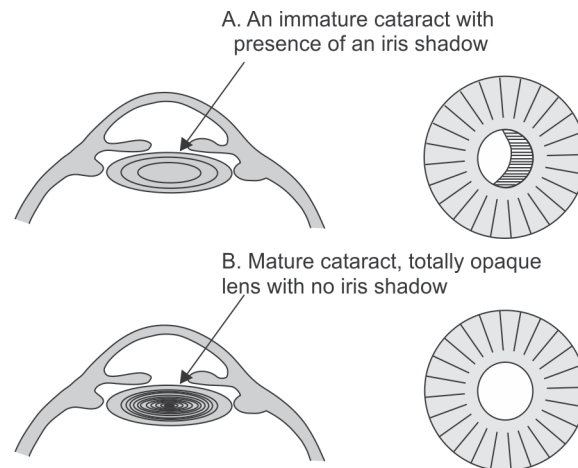


Fig. 5.2 : Use of iris shadow to diagnose maturity of cataract

Q.20. How will you test for iris shadow?

Ans. A torch light is thrown from the temporal side of the eye and we look for the iris shadow over the lens surface of the same side.

Q.21. Do you get an iris shadow in a normal crystalline lens?

Ans. No, it is present only in a case of IMSC.

Q.22. If PR is absent in the nasal quadrant what is the inference?

Ans. Temporal quadrant of the retina is damaged.

Q.23. Name one such condition (Inaccurate PR)

Ans. Retinal detachment.

Q.24. What are Purkinje images? How will you test it?

Ans. If a strong torch light is thrown on the eye - 4 images are formed – (Fig. 5.3)

1st (erect) – from anterior surface of cornea.

2nd (erect) – from posterior surface of cornea.

3rd (erect) – anterior surface of lens.

4th (inverted) – from posterior surface of lens.

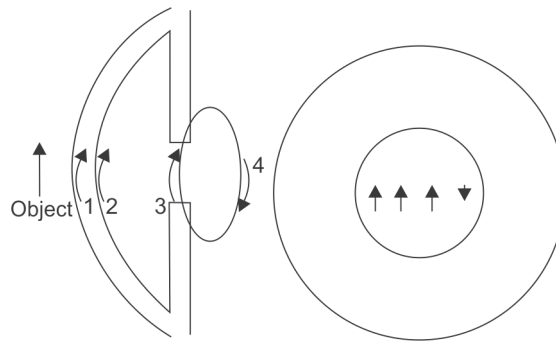


Fig. 5.3 : Showing Purkinje Images

Q.25. How many images will you get in a case of aphakia?

Ans. Two i.e. 1st and 2nd images are present; 3rd, 4th are absent (no lens).

Q.26. What will happen in a case of pseudophakia?

Ans. All 4 images will be present.

Q.27. What is the colour of the lens in IMSC/MSCHMSC?

Ans.

- Greyish white – IMSC
- White or Pearly white – MSC
- Milky white/Presence of calcified spots over anterior lens surface – HMSC

Q.28. Comment about the depth of anterior chamber in your case.

Ans.

- Normal – IMSC/MSCHMSC
- Deep - HMSC (Sclerotic)
- Shallow – IMSC (Intumescent stage), HMSC (Morgagnian)

Q.29. What complications can occur if the cataract becomes hypermature?

Ans.

- Secondary glaucoma
- Subluxation
- Dislocation in AC or PC
- Uveitis

Q.30. How will you confirm your diagnosis?

Ans. Slit lamp examination

Q.31. What is the literal meaning of cataract?

Ans. Waterfall



Q.32. What is the pathogenesis of cataract?

Ans. Two main processes (especially senile cataract)

1. Hydration
2. Sclerosis

Q.33. What is hydration?

Ans. Hydration

- Increased hydration leads to lamellar separation and collection of protein-deficient fluid between lens fibers.
- Leads to increased scattering of light and loss of transparency.
- Hydration also leads to denaturation of lens proteins and results in irreversible opacification.
- Mechanisms of increased hydration are:
 1. Failure of active pump mechanism
 2. Increased leakage across posterior or anterior capsule
 3. Increased osmotic pressure

Q.34. What is sclerosis?

Ans. • It is part of normal aging phenomenon, seen mostly in senile cataract and involves predominantly the nucleus (condensation of lens nucleus).
 • Increased compaction of lens proteins and fibers lead to increased scattering of light and loss of transparency.

Q.35. What are the symptoms of cataract?

Ans. • Dimness of vision
 • White reflex in the pupillary area (late case)
 • Frequent change of glasses
 • Unilateral polyopia/diplopia
 • Coloured halos
 • Scattering of light

Q.36. What is the cause of diplopia/polyopia in a case of cataract?

Ans. Irregular refraction by different parts of the lens, so that several images are formed of each object.

Q.37. What are the other causes of unilateral diplopia?

Ans. • Subluxated lens
 • Presence of iridectomy holes (too close to pupil)
 • Iridodialysis



Q.38. What is the cause of coloured halos?

Ans. Diffraction of light rays by the refractive media of the eyeball.

Q.39. What are the conditions causing coloured halos?

- Ans.**
- IMSC
 - Angle closure glaucoma
 - Mucopurulent conjunctivitis
 - Corneal oedema

Q.40. What do you mean by coloured halos?

Ans. Some patients with IMSC complain of rainbow coloured halos while looking at a light source. This is due to breaking of white light into coloured spectrum due to presence of water droplet in the lens.

Q.41. How will you differentiate between a rainbow halo of acute angle closure glaucoma with that of IMSC?

Ans. Fincham's stenopaedic slit test.

If a stenopic slit is passed across the pupil, glaucomatous halo remains intact, while a halo due to cataract is broken up into segments (Fig. 5.4).

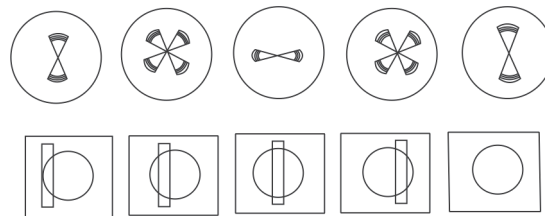


Fig. 5.4 : Emsley-Fincham stenopaedic - slit test demonstrating breaking up of halos due to immature cataract into different segments.

Q.42. What is polyopia? In which condition it is seen?

Ans. The patient complains of seeing multiple images of linear or crescent-shaped lights (moonlight, streetlight).

It is seen in incipient stages of immature cataract (due to irregular refraction by the lens owing to variable refractive index).

Q.43. What are the types of senile cataract?

- Ans.**
- a. Cortical cataract
 - Cuneiform
 - Cupuliform
 - b. Nuclear or sclerotic cataract



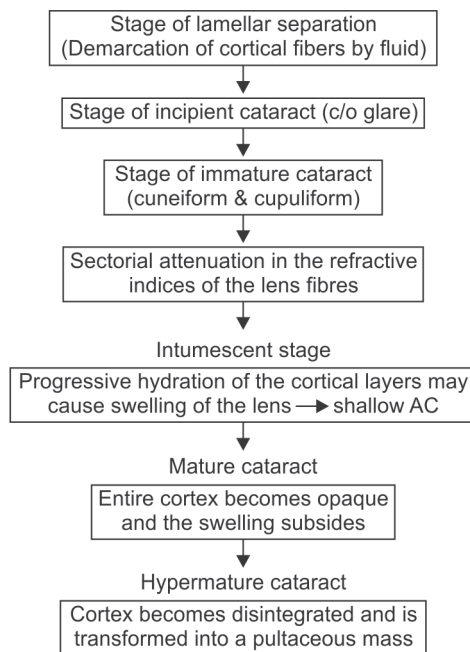
Q.44. Classify cataract according to stages of maturity?

Ans. According to stages of maturity, cataract can be classified as:

- Immature
- Intumescent
- Mature
- Hypermature
 - Morgagnian
 - Sclerotic

Q.45. What are the clinical stages of maturation of cortical cataract?

Ans.



Q.46. What is cuneiform cataract?

Ans. A type of IMSC .

Wedge shaped spokes of opacity with clear areas between them in periphery of the lens cortex.

Base of the wedge is in periphery while apex projects in pupil (causes diffraction of rays and monocular diplopia/polyopia).



Q.47. What is cupuliform cataract? (PSC)

Ans. A type of IMSC.

A disc or saucer shaped area of the cortex beneath the posterior capsule undergoes opacification.

The vision is worst in bright ambient illumination when the pupil is constricted(as the opacity is central).

Q.48. What is intumescent cataract?

Ans.

- Sometime during the course of maturation the lens imbibes lot of fluid and becomes swollen
- Anterior chamber becomes shallow
- Angle of anterior chamber may close resulting in glaucoma (Phacomorphic Glaucoma)

Q.49. Do you get intumescent stage in all cases?

Ans. No, only in some cases we get this condition.

Q.50. How will you manage such case?

Ans. Cataract surgery after control of IOP with antiglaucoma medications.

Q.51. What are the types of HMSC?

Ans.

- Sclerotic
- Morgagnian

Q.52. What are the features of sclerotic cataract?

Ans.

- The lens becomes inspissated and shrunken, due to water leakage
- Yellow in appearance
- Anterior capsule becomes thickened due to continued proliferation of the anterior cortical cells
- Tremulousness of iris
- Deep AC
- Degeneration of suspensory ligaments (may cause subluxation of lens)

Q.53. What are the features of Morgagnian cataract?

Ans.

- Cortex becomes fluid (milky)
- Nucleus may sink to bottom of the lens
- Shallow AC
- Increased IOP
- Nucleus is seen as brown mass altering its position with position of the head



Q.54. What are the features of cupuliform cataract?

- Ans.**
- Dense aggregation of opacities, often forming plaque, just beneath the capsule, usually in the posterior cortex
 - Progresses towards the equator
 - On distant direct ophthalmoscopy, it appears as dark shadow against a red fundal glow
 - Considerable decrease in vision, specially at day time (exposure to sunlight), being near the nodal point of the eye

Q.55. What is senile nuclear sclerosis or nuclear cataract?

Ans. The central nuclear fibres become sclerosed with age and is termed as 'nuclear sclerosis'. As time progresses, nucleus becomes diffusely cloudy spreading towards cortex nuclear cataract.

Q.56. What are the grading of nuclear sclerosis?

- Ans.**
- a. Grade I + - Grey/Greyish yellow
 - b. Grade II - Yellow
 - c. Grade III - Brown (cataracta brunescens)
 - d. Grade IV - Brown to Black (cataracta nigra -Hard)

Q.57. What is the indication of surgery in a case of IMSC?

Ans. When the vision is reduced to such an extent that the patient cannot carry out his day to day work efficiently in spite of full correction with spectacles.

Q.58. What are the investigations to be done before cataract surgery?

Ans. Systemic

- Blood Sugar (PP)
- BP
- ECG

Ocular

- Macular function test
- Biometry
- Syringing
- Tonometry
- USG B-scan in MSC/HMSC



Q.59. What are the different types of surgery done for cataract?

- Ans.**
- ECCE with PCIOL
 - SICS with PCIOL
 - Phacoemulsification
 - ICCE

Q.60. What are the advantages of SICS over ECCE + PCIOL?

- Ans.**
- Early rehabilitation
 - Less astigmatism
 - Safe as surgery is done in a closed chamber

Q.61. Why suture is not put in SICS?

- Ans.** Self sealing valve action (of the tunnel wall).
(see chapter 18)

PSEUDOPHAKIA (LONG CASE)

Q.62. What is your case?

- Ans.** A case of pseudophakia - RE/LE (mention age and sex of the patient).

Q.63. What is pseudophakia?

- Ans.** It means an eyeball with an artificial lens(IOL).

Q.64. What are the points in favour of your diagnosis?

- Ans.**
- H/O cataract operation
 - Limbal scar mark (may or may not be present)
 - Shiny reflex at pupillary area
 - Anterior chamber deeper than normal
 - Patches of iris atrophy (may or may not be present)

Q.65. Why anterior chamber is deeper than normal?

- Ans.** Normal crystalline lens thickness is 3.5 mm but IOL thickness is 2.5 to 3 mm.

Q.66. What is the normal AC depth?

- Ans.** 2.5 to 3 mm (central).



Q.67. What are the advantages of pseudophakia over aphakia?

Ans.

	<i>Pseudophakia</i>	<i>Aphakia</i>
Image magnification	0 - 2%	25 - 30%
Visual rehabilitation	Early	Late (after 6 weeks)
Optical aberration	Minimum	Much Spherical aberration - pin cushion effect Prismatic aberration - Jack-in-the-box phenomenon
Field of vision	Normal	Reduced
Cosmetic	Well accepted	Poor (Heavy and thick lens)

Q.68. How do you measure IOL power?

Ans. By **biometry**.

Q.69. What are the components of biometry?

- Ans.** a. **Keratometry** - Both vertical and horizontal curvature of cornea (in Dioptre) measured with Keratometer and average is taken (K_1 and K_2).
- b. **A scan ultrasonography**- Axial length of the eyeball is measured.

Q.70. What is SRK formula?

Ans. This is a formula commonly applied for calculation of IOL power (SRK- Sander, Retzlaff and Kraff)

$$P = A - 2.5L - 0.9K$$

P - IOL Power; L - Axial length; K - Average keratometer reading; A- Specific constant which varies with implant type.

Q.71. If biometry finding is not available what IOL power you will prescribe?

Ans. Standard IOL power +19 D can be given

Based on spectacle lens power of the patient, that is,

$P = + 19D + (R \times 1.25)$, where, P = IOL Power; R = Basic refraction.



Q.72. What are the contraindications of IOL implantation?

Ans. Absolute –

Denial of patient for IOL insertion.

Relative –

- Recurrent severe iridocyclitis
- Corneal endothelial dystrophies
- Severe proliferative diabetic retinopathy
- Congenital cataract <2 yrs of age
- Sometimes in subluxated and dislocated lens

Q.73. How can you implant IOL in a case of subluxated lens?

- Ans.**
- With use of capsular tension ring (CTR) IOL can be placed in bag
 - Anterior chamber IOL

Q.74. What is ICCE?

Ans. Intracapsular Cataract Extraction. In this method, the entire lens including the capsule is removed by tearing the zonules.

Q.75. Why ICCE is becoming obsolete now?

- Ans.**
- a. Poor quality of vision
 - b. It needs large incision
 - c. Inability to implant a posterior chamber IOL
 - d. More chances of complications like –
 - Vitreous (intraoperative)
 - Retinal detachment
 - Cystoid macular oedema
 - Astigmatism

Q.76. Who performed the first successful IOL implantation?

Ans. Harold Ridley, a British Ophthalmologist (1949).

Q.77. Does a pseudophakic patient need glass?

- Ans.** Yes, patient may have residual refractive error which needs glass.
- Patient may need near addition (because of loss of accommodation)
 - Correction of residual spherical error (due to under or over correction of IOL power either intentional or accidental because of inaccurate calculation).
 - Correction of postop astigmatism if any



Q.78. In which condition pseudophakic patient does not need any corrective glass?

Ans. If a multifocal IOL is put then there is no need of glass.

Q.79. What are the different types of IOL implantation?

Ans. • ACIOL (Anterior Chamber IOL)
• PC IOL (Posterior Chamber IOL)

Q.80. What are the different position of AC/PC IOL you know?

Ans. AC IOL	PC IOL
<ul style="list-style-type: none"> • Angle-supported lens • Iris-supported lens 	<ul style="list-style-type: none"> • In bag • Sulcus fixated lens • Scleral fixation lens

Q.81. What are the problems with ACIOL?

Ans. • Corneal endothelial decompensation.
• UGH syndrome (Uveitis, Glaucoma, Hyphaema).
• CME
• Decentration of lens

APHAKIA (LONG CASE)

Q.82. What is your case?

Ans. A case of aphakia RE/LE (Mention age and sex of the patient).

Q.83. What are the points in favour of your diagnosis?

Ans. • H/O Cataract surgery
• High plus power glass, poor vision without glass
• Deep AC
• Iridodonesis
• Peripheral Iridectomy (1/2)
• Jet black pupil
• Absence of 3rd and 4th Purkinje image

Q.84. What is iridodonesis?

Ans. Tremulousness of iris (due to lack of support of lens).

Q.85. Why pupil is Jet black?

Ans. Due to loss of reflection of light from the surface of the lens.

Q.86. What is aphakia?

Ans. • Literal meaning – Absence of lens.



- Anatomically – Absence of crystalline lens from the eyeball.
- Optically – Absence of lens in the pupillary area.

Q.87. What are the causes of aphakia?

- Ans.**
- Operative – Cataract operation (ICCE/needling/ECCE)
 - Traumatic/Spontaneous dislocation into the vitreous
 - Congenital aphakia (very rare)

Q.88. What are the conditions where you will find iridodonesis?

- Ans.**
- Aphakia
 - Subluxation/dislocation of the lens
 - Sclerotic HMSC
 - Buphthalmos

Q.89. What are the different modalities for management of aphakia?

- Ans.**
- a. Spectacles
 - b. Contact lens
 - c. Secondary IOL implantation (AC IOL/PC IOL)

Q.90. What is the usual power of glass in aphakia?

- Ans.**
- For distance +10 DSph along with + cylinder at 180°
 - For near vision – add +3 DSph

Q.91. What are the drawbacks of aphakic glass?

- Ans.**
- Image magnification of 25 – 30%
 - Spherical aberration (Pincushion effect)
 - Lack of physical coordination in finer movements
 - Cosmetic defect due to heavy plus lens
 - Prismatic aberration (Roving ring scotoma)
 - In monocular cases - **high anisokonia → diplopia**
 - Restricted field of vision

Q.92. What are the optical defects in aphakia?

- Ans.**
- High hypermetropia
 - Astigmatism (against the rule)
 - Loss of accommodation

Q.93. What are the advantages of contact lens in aphakia?

- Ans.**
- Image magnification – 7% – 8%
 - Aberrations are less
 - In monocular cases – no diplopia
 - Cosmetically well accepted
 - Wider and better field of vision



Q.94. What is *anisokonia*?

Ans. Size of image formed on retina is unequal in two eyes. e.g. anisometropia
(Upto 5% difference can be adjusted, if it is more then there will be diplopia).

Q.95. What is dioptric power of emmetropic eye?

Ans. Total + 58 to + 60D

Q.96. What is the contribution of lens and cornea?

Ans. Lens + 15 to + 18D
Cornea + 43 to + 45D

Q.97. What is *after cataract*?

Ans. It is a membranous white opacity formed by the remnant of anterior and posterior capsule following ECCE. It is also known as **posterior capsular opacification [PCO]**.

Q.98. What is PCO (posterior capsular opacification)?

Ans. It is the new terminology for after cataract(see above).

Q.99. What are factors influencing PCO?

Ans.

- Age of the patient
- History of intraocular inflammation
- Presence of pseudo exfoliation (white dandruff like material over iris and lens)
- Lens implant design
- Lens surface modification
- Lens optic material
- Time elapsed since surgery

Q.100. What is the mechanism of formation of *after cataract*?

Ans. In ECCE, a part of anterior and posterior capsule is left in situ. If the cataract is not mature, clear cortex sticks to the capsule which becomes partially absorbed by the action of the aqueous. But if it is shut off by adhesion of the anterior capsule to the posterior capsule, the cubical cells lining the anterior capsule continue to form new lens fibres, which are abnormal (opaque). This opaque membranous structure is known as after cataract.

Q.101. What are the types of after cataract?

Ans.

- Soemmering's ring
- Elschnig's pearl



Q.102. What is Soemmering's ring?

Ans. It is the ring behind the iris formed by the lens fibres enclosed between the two layers of lens capsules.

Q.103. What is Elschnig's pearl?

Ans. The subcapsular cuboidal cells proliferate and instead of forming lens fibres, develop into large balloon like cells which sometimes fill the pupillary aperture. The cells look like pearl and known as Elschnig's pearl.

Q.104. What is the treatment of after cataract?

Ans. a. Nd: YAG -Laser capsulotomy

- Done in case of PCO after ECCE+PCIOL/SICS + PCIOL/Phaco + PCIOL
- Painless and is performed as an OPD procedure.

b. Needling- In congenital cataract

Q.105. What are the complications of the above procedure?

Ans.

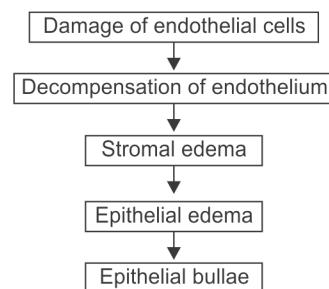
- Transient elevation of IOP (prophylactic antiglaucoma is given)
- Hyphema
- Risk of Retinal detachment
- Macular edema

Q.106. What is ABK?

Ans. Aphakic bullous keratopathy.

Q.107. What is the pathophysiology of ABK?

Ans. It commonly occurs after complicated cataract surgery.



Q.108. What is PBK?

Ans. Pseudophakic bullous keratopathy (mechanism is same as ABK).



Q.109. What is CME?

Ans. Cystoid macular edema

Fluid accumulates in the outer plexiform and inner nuclear layer of retina

Q.110. When does it occur?

Ans. 4 to 8 weeks after cataract surgery

Q.111. What are the causes of CME?

- Ans.**
- Vitreous loss
 - Iris in wound
 - Complicated surgery (post operative inflammation)

Q.112. How will you diagnose CME?

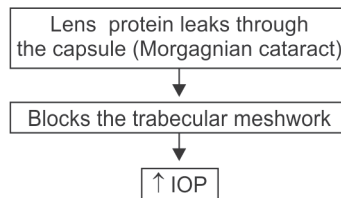
- Ans.**
- Ophthalmoscopy
 - FFA-Flower petal appearance

Q.113. What is the treatment of CME?

- Ans.**
- Topical corticosteroids
 - Topical NSAIDS
 - Oral acetazolamide

Q.114. What is phacolytic glaucoma?

Ans.



Q.115. What is phacomorphic glaucoma?

Ans. In Intumescent stage of cataract and in HMSC (Morgagnion) the lens pushes forward the iris, ciliary body → angle closure → glaucoma.

Q.116. What is the treatment of phacomorphic Glaucoma?

Ans. Conservative management (Diamox, mannitol) to decrease the IOP → Extraction of cataractous lens.

Q.117. What is phacodonesis? What is the cause?

Ans. Motility of lens along with the movement of the globe. Blunt trauma can cause zonular rupture and cause it.



Q.118 Can you implant an PCIOL in a case of zonular dialysis?

Ans. Yes; by using capsular tension ring (CTR)

Q.119. Describe the design and shape of IOL?

Ans. Two parts:

- Optic – optical part
- Haptic – part for fixing the IOL

Q.120. What are the sites where IOL can be implanted?

Ans.

- Capsular bag (preferred)
- Ciliary sulcus
- Anterior chamber (if posterior capsule is ruptured)

Q.121. Where is posterior chamber IOL placed?

Ans. Either in capsular bag or on the ciliary sulcus.

Q.122. Draw a diagram of PCIOL and show its different parts?

Ans. Optic: 5.5 – 7 mm diameter, made of PMMA [Polymethyl methacrylate]

- Planoconvex/biconvex
- 2 minute holes at the periphery of optic for dialing

Haptics-PMMA/nylon/prolene (Fig. 5.5a and b).

PMMA lens can be single piece or multiple piece.

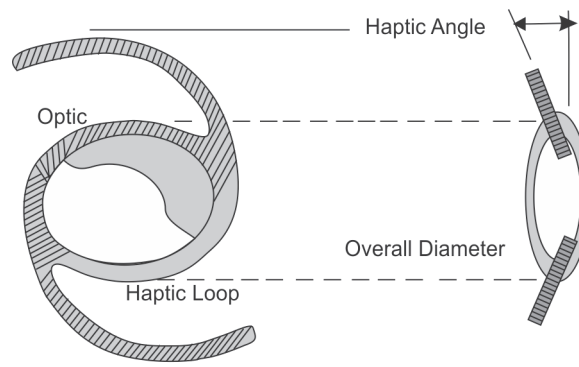


Fig. 5.5a : Intraocular lens designs, (Posterior chamber)

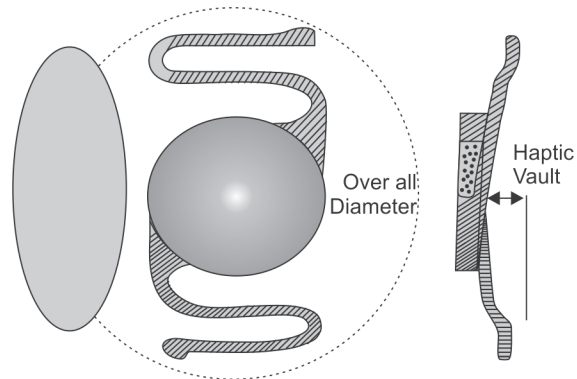


Fig. 5.5b: Intraocular lens designs (Anterior chamber)

Q.123. What is the overall diameter of IOL?

Ans. 12 - 14 mm

Q.124. How the IOL is sterilized?

Ans. ETO (ethylene trioxide)

Q.125. What are the different types of cataract operations?

- Ans.**
- a. Extra capsular cataract extraction (ECCE):
 - Conventional large incision ECCE with suture (8–10mm)
 - Small incision cataract surgery (SICS) without suture (5.5-7 mm)
 - Phacoemulsification (2.8-3.2 mm)
 - Phakonit (0.9-1.2 mm)
 - b. Intracapsular cataract extraction (ICCE)

Q.126. What are the preparatory steps before cataract operation?

- Ans.**
- Cleaning of the skin of the eyelids and adjacent area with Povidone iodine (5%) and spirit swabs
 - Application of head towel and eye sheets

Q.127. What is the anaesthesia used for ocular surgery?

- Ans.**
- Local anaesthesia
 - Topical anaesthesia
 - General anaesthesia



Q.128. What are the various types of local anaesthesia?

Ans. Peribulbar (commonest)
Retrobulbar (RB)
Facial

Q.129. Describe the method of peribulbar anaesthesia.

Ans. Anaesthetic agents:

2% lignocaine hydrochloride (with or without adrenaline)
+ 0.5% bupivacaine in a ratio of 3 : 2.
Injection hyaluronidase (1500 I U) is added to 1 bottle of 30 ml 2% xylocaine.

Technique:

- The above mixture is taken in 10 cc syringe (8-10 ml).
- ½ inch 26 gauge needle used to inject it.
- The patient is asked to look to the roof.
- Injection is given at two sites.
- Inferior site - at the junction of lateral 1/3 and medial 2/3 at lower lid. The needle is directed towards the floor of the orbit.
- Superior site - beneath the superior orbital notch (junction of medial 1/3 and lateral 2/3 of upper lid) with needle directed towards the roof of the orbit.
- Massage of the globe done with the fingers or super pinky ball.

Q.130. Why is hyaluronidase mixed?

Ans. It helps to spread the anaesthetic solution in periocular tissue.

Q.131. What are the advantages of peribulbar anaesthesia?

Ans.

- Less chance of retrobulbar haemorrhage (RBH).
- Less chance of globe perforation.
- Less chance of optic nerve and CNS complications
- Provides good hypotony.

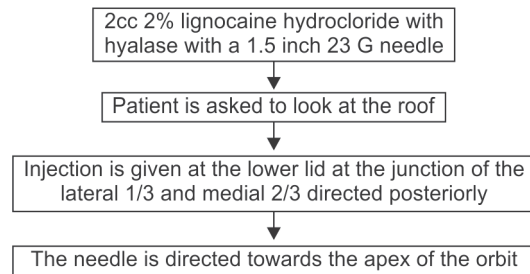
Q.132. What are the disadvantages of peribulbar anaesthesia?

Ans. Requires prolonged compression for effectiveness.
Causes chemosis that may interfere surgery.



Q.133. What is Retrobulbar (RB) anaesthesia?

Ans.



Q.134. What are the advantages of RB block?

- Ans.**
- Rapid anaesthesia
 - Very little anaesthetic agent is required

Q.135. What are the complications of RB block?

- Ans.**
- RB haemorrhage
 - CNS toxicity
 - Globe perforation

Q.136. What are the features of RB haemorrhage?

- Ans.**
- Proptosis
 - Eye lid chemosis
 - Sub conjunctival haemorrhage
 - Elevated IOP

Q.137. Why RB block is not used much now a days?

- Ans.**
- 2nd block is needed (facial block)
 - More chance of complications

Q.138. What is the purpose of facial block?

Ans. For akinesia of orbicularis oculi, facial block is given.

Q.139. What are the different methods of facial block?

- Ans.**
- Van Lint Akinesia
 - 'O' Brien akinesia
 - Atkinson akinesia
 - Nathbath block

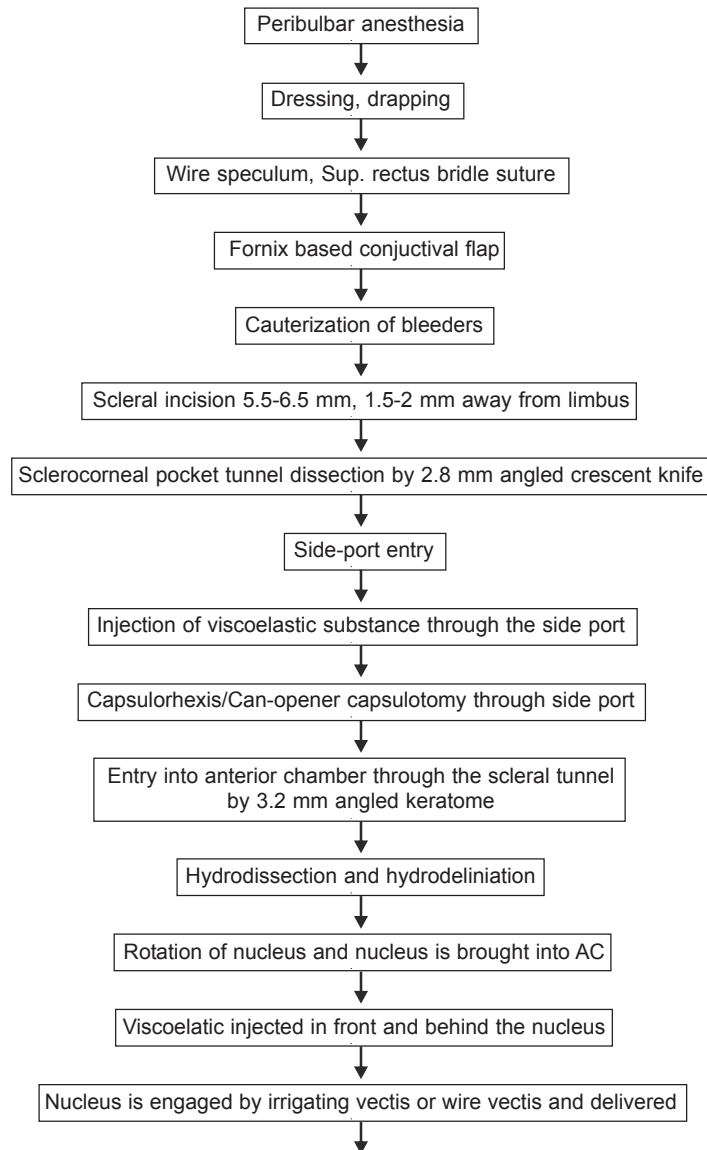
Q.140. What is 'O' Brien anaesthesia?

Ans. 4-6 ml of local anaesthetic is injected in front of the tragus of the ear over the condyloid process of the mandible.



Q.141. What are the steps of small incision cataract surgery (SICS)?

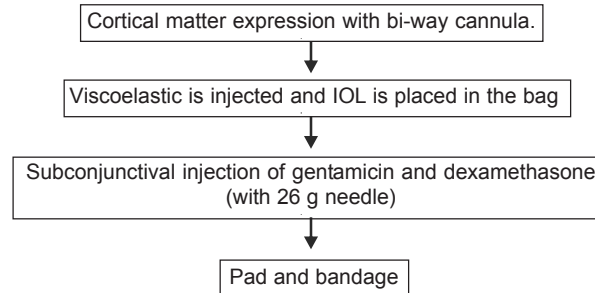
Ans.



Contd...



Contd...



Q.142. What complications can happen while making sclero-corneal tunnel?

Ans. • Premature entry into AC
• Button holing of sclera

Q.143. How will you make side port entry and what is its advantage?

Ans. It is made with MVR knife
It helps in removal of subincisional cortex

Q.144. What complication can occur during side port entry?

Ans. Leaking side port, iris prolapse

Q.145. What complications can occur during delivery of nucleus?

Ans. • Zonular dialysis
• Iridodialysis
• Too small incision – difficulty in nucleus delivery
• Rubbing the corneal endothelium by nucleus

Q.146. How will you prevent the above complication?

Ans. • Adequate tunnel incision
• Use of plenty of viscoelastics

Q.147. What is the usual site of incision in SICS?

Ans. 12 o' clock

Q.148. What are the other sites of SICS incision?

Ans. Superotemporal – RE
Superonasal – LE

Q.149. Describe the scleral tunnel incision?

Ans. Types-straight/frown/smile
Length : 5.5 – 6.5 mm
Breadth : 2.5 – 3 mm (1 mm within cornea)



Q.150. Which one is better?

Ans. Frown

Q.151. What are the steps of sclerocorneal tunnel?

Ans. 3 steps:

Step 1: External scleral incision (half thickness of sclera)- made by no 15 disposable surgical blade or crescent knife.

Step 2: Horizontal part of tunnel made by crescent knife.

Step 3: Oblique incision, at the end of tunnel using 3.2 angled keratome to enter into the AC.

Q.152. What is the shape of tunnel?

Ans. Funnel shaped i.e. internal incision is 20 % wider than external incision.

Q.153. What is hydrodissection?

Ans. BSS/Ringer lactate fluid is injected just beneath the anterior capsule to separate the capsule from cortex and nucleus .

Q.154. What is hydrodelineation?

Ans. It is done to reduce the bulk of epi nucleus before delivery of nucleus.

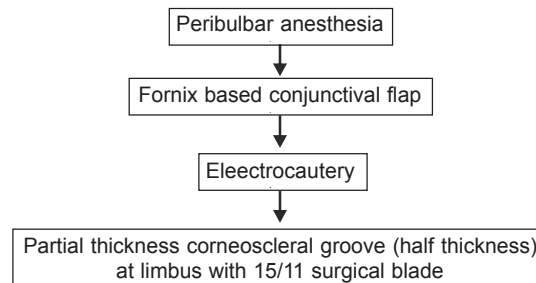
After good hydroprocedure the nucleus become freely mobile in the bag.

Q.155. How does superior rectus bridle suture help?

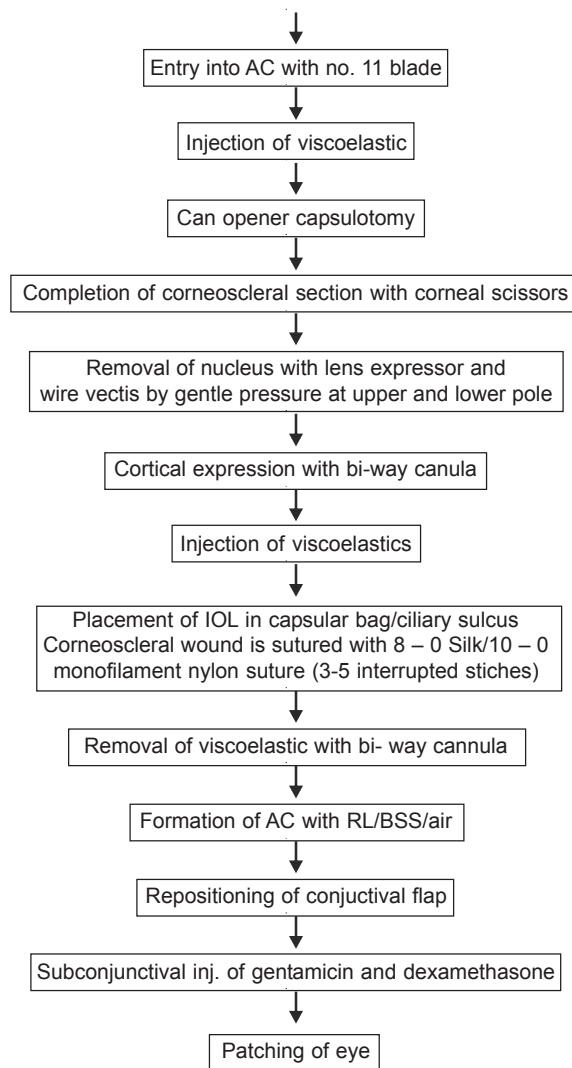
Ans. • Helps in maneuvering the globe forward and downwards during surgery
• Helps to give counter force during nucleus deliver
• Many surgeons do not use it now a days

Q.156. Describe the steps of ECCE + PCIOL?

Ans.



Lens



Q.157. What is phacoemulsification ?

Ans. It is an ECCE technique in which cataract operation is done through a 2.8 mm incision by a instrument called phacoemulsifier. The nucleus is emulsified by ultrasonic vibration of a titanium tip. The emulsified lens is removed by aspiration-infusion.



Q.158. What is the vibration speed of the phaco tip?

Ans. 28000- 60000 cycles per second.

Q.159. What is the dye used for anterior capsular staining in CCC?

Ans. Trypan blue. It delineates the capsule so that rhexis becomes easy.

Q.160. In which type of cataract capsular staining is necessary?

Ans. HMSC (morgagnian cataract)/MSC

Q.161. What are the different types of lens used in phacoemulsification?

Ans.

- Foldable acrylic lens
- Fluorinated PMMA lens
- Hydrogel IOL

Q.162. What are the advantages of CCC?

Ans.

- Less traction on zonules
- Regular margin of anterior Capsule prevents posterior Capsular tear
- Hydroprocedure is safe
- IOL can be placed over the anterior capsular rim in case there is PC rent

Q.163. What are the advantages of phaco over manual SICS?

Ans.

- Early rehabilitation
- Less astigmatism
- Sutureless surgery

Q.164. What are the disadvantages of phaco?

Ans.

- Steep learning curve
- Costly machine
- Proper patient selection is necessary

Q.165. What is phaconit?

Ans. It is a modified phaco in which surgery is done through a 0.9 mm incision with rollable IOL.

Q.166. What are the complications of cataract surgery?

Ans. **Preoperative complications** (complications of anaesthesia) include the following:

- Retrobulbar or peribulbar haemorrhage
- Accidental perforation of globe with intraocular injection of anaesthetic



- Accidental injection into optic nerve sheath with intracranial spread
- Anaphylactic shock
- Vasovagal reflex with collapse

Perioperative or intraoperative complications-

Poor operative preparation

- Excessive bleeding from conjunctiva if conjunctival flap is made and the patient is on anticoagulant
- Rise in BP or attack of angina if patient forgets to take usual dose of medications
- Hypoglycemic attack if patient is fasting and has taken his antidiabetic medications
- Patient starts moving and coughing during the surgery if not properly counselled preoperatively

Poor surgical technique

- Damage to superior rectus muscle while passing bridle suture (post-op ptosis).
- Poorly constructed wound with irregular edges (astigmatism).
- Damage to delicate intraocular tissue especially corneal endothelium, iris, lens zonules and posterior capsule.

Other complications

- Subluxations of lens
- Posterior dislocation of lens
- Rupture of posterior capsule or posterior capsular rent (PC rent) with vitreous loss
- Nucleus dropping back into the vitreous cavity (through PC rent)
- Expulsive haemorrhage (from choroidal vessels)
- Failure to implant the IOL in capsular bag

Post operative complications:

Early postoperative complications (within first few days to 4 week)

- Striate keratopathy
- Iris prolapse
- Endophthalmitis
- Uveitis
- Retained lens matter
- Corneal edema



- Shallow AC (Wound leak)
- Wound dehiscence
- Hyphaema
- Toxic anterior segment syndrome
- Astigmatism
- Retinal detachment
- Retinal tear or vitreous haemorrhage
- Refractive surprise (high refractive error)

Late postoperative complications (after 1 month to years)

- Endophthalmitis
- Bullous keratopathy
- Retinal detachment
- Cystoid macular edema
- Exacerbation of diabetic retinopathy
- Displacement of intraocular lens
- Persisting astigmatism
- Secondary glaucoma
- PCO
- Anterior capsular phimosis

COMPLICATED CATARACT (LONG/SHORT CASE)

Q.167. What is complicated cataract?

Ans. It results from disturbance in lens metabolism due to the inflammatory and degenerative condition of eye.

Q.168. What are the characteristic features of complicated cataract?

Ans. Posterior subcapsular cataract (PSC)

Slit lamp –

- Bread crumb appearance
- Polychromatic lustre (cataract may remain stationary for long time/progresses peripherally or axially until entire lens is involved)

Q.169. What are the symptoms of complicated cataract?

Ans. Dimness of vision mainly at day time
(Even in the early stages vision is impaired because of the position of the opacity near the nodal point of the eye)



Q.170. Name some conditions where you get complicated cataract?

- Ans.**
- Iridocyclitis
 - Degenerative myopia
 - Ciliary body tumor
 - Choroiditis
 - Retinis pigmentosa
 - Anterior segment ischemia
 - Retinal detachment

Q.171. How will you treat a complicated cataract?

Ans. Cataract surgery (SICS/Phaco) under cover of steroid

Q.172. What is true diabetic cataract?

Ans. Snow flake cataract (minute white dots of varying size like snow flakes)

Q.173. What is rosette cataract?

Ans. It looks like flower petal, seen after concussion injury

Q.174. In which condition oil droplet cataract is seen?

Ans. Galactosemia

Q. 175. What is sunflower cataract?

Ans. It is seen in Wilson's disease

Q.176. Name few cataract producing toxic agents.

- Ans.**
- Corticosteroid
 - Chlorpromazine
 - Chloroquin
 - Iron

Q.177. Name some systemic diseases which are associated with cataract:

- Ans.**
- Diabetes mellitus
 - Hypoparathyroidism
 - Dystrophia myotonica
 - Down syndrome
 - Galactosemia

Q.178. What is Ectopia lentis?

Ans. This is a subluxation or dislocation of lens usually upwards and bilateral.



Q.179. What are the causes of ectopia lentis?

- Ans.**
- Familial
 - Marfan's syndrome
 - Secondary to an eye disease — uveitis, HMSC, pseudoexfoliation syndrome, secondary to trauma.

Q.180. What is the direction of subluxation in Marfan's syndrome?

- Ans.** Superotemporal (inferotemporal in homocystineuria)

Q.181. What is subluxation of lens?

- Ans.** Partial dislocation of lens due to tearing of few fibres of suspensory ligament. Here the lens lies in the pupillary region.

Q.182. What is dislocation of lens?

- Ans.** Displacement of the lens from the pupillary region due to complete tearing of suspensory ligament.

CONGENITAL CATARACT (LONG/SHORT CASE)

Q.183. What are the clinical types of congenital cataract?

- Ans.**
- Embryonic nuclear cataract
 - Zonular cataract
 - Sutural cataract
 - Anterior polar cataract
 - Posterior polar cataract
 - Coronary cataract
 - Blue dot punctuate cataract
 - Total congenital cataract

Q.184. Describe the etiology of congenital cataract.

- Ans.**
- Idiopathic (50%)
 - Hereditary
 - **Maternal factors-**
 - TORCH infection during pregnancy
 - Maternal malnutrition
 - Drug induced
 - **Infantile factors-**
 - Anoxia (secondary to placental haemorrhage)
 - Metabolic disorder- Galactosemia, Hypoglycemia



- Birth trauma
- Malnutrition

Q.185. Name two syndrome which are associated with cataract?

- Ans.**
- Down's Syndrome
 - Lowe's Syndrome

Q.186. What are the indications of surgery in developmental cataract?

- Ans.**
- Poor vision
 - Development of squint which was previously not there.
 - Nystagmus

Q.187. Which is the commonest congenital cataract?

- Ans.** Blue dot cataract

Q.188. What are the characteristic features of zonular cataract?

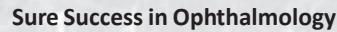
- Ans.**
- 50% of all visually significant congenital cataract are of this type
 - Strong hereditary tendency, autosomal dominant
 - Vitamin D deficiency
(The development is interfered at later stage and a zone around embryonic nucleus, usually in area of fetal nucleus becomes opacified)
 - Opacity is usually sharply demarcated
 - Area of lens within and around the opacity is clear
 - Linear opacities like spoke of a wheel (riders) may run outward towards the equator

Q.189. What are the indications of surgery in congenital cataract?

- Ans.**
- If the vision is $<6/18$ in a patient with unilateral cataract.
 - Size of the opacity - if the opacity is 2-3 mm in pupillary axis.

Q.190. What are the poor prognostic factors?

- Ans.**
- Unilateral cataract
 - Total cataract
 - An early age of onset
 - Late age of presentation
 - Associated ocular disorder (nystagmus/squint)



Ans.

More than 2 years — $\left. \begin{array}{l} \rightarrow \text{ECCE} \\ \rightarrow \text{SICS} \\ \rightarrow \text{Phaco} \end{array} \right\} \text{with IOL implantation}$

Ans.

- PCO (high incidence)
- More chance of post- op uveitis and fibrinous reaction.

Ans.

- Aphakic glass
- Contact lens
- Secondary PC/AC IOL implantation after 2 years.

Ans.

- Primary posterior capsulorhexis along with anterior vitrectomy (<5 yrs age)
- Optic capture
- Use of Heparin/Fluorine - coated lens

Ans.

- To prevent iris prolapse
- To prevent pupillary block glaucoma (In ICCE)
- To prevent peripheral anterior synechiae

Ans. a. PBHI (Peripheral button hole iridectomy for glaucoma and cataract surgery. In ACG YAG laser peripheral iridotomy is preferred (Fig. 6.6)
b. CI (Complete iridectomy)
c. Optical iridectomy



d. For removal of foreign body, cyst or tumour of the iris

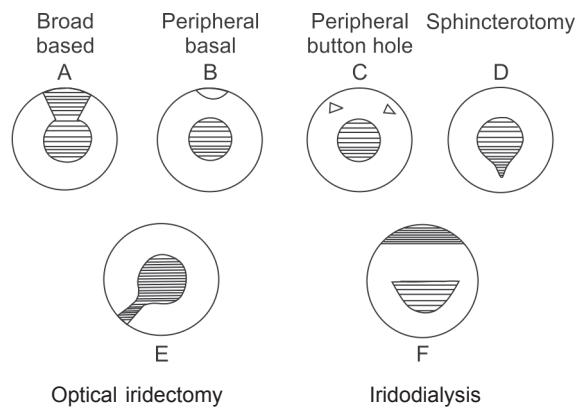


Fig. 5.6 : Types of Iridectomy

Chapter

6

Uvea

Q.1. What is Uvea? What are the parts of Uvea?

Ans. It is the vascular tunic of eye ball (means 'grapes')

- Iris – anterior most part of uvea. It is a free circular diaphragm with a central opening called pupil.
- Ciliary body – intermediate part.
- Choroid – posterior part.

Q.2. Describe the structure of iris.

Ans. It consists of four layers from anterior to posterior. (Fig. 6.1)

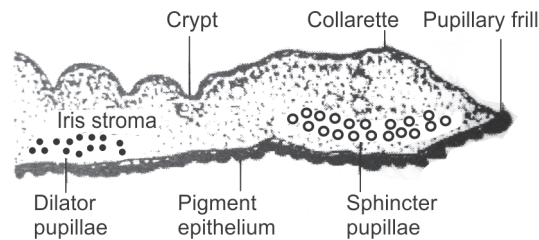


Fig. 6.1 : Structure of Iris

1. **Anterior endothelial layer** – single layer of cell covering the anterior surface of the iris.
2. **Stroma**—
 - Contains branched connective tissue cells usually pigmented, but largely unpigmented in blue iris.
 - Rich blood supply, blood vessels run in a radial direction forming the minor circle of iris.
 - The tissue spaces communicate directly with the anterior chamber through the crypts found mainly near the ciliary border.
3. **Posterior two layers of epithelium** – the stroma is covered on its posterior surface by two layers of pigmented epithelium, which are developmentally



derived from retina and continuous with each other at pupillary margin.

Anterior layer consists of flattened cells and posterior of cuboidal cells.

4. **Smooth muscle** – from anterior epithelial layer, two unstriated muscles arise which are dilator and constrictors of the pupil.

Sphincter pupillae – a circular bundle running around the pupillary margin.

Dilator pupillae – arranged radially near the root of the iris.

Q.3. What is the nerve supply of iris?

Ans. **Sensory** – nasociliary nerve, a branch of 5th cranial nerve (ophthalmic division of trigeminal).

Sphincter pupillae – parasympathetic (oculomotor nerve).

Dilator pupillae – cervical sympathetic chain.

Q.4. What is the shape of ciliary body?

Ans.

- In A-P section – it is shaped roughly as an isosceles triangle with the base forward.
- The iris is attached about the middle of the base.

Q.5. What are the muscles of the ciliary body?

Ans. The chief mass of the ciliary body is composed of unstriated muscle fibres, called ciliary muscle. It has 3 parts with a common origin circumferentially at the scleral spur.

- **Meridional** – the greatest part, running anteroposteriorly, inserted at the suprachoroid.
- **Circular** – concentrically with the root of the iris.
- **Radial**

Q.6. What are the different parts of the ciliary body?

Ans. The inner surface of the ciliary body is divided into 2 parts:

- Pars plana- posterior part (safe zone of the eye).
- Pars plicata-the anterior part; about 70 plications are visible around the circumference macroscopically. Microscopically, many smaller folds, ciliary processes are seen.
- Ciliary body is covered by the two layers of epithelium continuous with the iris anteriorly and retina posteriorly. Outer pigmented layer forms the pigment epithelial layer



of retina and inner unpigmented layer forms rest 9 layers of retina. So there is a potential space between pigment layer and other 9 layers of retina.

Ciliary body extends backwards as far as the ora serrata, at which point the retina begins.

Q.7. Describe the structures of ciliary processes.

Ans. This consists essentially of tufts of blood vessels, like glomeruli of kidney. They are covered by the epithelium, which belongs to the similar layers as in the iris. The outer layer corresponding to the anterior layer of the iris, consists of flattened cells; the inner layer of cuboidal cells. But only the outer layer of ciliary body is pigmented, inner layer is unpigmented.

Q.8. What is the nerve supply of ciliary body?

Ans.

- Sensory – 5th cranial nerve
- Motor – 3rd cranial nerve and sympathetic nerve

Q.9. What are the functions of iris?

Ans.

- It controls the amount of light entering into the eye.
- It regulates the flow of aqueous from posterior to anterior chamber.

Q.10. What are the functions of ciliary body?

Ans.

- Formation of aqueous humor by the ciliary processes.
- Ciliary muscle helps in accommodation for near work.
- Ciliary muscle helps in opening up of Schlemm's canal and thus facilitates in aqueous outflow.

Q.11. Describe the structure of the choroid.

Ans.

- It is an extremely vascular membrane in contact with the sclera.
- There is a potential space between the two known as suprachoroidal space.
- It is richly supplied by blood vessels.
- On the inner side it is covered by elastic membrane of Bruch.

Q.12. Describe the blood supply of the choroid.

Ans. From outside inwards:

- Haller's layer
- Sattler's layer
- Choriocapillaries



They are supplied by the branch of ophthalmic artery:

- **Short posterior ciliary arteries** (10-20 in numbers).
- **Long posterior ciliary arteries**—2 in number, each divides into 2 to form 'Major arterial circle' of the iris in ciliary body.
- **Anterior ciliary arteries**—Terminal branches of 2 muscular arteries of each rectus muscle. (except lateral rectus – 1 muscular artery).

Venous drainage – The venous blood of uvea is collected by a series of veins into the vortex veins (4 in number), located behind the equator of the globe.

The vortex veins drain into the superior and inferior ophthalmic vein.

ANTERIOR STAPHYLOMA (LONG CASE)

Q.13. What is your case?

Ans. It is a case of anterior staphyloma of RE/LE (mention age and sex).

Q.14. What are the findings in favour of your diagnosis?

Ans.

- Vision- no PL/PL only/ PR inaccurate.
- Cornea- globular bulging of cornea with pigmented scar which has lobulated appearance with bluish colour.
It may extend into surrounding part of sclera (if it is, partial part of the cornea is visible).
- Anterior chamber, iris, pupil, lens not visible.
- Digital tension high/normal.

Q.15. What is the literal meaning of staphyloma?

Ans. Bunch of grapes.

Q.16. Define staphyloma?

Ans. It is an ectasia or bulging of the outer coats (cornea, or sclera or both) of the eye ball lined by uveal tissue.

Q.17. What are the clinical types of staphyloma?

Ans. (Fig-6.2)

- Anterior staphyloma
- Intercalary staphyloma
- Ciliary staphyloma
- Equatorial staphyloma



- Posterior staphyloma (Pathological myopia)

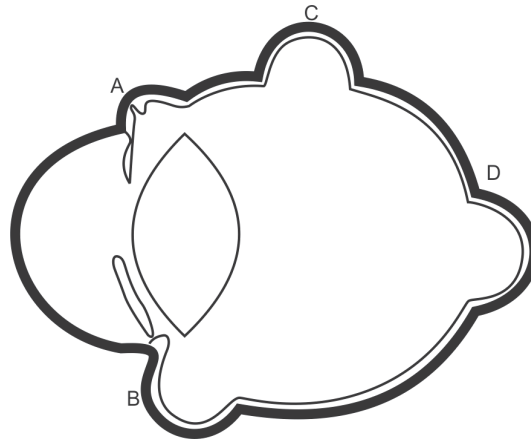


Fig. 6.2 : Staphylomas : A. Intercalary; B. Ciliary; C. Equatorial; D. Posterior.

Q.18. What is intercalary staphyloma?

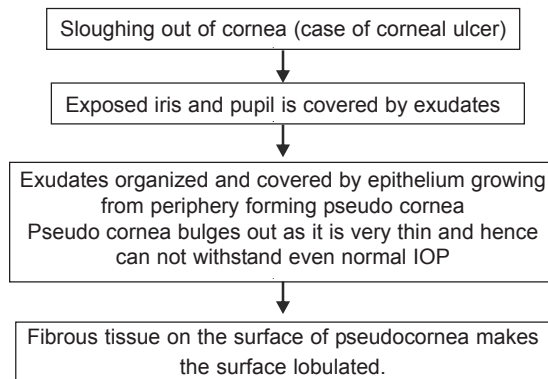
Ans. Ectasia between limbus and ciliary body.

Q.19. What are the causes of staphyloma?

- Ans.**
- Following perforation of a corneal ulcer
 - Scleritis
 - Perforating injury of the eye
 - Secondary glaucoma and high myopia

Q.20. What is the mechanism of formation of anterior staphyloma?

Ans.



**Q.21. What are the complications of anterior staphyloma?**

- Ans.**
- Rupture due to trauma or strain
 - Sepsis leading to panophthalmitis
 - Secondary glaucoma due to closure of AC angle
 - Absolute glaucoma (painful blind eye)

Q.22. What are the treatment options for anterior staphyloma?

- Ans.**
- Partial anterior staphyloma → iridectomy and trabeculectomy to treat secondary glaucoma
 - Total anterior staphyloma (painful blind eye) → Enucleation
 - For cosmetic reason → Staphylectomy → cosmetic shell
Enucleation → Acrylic Implant → Cosmetic shell.

Q.23. What is enucleation?

- Ans.** It is a destructive surgery where the whole globe along with part of optic nerve is sacrificed.

Q.24. What are the indications of enucleation?

Ans. Absolute:

- Intraocular malignancy:
 1. Retinoblastoma – in children
 2. Malignant melanoma – in adult
- Sympathetic ophthalmia

Relative:

- Ciliary staphyloma
- Huge anterior Staphyloma phthisis bulbi
- Phthisis bulbi
- Painful blind eye – such as thrombotic secondary glaucoma
- To collect eye from the cadaver for eye bank (for keratoplasty)

Q.25. What are the contraindications of enucleation?

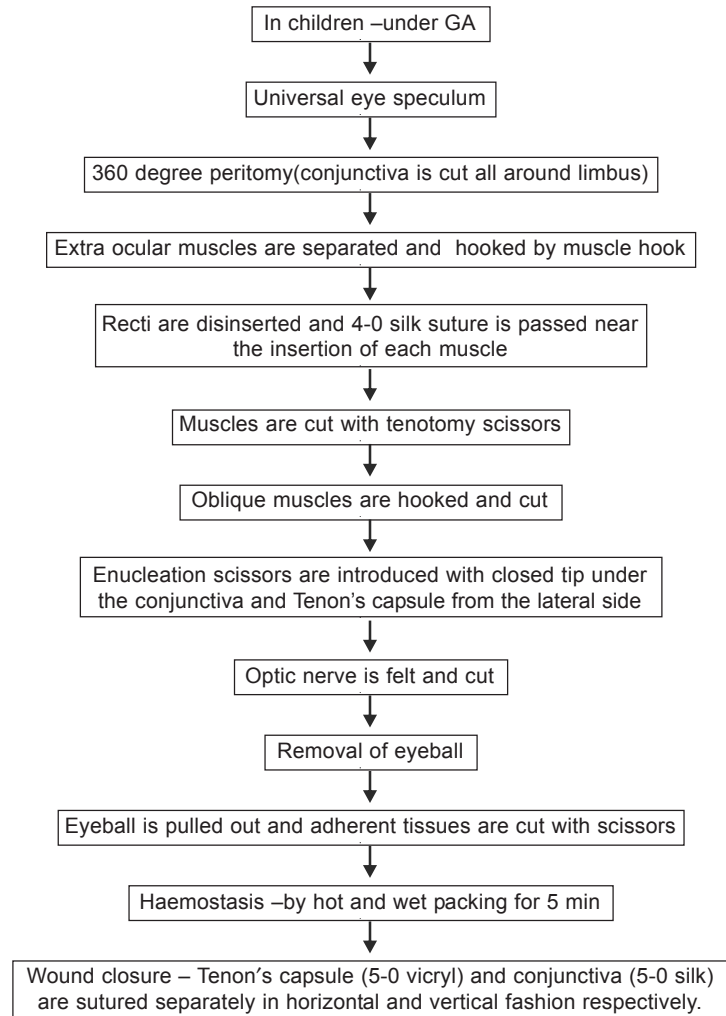
- Ans.** Panophthalmitis (because infection may spread to brain through meningeal sheaths leading to meningitis or encephalitis).



Q.26. Describe the steps of enucleation?

Ans.

Anaesthesia- peribulbar block (2% lignocaine)





Q.27. Why evisceration is not done?

Ans. Chance of sympathetic ophthalmia.

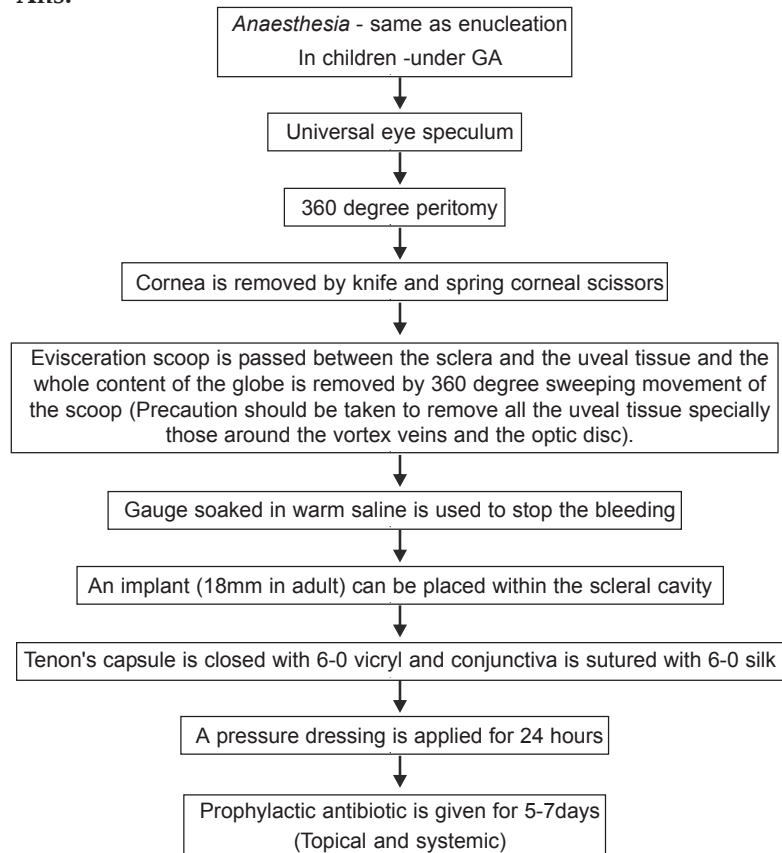
Q.28. What are the indications of evisceration?

Ans.

- Panophthalmitis.
- Expulsive haemorrhage.
- Painful blind eye following intraocular infection.

Q.29. Describe the steps of evisceration.

Ans.



Q.30. When will you fit artificial eye after evisceration enucleation?

Ans. After 4-6weeks.



Q.31. What is frill excision of sclera?

Ans. In evisceration a collar of sclera is left around the optic nerve so as to leave the optic nerve sheath undisturbed (in conventional evisceration almost the whole sclera is left out so there is more reaction).

IRIDOCYCLITIS (LONG CASE)

Q.32. What is your case?

Ans. A case of acute/chronic/resolved iridocyclitis RE/LE (mention age and sex).

Q.33. What are the features of acute iridocyclitis?

Ans. Symptoms:

- Pain around eye
- Blurring/Dimness of vision
- Redness of eye
- Intolerance to light
- Watering (may or may not)

Signs -(Fig 6.3)

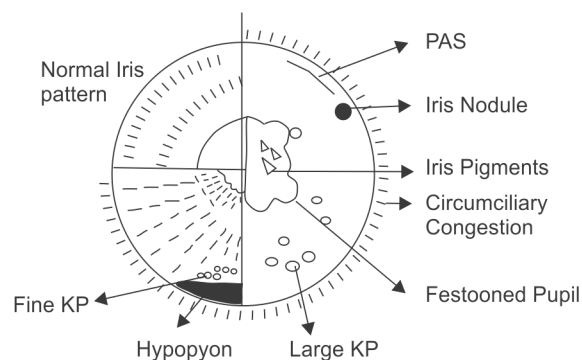


Fig. 6.3 : Various signs of iridocyclitis

- V/A – may reduce to HM or FC
- Ciliary congestion – present
- Cornea – K. P. (seen by slit lamp)
- AC – hazy (due to flare, cells)
- Iris – Loss of iris pattern



- **Pupil:**
 - Presence of synechiae
 - Smaller size pupil
 - Festooned pupil
- Ciliary tenderness present
- Lens – Iris pigments on lens surface
- Posterior synechiae
- IOP – Normal/Increased/low

Q. 34. What is aqueous flare?

Ans. Plasma protein leaks through damaged capillaries (seen with slit lamp) → causes Tyndall effect.

Q.35. What are the abnormal contents of the AC?

- Ans.**
- Blood (hyphaema)
 - Pus (hypopyon)
 - Dislocated lens (trauma)
 - Albuminous material (aqueous flare)
 - Vitreous (trauma)
 - FB in AC
 - Anterior chamber IOL
 - Air bubble after intraocular surgery

Q.36. What is KP (keratic precipitates)?

Ans. These are clumps of leucocytes adherent to the corneal endothelium. The inflammatory cells in the aqueous wander by convection current and stick to the edematous endothelium of the cornea.

Q.37. What is Arlt's triangle?

Ans. KPs arrange in triangular area at the lower part of the cornea due to gravitation.

Q.38. What are the different types of KPs?

- Ans.**
- a. Fine/coarse (lymphocytes and plasma cells) – found in allergic or acute iridocyclitis.
 - b. Mutton fat (Macrophage and epitheloid cells) – found in granulomatous uveitis – e.g. TB, sarcoidosis.

Q.39. How do you identify fresh KPs/Old KPs?

- Ans.**
- Fresh KPs – Solid, round, white, fluffy
 - Old KPs – Faded, shrunken, pigmented, crenated edge

Q.40. Why the pupil is constricted in acute uveitis?

- Ans.**
- Water logging of the iris



- Toxin acts as irritant to iris muscles (sphincter is more powerful than dilator muscles)
- Radial arrangement of the iris vessels

Q.41. What is muddy iris?

Ans. In uveitis/iridocyclitis there is extravasation of protein rich fluid into the iris tissue spaces leading to loss of iris pattern.

Q.42. What is the normal pattern of the iris?

Ans. See Ans. 43

Q.43. What are the features of anterior surface of iris?

Ans. Features of anterior surface:

- Presence of collarette
- Crypts and radial striations

Q.44. Why anterior chamber is hazy?

Ans. Protein rich fluid escapes into the AC making it hazy.

Q.45. What are the causes of raised IOP in uveitis?

- Ans.**
- Albuminous aqueous block the AC angle
 - Posterior synechiae
 - Pupillary block due to ring synechiae or occlusio pupillae
 - Trabeculitis/trabecular edema
 - Peripheral anterior synechiae
 - Steroid induced glaucoma

Q.46. What are the cause of low IOP in acute uveitis?

Ans. In acute iridocyclitis, there is ciliary body shock (less aqueous production).

Q.47. What is synechiae?

Ans. Adhesion of iris with the lens (posterior synechiae) or cornea (anterior synechiae).

Q.48. Define occlusio pupillae.

Ans. Fibrinous exudates get organized and cover the entire pupil.

Q.49. What is seclusio pupillae?

Ans.

Total 360° posterior synechiae, preventing aqueous flow from posterior to anterior chamber



Forward bowing of peripheral iris



Iris bombe

**Q.50. What is the treatment of iris bombe?**

Ans. YAG laser iridotomy.

Q.51. What is festooned pupil?

Ans. In chronic iridocyclitis with posterior synechiae, mydriatic causes irregular dilatation of pupil known as festooned pupil.

Q.52. What are the nodules seen in uveitis?

Ans.

- Koeppe's nodule - aggregate of epithelioid cells and giant cells, surrounded by lymphocytes seen at pupillary border. May be seen in granulomatous and non-granulomatous uveitis.
- Busacca's nodule - Similar nodule seen on the surface of iris; found in granulomatous uveitis.

Q.53. How will you investigate the case?

Ans.

- Routine haemogram
- Rheumatoid factor
- ANF, ANA
- VDRL, FT ABS
- HLA typing
- Mantoux test
- X-ray chest
- Kveim test (for sarcoidosis)
- ELISA test for TB, toxoplasmosis

Q.54. How will you treat the case?

Ans. *Management of acute iridocyclitis -*

Local:

- Atropine 1% drop or ointment - 3 times daily
- Corticosteroid drops – Betamethasone/Dexamethasone/Prednisolone acetate etc. 4 to 6 hourly depending upon the severity of the inflammation
- Hot compress – 2 to 4 times daily
- Use of dark glasses

Systemic:

- Anti inflammatory-Aspirin/Indomethacin/Corticosteroid etc
- Antiglaucoma – If IOP is raised:
 - Tab. Acetazolamide (250 mg) 3 -4 times daily
 - Timolol maleate eye drop 0.5% BD
- Systemic antibiotics/anti TB drugs in selected cases
- Immunosuppressive agents – rarely



Q.55. What is the role of atropine in iridocyclitis?

- Ans.**
- Keeps the iris and ciliary body at rest.
 - Decreases hyperemia.
 - Prevents formation of posterior synechiae and breaks down early synechiae.

Q.56. What are the different routes through which corticosteroids can be given?

- Ans.** Topical drops, oral, subconjunctival, subtenon, intravitreal injection.

Q.57. What is chronic anterior uveitis?

- Ans.** The inflammation of anterior uvea that lasts longer than 3 months is termed as chronic anterior uveitis.

Q.58. What are the complications of iridocyclitis?

- Ans.**
- Posterior synechiae
 - Seclusio pupillae
 - Iris bombe
 - Occlusio pupillae
 - Secondary glaucoma
 - Cyclitic membrane (exudative membrane covering the ciliary body and post lens surface)
 - Complicated cataract
 - Cystoid macular edema (due to release of toxin)
 - Phthisis bulbi
 - Band keratopathy
 - Tractional RD – due to traction on retina by cyclitic membrane

Q.59. What is hypertensive uveitis?

- Ans.** Increased viscosity of the plasmoid aqueous and blockage of trabecular meshwork by inflammatory cells causes an elevated IOP. This is known as hypertensive anterior uveitis.

Q.60. What type of IOL is preferred for cataract surgery in case of iridocyclitis?

- Ans.** Heparin coated IOL (it reduces inflammatory deposits on lens surface).

Q.61. Classify uveitis anatomically.

- Ans.**
- Anterior uveitis:
 - Iritis



- Iridocyclitis
- Anterior cyclitis
- Intermediate uveitis:
 - Posterior cyclitis
 - Pars planitis
- Posterior uveitis:
 - Choroiditis
 - Chorioretinitis
 - Retinochoroiditis
- Panuveitis (whole uveal tract is involved)

Q.62. Classify uveitis clinically.

- Ans.**
- Acute uveitis – Sudden onset, persists for less than 6 weeks
 - Chronic uveitis – If uveitis persists for more than 6 weeks

Q.63. How will you classify uveitis aetiologically?

- Ans.**
- Idiopathic
 - Exogenous – external trauma
 - Endogenous – Infective
 - Immune related

Q.64. What is granulomatous uveitis?

- Ans.**
- Uveitis characterized by collections of epithelioid and giant cells surrounded by lymphocytes in nodular form, caused by microbial infection
 - Insidious onset
 - Medium and large KPs
 - Iris nodules

Q.65. What is mutton fat KP?

- Ans.** These are large, creamy white KPs, seen in granulomatous uveitis.

Q.66. What is non-granulomatous uveitis?

- Ans.**
- Uveitis consisting cellular infiltrate of lymphocytes and plasma cells
 - Allergic in nature
 - Acute onset
 - Small KPs

Q.67. What is masquerade syndrome?

- Ans.** Conditions which mimic uveitis, e.g.



Malignancy:

- Retinoblastoma
- Leukemia
- Malignant melanoma
- Lymphoma
- Juvenile Xanthogranuloma

Q.68. What are the indications of immunosuppressive agents therapy in uveitis?

- Ans.**
- VKH syndrome
 - Sympathetic Ophthalmia
 - Behcet's syndrome

Q.69. What is panophthalmitis?

Ans. Suppurative uveitis involving all the coats of eyeball is known as panophthalmitis.

Q.70. Enumerate the etiology of panophthalmitis?

- Ans.**
- Penetrating ocular injury, esp. with retained intraocular foreign body
 - Post-operative bacterial or fungal infections

Q.71. What are the clinical features of panophthalmitis?

- Ans.**
- Constitutional symptoms: fever, headache, vomiting
 - Severe ocular pain
 - Marked diminution of vision
 - Proptosis with intense swelling of the lids
 - Cloudy cornea
 - Chemosis of conjunctiva
 - Both ciliary and conjunctival congestion
 - Restricted and painful ocular movements
 - Massive hypopyon
 - Eye is very tender
 - IOP is often raised
 - Purulent retinochoroiditis ; vitreous cavity becomes a bag of pus
 - In severe cases, eyeball may rupture near the limbus

Q.72. How will you treat a case of panophthalmitis?

- Ans.** Emergency; requires immediate treatment.
- Perforation following ocular trauma is treated immediately; subconjunctival injection of Gentamicin/Amikacin given along with systemic antibiotics.



- Post-operative sepsis- intravitreal injections of antibiotics and vitrectomy
- Severe cases- evisceration

Q.73. What is endophthalmitis?

Ans. Intraocular inflammation involving the vitreous, anterior chamber, retina and choroid is known as endophthalmitis.

Q.74. What are the causes of endophthalmitis?

- Ans.**
- Infectious
 - Exogenous:
 - a. Postoperative
 - b. Post-traumatic
 - c. Bleb infection
 - Endogenous
 - Noninfectious (sterile)

Q.75. How will you treat a case of post surgical endophthalmitis?

- Ans.**
- Broad spectrum antibiotic coverage by intravenous route for both Gram positive and Gram negative organisms:
 - Intravitreal- Vancomycin/Amikacin
 - Subconjunctival- Vancomycin/Ceftazidime
 - Topical- Vancomycin/Amikacin
 - Severe cases- pars plana vitrectomy

Q.76. What is pars planitis?

Ans. Uveitis involving vitreous, ciliary body and peripheral retina, also known as intermediate uveitis.

Q.77. What are the clinical features of pars planitis?

- Ans.**
- Chief presenting symptom: presence of floaters in both eyes
 - Anterior segment usually quiet
 - Anterior vitreous contains numerous cells
 - Snowball opacities near inferior retina (by indirect ophthalmoscopy)
 - Peripheral vasculitis associated with sheathing, exudation and vascular occlusion



Q.78. What is the treatment of pars planitis?

- Ans.**
- **Corticosteroid:** Sub-Tenon route; First line of treatment
 - Pars plana vitrectomy with induction of posterior hyaloid separation, photocoagulation
 - Systemic immunosuppressive agents, if all above fail

Q.79. Name some joint disorders which are associated with uveitis?

- Ans.**
- Ankylosing spondylitis
 - Juvenile rheumatoid arthritis
 - Reiter syndrome

Q.80. Name few congenital anomalies of the uveal tract.

- Ans.**
- Heterochromia
 - Anomalies of pupil: corectopia, polycoria
 - Aniridia
 - Persistent pupillary membrane
 - Coloboma of uveal tract
 - Cysts of iris

Q.81. What is aniridia?

Ans. Rare condition where iris is absent.

Q.82. What is iris coloboma?

Ans. Nonclosure of the fetal fissure in the inferior part of the eye results in a pear shaped pupil, known as iris coloboma.

Q.83. What is choroiditis (posterior uveitis) ?

Ans. Inflammation of choroid.

Q.84. What are the types of choroiditis?

- Ans.** A. According to the number and location of the areas involved.
1. Disseminated or diffuse choroiditis
 2. Multifocal choroiditis
 3. Central choroiditis
 4. Juxtapapillary choroiditis (of Jensen)



B. According to clinical presentation:

1. Granulomatous
2. Non granulomatous

Q.85. What are the clinical features of posterior uveitis?

Ans. Symptoms:

- Presence of 'floaters'
- Painless diminution of vision
- Photophobia
- Redness of eye (if anterior segment is involved)

Signs:

- Detected inflammatory cells and opacities in the vitreous (vitritis)
- Exudation or infiltration in the retina or choroid
- Oedema of retina and choroids
- Sheathing of vessels

Q.86. What is rubeosis iridis?

Ans. Neovascularisation of iris (NVI)

Q.87. What are the causes of rubeosis iridis?

- Ans.**
- Proliferative diabetic retinopathy
 - Central retinal vein obstruction (CRVO)
 - Chronic uveitis

Chapter

7

Glaucoma

Q.1. Describe the formation of aqueous Humor.

Ans. Active secretion:

- 80% aqueous humor is secreted by the non-pigmented ciliary epithelium.
- It is an active metabolic processes which needs a number of enzymatic system.



It depends on blood pressure in the ciliary capillaries, the plasma oncotic pressure and the level of IOP.

Q.2. Describe the drainage of aqueous.

Ans. (Figs 7.1a, 7.1b)

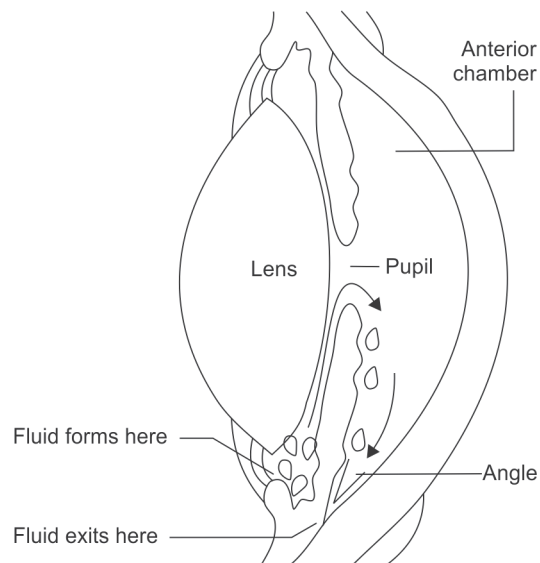


Fig. 7.1a : Drainage of Aqueous

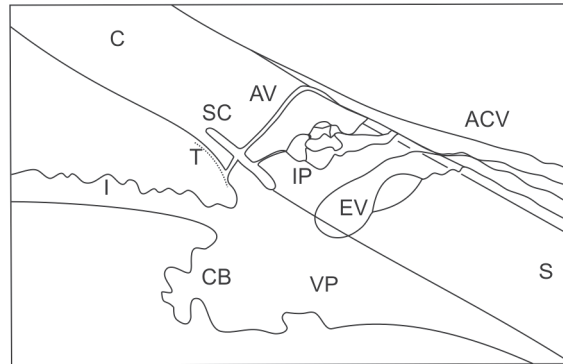
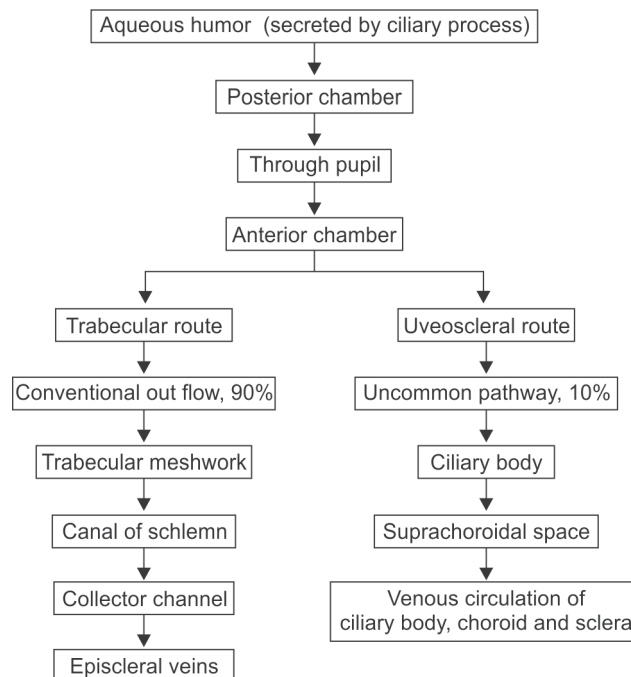


Fig. 7.1b : The region of the Angle of the Anterior Chamber.

CB : Ciliary Body , I : Iris, T : Trabecular Meshwork, SC : Schlemm's Canal
 IP : Intra scleral Plexus , AV : Aqueous Vein, EV : Efferent Vein, ACV : Anterior Ciliary Vein, VP : Venous Plexus
 C : Cornea, S : Sclera

Flow chart depicting drainage of aqueous humour





Q.3. What is blood aqueous barrier?

Ans. It is formed by tight junction between the cells of non pigmented epithelium and capillary endothelium of ciliary body . It allows only selected pharmacological molecules to pass through the barrier, which is useful for intraocular infiltration.

Q.4. What is the normal IOP ?

Ans. 10 mm of Hg to 21 mm of HG (mean 16 mm of HG).
(21 mm of Hg is considered the upper limit of normal).

Q.5. What factors determine the level of IOP?

Ans.

- Rate of aqueous secretion
- Resistance encountered in the outflow channels
- Level of episcleral venous pressure

Q.6. What is diurnal variation of IOP?

Ans. Physiological variation of IOP by 3-4 mmHg in morning and evening (high in the morning).

Q.7. What is the diurnal fluctuation in normal eye?

Ans. 4 mm of Hg

Q.8. What is the diurnal variation in glaucomatous eye?

Ans. 8 mm of Hg

Q.9. Define Glaucoma.

Ans. It is a chronic, progressive optic neuropathy caused by a group of ocular condition which leads to damage of optic nerve with loss of visual function .

Q.10. What is the pathogenesis of glaucomatous damage?

Ans.

- The Indirect Ischaemic theory – high IOP causes death of nerve fibre by interfering with the microcirculation of the optic nerve head.
- Direct mechanical theory – high IOP directly damages the residual nerve fibres as they pass through the optic nerve head (stasis of axoplasmic flow).

Q.11. What are the causes of glaucoma?

Ans. a. Obstruction of flow of aqueous humor from posterior chamber to anterior chamber, e.g. pupillary block glaucoma.



- b. Hypersecretory Glaucoma - Excess secretion of aqueous humour, e.g. epidemic dropsy.
- c. Blockage of angle due to peripheral anterior synechiae - PACG.
- d. Blockage of angle of AC due to sclerosis of the trabecular meshwork – POAG.
- e. High episcleral venous pressure e.g. - Sturge Weber syndrome.

Q.12. Classify Glaucoma.

Ans. Primary:

- a. Congenital
- b. Open angle
- c. Closed angle

Secondary:

- a. Steroid induced
- b. Neovascular
- c. Post-traumatic
- d. Lens induced
- e. Inflammatory – corneal ulcer, iridocyclitis
- f. Hypopyon/Hyphaema
- g. Epidemic dropsy
- h. Vitreous haemorrhage/I.O. tumor
- i. Aphakic glaucoma

Q.13. Why is it called primary glaucoma?

Ans. It occurs in absence of any ocular or systemic disorders.

- Obstruction is confined to anterior chamber or conventional outflow pathway.
- These are bilateral and probably genetically predisposed.

Q.14. Describe the anatomy of anterior chamber (AC).

Ans. AC is bounded:

- Anteriorly by the posterior surface of the cornea.
- Posteriorly by the anterior surface of iris, and part of anterior surface of lens.

The peripheral recess of the AC is known as *angle of AC*, which is bounded anteriorly by the corneo -sclera and posteriorly by the root of the iris and the ciliary body.



Q.15. What is the volume of AC?

Ans. 0.25 ml

Q.16. What is the depth of the AC?

Ans. 2.5-3 mm (central)

Q.17. What is the volume of posterior chamber?

Ans. 0.06 ml

Q.18. What is the most common risk factor for glaucoma?

Ans. Raised IOP

Q.19. What are the symptoms of POAG?

Ans. Generally asymptomatic:

- Painless progressive dimness of vision
- Non-specific complaints e.g headache
- Frequent change of presbyopic glass
- Difficulty in dark adaptation
- Diminished field of vision

Q.20. What are the classical triad of POAG?

Ans.

- Raised IOP
- Cupping of the optic disc
- Characteristic visual field defects

(at least 2 of the 3 signs must be present to make a diagnosis of POAG)

Q.21. What are the optic disc findings in POAG?

Ans.

- Cup : disc ratio > 0.3
- Asymmetry of cupping between the two optic nerve head > 0.2
- Narrowing, notching or pallor of the neuroretinal rim (NRR)
- Disc haemorrhage
- Vascular changes
- Laminar dot sign
- Peripapillary atrophy



Q.22. Which diameter is measured for C:D ratio?

Ans. Vertical diameter of the cup. (Fig .7.2)

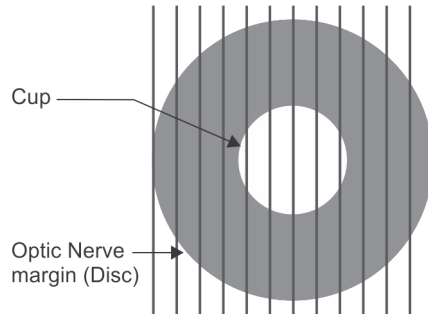


Fig. 7.2 : Diagram of optic nerve head

Q.23. What are the predisposing factors for POAG?

Ans.

- Genetic predisposition
- Race – Black
- Age – Elderly (4–6 decade)
- Myopia
- Diabetes mellitus

Q.24. What is the pathogenesis of POAG?

Ans. Increase resistance to the outflow of the aqueous at the TBM, especially at the juxtacanalicular region, this is partially due to age related changes in the tissue.

Q.25. What are the vascular changes on the optic nerve head in a case of POAG?

Ans.

- Baring of the circumlinear vessels
- Overpass of the central vessels

Q.26. What are the visual field defects seen in POAG?

Ans. (Fig 7.3)

- Baring of the blind spot
- Relative paracentral scotomas
- **Seidel's scotoma** – A sickle shaped defect extending from the blind spot above or below or both
- **Bjerrum's scotoma/arcuate scotoma**-appears to start at the superior or inferior poles of the blind spot and reach over macular area, widening as they curve down or up to end as a horizontal line nasally which never crosses the horizontal divide of the visual field.

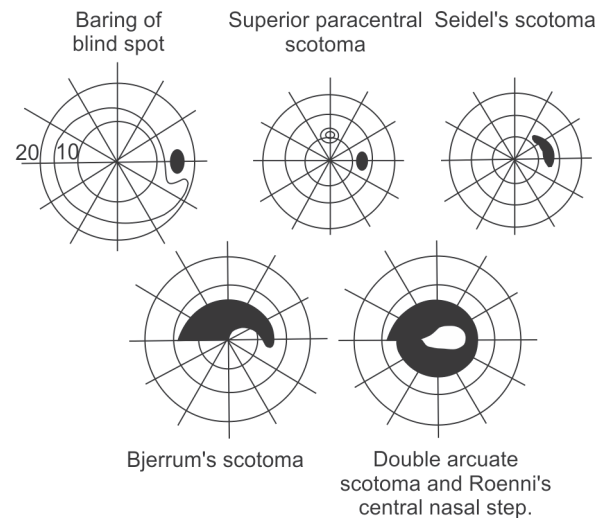


Fig. 7.3 : Field defects in POAG

- Double arcuate or ring scotoma—advanced glaucomatous defect. Superior and inferior arcuate scotoma may join to form ring scotoma.
- Roenne's nasal step—upper or lower area ends as a horizontal line nasally, which is sharp.
- End stage - Residual temporal or central patch of island may remain.

Q.27. Which is Bjerrum's area?

Ans. Field defects in glaucoma are initially seen in this area (10-20° from fixation).

Q.28. What is blind spot?

Ans. An area of blindness in the visual field corresponding to the site of optic nerve which is devoid of photoreceptors.

Q.29. What are the minimum investigations to be done in POAG?

Ans.

- Applanation/Schiotz tonometry
- Gonioscopy
- Automated perimetry

Q.30. Name some modalities for early detection of POAG.

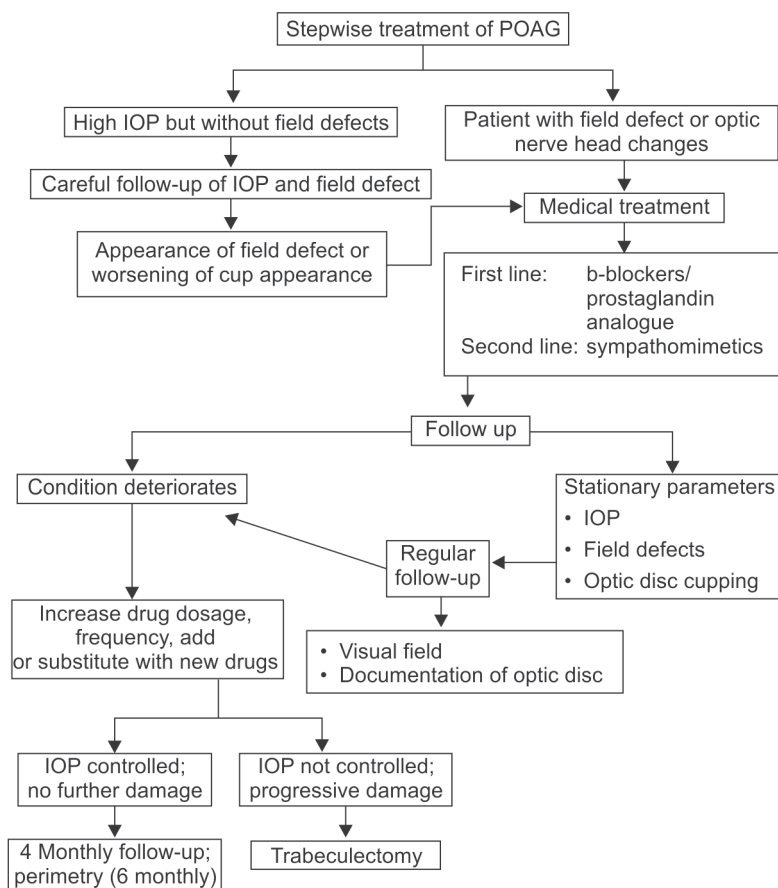
Ans.

- Short wave length AP
- Scanning laser ophthalmoscopy (SLO)
- GDX
- Polarimetry



Q.31. How will you treat a case of POAG?

- Ans.**
- Medical
 - Surgical
 - Laser
 - Combination therapy





Q.32. Describe the common antiglaucoma medications in brief.

Ans.

Antiglaucoma Medications				
Pharmacological group	Name of drug	Ocular side-effects	Systemic side-effects	Contraindications
Parasympatho mimetics	Pilocarpine 2% – 4%	Fluctuating and blurred vision, miosis, browache, accommodative spasm	Increased sweating and salivation, urinary frequency, diarrhoea, bronchospasm	Uveitis, posterior polar cataracts
Adrenergic antagonists				
Non-selective betablockers	Timolol 0.25 – 0.5%	Stinging sensation	Bronchospasm bradycardia, decreased cardiac output, hypotension, depression, impotence, altered lipid profile	Asthma, COPD, CCF, sinus bradycardia, a-v blocks. Sinus bradycardia.
Selective beta blockers	Betaxolol	Stinging sensation	Less pulmonary side-effects.	
Sympatho mimetic				
Non-selective	Dipivefine 0.1%	Follicular conjunctivitis, BP, rebound congestion, macular oedema in aphakia	Increase BP, arrhythmias	
Selective	Brimonidine	Conjunctival blanching, ocular allergy.	Drowsiness, fatigue, blood pressure changes	
Carbonic anhydrase inhibitors	Acetazolamide (250 mg)		Parasthesia of finger and toes. K-depletion. Urinary frequency. Abdominal discomfort. cramps nausea, diarrhea.	Hypersensitivity to sulphonamides, eye injury, Ocular infection or inflammation.
Parasympatho-mimetics	Pilocarpine 2-4%	Fluctuating and blurred vision, miosis, browache, accommodative spasm. Stringing sensation	Increased sweating and salivation, urinary frequency, diarrhoea, bronchospasm	Uveitis, posterior polar cataract

Q.33. What surgery is done for POAG?

Ans. Trabeculectomy

Q.34. When is surgery advised?

Ans.

- If medical treatment fails
- Non-compliance to the medication



Q.35. What type of laser is used?

Ans. Argon laser

Procedure – Argon Laser Trabeculoplasty (ALT)

Q.36. Is it necessary to have high IOP to diagnose a case of glaucoma ?

Ans. No

In normal tension glaucoma (NTG) the IOP is normal or low

Q.37. Define PACG.

Ans. PACG includes a spectrum of condition in which peripheral iris moves forwards to block the TBM at the angle causing a rise of IOP.

Q.38. Which sex is mostly affected in PACG?

Ans. Females (4 times)

Q.39. What are the predisposing factors for PACG?

Ans. Anatomical factors

- Short eye (hypermetropia)
- Smaller corneal diameter
- Shallow AC
- Relative anterior positioning of lens -iris diaphragm
- Bigger size of lens

General factors

- Age – 5th decade
- Sex – F:M – 1:4
- Personality – nervous patient
- Season – rainy season
- Family history – may be inherited
- Race – more common in South east Asians

Q.40. What are the precipitating factors of PACG?

Ans. In a predisposed eye, any of the following factors may precipitate an attack:

- Dim illumination
- Emotional stress
- Use of mydriatic drugs, e.g. atropine, cyclopentolate, tropicamide

Q.41. What are the causes of shallow AC?

Ans.

- Primary angle closure glaucoma
- Intumescent cataract



- Hypermetropia
- Hypermature Morgagnian cataract
- Post operative shallow AC due to wound leak

Q.42. What are the causes of deep AC?

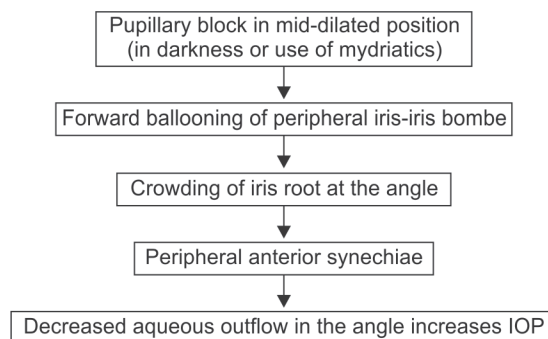
- Ans.**
- Aphakia
 - Myopia
 - Buphthalmos
 - Keratoconus

Q.43. What is the normal degree of anterior chamber angle?

Ans. $35^{\circ} - 45^{\circ}$

Q.44. What is the mechanism of angle closure?

Ans.



Q.45. What are the stages of ACG?

- Ans.**
- Prodromal stage
 - Phase of constant instability
 - Acute congestive attack
 - Chronic angle closure
 - Absolute glaucoma

Q.46. What are the factors which precipitate an acute attack?

- Ans.**
- Dim illumination (cinema hall)
 - Emotional stress
 - Use of mydriatic

Q.47. What are the clinical features of ACG?

Ans. Symptoms

- Acute intense pain radiating along the distribution of 5th cranial nerve



- Headache, nausea, vomiting (often mistaken for acute abdomen)
- Decreased vision
- Redness, lacrimation, photophobia

Signs

- Lid oedema
- Ciliary congestion
- Hazy, cloudy, insensitive cornea
- Shallow AC
- Mid-dilated vertically oval, non-reacting pupil
- Raised IOP (40-60mm Hg)
- VA- HM/ PL/PR
- Fundus- Not visualized due to corneal oedema
- Instillation of glycerine drop clears cornea
- Fundus- hyperemic disc, disc haemorrhage, arterial pulsation, no cupping

Q.48. What is the finding in the fellow eye?

- Ans.**
- Shallow AC
 - Narrow angle

Q.49. How the angle of anterior chamber is visualized?

Ans. The method is called gonioscopy (by using goniolens).
(Fig. 7.4)

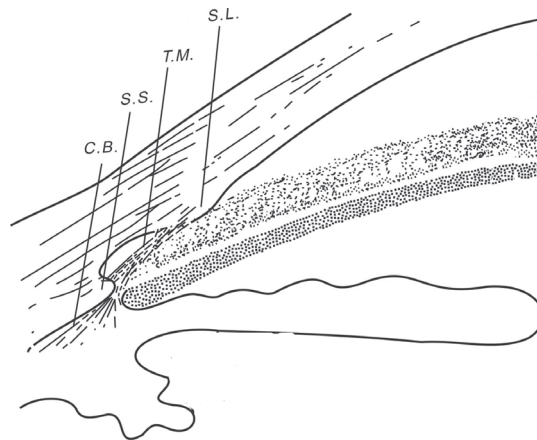


Fig. 7.4 : Gonioscopic view of anterior chamber angle

CB – Ciliary body band	SS – Scleral spur
TM – Trabecular meshwork	SL – Schwalbe's line



Q.50. What are the different types of gonioscopes?

Ans. Direct – Koeppe lens

Indirect – Goldmann and Zeiss lens

Q.51. Why is angle of anterior chamber not visible directly?

Ans. Because of total internal reflection of light (emitted from angle structures) at anterior surface of cornea it is not possible to visualize the angle of anterior chamber directly.

Q.52. Describe the angle structures.

Ans. (Seen from behind forwards)

- Root of the iris
- Ciliary body band
- Scleral spur
- Trabecular meshwork and Schlemm's canal
- Schwalbe's line

Q.53. What is trabecular meshwork (TBM)?

Ans. At the periphery of the angle, between Schlemm's canal and the recess of the AC, there lies a loose meshwork of tissues, which is known as TBM.

Q.54. What are the different parts of trabecular meshwork?

Ans.

- **The uveal meshwork** – the innermost portion ; extends from root of iris to Schwalbe's line.
- **The corneoscleral meshwork** – Larger middle portion , extends from scleral spur to Schwalbe's line.
- **The juxtacanalicular meshwork** – Narrow outer part, links the corneoscleral meshwork with the endothelium. It offers the major part of normal resistance to aqueous flow.

Q.55. What is Schlemm's canal?

Ans. Circular venous sinus within the inner layer of the sclera (scleral sulcus) at the angle is called the canal of Schlemm:

- It is of great importance in the drainage of the aqueous.
- Collector channels, 25–30 in number, leave SC (Schlemm's canal) to terminate in the episcleral veins.

**Q.56. What are the methods of measuring AC depth?**

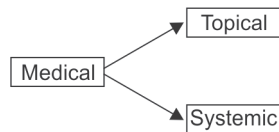
- Ans.**
- Oblique torch light illumination
 - Van Herrik's slit lamp grading

Q.57. How to assess AC depth with torch light?

- Ans.**
- If we throw light over temporal limbus, nasal part of limbus and nasal half of iris will be illuminated.
 - If AC is shallow, only part of nasal iris will be illuminated.

Q.58. How will you treat acute attack?

Ans. Usually the IOP is >50 mm of Hg.



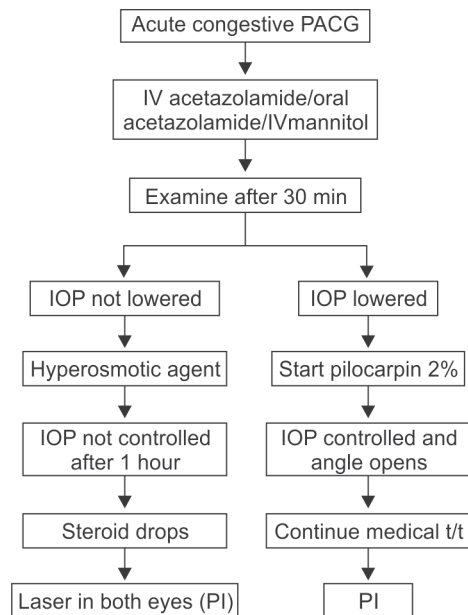
- Mannitol (20%) I.V 1.5-2 gm/kg body weight (rapid drip) or Acetazolamide I.V 500 mg
- If the pupil is still blocked- press over central part of cornea with a sterile cotton bud to open the peripheral block
- Oral glycerine (50%) with lemon juice TDS can be given
- Pilocarpine 2% drop is started after the IOP is reduced by Mannitol (in a very high IOP Pilocarpine can not act on sphincter muscle as it becomes ischaemic)
- Beta blockers- BD
Timolol maleate 0.5%
Betoxolol 0.5
- Analgesic systemically
- Steroid drops to reduce congestions
- LASER treatment – once the IOP is reduced and eye becomes quite – YAG LASER peripheral iridotomy is done
- Surgical treatment – if the medical and LASER treatment fails to control the IOP, then trabeculectomy is done



Q.59. How will you treat the fellow eye?

Ans. YAG Laser iridotomy/Surgical iridectomy (PI).

MANAGEMENT OF ACUTE PACG



Q.60. Why medical and LASER treatment may fail?

Ans. If there is formation of PAS (> 50% of angle).

Q.61. What are the after effects of an acute attack?

Ans.

- Reduced vision
- Corneal edema
- Shallow AC
- Iris – sector atrophy
- Pupil – irregular
- Anterior capsular opacity, glaucomflecken
- Optic disc – hyperemic

Q.62. What is plateau iris configuration?

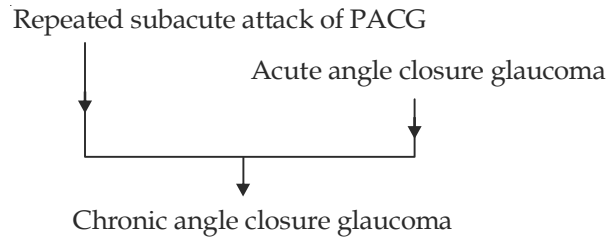
Ans. An anterior insertion of iris root with narrow angle, but relatively flat iris surface.

It can cause glaucoma if pupil is dilated.



Q.63. What is chronic angle closure glaucoma?

Ans. If the IOP is chronically raised in the eyes having synechial closures (> 50% of angle).



Q.64. What is absolute glaucoma?

Ans.

- It is the end stage of all types of glaucoma, characterized by high IOP with no PL.
- It can lead to atrophic bulbi due to atrophy of ciliary body.

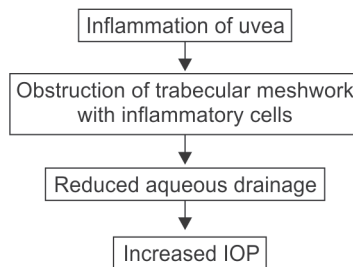
Q.65. What is secondary glaucoma?

Ans. Glaucoma due to some other reasons (angle closure glaucoma/open angle glaucoma)

- Ocular infection
- Inflammation
- Lens induced
- Trauma
- Surgery

Q.66. What is hypertensive uveitis?

Ans.



Q.67. How will you treat absolute glaucoma?

Ans.

- Cyclocryotherapy → cyclodestruction of ciliary body → reduced IOP
- Retro bulbar injection of 70% alcohol to destroy ciliary ganglion



Q.68. What are the causes of lens induced glaucoma?

Ans. Secondary angle closure glaucoma:

Intumescent cataract
Traumatic rupture of lens capsule } **phacomorphic glaucoma**
Anterior subluxation or
dislocation of lens

Secondary open angle glaucoma:

Hyperature cataract (phacolytic glaucoma)

Q.69. What is phacolytic glaucoma?

Ans.

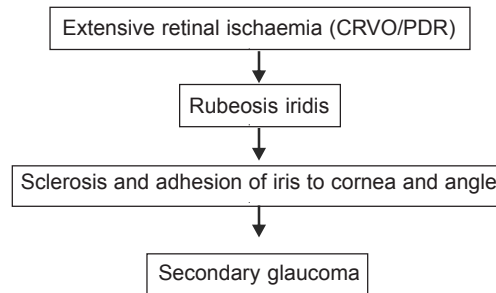
- Escape of lens protein into the aqueous from a trauma or hyperature senile cataract, blocks the angle.
- Cortical lens matter are engulfed by lymphocytes and blocks the trabecular meshwork.

Q.70. What is the treatment of lens induced glaucoma?

Ans. Reduction of IOP by medical therapy → extraction of lens

Q.71. What is neovascular glaucoma?

Ans.



Q.72. What is aphakic or pseudophakic glaucoma?

Ans.

- Commonest form of secondary glaucoma
- Rise of IOP after cataract surgery
- Mostly due to surgical complications.

May be due to:

- a. Use of viscoelastic
- b. Distortion of angle structures due to tight limbal sutures
- c. Severe post operative inflammation
- d. AC lens
- e. Retained cortical matter

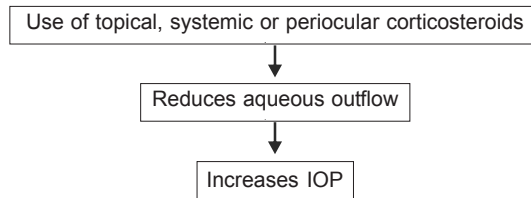


Q.73. How to treat secondary glaucoma?

- Ans.** a. Treat the cause of glaucoma.
b. Treat glaucoma medically/surgically (same as POAG, PACG).

Q.74. What is steroid induced glaucoma?

Ans.



Q.75. Does it occur in all cases?

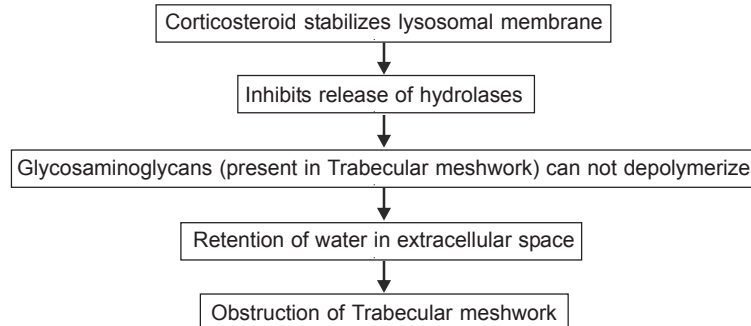
Ans. No. This tends to occur in the eyes genetically predisposed individuals (5–6% of normal population) after 4–6 weeks of treatment.

Q.76. What are the clinical features of steroid induced glaucoma?

Ans. Same as primary open angle glaucoma.
If IOP is very high → corneal edema → coloured halos.

Q.77. What is pathogenesis of steroid induced glaucoma?

Ans.



Q.78. How to treat the condition?

- Ans.** a. Stop the steroid, start antiglaucoma drugs
b. Trabeculectomy

Q.79. What is malignant glaucoma?

Ans. Normal flow of aqueous is blocked at the bend of ciliary body, lens or anterior vitreous face, causing misdirection posteriorly of aqueous into the vitreous → pushes iris, lens forward → pupillary block.



Q.80. What are the clinical features of malignant glaucoma?

Ans. The condition is seen after trabeculectomy / cataract surgery.

- AC- shallow or flat
- Increased IOP (very high)

Q.81. What are the treatment modalities of malignant glaucoma?

- Ans.**
- a. Cycloplegic agent – Atropine
 - b. Mannitol
 - c. Beta-blocker
 - d. YAG laser hyaloidotomy

Q.82. What is the surgical management of glaucoma?

- Ans.**
- Surgical iridectomy
 - Filtering operation – trabeculectomy

Q.83. Define congenital glaucoma.

- Ans.**
- It is defined as glaucoma appearing between birth and age of 3–4 years.
 - Up to this age the eyeball is distensible so that the eye enlarges progressively when the IOP is increased.
 - Usually bilateral; also called Buphthalmos (eye of an ox).
 - It may occur without any ocular finding (*primary congenital glaucoma*)
Or may be associated with some ocular defects (*secondary congenital glaucoma*)

Q.84. What is the pathogenesis of primary congenital glaucoma?

Ans. It is due to failure or abnormal development of trabecular meshwork.

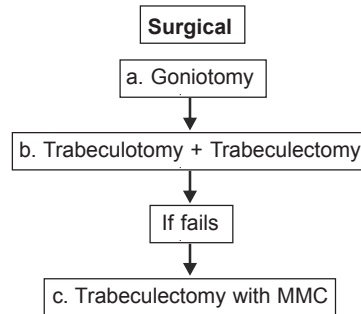
Q.85. What are the clinical features of congenital glaucoma?

- Ans.**
- Enlarged cornea
 - Haziness due to corneal edema
 - Watering
 - Photophobia
 - Corneal opacities (Haab's striae due to rupture of Descemet's membrane)
 - Thinner bluish sclera
 - Deep AC
 - Iridodonesis
 - Myopic shift
 - Deep cupping



Q.86. What is the treatment of congenital glaucoma?

Ans. Medical treatment is not very effective.



Q.87. What is juvenile POAG?

Ans. POAG developing between 10–35 years is termed juvenile POAG.

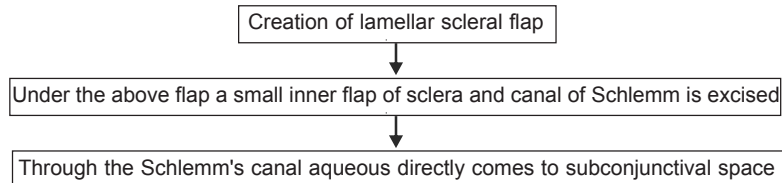
(4–10 years, developmental glaucoma).

Q.88. What is trabeculectomy?

Ans. It is a glaucoma surgery in which a portion of trabecular meshwork is excised along with iridectomy. It is a guarded filtration surgery.

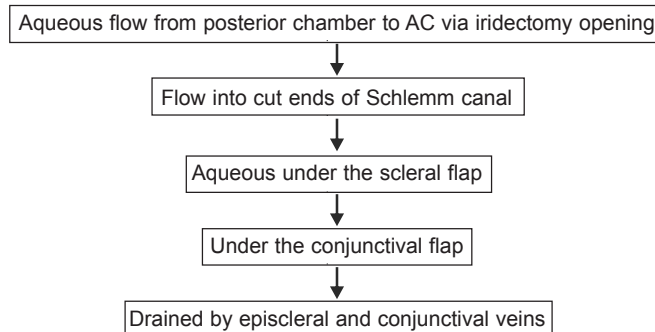
Q. 89. What is the principle of trabeculectomy?

Ans.



Q.90. What is its mechanism of action?

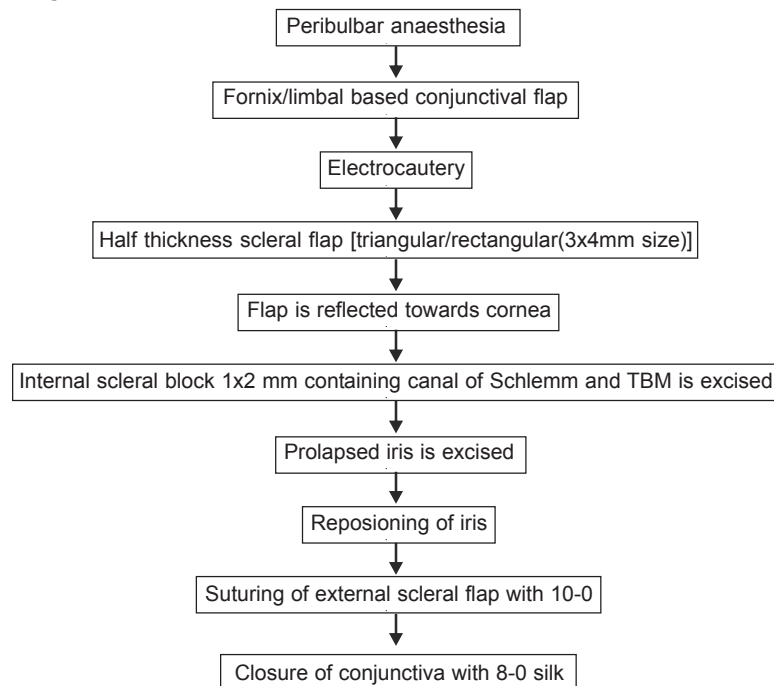
Ans.





Q.91. Describe the steps of trabeculectomy?

Ans.



Q.92. What is the ideal site for trabeculectomy?

Ans. 12 o' clock (preferred)

Other sites-supero nasal and supero temporal

Q.93. What are the different types of tonometry you know?

Ans. Digital tonometry

Indentation Tonometry

- Schiötz tonometry

It uses a plunger with a pre-set weight to indent the cornea. The amount of indentation is converted into millimeters of mercury by the use of tables.

Applanation Tonometry

- Goldmann applanation tonometer (attached to the slit lamp). The tear film is stained with Fluorescein dye and by adjusting the two mires IOP is measured (Fig. 7.5a and b).

Gives measurement of how much pressure is applied to make cornea flat.

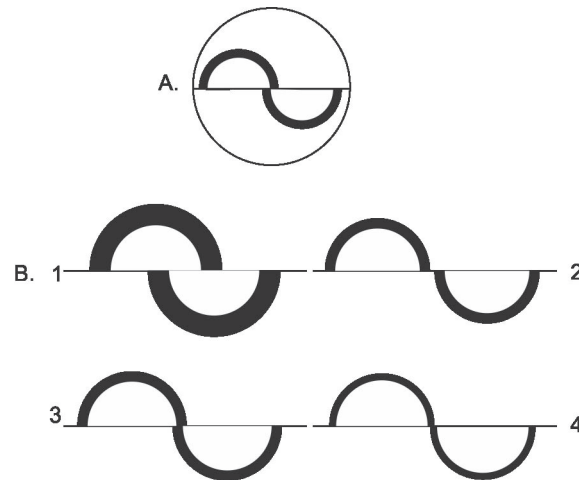


Fig. 7.5a : Fluorescein stained mires of the applanation tonometer when perfectly aligned.

Fig. 7.5b : 1 and 2. Not aligned; 3. Too thick mires, falsely low IOP will be recorded; 4. Too thin mires, falsely high IOP will be recorded.

- Perkins tonometer (hand held)
- The air-puff tonometer (non contact). Useful for mass screening
- The Tono-Pen

Q.94. Which tonometry is more accurate? Why?

Ans. Goldmann applanation tonometry.
Schiotz tonometry is less accurate as it is affected by corneal curvature, thickness and ocular rigidity.

Q.95. How will you measure IOP in presence of corneal oedema or corneal scarring?

Ans.

- Mackay – Marg
- Tonopen
- Pneumotonometer

Q.96. How will you measure IOP in an AIDS patient?

Ans. By non contact tonometry (air puff tonometer)

Q.97. What is the relationship between central corneal thickness (CCT) and measurement of IOP ?

Ans. A thick cornea will lead to over and a thin cornea to under-estimation of IOP.

Chapter

8

Retina and Vitreous

Q.1. What is the literal meaning of retina?

Ans. Net.

It is the innermost nervous tunic of eye.

Q.2. What is the thickness of retina?

Ans. Ora serrata – 100 micron
Macula – 350 micron
Equator – 150 micron
Fovea – 90 micron

Q.3. What are the layers of the retina?

Ans. (Fig. 8.1)

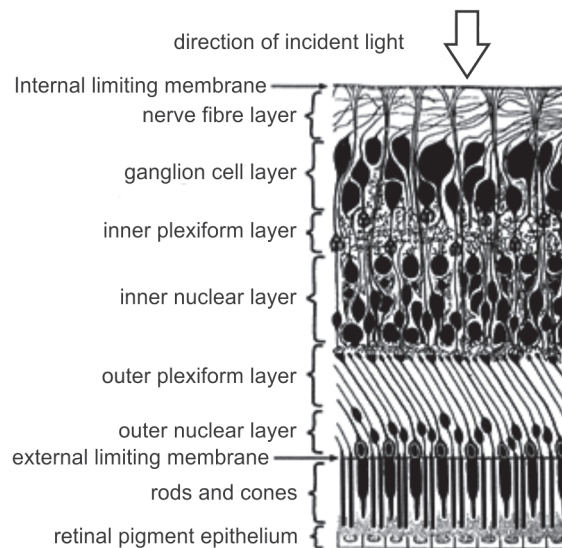


Fig. 8.1 : Layers of retina



Retina consists of ten layers.

From outside inwards :

- Retinal pigment epithelium (RPE)
- Layer of rods and cones
- External limiting membrane
- Outer nuclear layer
- Outer plexiform layer
- Inner nuclear layer
- Inner plexiform layer
- Ganglion cell layer
- Nerve fibre layer
- Internal limiting membrane

Q.4. How many rods and cones are there in retina?

Ans.

- Rods – 125 millions
- Cones – 7 millions

Q.5. What is the function of rods and cones?

Ans.

- Cones – acuity of vision, color vision, bright light vision
- Rods – scotopic vision (dim light vision)

Q.6. What is ora serrata?

Ans. It is the anterior termination of retina located 8 mm from limbus.

Q.7. What is macula lutea?

Ans. (Fig. 8.2)

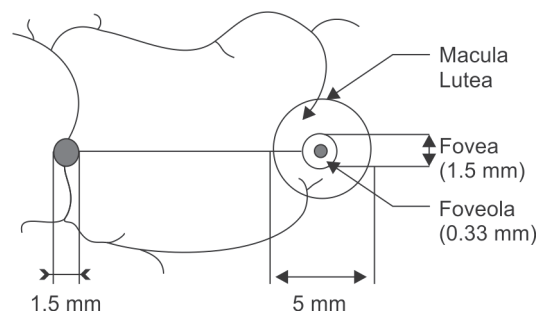


Fig. 8.2 : Relative position of macula and size of its different parts.

It is the central retina 5.5 mm in diameter. It contains yellow pigment xanthophylls. It is also called yellow spot.



Q.8. What is foveal centralis ?

Ans. It is the depressed central area of macula. It is 2 disc diameter (3 mm) temporal to the disc and half disc diameter below the horizontal midline.

It measures 1.5 mm. Its central depression is called foveola. The fovea is supplied by choriocapillaries.

Q.9. Why is the visual acuity maximum at the foveola?

Ans. One cone is connected to one ganglion cell unlike other parts of retina (1:100).

Q.10. Which photoreceptors are present in fovea?

Ans. Cones.

Q.11. What are the locations of the rods?

Ans. They are situated in peripheral retina.

Q.12. What is the diameter of the optic disc?

Ans. 1.5 mm (1500 micron).

Q.13. Which area of the retina represents the blind spot ? Why is it so named?

Ans. Optic disc. As there is no photoreceptor in the optic disc, when light falls on it, no visual impression is excited.

Q.14. Which layer is present in optic disc?

Ans. Only the nerve fibre layer is present.

Q.15. What is optic neuritis?

Ans. It is an inflammation of optic nerve which may be due to demyelinating disorder, local or systemic infection, immune mediated or metabolic causes.

Q.16. What are the clinical features of optic neuritis?

Ans.

- Marked loss of vision
- Ocular pain (in upward gaze)
- Presence of RAPD
- Local tenderness
- Decreased color vision
- Visual field defect- centrocaecal ,arcuate scotoma

Q.17. What are the ophthalmoscopic finding of optic neuritis?

Ans.

- Hyperemic swollen disc
- Peripapillary flame shaped haemorrhage
- Disc margin blurred

(In retro bulbar neuritis-disc is normal)

**Q.18. What is papilloedema?**

Ans. It is defined as oedema of the optic disc or nerve head due to raised intra cranial pressure.

Q.19. How will you differentiate between papilloedema and papillitis?

Ans.

	<i>Papillitis</i>	<i>Papilloedema</i>
Visual acuity	Markedly decreased	Usually normal
Pupillary reaction	RAPD	Normal
Color vision	Impaired	Normal
Disc oedema	Moderate (2-3D)	Upto 6D
Scotometry / Perimetry	Relative Scotoma	Enlargement of blind spot

Q.20. What is diabetic retinopathy(DR)?

Ans. It is a microangiopathy affecting pre-capillary arterioles, capillaries and venules leading to occlusion and leakage → retinal ischaemia and hypoxia → neovascularisation → arteriovenous shunt formation.

Q.21. What is the fundus picture of diabetic retinopathy?

- Ans.**
- Non-proliferative diabetic retinopathy (NPDR)
 - Micro aneurysms – specially in macular region
 - Dot and blot haemorrhage
 - Hard waxy exudate
 - Cotton wool spots
 - Dilatation and irregularities of vessels
 - Intra retinal micro vascular abnormalities (IRMA)
 - Proliferative diabetic retinopathy(PDR)
 - Neovascularisation of disc/other parts of retina
 - Vitreous haemorrhage
 - Neovascularisation of iris (rubeosis iridis)

Q.22. What are the clinical features of diabetic maculopathy?

- Ans.**
- Focal or diffuse macular edema/hard exudates leading to retinal thickening.
 - Macular ischemia



Q.23. What are the risk factors for development of DR?

- Ans.**
- Duration of diabetes (longer the duration greater the risk)
 - Glycemic control (poor control of blood glucose → more risk)
 - Hypertension

Q.24. What is the treatment of DR?

- Ans.**
- Control of blood sugar and blood pressure
 - Periodic fundus examination
 - Fluorescein angiography
 - Photocoagulation (diode laser)

Q.25. What are the clinical features of CRAO?

- Ans.**
- Dramatic, unilateral, sudden, painless loss of vision (exception – presence of cilioretinal arteries).
 - Blockage occurs by an embolus/atherosclerosis of CRA at the lamina cribrosa.
 - Marked afferent pupillary defect.
 - Fundus – Opaque retina, cherry red spot at the macula, segmentation of the blood column in the veins (Cattle trucking).

Q.26. What is the management of CRAO?

- Ans.**
- Massaging the globe
 - Paracentesis
 - Inhalation of amyl nitrite

Q.27. What are the clinical features of CRVO?

- Ans.**
- Sudden, unilateral, painless loss of vision (not as severe as CRAO).
 - Occurs due to atherosclerosis of adjacent CRA that presses the central vein in the common sheath (just behind the lamina cribrosa).
 - Fundus – Dilated, tortuous retinal veins, diffuse haemorrhages in all quadrants of the retina, cotton wool spots.

Q.28. What are the clinical features of BRVO?

- Ans.**
- Sudden painless decrease in VA.
 - A single branch of the central vein is affected.
 - Fundus – Haemorrhages and oedema (to the area supplied by the vein).



Q.29. What are the treatment modalities of venous occlusion?

Ans.

- Investigations of the cause (Hypertension, glaucoma, hypercoagulable state)
- FFA
- Photocoagulation

Q.30. What is CSR?

Ans. Central serous Retinopathy

Q.31. What are the clinical features of CSR?

- Ans.**
- Commonly occurs in young male
 - Sudden D/V
 - Positive scotoma and metamorphopsia
 - Fundus – Circular swelling in the macular area due to RPE/neurosensory retina detachment
 - Usually self resolving
 - Recurrence can occur

Q.32. What is the aetiopathogenesis of CSR?

Ans. Leakage from the choriocapillaries

Q.33. What is the management of CSR?

- Ans.**
- DFA
 - Photocoagulation of leaking points (selective case)

Q.34. What is ARMD?

Ans. Age related macular degeneration (>50 yrs)

Q.35. What are the clinical features of ARMD?

- Ans.**
- Decreased VA (bilateral)
 - Presence of drusens (macula)
 - Geographical atrophy of RPE
 - Subretinal neovascularisation

Q.36. What are the types of ARMD? Which type is more harmful?

Ans. Dry (non-exudative)
Wet (exudative) → more harmful

Q.37. What are the T/t modalities of ARMD?

- Ans.**
- Fluorescein angiography
 - Photocoagulation
 - Intravitreal steroid/anti-VEGF
 - Photodynamic therapy



Q.38. What is ROP?

Ans. Retinopathy of prematurity. It is a fibrovascular proliferation of retina in premature low-birth weight infant.

Q.39. What are the risk factors of ROP?

Ans.

- Low birth weight (< 1.5 kg)
- Oxygen therapy
- Septicaemia
- Acidosis
- Premature (< 28 weeks)

Q.40. When to screen the infants?

Ans. 1 month after birth

Q.41. What is retinal detachment (RD)?

Ans. Separation of the neurosensory retina from underlying retinal pigment epithelium with accumulation of fluid in the potential space between the two layers.

Q.42. What are the different types of RD?

Ans.

- Rhegmatogenous RD
 - presence of break in the retina, e.g in trauma, myopia.
- Exudative RD-no break; retina or choroidal pathology, e.g malignant melanoma.
- Tractional RD - vitreous tractional band, e.g PDR .

Q.43. What is the commonest ocular malignancy in children?

Ans. Retinoblastoma (glioma retinae)

Q.44. From which layer of retina it arises?

Ans. It is a proliferation of neural cells which fails to evolve normally.

Q.45. Which age group is mostly affected?

Ans. Infant and very young children

Q.46. What is the chromosomal abnormality related to retinoblastoma?

Ans. Deletion or mutation of the q14 band of chromosome 13 which is responsible for controlling retinal cell division.

Q.47. How does it clinically present?

Ans.

- Leukocoria (yellowish white pupillary reflex)
- Cataract



- Squint
- Secondary glaucoma
- Hypopyon

Q.48. What are the treatment modalities of retinoblastoma?

Ans. Depending upon the size, location, associated signs, following treatment modalities are present-

- Cryotherapy
- Photocoagulation
- Brachytherapy
- Chemotherapy
- Enucleation – optic nerve is cut as long as possible
- Exenteration (Removal of all the contents of the orbit)

Q.49. What are the differential diagnosis of retinoblastoma?

Ans. Several conditions occurring in children termed as pseudoglioma—
Congenital cataract, Toxocariasis, PHPV.

Q.50. What are the causes of cherry red spot?

Ans.

- Central retinal artery occlusion (CRAO)
- Tay-Sach's disease
- Niemann- Pick's disease
- Gaucher's disease
- Berlins oedema

VITREOUS

Q.51. What is vitreous? Where is it present?

Ans. It is a jelly like transparent substance. It fills the posterior 4/5 th of the eye ball . It occupies the space between the lens and the retina.

Q.52. What is PHPV?

Ans. Persistent hyperplastic primary vitreous.
It is one of the cause of leukocoria (primary vitreous disappears by 8th month of gestation. If it persists – PHPV results.)



Q.53. What is vitreous degeneration? What are the causes?

Ans. Vitreous liquefaction is the most common degeneration.

Causes:

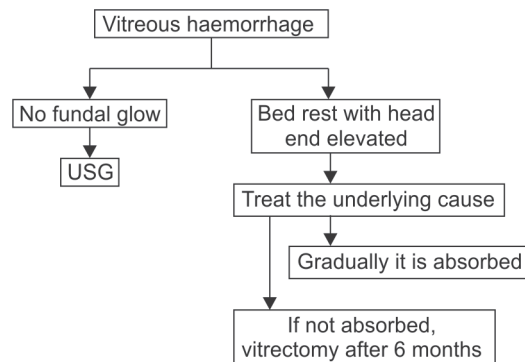
- Old age – senile
- Myopia
- Trauma

Q.54. What are the causes of vitreous haemorrhage?

- Ans.**
- Trauma (common in young)
 - Proliferative diabetic retinopathy (adult)
 - Eales's disease (young)
 - CRVO
 - BRVO
 - RD
 - Sick cell retinopathy

Q.55. What are the clinical features of vitreous haemorrhage?

- Ans.**
- **Symptoms:**
 - Sudden painless loss of vision
 - Floaters
 - Photopsia
 - **Signs:**
 - RAPD (if massive haemorrhage)
 - RBC in vitreous (S/L)
 - Fundal glow absent or poor – Black reflex seen by ophthalmoscope





Prognosis – depends on underlying disease. If no recurrent bleeding – good prognosis

If bleeding recurs, poor prognosis .

Q.56. What is the source of vitreous haemorrhage?

Ans. • Abnormal retinal vascularity – PDR
• Normal retinal vascularity – RD

Q.57. How will you manage vitreous haemorrhage?

Ans. Work up :

- History – diabetes, hypertension, trauma.
- Complete ocular examination
 - Anterior segment
 - Indirect ophthalmoscopy
 - Examination of other eye
- USG -B scan

Treatment:

Bed rest with head end elevated.

Treatment of the precipitating factor.

Vitrectomy (if haemorrhage does not resolve).

Chapter

9

Orbit and Globe

Q.1. What are the bones which form bony orbit?

Ans. Seven bones.

Frontal, zygomatic, maxillary, sphenoid, ethmoid, palatine, lacrimal.

Q.2. Which bones form the floor of the orbit?

Ans. Maxillary, zygomatic, palatine.

Q.3. Which bones form the roof of the orbit?

Ans. Frontal, lesser-wing of sphenoid.

Q.4. Which bones form medial wall of orbit?

Ans. Sphenoid, ethmoid, lacrimal, maxillary.

Q.5. What is proptosis?

Ans. Protrusion of the globe forward.

Q.6. Name some important causes of proptosis?

Ans. Children –	Orbital cellulitis
	Retinoblastoma
Adult –	Pseudotumour
	Meningioma

Q.7. What is exophthalmos?

Ans. Passive displacement of the globe forward due to dysthyroid condition.

Q.8. What is enophthalmos?

Ans. Retraction of the globe deep into the orbit, e.g. old age, blow out fracture, etc.



Q.9. What are the spaces of the orbit?

Ans. 4 spaces (Fig-9.1)

1. Subperiosteal- b/w bone and periorbita.
2. Peripheral-b/w Periorbita and extraocular muscles.
3. Central space - enclosed by 4 muscles.
4. Tenon space-b/w sclera and Tenon capsule.

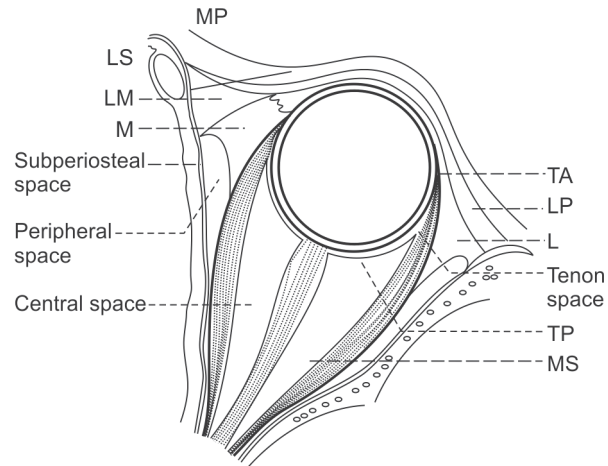


Fig. 9.1 : Horizontal Section through the orbit

L : Lateral Check Ligament, LM : Lacrimal portion of orbicularis muscle, LP : Lateral Palpebral ligament, LS : Lacrimal Sac, M : Medial Check Ligament, MP : medial palpebral ligament dividing into a superficial & a deep band, MS : Muscle sheath, TA : Anterior part of Tenon capsule, TP : Posterior part of Tenon Capsule.

Q.10. What is blow-out fracture of the orbit?

Ans. Blunt trauma to orbit with fist or cricket ball can cause it. Eye ball sinks in maxillary antrum.

Q.11. Which wall of orbit is usually affected?

Ans. Floor of the orbit (weakest wall), followed by medial wall.

Note : All the walls of orbit are related to paranasal sinuses except lateral wall (thickest) which is related to temporal lobe of brain.

Q.12. Which is the thickest wall?

Ans. Lateral wall.

Q.13. What is the shape of the orbit?

Ans. Pyramidal.



Q.14. Which structure forms the apex of the orbit?

Ans. Optic foramen – part of greater wing of sphenoid.

Q.15. What is the relation of the paranasal sinuses with the orbit?

Ans. All the walls of the orbit are related to paranasal sinuses except lateral wall, which is related to temporal lobe of brain.

Q.16. What are the contents of the orbit?

Ans.

- Eyeball along with Tenon capsule
- Retrobulbar fat
- Ophthalmic vessels
- 3th, 4th, 5th, 6th cranial nerve, ciliary ganglion
- Intraorbital part of optic nerve
- Lacrimal gland and lacrimal sac

Chapter

10

Neuro-ophthalmology and Pupil

Q.1. What are the pupillary reflexes?

Ans. Light reflex, near reflex and psychosensory reflex.

Q.2. Describe the pathways of light reflex.

Ans. Impulse from retina → Optic nerve → Optic tract → Pretectal nucleus in midbrain → Edinger westphal nucleus of both sides → Oculomotor nerve → Ciliary ganglion → Short ciliary nerves → Sphincter pupilae. (**Figs 10.1a, 10.1b**).

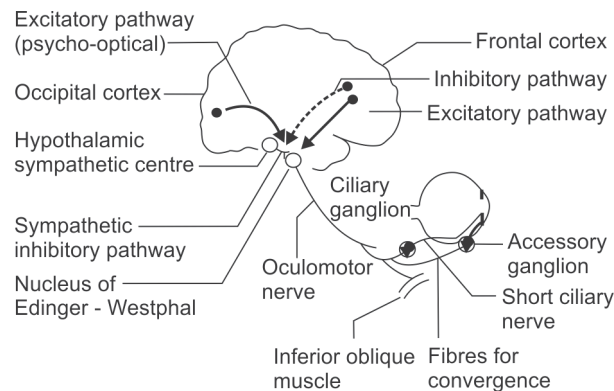


Fig. 10.1a : Parasympathetic Pupillary System

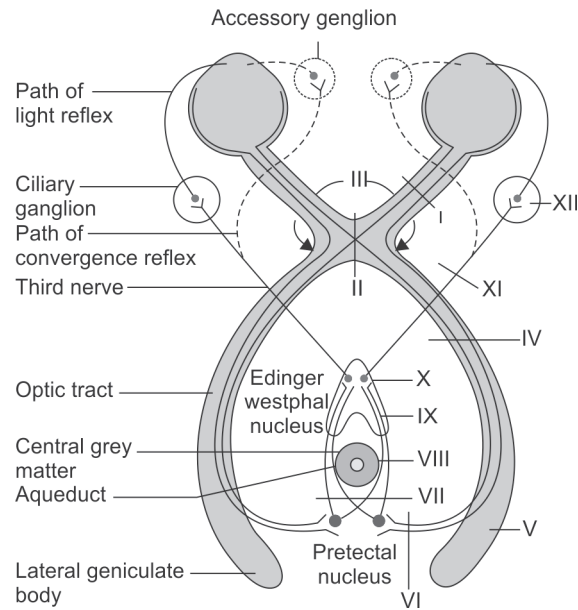


Fig. 10.1b : Pupillary pathways for the light reflex and associated lesions.

Q.3. What is near reflex? Describe pathways of near reflex?

Ans. Near reflex - constitutes contraction of pupil on seeing near object.

Impulses from medial rectus → Oculomotor Nerve → Mesencephalic root of trigeminal nerve → Nucleus of Perlia → Edinger westphal nucleus of both sides → Motor fibres of oculomotor nerve → Accesary ciliary ganglion → Sphinchter pupillae.

Q.4. Describe the pathways of accommodation reflex.

Ans. Impulses from retina → Optic N → Optic tract → LG body → Optic radiation → Striate cortex of occipital lobe → Parastriate area → Occipitomesencephalic tract → Nucleus of Perlia → Edinger westphal nucleus of both sides → Oculomotor Nerve → Accesary ciliary ganglion → Sphinchter pupillae and ciliary muscles.



Q.5. What is psychosensory reflex?

Ans. Dilatation of pupil (mydriasis) in response to psychic and sensory stimuli.

Q.6. What are the features of Adie's tonic pupil?

Ans.

- Delayed pupillary reaction
- Congenital anomaly
- Seen in recovered 3rd cranial nerve palsy
- More common in female
- Responds to 0.1% pilocarpine

Q.7. What is polycoria?

Ans. Multiple pupil

Q.8. What is anisocoria?

Ans. Size of the two pupils are different (diff of >1mm).

Q.9. What are the causes of miosis?

Ans.

- Drugs- miotics
- Iridocyclitis
- Infancy and senility
- In bright light
- Pontine haemorrhage
- Morphine/opium addicts

Q.10. What are the causes of uniocular dilated pupil ?

Ans.

- Topical mydriatics
- Optic atrophy
- Acute angle closure glaucoma
- 3rd N palsy
- Absolute glaucoma
- Topical sympathomimetics

Q.11. What are the features of Argyll-Robertson pupil?

Ans.

- Accommodation reflex is present
- Light reflex is lost
- Usually bilateral, pupil constricted
- Seen in neurosyphilis, tabes dorsalis, multiple sclerosis and diabetes mellitus

(mnemonic ARP—Accommodation reflex is present)



Q.12. What is Marcus Gunn pupil ?

- Ans.**
- It is a relative afferent papillary defect(**RAPD**)
 - Both pupil dilate when the light is moved from the unaffected eye to the affected eye
 - Usually seen in unilateral optic neuritis

Q.13. What is Hutchinson's pupil?

Ans. Widely dilated immobile pupil on the side of head injury.

Q.14. What is nystagmus?

Ans. The term is applied to bilateral, involuntary, rapid oscillatory movements of the eyes, independent of normal eye movements.

Q.15. What are the causes of nystagmus?

- Ans.**
- Congenital nystagmus.
 - Acquired nystagmus – Found in diseases of midbrain, cerebellum, vestibular tracts and semicircular canals.

Q.16. What are the clinical feature of 3rd nerve palsy?

- Ans.**
- Ptosis due to paralysis of levator muscle
 - Divergent squint with intorsion
 - Limitation of movements in all the directions except outwards
 - Pupil is semidilated and fixed
 - Loss of accommodation
 - Diplopia on raising the upper lid

Note : Sup. oblique depresses eye ball only in adducted position.

Q.17. What are the parts of the optic nerve?

Ans. *Four parts:*

- Intraocular part (1mm) – passes through the sclera, choroid and finally appears as the optic disc.
- Intraorbital part (30mm) – extends from back of the eyeball to the optic foramina.
- Intracanalicular part (6-9mm).
- Intracranial part (10mm) – it lies above the cavernous sinus.



Q.18. Describe the effects of lesions of visual pathway at different levels with a diagram.

Ans. (Fig-10.2)

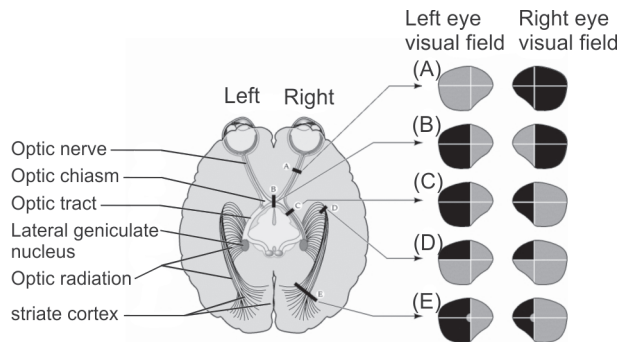


Fig. 10.2 : Diagram of visual path showing sites of lesions and the corresponding field defects.

- A – Ipsilateral blindness
- B – Bitemporal Hemianopia
- C – Homonymous Hemianopia
- D – Superior quadrantic Homonymous defects
- E – Homonymous Hemianopia (Sparing the macula)

1. **Lesions of the optic nerve** – complete blindness of the affected side with loss of direct reflex and consensual on the opposite side.

Causes:

Optic atrophy.
Optic neuritis.

2. **Lesions through proximal part of the optic nerve** – Ipsilateral blindness, contralateral hemianopia.

Loss of direct reaction on the affected side and consensual on the opposite side.

3. **Central chiasmal lesions** – Bitemporal hemianopia and bitemporal hemianopic paralysis of pupillary reflexes.

Causes:

Suprasellar aneurysms.
Pituitary tumor.
Craniopharyngioma.



4. **Lateral chiasmal lesions** – Binasal hemianopia.
Cause: Distention of 3rd ventricle, causing pressure on each side of the chiasma.
5. **Lesions of the optic tract** – Incongruous homonymous hemianopia with contralateral hemianopic pupillary reaction (Wernick's pupil) partial optic atrophy.
Causes:
Syphilitic or TB meningitis.
Tumors of thalamus.
6. **Lesions of LGB** – Homonymous hemianopia with sparing of pupillary reflexes.
7. **Lesions of optic radiations** – Features depend upon the site of lesion:
 - a. **Involvement of the total optic radiations** – complete homonymous hemianopia.
 - b. **Parietal lobe lesion affecting superior fibres of optic radiations** – Inferior quadrantic hemianopia (Pie on the floor).
 - c. **Temporal lobe lesions** – superior quadrantic hemianopia (Pie in the sky).
8. **Lesions of visual cortex** – Congruous homonymous hemianopia (usually spare the macula).
Cause: Occlusion of PCA.
9. **Lesion at tip of the occipital cortex** – congruous homonymous macular defect.
Causes:
Gunshot injury (head).
Head injury.

(LESION AT 8, 9 – PUPILLARY REACTION NORMAL, NO OPTIC ATROPHY)

Q.19. What will be the pupillary reaction in lesions of optic radiations?

Ans. Normal as the fibres of the light reflex leave the optic tract before optic radiations.

Q.20. What are the common lesions of optic radiations?

Ans.

- Vascular occlusions
- Trauma
- Tumors



Q.21. What is cortical blindness?

Ans. Bilateral loss of vision with normal pupillary response due to occipital lobe lesions.

Q.22. What are the causes of cortical blindness?

- Ans.**
- Vascular lesions of bilateral occipital lobe
 - Head injury
 - Tumors

Q.23. What is altitudinal hemianopia?

Ans. Hemianopia involving the inferior or superior half of the visual field.

- AION (anterior ischemic optic neuropathy)- DM, giant cell arteritis, hypertension
- BRVO
- CRAO

Q.24. What is toxic amblyopia?

Ans. Visual loss due to damage of optic nerve fibres due to some exogenous or endogenous toxins. It is a misnomer

e.g. Toxic amblyopia (tobacco, alcohol)

- Methyl alcohol amblyopia
- Quinine amblyopia

Q.25. What is amaurosis fugax?

Ans. Sudden temporary painless monocular visual loss due to a transient failure of retinal blood supply

e.g. Embolism of retinal vessels

Papilloedema

Q.26. What is amblyopia?

Ans. Dimness of vision in one or both eye in absence of any ophthalmoscopic or other marked ocular defects. It is also called lazy eye

e.g. Amblyopia due to squint, cataract, anisometropia, ptosis (congenital). Macular fixation starts from 6 wks and is completed by 6 yrs. During this period, if there is any obstruction in vision, macular development is not completed → amblyopia develops. (stimulus deprivation amblyopia)

Chapter

11

Squint

Q.1. Name the extraocular muscles of the eye ball.

Ans. (Fig-11.1)

4- Recti

2- Oblique

Recti-Superior Rectus

Inferior Rectus

Medial Rectus

Lateral Rectus

Oblique- Superior oblique

Inferior oblique

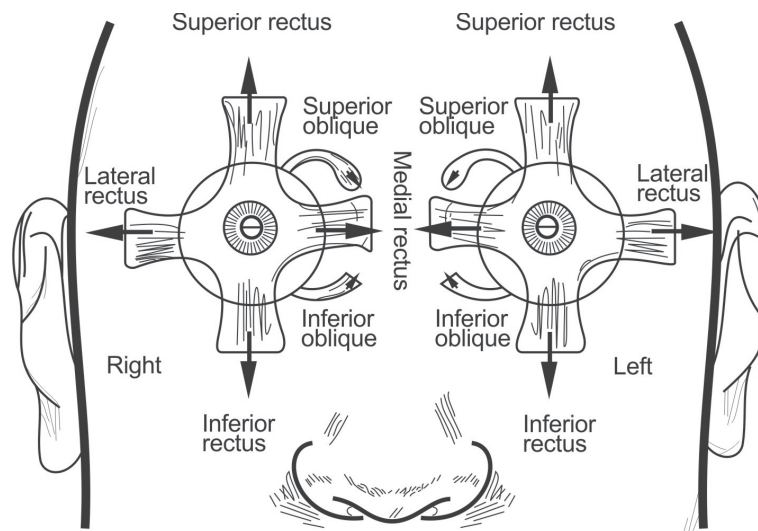


Fig. 11.1: Shows extraocular muscles



Q.2. What is the origin of recti muscles?

Ans. All 4 recti muscles originate from common tendinous ring (annular tendon of Zinn), at the apex of the orbit.

Q.3. What is the distance of the recti insertion from limbus?

Ans. a. Superior rectus-7.7 mm (Pnemonic – SLIM)
b. Lateral rectus - 6.9 mm
c. Inferior rectus-6.6 mm
d. Medial rectus- 5.5 mm

Q.4. Which is strongest recti?

Ans. Medial rectus

Q.5. Which is largest extra ocular muscle?

Ans. Superior oblique

Q.6. Describe the origin and course of superior oblique muscle.

Ans. Arises from annular tendon of Zinn → Runs to the trochlea → becomes tendinous → reflected backward from superior rectus muscle.

Q.7. Where is trochlea situated?

Ans. At the upper and the inner angle of the orbit.

Q.8. Which extraocular muscle does not arises from common tendinous ring?

Ans. Inferior Rectus. It arises anteriorly from the lower and inner orbital walls near the lacrimal fossa. It is the shortest muscle. Inserted in lower and outer quadrant of eye ball behind equator.

Q.9. Tell the nerve supply of extraocular muscles.

Ans. All the muscles are supplied by the 3rd nerve except, Lateral rectus (6th) and Superior oblique (4th).
(Remember All 3, SO4, LR6).

Q.10. Enumerate the actions of extraocular muscles.

Ans. Primary Position :

- Medial Rectus – Adduction
- Lateral Rectus – Abduction
- Superior rectus – Elevation, adduction, intorsion
- Inferior rectus – Depression, adduction, extorsion
- Superior oblique – Depression, abduction, intorsion
- Inferior oblique – Elevation, abduction, intorsion



- Remember:
 - All superiors are intorters (SIN)
 - All inferiors are extorters
- All recti are adductor (RAD) (except lateral rectus)
- All obliques are abductors.

Q.11. What are the extra ocular movements?

- Ans.**
- Duction - uniocular movement
 - Version-binocular movements
 - Adduction
 - Abduction
 - Depression
 - Elevation
- (see chapter 1)

Q.12. What is synergist/yoke muscle?

- Ans.** The two muscles that work together.
- e.g., Dextroversion $\left. \begin{array}{l} \text{RLR} \\ \text{LMR} \end{array} \right\} \text{ yolk muscles}$

Q.13. What is squint?

- Ans.** Squint or strabismus is the condition where the visual axes of the two eyes do not meet at the point or object of regard.

Q.14. Classify Squint.

- Ans.**
- a. Manifest squint (heterotropia)
 - b. Latent squint (heterophoria)
 - c. Apparent squint

Q.15. What is diplopia?

- Ans.** Double vision due to break down in fusional capacity of the binocular system.

Q.16. What is monocular diplopia?

- Ans.** Diplopia persists on occlusion of one eye.

Q.17. What are the causes of monocular diplopia?

- Ans.**
- IMSC
 - Ectopia lentis
 - Iridectomy (close to pupil)

Q.18. What are the causes of binocular diplopia?

- Ans.** Diplopia occurs when both the eyes are open and disappears on occlusion of either eye.



Q.19. Name two conditions where you will get binocular diplopia.

Ans.

- Paralytic squint
- Anisometropia

Q.20. What is orthophoria?

Ans. It is a condition when visual axis of both eyes remain parallel for all cardinal gazes (normal).

Q.21. What is heterophoria (latent squint)?

Ans. It is a condition when there is tendency of one eye for deviation, if the fusion is broken.

Q.22. What is comitant squint?

Ans. In this condition, although the eyes are misaligned, they retain their abnormal relation to each other in all direction of gaze. Here the afferent path or the central mechanism mediating fixation fusion reflexes are abnormal.

Q.23. What is Incomitant squint?

Ans. Efferent pathway is abnormal. The eyes are misaligned but it is not same in all direction of gaze, e.g. paralytic squint.

Q.24. What is heterophoria or latent squint?

Ans. If the fusion mechanism is well developed and the deviation is slight, visual alignment may be maintained in normal circumstances by a continued effort of fusion. So the squint remain latent and only become manifest if fusion is broken (as by covering one eye).

Q.25. What are the symptoms of latent squint?

Ans.

- Asthenopia (Eye strain) and headache after prolong near work.
- Blurring of vision
- Diplopia

Q.26. What are the various tests which are used to diagnose squint?

Ans.

- Hirschberg test (corneal reflex test)
- Cover test
- Maddox rod test
- Maddox wing test
- Prism bar cover test

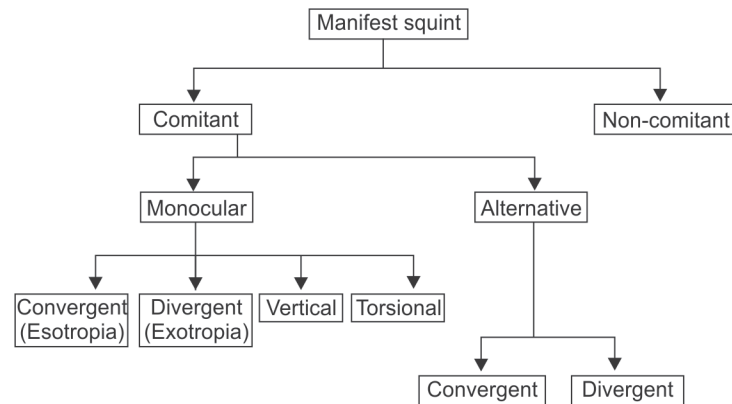


Q.27. How will you manage latent squint?

- Ans.**
- Correction of refraction error
 - Convergence exercise
 - Exercise with prism
 - Exercise with Synaptophore
 - Improvement of general health
 - Mild degree of phoria → no treatment

Q.28. What is manifest squint?

Ans. If the maintenance of alignment is impossible, a true or manifest comitant squint develops.



Q.29. How will you manage concomitant squint?

- Ans.**
- 'O' – Optical (refraction)
 - 'O' – Occlusion (for amblyopia)
 - 'O' – Orthoptic exercise
 - 'O' – Operative

Q.30. How will you manage paralytic squint?

- Ans.**
- Investigation
 - CT
 - MRI
 - Blood Sugar
 - B.P
 - Occlusion of the worse eye
 - Prism correction
 - Vit B complex injection
 - Surgery after 6 month (if the condition does not subside)

Chapter

12

Refraction

Q.1. What is the visual spectrum of light?

Ans. 400–700 nm
($< 400\text{nm}$ – ultraviolet, $> 700\text{nm}$ –infrared)

Q.2. What is the nodal point of eye ball?

Ans. It is the optical centre in the reduced eye or schematic eye.
It lies in the posterior part of the crystalline lens.

Q.3. What is a reduced eye?

Ans. In a reduced eye the entire optical system of the eye behaves as a single lens.

Q.4. What is a visual axis?

Ans. It is the line joining the fixation point, nodal point and the fovea.

Q.5. What is the optical axis of the eye?

Ans. Line joining the fixation object, nodal point and the fovea.

Q.6. What is the visual angle?

Ans. Angle subtended by the object at the nodal point.

Q.7. What is angle Kappa?

Ans. Angle between visual axis and pupillary line.

- If it is positive – Pseudoexotropia
- If it is negative – Pseudoesotropia

Q.8. What is emmetropia?

Ans. It is the normal condition in which the incident parallel rays come to a perfect focus upon the retina, when accommodation is at rest. (Fig-12.1 a and b)

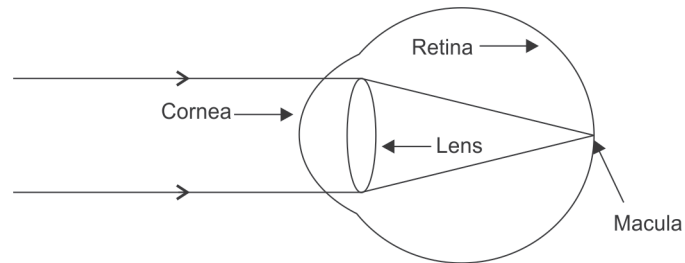


Fig. 12.1a : The emmetropic eye

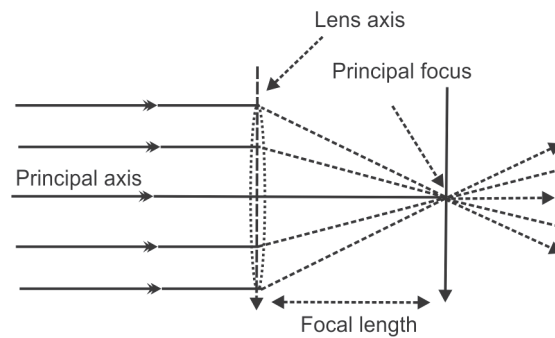


Fig. 12.1b : Light ray

Q.9. What is ametropia?

Ans. The condition in which incident parallel rays of light do not come to a focus upon the light sensitive layer of retina. (Fig-12.2)

- If it focuses – Infront of retina— Myopia



- Behind the retina—Hypermetropia.

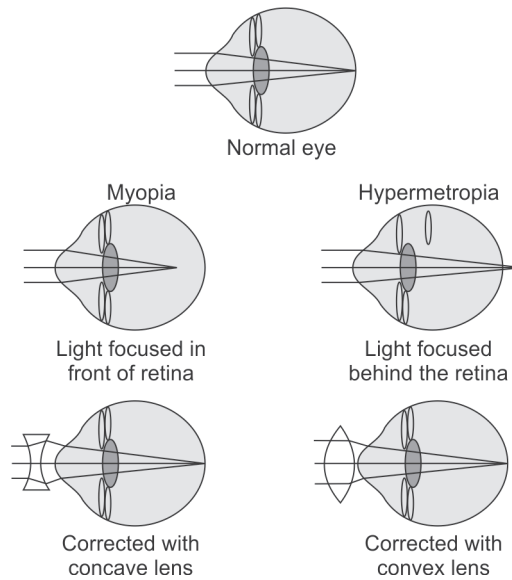


Fig. 12.2 : Refraction

Q.10. What are the etiological types of ametropia?

Ans. Axial ametropia:

- Axial length too long – myopia
- Axial length too short – hypermetropia

Curvature ametropia:

- More curved cornea- myopia
- Less curved cornea- hypermetropia

Index Ametropia:

- High Refractive index of lens – Index myopia
- Low Refractive index of lens – Index hypermetropia

Positive ametropia:

- Forward displacement of lens – myopia
- Backward displacement of lens – hypermetropia

Q.11. Define myopia.

Ans. It is that dioptric condition of the eye in which incident parallel rays come to a focus anterior to the light sensitive layer of the retina when accommodation is rest .



Q.12. What is the commonest etiology of myopia/hypermetropia?

Ans. Axial Length—too long or too Short.

Axial length of eye ball is 24 mm. For each 1 mm increase or decrease there will be 3D myopia or hypermetropia.

Q.13. What are the clinical types of myopia?

Ans.

- Simple Myopia
- Congenital myopia/developmental myopia
- Pathological myopia

Q.14. What is simple myopia?

Ans. It is the commonest etiological type:

- Does not progress after adolescence
- No degenerative changes in fundus
- Usually upto -5D to -6D

Q.15. What is congenital/developmental myopia?

Ans. Present at birth (abnormally long eye at birth)

Refractive error upto – 10 D

Q.16. What are the features of pathological myopia?

Ans.

- Hereditary
- Appears in childhood and steadily progress upto 25 years or beyond
- Range -10 D to -20 D
- Characteristic degenerative changes seen in fundus
- Prognosis is poor

Q.17. What are the fundus changes seen in pathological Myopia?

Ans.

- Disc size appears larger
- Pale and larger physiological cup
- Temporal crescent around the disc
- Posterior staphyloma
- Macula- Foster Fuch's spot
- Vitreous- Degenerative changes
- Retinal tear, hole, detachment

Q.18. What are the complications of myopia?

Ans.

- More incidence of peripheral retinal degeneration, e.g Lattice degeneration
- Retinal breaks with or without vitreous haemorrhage
- Retinal detachment
- Degeneration of vitreous gel
- Posterior polar cataract
- Higher incidence of POAG

**Q.19. What are the treatment options of myopia?**

- Ans.**
- Optical

 - Glass
 - Contact lens
 - Surgical:
 - Radial Keratotomy (RK)
 - Photorefractive keratotomy (PRK)
 - LASIK
 - Phakic IOL
 - Keratophakia
 - Keratomileusis
 - Removal of crystalline lens (> 21 D Myopia)
 - Scleroplasty
 - Intracorneal ring
 - General health and ocular hygiene , outdoor physical activity
 - Genetic counseling in pathological myopia

Q.20. What is LASIK?

Ans. Laser- assisted in situ keratomileusis.
Excimer laser is used to ablate corneal stroma. Upto -10 D of myopia can be corrected.

Q.21. At what age LASIK is done?

Ans. > 18 years age , when the progression of refractive error stops.

Q.22. What is pseudomyopia?

Ans. Spasm of accommodation causes it.

Q.23. Name two drugs which produces transient myopia.

- Ans.**
- Acetazolamide
 - Pilocarpine

Q.24. How does a high myopic eye usually look?

- Ans.**
- Eyes are prominent
 - Large pupil
 - Deep AC

Q.25. When you call it as high myopia?

Ans. >6 D

Q.26. What is hypermetropia?

Ans. It is a refractive error where parallel rays of the light coming from infinity come to focus posterior to the light sensitive layer of the retina, when accommodation is at rest.



Q27. What are the different types of hypermetropia?

- Ans.**
- Facultative hypermetropia — Corrected by effort of accommodation.
 - Absolute hypermetropia — can not be corrected by accommodation.
 - Latent hypermetropia— corrected by tone of the ciliary muscle. (generally amounts to one dioptre)
Unmasked by cycloplegics e.g atropine.
 - Total hypermetropia— Latent + manifest hypermetropia.
 - Manifest hypermetropia— facultative + absolute.

Q.28. What are the aetiological classification of hypermetropia?

- Ans.**
- Axial hypermetropia - 1mm shortening of the globe is equal to +3D hypermetropia.
 - Curvature hypermetropia—If the cornea/lens is flattened, 1mm flattening produces + 6D hypermetropia.
 - Index hypermetropia— increase of refractive index of cornea in old age .

Q.29. What are the problems of hypermetropia?

- Ans.**
- Early onset presbyopia.
 - Higher incidence of angle closure glaucoma.
 - Convergent squint.
 - Amblyopia (common in unocular high hypermetropia).

Q.30. What is the usual power of a newborn?

- Ans.** Newborns are almost invariably hypermetropic (average: 2.5D).

Q.31. What are the treatment options of hypermetropia?

- Ans.**
- Optical

 - Spectacle correction
 - Contact lens
 - Surgical—
 - PRK
 - LASIK (upto +6D)
 - IOL implant

Q.32. What is astigmatism?

- Ans.** It is a type of refractive error in which incident parallel rays do not come to a point focus upon the retina, instead they may form a line as the refraction varies in different meridian of the eye.

**Q.33. Classify astigmatism.**

- Ans.**
- Regular astigmatism— Cornea is more curved in one meridian.
 - Irregular astigmatism— Due to irregularities in curvature of the meridians and can't be corrected with glass.

Q.34. What are the different types of regular astigmatism?

- Ans.**
- Simple —One of the principal meridian is emmetropic and other is myopic or hypermetropic (simple myopic/ simple hypermetropic).
 - Compound — Both the principal meridians are either myopic or hypermetropic (compound myopic/ compound hypermetropic).
 - Mixed— If one meridian is myopic and other is hypermetropic.

Q.35. What is 'with the rule' astigmatism?

- Ans.** In regular astigmatism if the cornea is more curved in vertical axis (remember plus 90°/minuses 180°).

Q.36. What is 'against the rule' astigmatism?

- Ans.** If the horizontal curvature is greater than vertical (remember the opposite rule).

Q.37. What are the problems of astigmatism?

- Ans.** Asthenopia or headache.

Q.38. What are the causes of irregular astigmatism?

- Ans.**
- Scarring of the cornea
 - Keratoconus
 - Lentoconus
 - Incipient cataract

Q.39. What are the treatment options of astigmatism?

- Ans.**
- Regular astigmatism— Sphero cylindrical lens
 - Irregular astigmatism— Contact lens
 - Surgery:
 - Astigmatic keratotomy
 - Excimer laser
 - Removal of tight sutures after cataract/PK

Q.40. What is anisometropia?

- Ans.** This is the condition in which the refractive status of the two eyes show a considerable difference (> 2.5 D).



Q.41. What are the problems of anisometropia?

- Ans.**
- Eyestrain due to imperfect binocular vision due to an effort to fuse the images.
 - Uniocular vision—(worse eye vision will be suppressed).
 - Squint—Convergent in children.
— Divergent in adults.
 - Diplopia—Due to unequal image sizes.

Q.42. How will you treat anisometropia?

- Ans.**
- Contact lens
 - Iseikonic glass
 - T/t of amblyopia
 - Refractive surgery

Q.43. What is anisokonia?

- Ans.** Unequal image size in two eyes. Up to 5% difference in image size is tolerable.

Q.44. What is accommodation?

- Ans.** It is the power of changing the focus by which near object is seen clearly.

Q.45. What is range of accommodation?

- Ans.** It is the range of clear vision (that is range between far point and near point).

Q.46. What are the three changes occur in accommodation?

- Ans.**
- Bulging of anterior lens capsule forward
 - Contraction of ciliary muscles and relaxation of ciliary body
 - Convergence of eyes

Q.47. What is pseudo-accommodation?

- Ans.** If a special foldable silicon lens is placed in the capsular bag, in some patients they behave as an accommodating IOL.

Q.48. What is presbyopia?

- Ans.** It is a condition seen in advancing age due to physiological loss of accommodation leading to failure to see near objects. It occurs due to decreased elasticity and plasticity of lens.



Q.49. What are the causes of frequent change of glasses?

- Ans.**
- POAG
 - Keratoconus
 - Nuclear cataract
 - Hyperglycemia/Hypoglycemia

Q.50. What is asthenopia?

Ans. Ocular or periocular discomfort, browache and headache associated with prolonged use of eyes especially for near work.

Q.51. What are the causes of asthenopia?

- Ans.**
- Uncorrected refractive error
 - Presbyopia
 - Anisometropia
 - Heterophoria
 - Convergence insufficiency

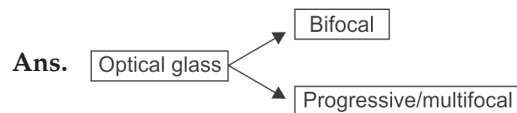
Q.52. What is near point ?

Ans. Nearest point at which an object is clearly seen. It varies with age. It is 7-10cm upto 10 years of age and 25 cm at 35 years of age.

Q.53. What is far point of eye?

Ans. Farthest point at which an object placed is clearly seen by an eye. (Far point for an emmetropic eye is at infinity. In myopia – far point is in front of the eye . In hypermetropia it is behind the eye, i.e. virtual).

Q.54. How will you treat presbyopia?



- Surgical-
 - LASIK
 - Bifocal/multifocal IOL is implanted after lens extraction.
 - Monovision with IOL, i.e. one eye is corrected for distance and the other for near after cataract surgery.



Q.55. What is bifocal lens?

Ans. It contains two optical corrections. The upper part is for distant vision and lower half is for near vision.

Q.56. What is high index lens? In which case it is used?

Ans. These are made of glass/plastic having a Refractive Index of 1.56-1.67. So the lenses are thinner, flatter and lighter. These are used in patients having high refractive error.

Q.57. What is the refractive index of ordinary spectacle lens?

Ans. Crown glass is used for ordinary spectacle lens which has a refractive index of 1.52.

Q.58. How does photo chromatic lenses help?

Ans. They offer protection from harmful ultraviolet rays.

Q.59. How does anti-reflective coating on spectacle lens help?

Ans. It improves patient's clarity of vision by reducing glare from oncoming headlight, computer screen.

Q.60. What is the axial length of a patient who does not require aphakic correction after surgery?

Ans. 31 mm. As parallel rays will focus upon retina without correcting lens (-21D myopia).

Q.61. Which one is better option for myopic—spectacle or contact lens?

Ans. Contact lens have the advantage of providing a wider field and larger image size compared to glasses.

Q.62. A patient has got high myopia, will you put IOL after cataract surgery?

Ans. Yes. As in such cases the vitreous is likely to be fluid and there is increased tendency of retinal detachment, minus powered concave IOLs are becoming popular.

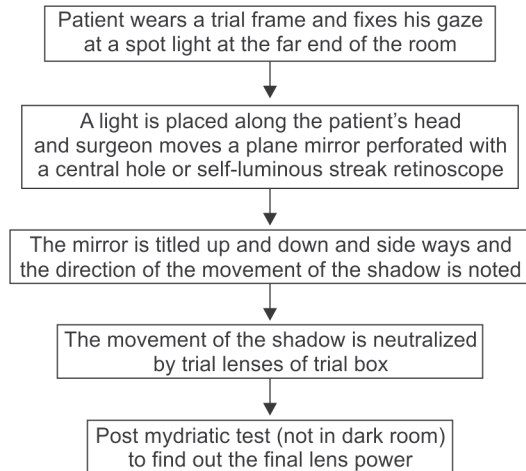
Q.63. What is retinoscopy?

Ans. It is a procedure which determines the refractive status of the eye accurately.



Q.64. How retinoscopy is done?

Ans. It is a dark room procedure.



Q.65. In which case retinoscopy should be done with cycloplegics?

Ans.

- In children (as accommodation is strong).
- In young adult – Homatropine 2% drop.
- In old age – better not to dilate as there is chance of acute attack of ACG (if AC is shallow).

Q.66. Which instrument is a substitute for retinoscopy?

Ans. Autorefractometer.

Q.67. What is contact lens (CL)?

Ans. These are thin optical corrective lenses worn on the eye, resting on the surface of the cornea.

Q.68. What are the indications of contact lens use?

Ans. Ideal correction for ametropia especially high errors of refraction (myopia, aphakia).

Q.69. What are the advantages of contact lens over spectacle use?

Ans.

- Prismatic effect of spectacles are eliminated
- Increase field of clear vision
- Size of the image is almost same to that of the emmetropic eye
- Eliminates high errors of astigmatism (Keratoconus)



Q.70. What are the disadvantages of contact lens?

Ans.

- Need special care to avoid infection
- Allergy
- Corneal hypoxia

Q.71. What are the various types of CL available in market?

Ans.

- Soft CL
- Rigid gas – permeable (RGP) CL
- Hard CL

Q.72. Which type of CL is mostly responsible for microbial keratitis?

Ans. Soft CL, especially high water content extended wear variety.

Q.73. What is the water content of soft CL?

Ans. As much as 79%

Q.74. What are low vision aids?

Ans. These are used in patients having low vision, which cannot be fully corrected by any other means.

Q.75. What are the different types of LVA available?

Ans.

- Hand – held magnifier
- Telescopes
- Videomagnifier

Chapter

13

Lacrimal Apparatus

Q.1. Describe the anatomy of lacrimal apparatus.

Ans. (Fig. 13.1)

It consists of two parts:

- lacrimal glands
- lacrimal passages

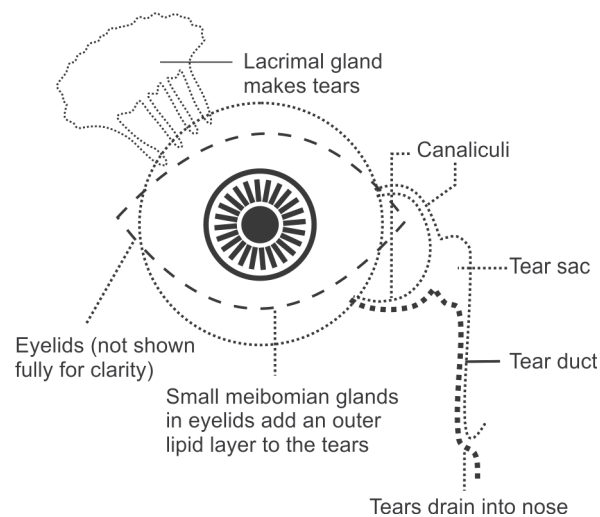


Fig. 13.1 : Eye and Tear production

Lacrimal Gland

Superior or orbital gland: Almond shaped, situated in lacrimal fossa at the outer part of the orbital plate of frontal bone, through which passes 10-12 lacrimal ducts to open on the surface of the conjunctiva at the lateral aspect of superior fornix.

Inferior or Palpebral gland: 1-2 lobules on the course of the ducts of the superior portion.



Accessory lacrimal or Krause gland (42 in superior fornix, 6-8 in the lower fornix) situated below the surface of conjunctiva between the fornix and tarsal edge.

Lacrimal Passage

Lacrimal puncta – situated 6mm from the medial canthus near the posterior border of the free margin of the lid.

Lacrimal canaliculi- direction- vertically 1-2mm then turns at right angles at ampulla and runs horizontally 6-7mm.

Common canaliculus- upper and lower canaliculi join to form common canaliculus which opens in the lacrimal sac guarded by mucosal fold (valve of rosenmuller).

Lacrimal sac- situated in lacrimal fossa, 15mm length vertically, 5-6mm width.

Nasolacrimal duct (NLD) -12-24mm long, 3mm in diameter. It opens in the inferior meatus of the nose.

Q.2. What is the position of the lacrimal sac?

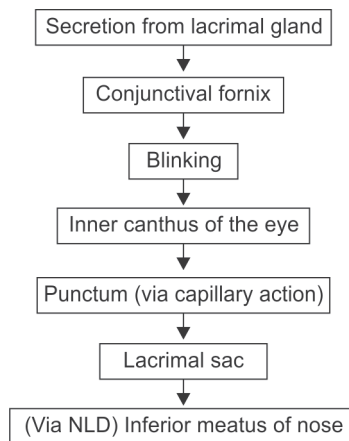
Ans. It lies in the **lacrimal fossa** formed by the lacrimal bone and the frontal process of the maxilla.

Q.3. What is the lining epithelium of the lacrimal sac and NLD?

Ans. Columnar epithelium.

Q.4. Describe how the tear is drained.

Ans.



Q.5. Where does NLD open?

Ans. In the outer wall of inferior meatus of nose.



Q.6. What are the different layers of tear film?

Ans. (Fig. 13.2)

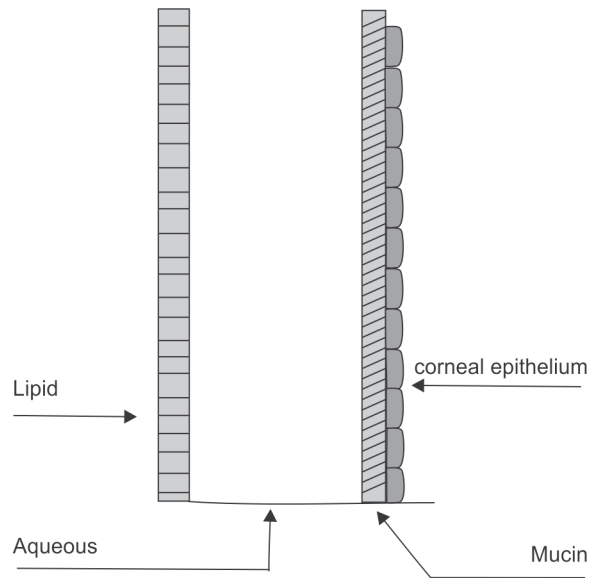


Fig. 13.2 : Layers of the tear film

- Lipid layer (superficial) – produced by meibomian gland and glands of zeiss and moll. It prevents evaporation of tears.
- Aqueous layer – Produced by accessory lacrimal gland. It is the middle layer. It provides optical media.
- Mucin layer – Innermost layer. Produced by conjunctival goblet cells. It binds tear film to conjunctiva and cornea.

Q.7. What does the tear contain?

Ans. It is alkaline in nature comprising mainly NaCl (98.2% water).
Enzyme – lysozyme, betalysin, lactoferrin.

Q.8. What are the function of tear?

- Ans.**
- Keeps ocular surface moist.
 - Provides nutrition to corneal epithelium.
 - Washes the eye constantly by removing FB.
 - Maintain transparency of cornea.
 - Prevents infection due to presence of antibacterial substances (Lysozyme).



Q.9. What is lacrimation and what is epiphora?

Ans. Lacrimation:

Excessive secretion of tears.

Causes – Psychic stimulation

Irritation of conjunctiva/cornea – FB or inflammation.

Epiphora – obstruction in tear drainage pathway.

Causes:

- Occlusion or congenital absence of punctum.
- Canalicular block.
- NLD block – congenital or due to chronic dacryocystitis.
- Growth in the lacrimal sac.
- Lacrimal pump failure.

Q.10. What are the causes of lacrimal pump failure?

- Ans.**
- Senile lid laxity.
 - Orbicularis oculi weakness (7th nv. palsy).

Q.11. Define dacryocystitis.

Ans. It is an inflammation of the lacrimal sac.

Q.12. Classify dacryocystitis.

Ans. *Classification* is as follows:

- **Congenital dacryocystitis.**
- **Acquired dacryocystitis** – two types
 - a. Chronic dacryocystitis
 - b. Acute dacryocystitis

Q.13. What is DCR?

Ans. Dacryocystorhinostomy. It is a surgical procedure in which an anastomosis is made between the medial sac wall and mucosa of the middle meatus of nose.

Q.14. What is the basic principle of DCT?

Ans. The lacrimal sac is removed (so watering will be there but reservoir of infection will not be there).

Q.15. What are the clinical features of congenital dacryocystitis?

Ans. Congenital dacryocystitis is due to incomplete canalization of lacrimal system, most often due to valve of Hasner.

Q.16. How will you manage a case of congenital dacryocystitis?

- Ans.**
- Hydrostatic sac massage (success rate 95%) and topical antibiotic (if purulent discharge is present).



- If sac massage fails and age > 1 year → Probing with syringing under GA.
- If the above T/t fails → balloon dacryoplasty of the sac/silicone tube placement.
- If all above fails → DCR done after 3 years.

Q.17. Why DCR is not done before 3 years?

Ans. As the development of orbital bone is not complete before 3 years so there is chance of failure of surgery.

Q.18. What is the method of sac massage ? How does it work?

Ans.

Common canaliculus is blocked to prevent the reflux by putting the little finger (should be done by mother, preferably when the baby is calm after having feed).



Stroking firmly downward to increase the hydrostatic pressure inside the sac 3-4 times daily.



Instillation of antibiotic eye drops and maintenance of hygiene.

Advantages of lacrimal massage:

- Empties the lacrimal sac and hence prevents stasis of secretions and bacterial growth.
- Helps in opening up the membranous blockage at the lower end of NLD.

Q.19. Why there is more failure of paediatric DCR operation than adult one? What are the measures that prevents failure of the operation in former age group?

- Ans.**
- Causes of failure – there is more reactive granulation tissue formation at the bony ostium.
 - Remedy – a large bony opening is to be made and both anterior and posterior flap should preferably be sutured.
 - Antimetabolites (Mitomycin C) can be applied which prevents the late failure due to scarring.

CHRONIC DACRYOCYSTITIS (LONG CASE/SHORT CASE)

Q.20. What is your case?

Ans. A case of chronic dacryocystitis (right/left).



Q.21. What are the points in favor of chronic dacryocystitis?

- Ans.**
- a. Watering (right/left) for days/months.
 - b. Swelling on the inner angle of the eye (may/may not).
 - c. Regurgitation of pus on pressure over the sac area (may/may not).
 - d. Conjunctival congestion near the inner canthus.

Q.22. How will you confirm your diagnosis?

Ans. By syringing of nasolacrimal duct (NLD)/patency test.

Q.23. What are the risk factors for chronic dacryocystitis?

- Ans.**
- Middle aged – more in female (M:F= 1:4).
 - Left eye more affected (RE:LE= 1:9).
 - Lower socioeconomic status.
 - DNS , hypertrophied inferior turbinate, nasal polyp, chronic rhinitis etc.

Q.24. What are the common organisms responsible for chronic dacryocystitis?

Ans. Pneumococcus, Streptococcus, Staphylococcus etc.

Q.25. What are the complications of chronic dacryocystitis?

- Ans.**
- Corneal ulcer (Hypopyon corneal ulcer).
 - Recurrent conjunctivitis.
 - Acute exacerbation of chronic dacryocystitis.
 - Lacrimal abscess, fistula, osteomyelitis, mucocele.

Q.26. What is lacrimal fistula?

Ans. In a case of chronic dacryocystitis if it bursts through skin during acute attack , small fistula may be present at the sac area. Discharge may come out through it.

Q.27. What are the preoperative investigations in chronic dacryocystitis?

- Ans.**
- Syringing—to locate the site of block.
 - Blood for TC, DC, ESR, Hb.
 - Bleeding and clotting time.
 - BP and PPBS.
 - ENT check up to exclude any contraindications for DCR—e.g. atrophic rhinitis, nasal polyp, DNS, hypertrophied inferior turbinate.
 - Children—General anaesthesia fitness. (TC, DC, Hb%, X-Ray chest, stool for OPC)



Q.28. What are the treatment options for the chronic dacryocystitis?

Ans. Surgery (Antibiotic eye drop is given till surgery is done).
 • DCR
 • DCT

Q.29. What are the full forms of DCR and DCT?

Ans. Dacryocystorhinostomy, Dacryocystectomy.

Q.30. What are contraindications of DCR?

Ans. a. Old age (sac is atrophic)
 b. Lacrimal sac tumor
 c. Gross nasal pathology
 d. Syphilis, TB or other diseases affecting the lacrimal sac
 DCT is done in all the above conditions.

Q.31. What is mucocoele?

Ans. In a case of chronic dacryocystitis a swelling just below the inner canthus may be found, it is a non inflammatory condition. On regurgitation test, milky/gelatinous mucoid fluid from lower punctum may come out. In encysted mucocoele → both the canalicular openings are blocked → no regurgitation occurs.

Q.32. What is functional blockage of lacrimal passage?

Ans. Epiphora in absence of any obstruction to the outflow of tears (syrringing is patent). It is due to weakness of orbicularis oculi causing lacrimal pump failure in old age.

Q.33. What operation is done in a canalicular block?

Ans. Conjunctival DCR. Anastomosis is made between conjunctival sac and nasal mucosa.

Q.34. What are the sites of obstruction in chronic dacryocystitis?

Ans. • Nasolacrimal duct (NLD)
 • Canalicular
 • Punctum

Q.35. What is done in DCR?

Ans. A communication is made between the lacrimal sac (medial wall) and middle meatus of nose by anastomosis between wall of lacrimal sac and nasal mucosa.



Q.36. What are the indications for DCR surgery?

- Ans.**
- Dacryocystitis with NLD block .
 - Mucocele of the lacrimal sac.
 - Recurrent attack of acute dacryocystitis.

Q.37. What are the contraindications of DCR?

- Ans.**
- Acute dacryocystitis.
 - Lacrimal sac tumor.
 - Children less than 3 years.

Q.38. What type of anaesthesia is given in DCR?

- Ans.**
- In adult infiltration anaesthesia.
 - In children – general anaesthesia.

Q.39. How will you give infiltration anaesthesia?

Ans. First injection- 2% lignocaine hydrochloride with adrenaline (5–6 ml) is injected at the junction of the inferior orbital margin and anterior lacrimal crest, and then along the line of incision to a point 3 mm above the medial palpebral ligament.

Second injection is made at the above point and needle is directed posteriorly for about 8 mm, and tissue around the fundus are injected with about 0.5 ml. The needle is then carried out further downwards to the upper half of posterior lacrimal crest and injected with 0.5 ml solution.

Q.40. Describe the steps of DCR surgery.

Ans.

Nasal pack – In DCR, before starting the actual operation, a nasal pack is given on the same side with a roller gauze soaked with 4 % lignocaine and adrenaline for haemostasis.



Incision - A curved incision is made 4 mm away from inner canthus, beginning 4 mm above the medial palpebral ligament extended 4 mm vertical downwards and 4 mm outwards along the line of anterior lacrimal crest. (remember rule of '4')



(Alternatively a straight incision 11 mm medial to the medial canthus can also be made). Care is taken to avoid injury to angular vein.



Dissection of skin , sub cutaneous tissue and orbicularis oculi along the line of incision with lacrimal dissector and retracted with Muller's retractor.



Contd...



Contd...

Medial palpebral ligament is exposed and disinserted from the anterior lacrimal crest by a rongeur or cut with surgical blade.



Creation of bony ostium – 15 × 15 mm bony ostium is made by removing bones forming lacrimal fossa . Nasal mucosa is exposed.



Lacrimal sac flap is made. Two horizontal 'H' shaped incision is made on the nasal mucosa and medial wall of nasal sac, and thereby creating two anterior flaps and two posterior flaps.



It is difficult to suture both anterior and posterior flaps, so only anterior flaps are sutured together while posterior flaps are removed from both sides.



Closure of incision – Fibres of orbicularis oculi is sutured with 6-0 vicryl with interrupted suture.



Skin is sutured by 5-0 or 6-0 silk.



Pressure bandage is applied for 48 hours.



Nasal pack is removed after 24 hours.

Q.41. How will you follow up the patient post operatively?

Ans.

- Broad spectrum antibiotic for 5-7 days.
- Suture removal after 1 week and syringing is done to check for patency.
- Regular follow up (syringing) till one year.

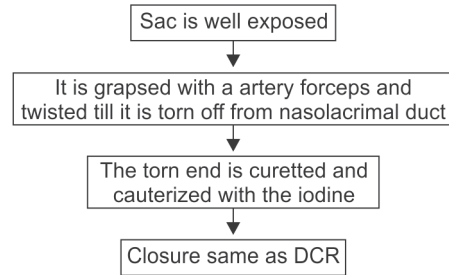
Q.42. What is DCT?

Ans. Dacryocystectomy .In this operation, lacrimal sac is dissected and excised.



Q.43. Describe the steps of DCT.

Ans. (initial few steps are same as DCR)



Q.44. What are the complications of DCR?

Ans.

- Bleeding- due to injury of the angular vein or vascular nasal mucous membrane.
- Blood clot may close the communication.
- Infection.

Q.45. How will you manage a failed DCR?

Ans.

- Re-DCR with mitomycin-C.
- Use of canalicular stents.

Q.46. What are the other types of DCR apart from the conventional DCR?

Ans.

- Endonasal DCR.
- Laser-assisted DCR.
- Transcanalicular laser DCR.

Q.47. What is endoscopic nasal DCR?

Ans. Done through nasal pathway, specially in a cosmetically conscious patient. It has poor surgical success rate.

ACUTE DACRYOCYSTITIS(SHORT CASE)

Q.48. What is acute dacryocystitis?

Ans. Suppurative inflammation of the lacrimal sac.

Q.49. What are the clinical features of acute dacryocystitis?

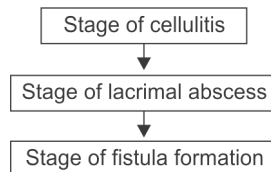
Ans.

- Painful swelling of lacrimal sac region.
- Fever, malaise.
- Redness, edema around lid, may spread to cheek.



Q.50. What are the stages of acute dacryocystitis?

Ans.



Q.51. What are the complications of acute dacryocystitis?

Ans. Complications of acute dacryocystitis -

- Lacrimal abscess, lacrimal fistula.
- Orbital/facial cellulitis.
- Cavernous sinus thrombosis.
- Osteomyelitis.

Q.52. How you will manage a case of acute dacryocystitis?

- Ans.**
- *Stage of cellulitis*
 - a. Hot fomentation.
 - b. Systemic anti-inflammatory and analgesic.
 - c. Systemic antibiotics and topical antibiotics.
 - *Stage of lacrimal abscess*
Antibiotics + drainage of pus.
Later DCT/DCR.

Chapter

14

Trauma

Q.1. What are the common foreign bodies found in the eye?

Ans. Iron, steel, vegetable matters- husks of seeds.

Less common- stone, glass, thorn.

Q.2. What are the probable locations of F.B in the eye?

Ans. Superficial:

Conjunctival

- Bulbar
- Palpebral - (Sulcus subtarsalis in the upper lid).

Corneal - Superficial/ intrastromal.

Deep:

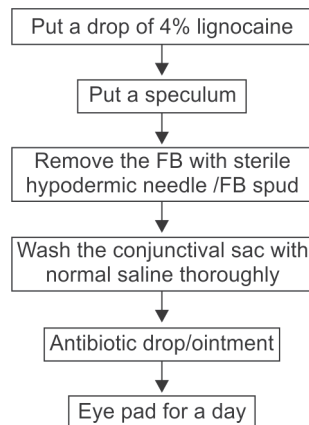
Anterior chamber, intralenticular, intravitreal, retina.

In the orbit (double perforation).

Q.3. How will you remove a superficial ocular FB?

Ans.

(Slit lamp / good torch light)





Q.4. What complications can occur from a corneal FB?

Ans.

- Infection.
- Corneal ulceration.
- Deep seated foreign body may escape into the AC during removal.

Q.5. What is the commonest mode of entering FB in the eye?

Ans. Industrial workers-especially working with grinding tools, lathe workers or hammering on a chisel.

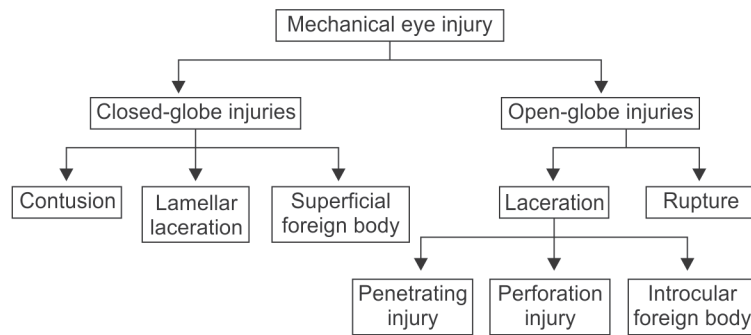
Q.6. What advice should you give to prevent these type of injury?

Ans.

- Comfortable goggles.
- Educational notice and lectures.

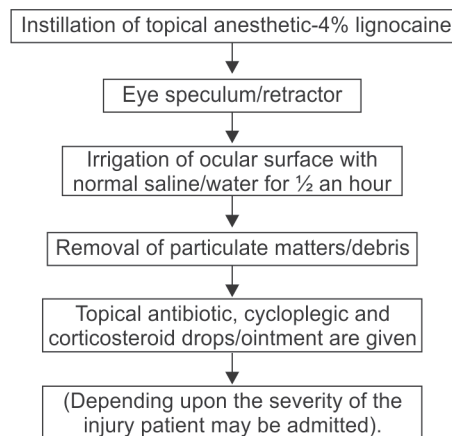
Q.7. Classify ocular trauma.

Ans.



Q.8. How will you treat chemical injury in emergency set up?

Ans.





Q.9. What are the usual offending chemical agents?

- Ans.**
- Lime (white washing/edible chuna)
 - Acid-toilet cleaner
 - Ammonia vapour
 - Nitric acid burn
 - Colours (holi)

Q.10. Which injury is more harmful acid/ alkali? Why?

Ans. Alkali.

It increases pH → saponification of the fatty acid of cell membrane → destruction of cells.

Ulceration and proteolysis continue till 2-3 weeks of injury.

Q.11. What is the mechanism of acid injury?

- Ans.**
- It causes coagulation and precipitation of proteins in cells.
 - It results in shortening of collagen fibre leading to increase IOP.

Q.12. What are the immediate effects of chemical injury?

- Ans.**
- Opacification and melting of cornea.
 - Chemosis of conjunctiva.
 - Limbal ischaemia.

Q.13. What are the late complications of chemical injury?

- Ans.**
- Severe dry eye.
 - Vascularised leucoma.
 - Symblepharon.
 - Persistent corneal epithelial defect.
 - Secondary glaucoma

Q.14. How will you prevent symblepharon?

Ans. It may be prevented by sweeping a glass rod well coated with a lubricant round the upper and lower fornices several times a day depending upon the severity of the case.

Fitting of a bandage contact lens separates the two mucosal surfaces and prevents adhesion.

Q.15. How will you manage late complications?

- Ans.**
- Penetrating keratoplasty.
 - Conjunctival limbal autograft.
 - Limbal stem cell grafting.



Q.16. What are the effects of blunt trauma of the eye?

Ans. Blunt trauma can cause concussion or contusion injury to the eye.

<i>Ocular tissue involved</i>	<i>Clinical manifestations</i>
Orbit	Blow out fracture on medial wall or floor Orbital hematoma Carotid cavernous fistula
Eye lids	Haematoma Avulsion of the lower lid
Conjunctiva	Subconjunctival haemorrhage Conjunctival laceration
Cornea	Abrasion Stromal oedema Blood staining of cornea (due to traumatic hyphaema) Corneal rupture
Anterior Uvea	Hyphaema Tear of the iris sphincter and iridodialysis Ante-flexion of the iris Retroflexion of the iris Iridoschisis Traumatic iridocyclitis
Ciliary body	Angle recession Cyclodialysis
Lens	Vossius ring Rosette cataract Subluxation of the lens Dislocation of the lens Rupture of the anterior or posterior capsule
Sclera	Rupture, commonly at the limbus or behind the insertion of the recti.
Vitreous	Haemorrhage Liquifaction of vitreous Posterior vitreous detachment

Contd...



Contd...

<i>Ocular tissue involved</i>	<i>Clinical manifestations</i>
Choroid	Choroidal rupture Suprachoroidal Haemorrhage
Retina	Retinal or subretinal haemorrhage Retinal edema, commotion retinae (Berlin's oedema) Retinal dialysis Macular oedema or hole
Optic nerve	Optic nerve avulsion Haemorrhage of the optic nerve sheath Traumatic optic neuropathy
Intraocular pressure	Hypotony Secondary glaucoma
Lacrimal apparatus	Injury to lacrimal canaliculus

Q.17. How will you manage a case of blunt trauma?

- Ans.**
- Management of emergency and life threatening situations – e.g. Respiratory distress, Cardiovascular compromise, massive bleeding and shock.
 - **Ocular examination:**
 - Inspect globe and adnexa—
Abnormal globe position
Subcutaneous emphysema
Abnormal lid position
Lacrimal drainage investigation
Particulate material on lid/face
Palpate orbital margins for fractures
 - Visual acuity
 - Pupillary reaction – look for sphincter tear, iritis and RAPD
 - Ocular motility – Restriction/diplopia
 - Visual fields – Confrontation perimetry
 - Inspect all ocular structures and identify the extent of injuries

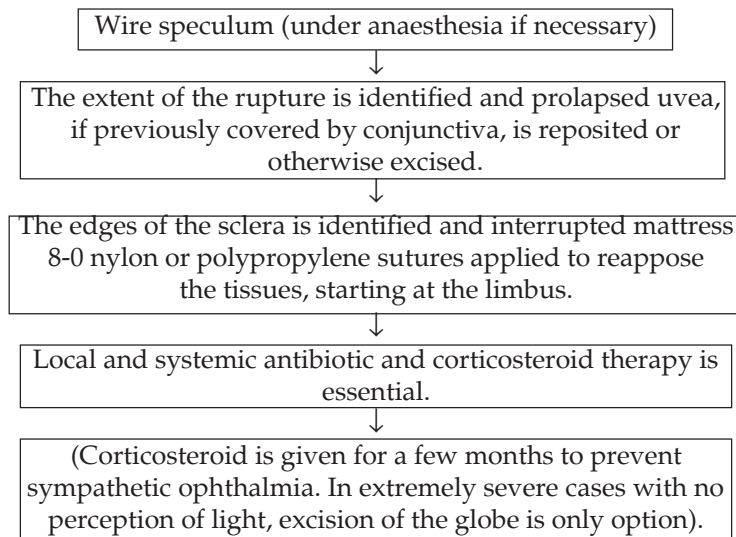


- IOP ↑ - Trabeculitis
- Angle recession
- ↓ IOP - Ciliary body injury
- Ruptured globe

Investigations:

- X-ray orbit – AP and lateral view to detect intra ocular foreign body/fracture of orbital bone.
- USG – to evaluate the posterior segment.
- CT – scan (preferred modalities nowadays) to detect radio opaque FB.
- MRI – should be avoided in metallic foreign body.

Treatment (Rupture of the globe)



Traumatic Iridocyclitis

Mydriatics
Topical steroids

Traumatic hyphaema – discussed later.

Lens:

- The treatment of cataract is on general lines unless rapid intumescence of the lens leads to a secondary glaucoma which may then require immediate treatment. Any surgical interference should be delayed for some months until the final outcome is apparent.



- A lens dislocated into the vitreous should be left there, but if uveitis or glaucoma supervene, extraction of the lens is necessary, together with vitrectomy.

Traumatic glaucoma - Antiglaucoma medications.

Q.18. What is the complication of retained intraocular (RIO) iron foreign body?

Ans. Ferrous ion causing irreversible degenerative changes in the ocular tissue, is called siderosis bulbi. Epithelial structures are more prone to be affected in siderosis bulbi. It can cause blindness.

Q.19. What is the effect of retained intraocular copper foreign body?

Ans. Chalcosis bulbi.

Q.20. Which type of copper cause it?

Ans. Copper alloy (brass)

Q.21. What reaction is produced by pure Copper?

Ans. Suppurative reaction → phthisis bulbi.

Q.22. What are the clinical features of chalcosis bulbi?

- Ans.**
- Cornea-Kayser Fleisher's ring, a golden brown ring at the level of descemet's membrane.
 - Lens-sun flower cataract.
 - Retina-golden plaques.

Q.23. What is Vossius ring?

Ans. Imprint of miosed pupil on the anterior surface of the lens.

Q.24. What are the different types of cataract seen after concussion injury?

- Ans.** It is due to imbibitions of fluid from the ruptured capsule.
- Total cataract
 - Rosette cataract (star shaped cataract in post cortex)
 - Late rosette (occurs 1-2 yrs after trauma)

Q.25. What are the common causes of hyphema?

- Ans.**
- Trauma
 - Surgical
 - Spontaneous-neovascularisation of iris-(diabetes malitus)
 - Clotting disorder-leukemia, haemophilia

Q.26. What is the common cause of hyphaema in young?

Ans. Trauma

**Q.27. How will you manage a case of hyphema?**

- Ans.**
- Hospitalisation of patient if hyphaema occupies >1/2 of AC.
 - Bed rest with head end elevation.
 - Topical steroid and cycloplegic(controversial).
 - If IOP is increased----->topical and systemic antiglaucoma medications.
 - Aspirin and anticoagulant should be discontinued.

Q.28. Why bed rest is important?

- Ans.** 72-96 hrs rest is necessary as there is chance of rebleeding during this period.

Q.29. What are the indications of surgical intervention?

- Ans.**
- If IOP>50mm hg for 5 days
 - If IOP>35 mm hg for 7 days
 - Eight- ball hyphema
 - Total hyphema

Q.30. What are the complications of hyphema?

- Ans.**
- Recurrent bleeding
 - Secondary glaucoma
 - Corneal blood staining

Q.31. What factors lead to blood staining of cornea?

- Ans.**
- Increased IOP
 - Rebleeding
 - Decompensated endothelium
 - Long standing large hyphema

Q.32. What is eight ball hyphaema?

- Ans.** Clotted blood-black/purple color due to impaired oxygenation (due to impaired aqueous circulation as a result of pupillary block).

Q.33. What is angle recession glaucoma?

- Ans.** Blunt trauma can cause rupture of the ciliary muscle causing angle recession of the AC.This is associated with scarring of the TBM and glaucoma.

Q.34. What is sympathetic ophthalmia?

- Ans.** This is a condition in which granulomatous uveitis attacks the sound eye after the injury to the other eye.

Q.35. What type of injury causes it?

- Ans.**
- Penetrating injuries
 - Intraocular surgeries (rarely)



Q.36. Injury of which structure is mostly associated with sympathetic ophthalmia?

Ans. Ciliary body (iris/lens capsule can also cause it)

Q.37. What is the time interval between injury and development of sympathetic ophthalmia?

Ans. 4-8 weeks.

Q.38. What is the aetiology of sympathetic ophthalmia?

Ans. Autoimmune.

Q.39. Who are more prone?

Ans. Children and young adults.

Q.40. What is the characteristic pathological feature of sympathetic ophthalmia?

Ans.

- Dalen-Fuchs nodules.
- Pigment epithelium of the iris and ciliary body proliferates to form nodular aggregation. These are seen scattered throughout the uveal tract .

Q.41. What are the characteristic features of sympathetic ophthalmia?

Ans.

- Plastic iridocyclitis present in the injured eye.
- Features in sympathizing eye
 - Photophobia
 - Accommodative paresis
 - Keratic precipitate
 - Retrolenticular flare and cells
 - Vitreous opacities/degenerations
 - Dalen-fuchs nodule
 - Papillitis

Q.42. What is the treatment of sympathetic ophthalmitis?

Ans.

- Prophylactic:
 - In every case of penetrating injury - long term topical and systemic corticosteroid.
 - If the injured or exciting eye has no useful vision → enucleation.
- Treatment of sympathizing eye:
 - Pulse steroid – Topical steroid – Cycloplegics

Q.43. Why sympathetic ophthalmia has become a rare entity nowadays?

Ans. Use of steroid has changed the picture.

Chapter

15

Community Ophthalmology

Q.1. Define blindness.

Ans. WHO definition-BCVA (Best corrected visual acuity) in better eye is $< 3/60$ and/or visual field restricted to 10° .
NPCB definition- BCVA in better eye is $<6/60$.

Q.2. What is economic blindness?

Ans. Visual acuity in better eye is $6/60$ or less and/or visual field restricted to 20° .

Q.3. What do you mean by visually handicapped?

Ans. When the VA in better eye is $6/18$ or less.

Q.4. What is avoidable blindness?

Ans. It includes both preventable and curable blindness.

Q.5. What is preventable blindness?

Ans. Blindness which can be prevented by removing the causative factors or treating them
e.g. Vitamin A deficiency
Diabetic retinopathy

Q.6. Name two important causes of curable blindness.

Ans.

- Cataract
- Glaucoma

Q.7. What is the commonest cause of reversible blindness?

Ans. Cataract

Q.8. What is low vision?

Ans. BCVA in the better eye is $3/60$ - $6/18$

Q.9. What is the magnitude of blindness in India?

Ans. 70 lakh (world 450 lakh)

Q.10. What is NPCB?

Ans. National Program for Control Of Blindness. It was launched in the year 1976.



Q.11. What are the objectives of NPCB?

Ans. To provide eye care facilities by increasing public awareness in under privileged areas .
To combat blindness caused by cataract.

Q.12. What is DBCS?

Ans. District Blindness Control Society. It implements the program for NPCB at district level.

Q.13. What is vision 2020?

Ans. The 'right to sight' is the program of WHO for the elimination of avoidable blindness by the year 2020 AD. It will be implemented through 4 'five years' plan. The 1st one has started in 2000 AD.

Q.14. What are the blindness identified as immediate priorities within the frame work of vision 2020?

Ans.

- Cataract
- Trachoma
- Onchocerciasis
- Childhood blindness
- Refractive error and Low vision

(In India onchocerciasis is not found. Three more conditions are included) –

- Diabetic retinopathy
- Glaucoma
- Corneal blindness

Q.15. What is the commonest cause of blindness in India?

Ans. Cataract

Q.16. What is the prevalence of cataract blindness in India?

Ans. 62% (National Survey 2001–2004)

Q.17. What is corneal transplant?

Ans. It is an operation by which a disc-shaped segment of a defective cornea is removed and replaced by an identical piece of donor cornea.

Q.18. Define eye bank.

Ans. It is an organisation which deals with the collection, storage and distribution of the donor (cornea) for the purpose of corneal grafting, research and supply of the eye tissue for other ophthalmic purposes.



Q.19. When should the eyeball (/cornea) be removed?

Ans. Within 6 hours of death

Q.20. Who can pledge to donate eyes?

Ans. Almost anyone at any age can pledge.

Q.21. Name the conditions where eye collection is absolutely contraindicated?

Ans.

- Active viral hepatitis
- Rabies
- Death due to unknown cause
- Tetanus
- AIDS
- Ocular malignancy

Q.22. In India where the first eye bank was established?

Ans. Madras (1945)

Q.23. When is the National Fortnight on Eye Donation observed?

Ans. 25th August to 8th September

Q.24. What is the significance of the period?

Ans. Late Prime Minister Shri Rajiv Gandhi pledged his eyes for donation on the 25th August to motivate people. Since then a fortnight is celebrated every year.

Q.25. Name few Eye Banks in West Bengal? How many eye banks are there in India?

Ans.

- RIO, Kolkata
- Atul Ballav Eye Bank (NRS Medical college, Kolkata)
- Disha Eye Hospital, Barrackpur
- Howrah Lion's Hospital

India – 600 Eye banks (Functioning – 156)

Q.26. How is the donor's cornea preserved after enucleation?

Ans. M-K Media –is used for short term preservation (upto 96 hrs at 4°C).

Q.27. What does MK stand for?

Ans. McCarey-Kaufman's medium

Q.28. From where will you get the donor cornea?

Ans. Donor cornea is obtained from the enucleated eye ball of a donor who has died recently.



Q.29. What is HCRP?

Ans. Hospital Cornea Retrieval Programme.

Ramayamma International Eye Bank initiated HCRP IN 1990 to concentrate on death that occurs in hospitals and encourage eye donations using a combined method of motivation and grief counselling. HCRP focuses on hospital because:

- Availability of medical history
- Availability of tissues from younger individuals
- Reduction of time interval between death and corneal excision

Q.30. What is School eye health screening (SES) programme?

Ans. SES was initiated under NPCB in 1994. The activities include-

- Identification of schools
- Collection of data regarding students
- Training of teachers (to detect any defect in VA)
- Screening of students by ophthalmic assistants/ ophthalmologists
- Prescription of correcting lens
- Providing free spectacles to children from poor socioeconomic strata
- Referring patient to a higher centre for further management if needed

Q.31. What is the magnitude of childhood blindness?

Ans. World – 1.5 million blind child

Asia – 1 million

Q.32. What are the causes of childhood blindness in India?

- Ans.**
- Vit A deficiency
 - Cataract
 - Refractive error and low vision
 - Corneal opacity
 - Congenital eye diseases

Chapter

16

Ocular Therapeutics

Q.1. What are the different modes of administrations of ocular medications?

- Ans.**
- a. Topical medications
 - Eye drops
 - Eye ointment
 - Gel
 - Ocusert
 - b. Periocular injections
 - Subconjunctival injection
 - Subtenon injection
 - Retrobulbar injections
 - Peribulbar injection
 - c. Intraocular injections
 - Intracameral(AC)
 - Intravitreal(IV)
 - d. Systemic administration

Q.2. Name one indication of subtenon injection?

Ans. Uveitis.

Q.3. Name one condition where intracameral injection of antibiotic is given.

Ans. At the end of cataract surgery.

Q.4. What are the indications of IV (intravitreal) injection?

Ans. IV antibiotics/steroid - In endophthalmitis
IV steroid/antiVEGF - In ARMD, Macular oedema

Q.5. Name some ocular antifungal drugs.

Ans. Nystatin- eye ointment
Amphotericin-B eye drops
Natamycin suspension
Ketoconazole, Fluconazole eye drops



Q.6. What are the common antiviral drugs used in ophthalmology?

Ans. Acyclovir, Idoxuridine

Q.7. What is cycloplegia?

Ans. Paralysis of the ciliary muscles → loss of accommodation.

Salient features of common cycloplegic and mydriatic drugs

<i>Name of Drug</i>	<i>Age of the patient when indicated</i>	<i>Dosage of instillation</i>	<i>Peak Effect of Action</i>	<i>Duration</i>
Atropine Sulphate (1% ointment)	< 5 years	TDS 3 day	2-3 days	10-15 days
Homatropine hydrobromide (2% drops)	5-8 years	One drop every 10 min for 3 times	60-90 min	4-5 days
Cyclopentolate hydrochloride (1% drops)	8-20 years	One drop every 15 min for 3 times	80-90 min	Upto 24 hours
Tropicamide (0.5%, 1% drops) as mydriatic	Used only	One drop every 15 min for 3 to 4 times	20-40 min	10-15 min.
Phenylephrine	Used only as mydriatic alone or in combination with tropicamide	One drop every 15 min for 3 to 4 times	30-40 min	4-6 hours

Q.8. In which condition you should avoid use of cycloplegics/mydriatics?

Ans.

- In shallow AC
- In patient with occludable iridocorneal angles
- Angle closure glaucoma

Q.9. What are the adverse reactions of atropine?

Ans.

- Blurred vision (loss of accommodation)



- Photophobia (due to mydriasis)
- Allergic reactions
- Transient stinging sensation

Systemic side effects

- Dry mouth
- Flushing of the skin
- Cardiovascular side effects - tachycardia, arrhythmias
- CNS effects - dizziness, headache, behavioral disturbances

Corticosteroids**Q.10. What are the common ophthalmic topical steroid preparations?**

- Ans. • Hydrocortisone – 0.5% suspension
– 0.2% solution
- Dexamethasone – 0.1% solution
 - Betamethasone – 0.1% solution
 - Fluomethanole – 0.1% suspension
 - Loteprednole – 0.5% suspension

Q.11. What are the systemic corticosteroids preparations commonly used?

- Ans. • Prednisolone
- Dexamethasone
 - Betamethasone

Q.12. What are the indications of steroid use in ophthalmology?

- Ans. Topical preparation is used in:
- Uveitis
 - Scleritis
 - Allergic conjunctivitis
 - Cystoid macular edema
 - Post operative (cataract, glaucoma, keratoplasty, surgery)

Systemic preparation is used in:

- Uveitis
- Papillitis
- Scleritis
- Orbital pseudotumor
- Post operative (cataract patients)



Q.13. What are the side effects of steroids use?

Ans. Topical steroid:

- Glaucoma
- Cataract
- Dry eye

Systemic corticosteroids:

Ocular:

- Cataract
- Glaucoma
- Papilloedema
- Activation of infection

Systemic:

- Peptic ulcer
- Hypertension
- Cushingoid state

Q.14. Name some topical ophthalmic NSAID preparations.

- Ans.**
- Indomethacin (0.1%)
 - Flurbiprofen (0.3%)
 - Ketorolac tromethamine (0.5%)
 - Diclofenac sodium (0.1%)

Q.15. What are the indications of NSAIDs in ophthalmology?

- Ans.**
- Uveitis
 - Scleritis
 - Episcleritis
 - Cystoid macular edema
 - Pre-operative cataract surgery
 - Vernal Keratoconjunctivitis

Q.16. What is the role of NSAID in preoperative cataract surgery?

Ans. Flurbiprofen drops 2-3 times before surgery are effective in maintaining the pupillary dilatation during the cataract surgery.

Q.17. Name two mast cell stabilizers which are used in ophthalmology?

Ans. Sodium cromoglycate
Lodoxamide



Q.18. Name one condition where we use mast cell stabilizers?

Ans. Vernal conjunctivitis

Q.19. What are the other methods of maintaining pupillary dilatation during cataract surgery ?

Ans.

- Use of adrenaline in irrigation bottle
- Use of viscoelastic
- Use of iris hook

Q.20. What are viscoelastic substances?

Ans. Viscoelastic substances are intraocular surgical devices, which are inert, sterile, transparent, and have certain properties like viscosity, viscoelasticity and pseudoplasticity.

Q.21. Name some viscoelastic substances.

Ans.

- Methyl cellulose (most commonly used)
- Sodium Hyaluronate (1%) – best viscoelastic
- Hypromellose - similar to methyl cellulose
- Chondroitin sulfate

Q.22. What are the uses of viscoelastic substances?

Ans. In cataract surgery

- Maintenance of AC
- Protection of corneal endothelium
- Coating the IOL

Other uses

- Glaucoma , keratoplasty, globe rupture repair surgery
- Coupling agent in gonioscopy

Q.23. What are the side effects of viscoelastics?

Ans.

- Post-operative rise in IOP if it is left in AC (So AC wash is a must at the end of the surgery)
- Toxic anterior segment syndrome (TASS)



Glaucoma: Newer drugs				
Generic name	Trade name	Dosing	Mechanism of action	Side effects
Brimonidine	Alphagan	0.2% BID/TID	A2 Agonist. 1. Decrease Aqueous Production. 2. Increase uveoscleral outflow	Allergy, periocular dermatitis, drowsiness
Apraclonidine	Iopidine	0.5% TID	A2 agonist, decrease Aqueous production	Intolerable allergy; Tachyphylaxis
Latanoprost	Xalatan	0.005% OD	Prostaglandin analogues increase uveoscleral flow	Iris pigmentation, conjunctival hyperemia, burning, eyelash growth and pigmentation, may worsen uveitis, possible drug related cystoids macular edema and reactivation of ocular Herpes simplex.
Bimatoprost	Lumigan	0.03% OD	Topical carbonic anhydrase inhibitor- decrease aqueous production	Ocular pain, blurred vision, Taste perversion. Other side of CAI as in previous table.
Travoprost	Travatan	0.004% OD		
Unoprostone Isopropyl	Rescula	0.12% BID		
Dorzolamide	Trusopt	2% TID		
Brinzolamide	Azopt	1% BID	DO	

**Antibacterial ocular medications, dosages and routes of administration**

<i>Antibiotic</i>	<i>Topical</i>	<i>Subconjunctival</i>	<i>Intra vitreal</i>
Cefazolin	5%	100 mg	2.25mg/ .1 ml
Ceftriaxone	10%	100 mg	3.0 mg
Ciprofloxacin	0.3%		0.1 mg
Ofloxacin	0.3%		0.1 mg
Moxifloxacin	0.5%		
Gatifloxacin	0.3%		
Vancomycin	2.5–5%	25 mg	1 mg/0.1 ml
Tobramycin	1–1.4%	20–40 mg	0.2 mg/0.1 ml
Gentamicin	0.3–1.4%	20–40 mg	0.1 mg/0.1 ml
Amikacin	1–2.5%	25–50 mg	0.2–0.4 mg/0.08 ml

Antiviral ocular medications

<i>Medication</i>	<i>Concentration</i>
5-iodo-2-deoxyuridine (IDU)	0.1% drops/ 0.5% ointment
Trifluorothymidine	1% drops
Adenine arabinoside	3% ointment
Acyclovir	3% ointment

Antifungal medications

<i>Drug</i>	<i>Topical</i>	<i>Subconjunctival</i>	<i>Intravitreal</i>
Amphotericin B	1 – 10 mg/ ml drops, 0.37 mg 2.5% ointment		5-10 microgm
Natamycin	5% suspension		25 microgm
Miconazole	1% drops, 2% ointment		
Ketoconazole	1% – 5%		0.5 microgm
Fluconazole	0.2% – 2%		0.1 microgm
Flucytosine	1%		



Different ocular viscosurgical devices (OVDs) used during cataract surgery

<i>Composition</i>	<i>Commercially available</i>	<i>Main properties</i>	<i>Indications for use</i>
Hydroxypropyl methyl cellulose 3%	Visilon, ocucoat	Viscoelastic	Extracapsular cataract surgery, conventional and manual SICS and phacoemulsification
Sodium hyaluronate 1%	Healon, Amvisc	Viscoelastic and cohesive	Especially if shallow anterior chamber
Sodium hyaluronate 1.4%	Healon G.V.	Cohesive	Small pupil, mature white cataract or in children
Sodium hyaluronate 2.4%	Healon 5	Viscoadaptive	Phacoemulsification of hard cataract and cases with small pupil
Sodium hyaluronate 3% and chondroitin sodium sulphate 4%	Viscoat	Viscoelastic with a high coating ability	Especially in cases with compromised corneal endothelial function

Chapter

17

Instruments

Universal Eye Speculum-(Fig. 17.1)

- Id points:
 - Two limbs attached with an adjusting screw
 - Why it is called universal?
 - It can be used for both the eyes
 - How to hold the instrument?
 - Convexity in front
 - Concavity backward
 - Screw faces outward and forward

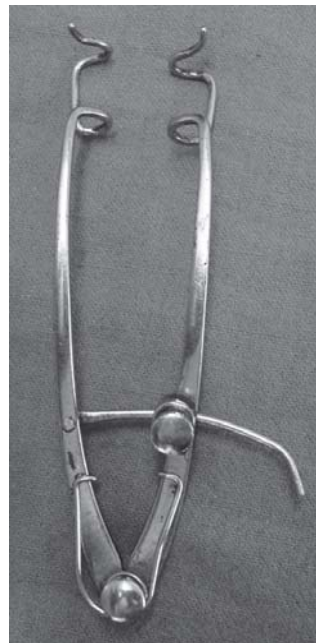


Fig. 17.1: Universal eye speculum



Barraquer Wire Speculum (fig-17.2)

- Id points:
 - No Screw

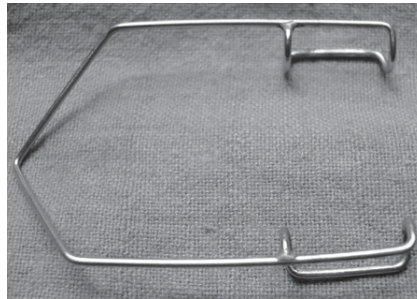


Fig. 17.2: Wire speculum

Speculum with Screw (fig-17.3)

- Similar to universal eye speculum with adjustable screw.
- What are its advantages?



Fig. 17.3: Speculum with screw



- Very light weight.
 - Gives minimum pressure on the globe.
- What are the uses of eye speculum?
 - Removal of conjunctival, corneal foreign body.
 - Removal of suture.
 - Pterygium operation.
 - Cataract operation.
 - Squint operation.
 - Enucleation, Evisceration.
 - Removal of conjunctival tumors/cysts.
 - Examination of the eye in a patient with blepharospasm.

Barraquer's Needle Holder (fig-17.4)

- *Id points:*
 - Smaller tipped fine needle holders.
 - Curved tip with groove for holding 10-0/8-0 sutures needle.
- What are its uses?
 - Suturing conjunctiva, cornea, extraocular muscles, sclera, etc.

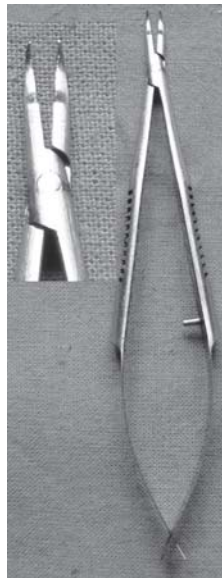


Fig. 17.4: Barraquer's needle holder



Artery forceps (fig-17.5)

- *Id points:*
 - Medium size, serrated with catch.
 - May be curved or straight.
- What are its uses?
 - To hold lid stitches and superior rectus stitch and then to fix the suture ends with head towel.
 - To crush lateral canthus in lateral canthotomy.
 - For hemostasis in DCR or DCT if angular vein is damaged.
 - To hold muscle stump during enucleation operation.
 - To hold lacrimal sac prior to excision in DCT.



Fig. 17.5: Artery forceps

Thermo Cautery (fig. 17.6)

- *Id points:*
 - A metallic ball (made of steel or brass) with pointed tip attached with a handle.

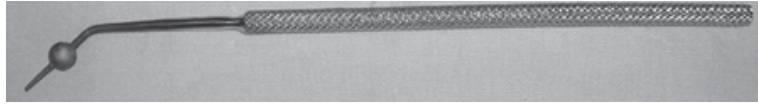


Fig. 17.6: Thermo cautery

- What are its uses?
 - To cauterize superficial bleeding points during cataract surgery, trabeculectomy.
 - To cauterize the bare sclera area in pterygium operation.
 - To cauterize the margin of progressive corneal ulcer and small iris prolapse.
 - Temporary punctal occlusion.
- What is the other mode of cauterization?
 - Wet field electrocautery (most commonly used during ocular surgery now a days).

Bard Parker Handle (fig. 17.7)

- *Id points:*
 - Flat handle, neck is short grooved. Blades are fixed in the neck of the handle.
- What are its uses?
 - To give incision on sclera in cataract surgery.
 - In trabeculectomy operation.
 - To scrap out the head of pterygium from cornea.
 - To give incision in DCR/DCT operation.
 - In chalazion operation
 - Sutures can be removed after different operations.



Fig. 17.7: BP handle



No 11 surgical blade (fig. 17.8)

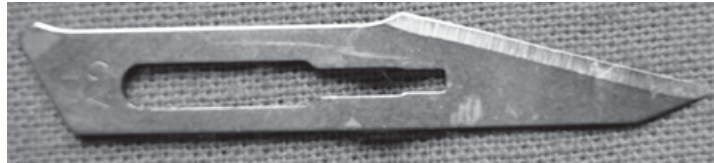


Fig. 17.8: 11 no. blade

No 15 surgical blade (fig. 17.9)

Where we use these in ophthalmology?
(See ans fig. 17.7).

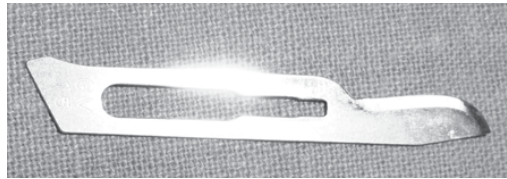


Fig. 17.9: 15 no. blade

Von Graefe's Cataract Knife (fig. 17.10)

(It is obsolete nowadays).

- *Id points:*
 - It has long handle with pointed, thin blade at one end. Blade has sharp edge.
 - Blade can be slide inside the handle if not in use.
- What are its uses?
 - Used in cataract surgery (ICCE) for giving corneo-scleral incision from inside to outside (ab-interno).

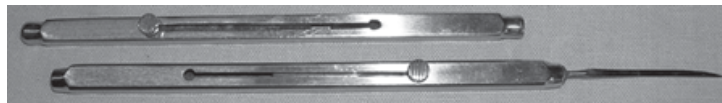


Fig. 17.10: Von Grafee Knife

**Straight Scissors (fig. 17.11)**

- *Id points*
 - Straight/Curved variety, pointed tips.
- Where it is used?
 - For cutting conjunctiva, suture in extra and intraocular operations.

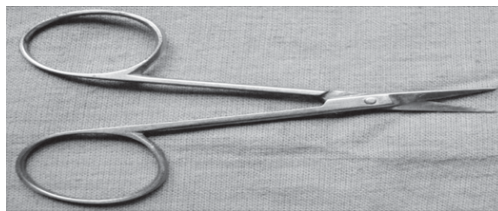


Fig. 17.11: Straight scissors

Tenotomy Curved Scissors (fig. 17.12)

- *Id points*
 - Curved, medium size blades with blunt tips
- Where it is used?
 - Tenotomy in squint and retina surgery.



Fig. 17.12: Tentomy curved scissors

Corneal Spring Scissors (universal) (fig-17.13)

- *Id points* –
 - Straight or curved blade.
 - Spring action in handle.
- Why it is universal?
 - It can be used to cut both Right and Left part of the sclerocorneal section.

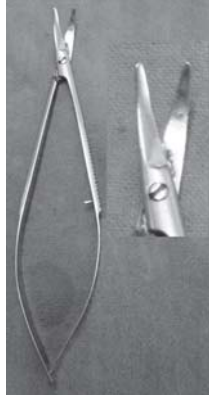


Fig. 17.13: Corneal spring scissors

- *What are its uses?*
 - Cutting of conjunctiva (peritomy)/Tenon's capsule from the limbus during ab externo incision.
 - For extension of corneo sclera incision.
 - For iridectomy.
 - Cutting of 8-0, 10-0 suture during suturing.

Corneal Scissors (unilateral) (fig. 17.14)

- *Id points:*
 - Small curved spring scissors with sharp blades.
- *Where it is used?*
 - Rt sided corneal scissors (to be hold in left hand) is used to cut the right half of the section (ECCE of RE/LE).
 - Lt sided corneal scissors (to be hold in right hand) to cut the left half of sclerocorneal section (ECCE of RE/LE).



Fig. 17.14: Corneal scissors unilateral direction (left)

**Vanna's Scissors (fig. 17.15)**

- *Id points:*
 - Small, fine blades.
 - Straight/curved/angular blades.
- *What are its uses?*

Use in cataract surgery:

- To cut anterior capsular tags.
- Open sky vitrectomy in case of vitreous loss due to PC rent.
- Iridectomy.
- To cut 10-0 sutures.

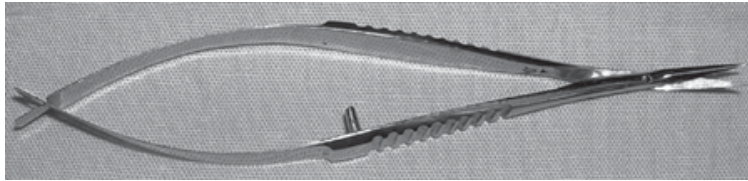


Fig. 17.15: Vanna's scissors

De-Wecker's Scissors (fig. 17.16)

- *ID points:*
 - Blade at right angles to the arms.
 - Blades have a special quick cutting spring action.
- *What are its uses?*
 - In all types of iridectomy.
 - Sphincterotomy at 6 o'clock position.

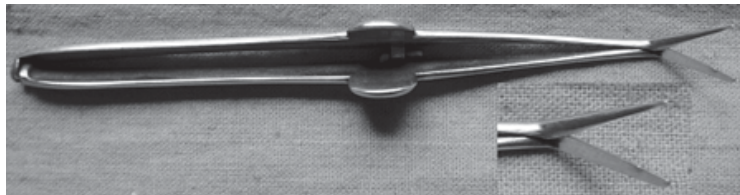


Fig. 17.16: De-wecker's scissors

Arruga's Intracapsular Forceps (fig. 17.17)

- *Id points:*
 - Cup on inner side of tip of each limb.

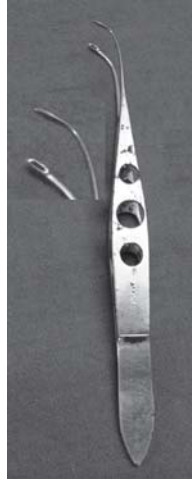


Fig. 17.17: Arruga's intracapsular forceps

- Where it is used?
 - To hold the lens capsule during removal of lens in ICCE.

Epilation Forceps (fig. 17.18)

- *Id points:*
 - Small stout forceps
 - Blunt and flat ends
 - Where it is used?
 - *To remove the trichiatic eyelashes.*
- What are the other methods of removal of trichiatic eye lashes?
 - Electrolysis
 - Cryotherapy
 - Diathermy

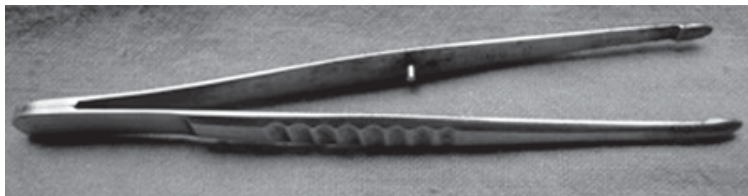
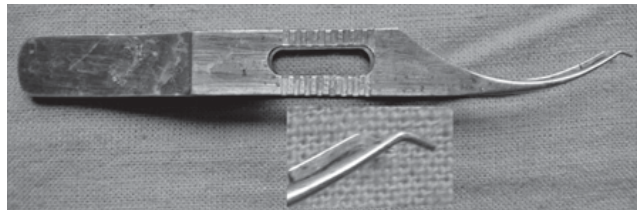


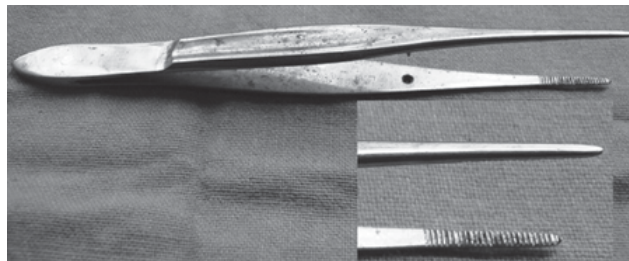
Fig. 17.18: Epilation forceps

**Colibri Forceps (fig. 17.19)**

- *Id points:*
 - Fine teeth (1×2).
 - Tying platform.
- What are its uses?
 - To hold the cornea or scleral edge for suturing during cataract, glaucoma, repair of corneal or sclera rupture and keratoplasty operations.

**Fig. 17.19:** Colibri**St. Martin's Forceps - (fig-17.20)**

- *Id points:*
 - Straight.
 - Stout.
 - Tooth (1×2)
- What are its uses?
 - To hold the delicate tissue like cornea without crushing.
 - To hold cornea/sclera during suturing.

**Fig. 17.20:** St. Martin forceps



Elschnig's Intracapsular Forceps (fig. 17.21)

- *Id points:*
 - Non-toothed, double curved with blunt tips.
- What are its uses?
 - Same as arruga's.



Fig. 17.21: Elschnig's intracapsular forceps

Iris Forceps (fig. 17.22)

- *Id points:*
 - Fine tooth(1x2) with curved or angled limbs.
- *Uses:*
 - To catch the iris during iridectomy.

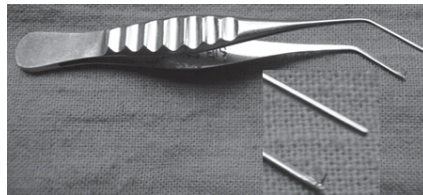


Fig. 17.22 : Iris forceps

Plain Smooth Forceps - (fig-17.23)

- Tip (toothless), shaft, handle.
- What are its uses?
 - Used to manipulate the delicate tissues and holding the sutures.

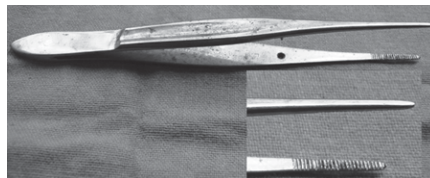


Fig. 17.23: Plain forceps

**Mc Pherson's Forceps (fig. 17.24)**

- *ID points:*
 - No tooth.
 - Angulation at the front.
- What are its uses?
 - To remove anterior capsule after capsulotomy/capsulorhexis.
 - To introduce IOL during cataract surgery.
 - As a suture tier during suturing.
- Why the limbs are fenestrated?
 - To have a better grip.
 - To make it lighter.

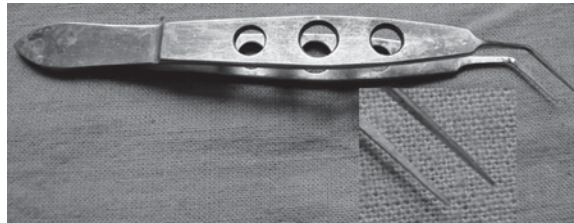


Fig. 17.24: McPherson's forceps

Capsulorhexis Forceps (fig. 17.25)

- *ID points:*
 - Non-toothed with curve sharp tip.
- What are its uses?
 - Used for continuous curvilinear capsulorhexis (CCC) in cataract surgery (Phaco, SICS).
- What is the alternative way to do CCC?
 - It can be done with a bent 26 gauze needle .

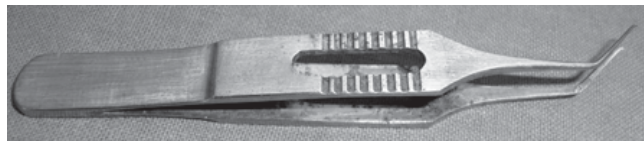


Fig. 17.25: Capsulorhexis



Scleral Fixation Forceps (fig. 17.26)

- *Id. points:*



Fig. 17.26: Scleral fixation forceps

- What are its uses?
 - For grasping conjunctiva, episcleral tissue during ocular surgery.
 - Fixing the globe to facilitate the surgical manoeuvres.
 - Lifting conjunctiva for subconjunctival injection.

Fixation Forceps (fig. 17.27)

- *Id points:*
 - Toothed (2×3).
- What are its uses?
 - Fixing the globe to facilitate the surgical manoeuvres.
 - To pass the superior rectus bridle suture during cataract surgery.

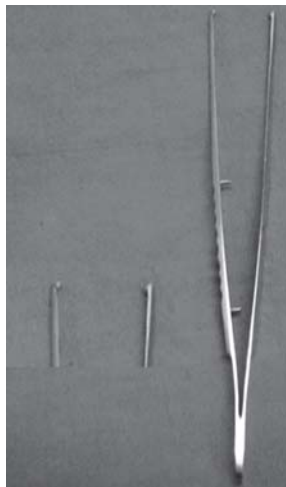


Fig. 17.27: Tooth forceps

**Dastoor Superior Rectus Forceps (fig.17.28)**

- *ID points:*
 - Stout forceps with 'S' shaped double curvature at the tip.
 - Handle is serrated or multiple large holes.
- Where it is used?
 - To catch the superior rectus for bridle suture in cataract surgery.



Fig. 17.28: Superior rectus holding forceps

Lens Holding Forceps (17.29)

- *Id points:*
 - Angled platform for holding the IOL.
- Where it is used?
 - To introduce the lens during cataract surgery.

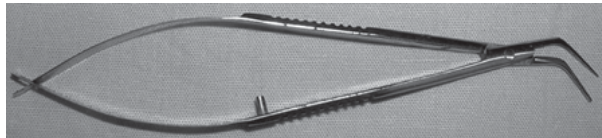


Fig. 17.29: Lens holding forceps

Suture Tier (fig. 17.30)

- *Id points:*
 - Straight limb with tying platforms.
 - Long fine limbs.
 - No tooth.
- Where it is used?
 - To hold the suture ends during suturing .
 - Removal of caterpillar hair.

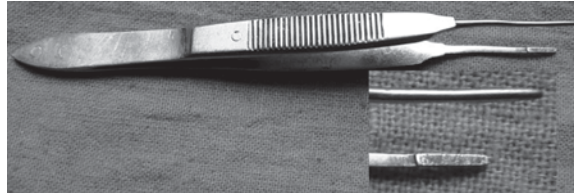


Fig. 17.30: Suture tier

Lens Expressor (fig. 17.31)

- *Id points:*
 - Flat metal handle with corrugation with a rounded curve at one end.
 - Plane of the handle is at right angle to the curvature of the hook.
 - Tip of the curve is knobbed.
- Where it is used?
 - Delivery of nucleus in ECCE/ lens in ICCE.
 - As muscle hook and retractor in DCR/DCT.

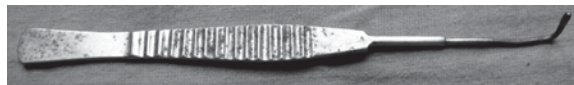


Fig. 17.31: Lens expressor

Irrigating Vectis (fig. 17.32)

- *ID points:*
 - Hollow vectis.
- Where it is used?
 - For nucleus delivery in SICS (connected with the irrigating system or with a 2 cc syringe).

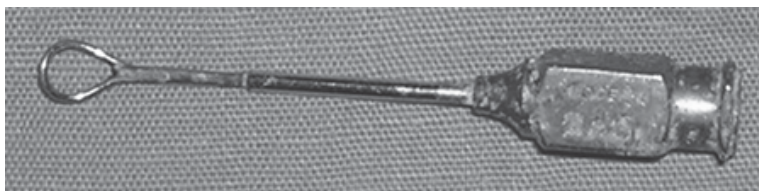


Fig. 17.32: Irrigating vectis



- **What complications can occur during nucleus delivery?**
 - Iridodialysis
 - Zonular dialysis

Wire Vectis (fig. 17.33)

- *Id points:*
 - Wire loop attached to a metallic handle.
- What are its uses?
- For removal of the nucleus in:
 - SICS
 - ECCE
 - ICCE (subluxated/dislocated lens).

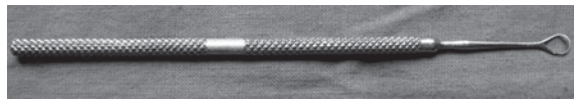


Fig. 17.33 : Wire vectis

Irrigation Aspiration Cannula (IA) (fig. 17.34)

- *Id points:*
 - Two way cannula.
 - One end is attached to blunt needle via a silicone tube.
- *Where it is used?*
 - Aspiration of lens cortex/viscoelastic in ECCE/SICS.
 - Cleaning of blood from AC.

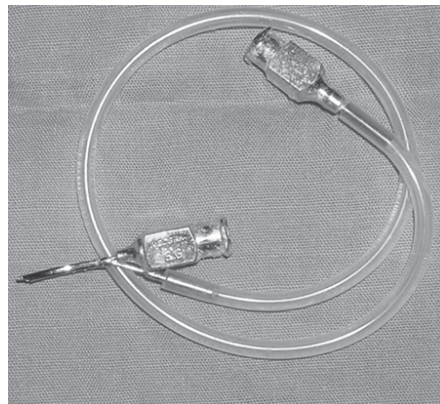


Fig. 17.34: IA Cannula



- *Direct Simcoe:*
 - Aspiration is through the silicone tube and irrigation through the main tube (surgeon holds the syringe attached to the silicone tubing used for aspiration).
- *Reverse simcoe* – Irrigation is through the silicone tube and aspiration through main tube.
- What complications can occur during IA?
 - PC rent.
 - Repeated iris prolapse.

Disposable Cystitome (17.35)

- *Id points:*
 - Made by bending 26G disposable needle (at tip and route of the needle).
- What is its use?
 - For doing capsulotomy/capsulorhexis during ECCE.

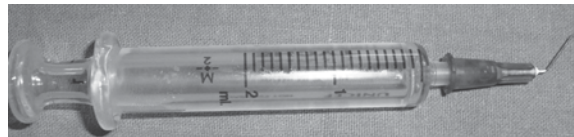


Fig. 17.35: 2 cc syringe with 26 G needle (cystitome)

Iris Repositor (fig. 17.36)

- *Id points:*
 - Its S or Z shaped.
 - Tip rounded.
- What are its uses?
 - Repositioning of iris after iridectomy or cataract operation.
 - To break synechiae at the pupillary margin.

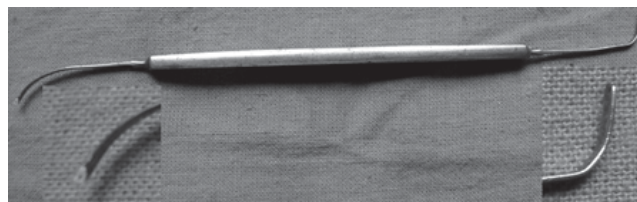


Fig. 17.36: Iris reposer

**Sinsky's Hook (Dialler) (fig. 17.37)**

- Long round solid handle.
- Angular fine hook.
- *What are its uses?*
 - To dial the IOL (during cataract surgery).
 - To rotate the lens nucleus.
 - To chop the nucleus in phacoemulsification.
 - Prolapsing nucleus into the AC after hydrodissection in SICS.
- *Why dialing is needed?*
 - If the IOL is not positioned properly there will be astigmatism in post-operative period.

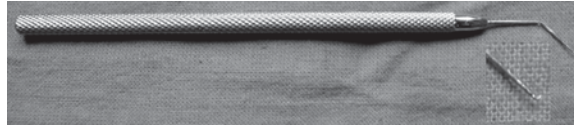


Fig. 17.37: Sinsky hook (dialler)

Crescent Knife - (fig-17.38)

- *Id Points:*
 - Thin crescentic blade with forward angulation (plastic handle).
 - Blunt-tipped, bevel up.
 - Cutting and splitting action at the tip and both the sides.
- *What are its uses?*
 - To make the horizontal part of the sclerocorneal tunnel in SICS/Phaco.
 - Lamellar dissection in lamellar keratoplasty, trabeculectomy.



Fig. 17.38: Crescent Knife

Keratome Knife - (fig-17.39)

- *Id points:*
 - Sharp cutting surfaces on both sides and sharp tip.
- *Where it is used?*
 - Last part of the sclerocorneal tunnel to enter into the AC (SICS, PHACO).



Fig. 17.39: Keratome Knife

Side Port Entry Blade (fig. 17.40)

- *Id points :*
 - Thin long straight blade.
 - Cutting edge on one side.
- *Where it is used?*
 - Making side port to enter into the AC in SICS/phaco-emulsification/vitrectomy.
- What is the purpose of making side port?
 - To inject viscoelastic.
 - To inject trypan blue dye.
 - To clean 12 'oclock cortical matter with IA cannula.



Fig. 17.40: MVR Knife

Desmarre's Upper Eye Lid Retractor (fig. 17.41)

- *Id points:*
 - Saddle shaped with long metallic handle.
 - Not self retaining.
- What are its uses?
 - While examining children.
 - In lid edema and blepharospasm.
 - Un-cooperative patients examination.

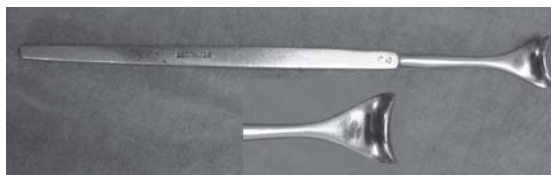


Fig. 17.41: Desmarre's Lid retractor

**Chalazion Clamp (fig. 17.42)**

- *Id points:*
 - It is a forceps with a large screw for fixing or tightening the limbs like a clamp.
 - One limb has a solid disc-shaped plate and the other limb has a ring at its end.
 - It is haemostatic (by pressure of the ring on the plate) and self retaining.
- *What are its uses?*
 - To fix the chalazion for surgery and also to ensure haemostasis.
 - To give the intralesional injection of steroids in chalazion after fixing it with the forceps.
 - Excision of a small granuloma or papilloma of the lid.



Fig. 17.42: Chalazion clamp

Chalazion scoop (fig. 17.43)

- *Id points:*
 - It is a small scoop with a sharp edge, attached to a handle.
 - The size of the scoop may vary.
- *What are its uses?*
 - To scoop out the granulation tissue after giving incision on chalazion.

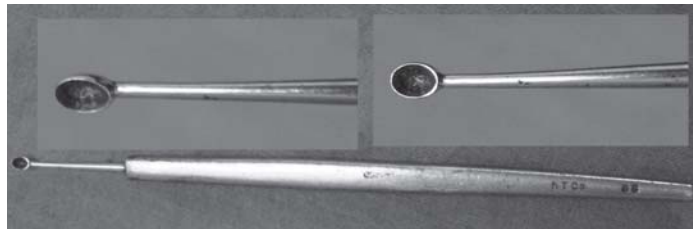


Fig. 17.43: Chalazion scoop



Nettleship Punctum Dialator (fig. 17.44)

- *Id points:*
 - Cylindrical corrugated metal handle.
 - Conical pointed end.
- *Where it is used?*

Dilatation of punctum and canaliculus during—

 - Syringing
 - Dacrocystography
 - Probing
 - DCT/DCR

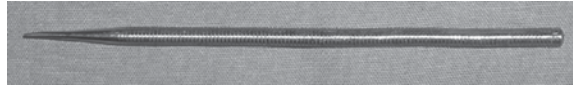


Fig. 17.44: Nettle's punctum dilator

Lacrimal Canula (fig. 17.45)

- *Id points:*
 - Long curved hypodermic needle with blunt tip.
- *What are its uses?*
 - Syringing the lacrimal passages.
 - As AC cannula for introducing air or BSS in the AC during intraocular surgery.

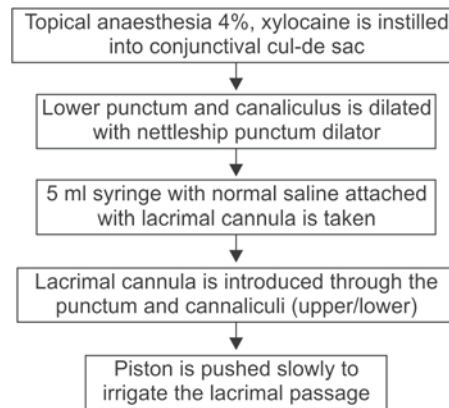


Fig. 17.45: Lacrimal cannula



Q. Describe the steps of syringing.

Ans.



Q. What are the interpretations of the results of lacrimal syringing?

Ans. Saline water passes freely to the nose to throat → No obstruction.

Fluid regurgitates through same punctum → blockage of common canaliculus.

Fluid comes out through upper punctum immediately → blockage at common canaliculus (CC).

Slow regurgitation → Blockage at the nasolacrimal duct (NLD)

Q. What is the T/t of NLD block?

Ans. DCR operation.

Q. What is the T/t of CC block?

Ans. Canaliculo-DCR (CDCR).

Q. What complications can occur during syringing?

Ans. False passage.

Q. What are the indications of syringing?

Ans. • Chronic dacryocystitis

- Congenital dacryocystitis.
- Post-op DCR.



Lacrimal Dissector with Scoop (Lang's) (fig. 17.46)

- *Id points:*
 - A cylindrical instrument
 - One end having elongated scoop.
 - Other end having pointed dissector.
- *What are its uses?*
 - Dissector end is used for-
 - Blunt dissection of lacrimal sac in DCR/DCT operation.
 - To open up nasolacrimal duct in DCT.
- *Scoop end is used:*
 - In DCT to scoop out the tissue remnants after excision of lacrimal sac.



Fig. 17.46: Lacrimal dissector with scoop

Rougine (fig. 17.47)

- *Id points:*
 - Non-corrugated handle.
 - A rectangular shaped small blade which is beveled on one side.
- *What are its uses?*
 - To do blunt dissection of soft tissue during initial steps of DCT/DCR.
 - To dissect the sac from the medial wall in DCT/DCR operation.



Fig. 17.47: Rougine

Cat's Paw Retractor-(fig-17.48)

- *Id points:*
 - Fork like instrument with distal end bent inwards.
 - Tips are a bit blunt.



- *Where it is used?*
 - In lacrimal sac operation
 - Lid surgery

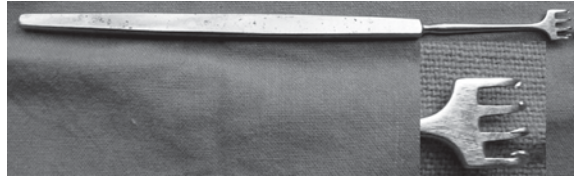


Fig. 17.48: Cat's-paw retractor

Bone Punch (fig. 17.49)

- *Id points:*
 - Spring handle with two stout blades.
 - (different sizes are available).
- What are its uses?
 - In DCR operation to break the bones while making the bony osteum.
- What are the bones cut to make the bony ostium?
 - Lacrimal bone.
 - Part of nasal bone
 - Frontal process of maxilla.

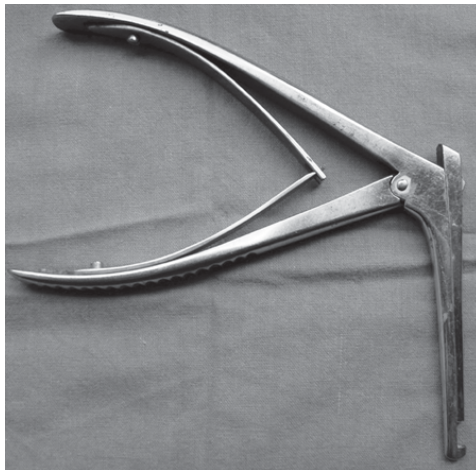


Fig. 17.49: Bone punch



Evisceration Scoop (fig. 17.50)

- *Id points:*
 - Spade shaped.
- Where it is used?
 - To scoop out contents of eyeball (lens, vitreous, uvea, retina)leaving only sclera behind during evisceration surgery.
- What are the indications of evisceration?
 - Expulsive haemorrhage.
 - Panophthalmitis.



Fig. 17.50: Evisceration scoop

Enucleation Scissors - (fig-17.51)

- *Id points:*
 - Curved scissors.
 - Long stout blades.(Biggest scissors used in ophthalmology.)
- Where it is used?
 - During enucleation to cut the optic nerve.
- What are the **indications of enucleation?**

In cases of:

- Malignant ocular tumour-e.g retinoblastoma in children, malignant melanoma in adult.
 - Grossly lacerated injury of the globe.
 - Painful blind eye.

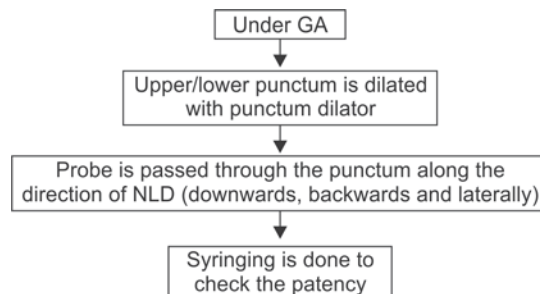


Fig. 17.51 : Enucleation scissors

- What structures are cut with this scissors?
 - Optic nerve with its meningeal sheaths.
 - Central retinal artery and vein.
 - Two long posterior ciliary arteries and long ciliary veins.
 - Two oblique muscles.

Lacrimal Probe (fig. 17.52)

- *Id points:*
 - Straight thin metal wire with blunt ends with flat central platform.
(probes are of different diameters).
- Where it is used?
 - In congenital dacryocystitis where probing is done to break the obstruction of the NLD.
 - During DCR operation while making the sac flap.
- How probing is done?





- What are the complications of probing?
 - False passage.

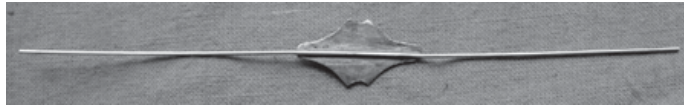


Fig. 17.52: Lacrimal probe

Entropion Clamp (fig. 17.53)

- Stout clamp with two limbs. One is solid semilunar plate like and the other one has 'U' shaped curved rim.
- *Where it is used?*
 - To fix lid during entropion surgery.



Fig. 17.53: Entropion clamp

- Solid plate will be facing towards the conjunctiva and the other one towards the skin (reverse of chalazion clamp).
(It is not used nowadays as it reduces the operative field).

**Calliper (castroveijo's) (fig. 17.54)**

- *Where it is used?*
To measure
 - The size of cornea.

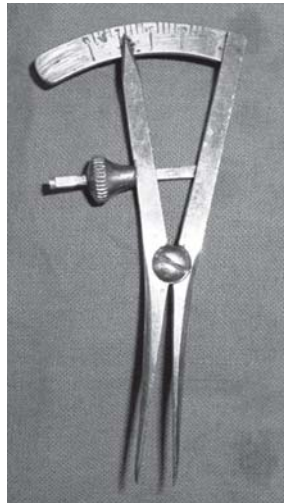


Fig. 17.54: Callipre

- Length of incision (Phaco/SICS).
- In trabeculectomy length of scleral flap.
- In squint operation amount of resection or recession of muscles.
- Size of donor and recipient corneal button in keratoplasty.

Muller's Self Retaining Haemostatic Retractor (fig. 17.55)

- *Id points:*
 - Consists of two limbs.
 - Each limb has three curved pin like structures with blunt tips.
 - Screw between two limbs.
- What are its uses?
 - In DCT/DCR operation to retract skin.
 - Haemostasis .



- What are its disadvantages?
 - Damage to skin (laceration).
 - Injury to angular vein.



Fig. 17.55: Mullers hemostatic self retaining retractor

Muscle Hook (fig. 17.56)

- *Id points:*
(Similar to the lens expressor in appearance)
 - Flat handle (no corrugation).
 - Plane of the handle is the same as that of the curvature of the hook.
 - Blunt guarding knob.

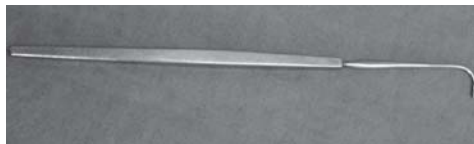


Fig. 17.56: Squint hook

- *Where it is used?*

For hooking extraocular muscles in

- In squint operation.
- In retinal surgery.

**Concave Spherical Lens (fig. 17.57)**

- *Id points:*
 - Minification of the object seen through it.
 - Images seen through it move in the same direction.
 - No distortion of the objects on rotation of the lens.
- What are its uses?

Correction of refractive error in:

- Myopia.

Instrumental:

- Direct ophthalmoscope.



Fig. 17.57: Spherical concave lens

Diagnostic:

- Hruby lens
- Central gonio lens.

Convex Spherical Lens (fig. 17.58)

- *Id points:*
 - Magnification of the objects seen through it.
 - Images seen through it moves in opposite direction.
 - Movement of the object is same in all meridian.
 - On rotation of lens no distortion of the image.
- What are its uses?

Correction of refractive error in:

- Hypermetropia
- Presbyopia
- Aphakia



- Low visual aids (LVA)



Fig. 17.58: Spherical convex lens

Concave cylindrical lens (fig. 17.59)

- *Id points:*
 - Minification of images.
 - Movement of objects in same direction (only in one meridian).
 - Distortion of images on rotation of the lens.
- What are its uses?
 - Myopic astigmatism (simple, compound, mixed).
- If the patient has irregular astigmatism which lens is preferred?
 - Contact lens.
 - Aphakia
 - Low vision aid.



Fig. 17.59 : Cylindrical concave lens



Used in instrument-

- Ophthalmoscopy
 - Direct
 - Indirect
- Microscope
- Corneal loupe

Convex Cylindrical Lens (fig. 17.60)



Fig. 17.60: Cylindrical convex lens

- *Id points:*
 - Magnification of the images.
 - Movement of objects in opposite direction (only in one meridian).
 - Distortion of the images on rotation of lens.
- What are its uses?
 - Used in hypermetropic astigmatism (simple compound and mixed).

Maddox Rod (fig. 17.61)

- *Id points:*
 - It consists of a series of high power plus cylinder of red glasses placed in a disc.
- *Principle:*
 - It converts points into line at right angle to axis of rods.



- *What are its uses?*
 - To detect latent squint (heterophoria) by dissociation of images.
 - Macular function test in opaque media.



Fig. 17.61: Maddox rod

Pin-hole (fig. 17.62)

- *Id points:*
 - It is a black colored opaque disc with central hole.
- *What are its uses?*
 - To differentiate whether impaired vision is due to refractive error or organic causes.



Figure 17.62: Pin hole

(In cases of refractive error vision improves).



In organic pathology, e.g. macular or neuroophthalmic disease vision remain same or deteriorate.

- To measure potential visual acuity following cataract surgery.
- *What is the principle of Pin-hole*

It acts like a Pin-hole camera.

(It allows only a small pencil of rays to pass through the axis of dioptric system of the eye so image is not blurred).

It differentiates whether the dimness of vision is due to refractive error or due to retinal or neuroophthalmologic diseases. In the first case VA will improve with PH but not in the others.

Stenopic Slit (fig. 17.63)

- *Id points:*
 - It is a black colored opaque disc with a slit in the centre.
- *Where it is used?*
 - For detection of axis of astigmatism
 - To differentiate the colored halos due to glaucoma or cataract.
 - Halos intact in glaucoma (edema in cornea).
 - Halos broken in immature cataract (water vacuole in lens).
 - As a low vision aids.

Sterilisation of Surgical Instruments

Q. How will you sterilize operation theatre (OT)?

Ans. With 40 % formalin.

Q. How will you sterilize Linen (gown, cap mask, drape etc)?

Ans. Autoclaving.

Q. How will you sterilize glassware (syringe)?

Ans. Hot air oven (dry heat).

Q. How the metal instruments are sterilized?

Ans. Sharp instruments – dry heat.

Moisture resistant instruments – autoclave.

Q. How the IOL is sterilized?

Ans. ETO (ethylene trioxide).

Q. What is the temperature and time required for autoclaving?

Ans. 121° C - 45 minutes.

Flash autoclave -



Q. What is sterilization?

Ans. The process by which by all microorganisms (including spores) are killed.

Q. What is disinfection?

Ans. Process by which all microorganisms are killed or removed (but not necessarily spores).
34°C – 20 minutes.



Figure 17.63: Stenopic slit

Chapter

18

Miscellaneous

VITAL STATISTICS OF AN EMMETROPIC EYE

Anteroposterior length	24 mm (approx)
Horizontal diameter	23.5 mm (approx)
Vertical diameter	23 mm (approx)
Volume of eyeball	7 cc
Total orbital volume	30 cc
Anterior corneal diameter	<ul style="list-style-type: none">• 11 mm (vertically)• 12 mm (horizontally)
Radius of curvature of cornea	<ul style="list-style-type: none">• 7.8 mm (anterior)• 6.5 mm (posterior)
Thickness of cornea	<ul style="list-style-type: none">• 0.52 mm (central)• 0.62 mm (peripheral)
Dioptric power of cornea	+43 D
Refractive index of lens	1.39
Dioptric power of lens	17 to 18 D (with accommodation at rest)
Radius of curvature of lens	<ul style="list-style-type: none">• 10 mm (anterior surface)• 6 – 9 mm (posterior surface)

CAUSES OF RED EYE

- | | |
|--|---|
| <ul style="list-style-type: none">• Conjunctivitis• Inflamed pinguecula• Episcleritis• Scleritis• Foreign body• Corneal ulcer | <ul style="list-style-type: none">• Subconjunctival haemorrhage• Acute iridocyclitis• Acute congestive glaucoma• Pterygium |
|--|---|



D/D OF CONJUNCTIVAL AND CILIARY CONGESTION

<i>Features</i>	<i>Conjunctival congestion</i>	<i>Ciliary congestion</i>
Site	More marked in the fornices	More marked around the limbus
Colour	Bright red	Purple or dull red
Arrangement of vessels	Superficial and branching	Deep and radiating from limbus
On moving conjunctiva	Congested vessels also move	Congested vessels do not move
On mechanically squeezing out the blood vessels	Vessels fill slowly from fornix towards limbus	Vessels fill rapidly from limbus towards fornices
Blanching, i.e. on putting one drop of 1 in 10000 adrenaline	Vessels immediately blanch	Do not blanch
Common causes	Acute conjunctivitis	Acute iridocyclitis, keratitis (corneal ulcer)

Causes of gradual painless and progressive diminution of vision (in adult)

Refractive error	Corneal dystrophy
Presbyopia	Retinitis pigmentosa
Age related cataract	Drug induced maculopathy (chloroquin)
Primary open angle glaucoma	
Keratoconus	
Age related macular degeneration	
Diabetic retinopathy	



Causes of Sudden Painless Diminution of Vision

<i>Unilateral</i>	<i>Bilateral</i>
Retinal Detachment	
Retinal vascular occlusion (BRVO, CRVO, CRAO)	Bilateral occipital infarction
Vitreous haemorrhage	Optic neuritis
Retinal haemorrhage	Diabetic retinopathy
Wet ARMD	Hypertensive retinopathy with macular star (grade 1V)
Subluxation or dislocation of the lens	Toxic optic neuropathy
Anterior ischaemic optic neuropathy (AION)	Posterior uveitis
Posterior ischaemic optic neuropathy	

Diminution of vision associated with pain and/or an acute red eye

Uveitis	Endophthalmitis
Corneal ulcer	Retrobulbar neuritis
Acute angle-closure glaucoma	

Causes of Transient Loss of Vision

- Papilloedema
- Amaurosis fugax
- Migraine

Causes of Tubular Vision or Generalized Constriction of Fields

- Advanced glaucoma
- Retinitis pigmentosa

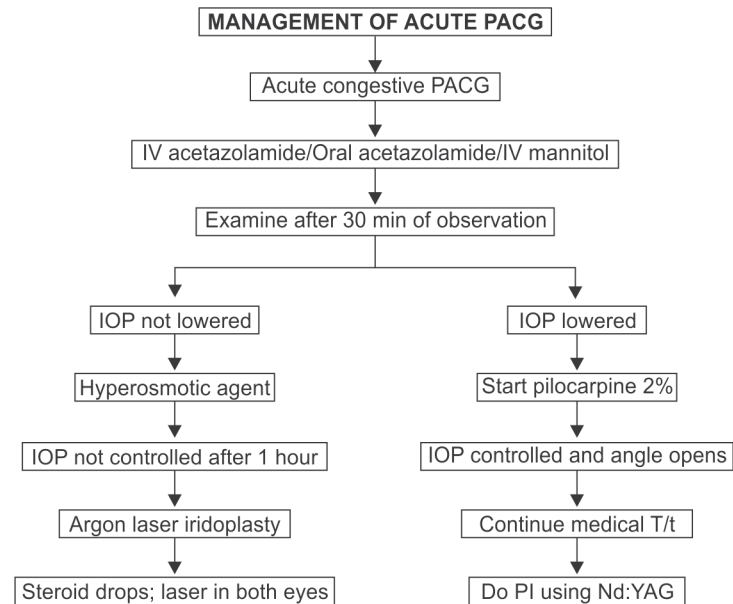


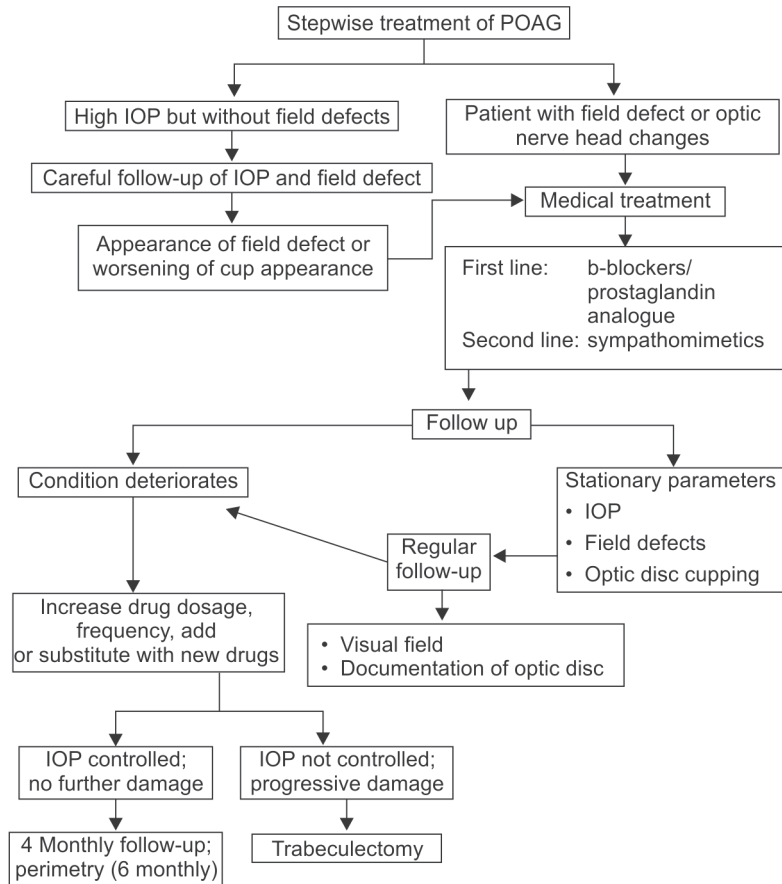
D/D OF PTERYGIUM AND PSEUDOPTERYGIUM

	<i>Pterygium</i>	<i>Pseudopterygium</i>
1. Aetiology:	Degenerative process	Inflammatory process
2. Age:	Usually occurs in elderly	Can occur at any age
3. Site:	Always situated in the palpebral aperture	Can occur at any site
4. Stages:	Progressive/ regressive / stationary	Always stationary
5. Probe test:	Probe cannot be passed underneath	Probe can be easily passed under its neck

WHO classification of vitamin A deficiency

<i>Classification</i>	<i>Primary signs</i>
X 1A	Conjunctival xerosis
X 1B	Bitot's spots with conjunctival xerosis
X 2	Corneal xerosis
X 3A	Corneal ulceration with xerosis (<1/3)
X 3B	Keratomalacia (>1/3 cornea)
XN	Night blindness
XF	Xerophthalmic fundus
XS	Corneal scars



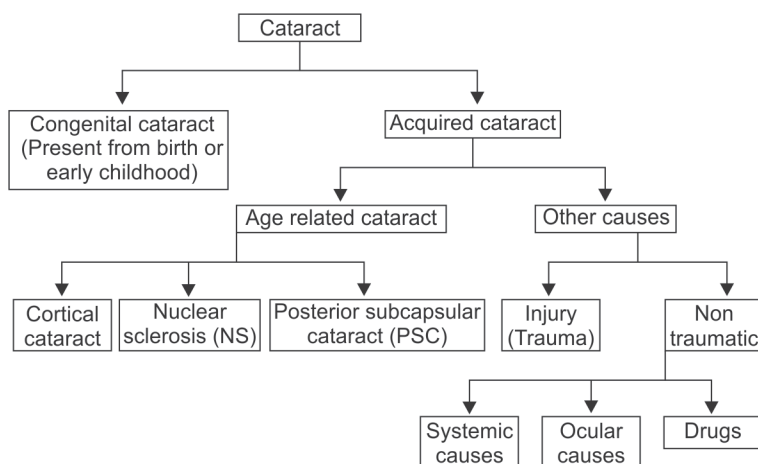


Aetiological classification of cataract

1. Congenital
2. Acquired
 - Senile
 - Complicated
 - Heat/ thermal
 - Toxic
 - Traumatic
 - Secondary
 - Radiation



Types of Cataract



Types of senile cataract

Morphological

- Posterior subcapsular cataract
- Nuclear cataract
- Cortical cataract or cuneiform

Stages of maturity

- Immature
- Intumescent
- Mature
- Hypermature

Differentiating Various Stages of Cataract

Features	Immature	Mature	Hypermature
Vision	6/9 - FC	HM - PL	HM - FC
Anterior chamber	Normal (shallow in intumescent)	Normal	Normal to deep



Contd...

<i>Features</i>	<i>Immature</i>	<i>Mature</i>	<i>Hypermature</i>
Color of lens	Grayish white	Pearly white	Milky white (with brown crescent of nucleus) or chalky white
Iris shadow	Seen	Not seen	Not seen
Distant direct ophthalmoscopy	Black patches against red glow	No red glow seen	No red glow seen
Purkinje-Samson images	4th image seen	4th image not seen	4th image not seen

Macular function tests

- Two point discrimination
- Maddox rod test
- Blue-field entoptoscopy
- Colour vision test
- Laser interferometry (LI)
- Potential acuity meter (PAM)
- Foveal ERG

Salient Features of Preoperative Work-up (cataract)

- BP
- PPBS
- Tonometry
- Syringing
- IOL Power calculation (Biometry)
 - Axial length measurement
 - Keratometry
- USG B-scan for posterior segment evaluation in cases where retina cannot be examined with ophthalmoscope (MSC/HMSC).



Cataract surgery options

- ECCE
 - Conventional
 - SICS
 - Manual SICS
 - Phacoemulification
 - Phacovit
- ICCE

ECCE vs ICCE

	<i>ECCE</i>	<i>ICCE</i>
Lens removal	Nucleus removed out of the capsule and cortex sucked out	Lens removed as single piece within its capsule
Posterior capsule and zonules	Intact	Removed
Incision	Smaller (8 mm)	Larger (10 mm)
Peripheral iridectomy	Not performed	Required to avoid pupillary block glaucoma
Sophisticated equipment	Required	Not required
Time taken	More	Less
IOL Implantation	Posterior chamber	Anterior chamber (with its associated risk of damage to corneal endothelium leading to Pseudo-phakic Bullous Keratopathy)
Expertise required	Difficult technique	Easier to learn
Cost	More	Less
Complications	After-Cataract	1. Vitreous prolapse and loss 2. Cystoid Macular Edema 3. Endophthalmitis 4. Aphakic Glaucoma 5. Fibrous and Endothelial ingrowth 6. Neovascular Glaucoma in PDR
Complications which are decreased	All the complications mentioned for ICCE	After-Cataract
Indications	A routine procedure for all forms of cataract (except where contra-indicated)	1. Dislocated Lens

Contd...



Contd...

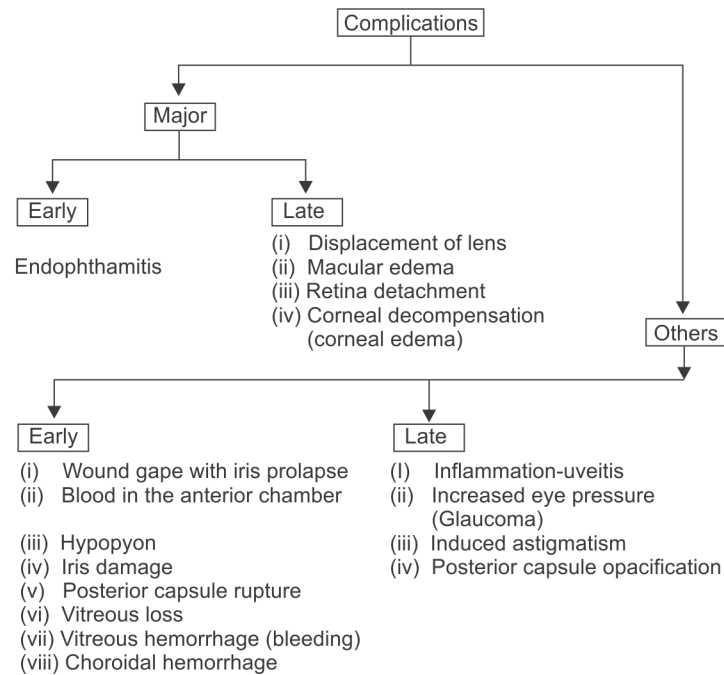
	ECCE	ICCE
		2. Subluxated Lens (>1/3rd zonules broken) 3. Chronic Lens Induced Uveitis 4. Hypermature Shrunken Cataract with thick anterior capsule 5. Intra-lenticular Foreign Body when integrity of posterior capsule is compromised.
Contraindications	1. Dislocated lens 2. Subluxated lens (>1/3rd zonules broken)	Young Patients (<35 years) who have strong attachment between lens and vitreous (Ligament of Weigert)
Intra capsular cataract extraction (ICCE)	i. Simple procedure ii. Not dependent on operating microscope iii. Sight restored with spectacles iv. Done in subluxated lens	i. Good BCVA but poor in quality ii. Patients blind without spectacles iii. Heavy spectacles, inconvenient with distortion of vision.
ICCE with Anterior chamber Intra - ocular Lens	i. Simple procedure	i. Requires operating microscope ii. More skill than ICCE iii. Higher incidence of late complications
Manual ECCE with Posterior chamber intra-ocular lens	i. Safer than ICCE ii. Better visual quality iii. Requires a relatively shorter learning curve v. Simple Instruments and equipment.	i. Suture related complications (Irritation, discomfort, Infection) ii. Delayed wound healing (1 to 2 months) and visual rehabilitation iii. Repeated follow-up visits
Manual sutureless Cataract Surgery (Late 1980's)	i. No suture - related complications ii. Minimal discomfort to the patient iii. Quick and good rehabilitation iv. Less follow up v. Simple instrument and equipment vi. Low cost	i. Longer learning curve ii. Difficult in complicated cases

Contd...



Contd...

	vii. Quick Procedure	
	viii. More affordable than phacoemulsification with similar results	
Suture less Phacoemulsification (scleral /corneal incision with foldable lens(1990's)	i. Can be done with topical anaesthesia	i. Expensive
	ii. Better quality of vision	ii. More difficult in MSC/ HMSC/Hard cataract
	iii. Minimal discomfort	iii. Complications more difficult to manage
	iv. Fast rehabilitation	
	v. Less tissue handling	



Types of anterior capsulotomy

1. Can opener
2. Envelope or endocapsular
3. Continuous curvilinear capsulorhexis(CCC)



D/D of acute ACG, conjunctivitis and acute iridocyclitis

Features	Acute ACG	Conjunctivitis	Acute iridocyclitis
History	H/O coloured haloes	Rarely H/O coloured haloes	No H/O coloured haloes
Pain	Marked pain, nausea, vomiting prostraton	Only discomfort	Marked pain along the distribution of trigeminal nerve
Redness	circumciliary congestion	Conjunctival congestion	Circumciliary congestion
AC	Shallow	Normal depth	Normal depth
IOP	Increased	Normal	Normal/high/low
Pupil size and shape	Mid-dilated, vertically oval	Normal	Normal/smaller
KPs, cells flare	Few	Nil	Present
Vision	Decreased	Normal	Decreased
Discharge	Lacrimation	Mucopurulent	Lacrimation
Hypopyon	None	None	May be present

Clinical features of bacterial corneal ulcer

Symptoms	Signs
Pain	Corneal ulcer with yellowish white opaque areas
Photophobia	Necrotic stromal infiltration
Decreased visual acuity	Mucopurulent exudates on the ulcer base
Redness	Anterior chamber reaction
Discharge	Hypopyon

Management of bacterial corneal ulcer

Lab diagnosis

- Gram's staining of corneal scrapings.
- Bacterial culture for both aerobic and anaerobic organisms (in blood, chocolate, Sabouraud's agar and thioglycolate broth).



Treatment

- Fortified cefazolin 5% and Tobramycin 1.3% or ciprofloxacin 0.3% (6-8 times a day).
- Cycloplegics.
- Antiglaucoma medications.
- Supportive treatment(analgesics and multivitamins).

Note:

1. Ulcer size, infiltration, hypopyon, IOP and symptoms.
2. Don't bandage the eye unless the ulcer is perforated.
3. Don't prescribe steroids for suspected corneal ulcer.

Management of fungal corneal ulcer

<i>Clinical features</i>	<i>Lab diagnosis by</i>
Signs more prominent than the symptoms Feathery infiltration Elevated ulcer margins	KOH wet mount Culture: Sabouraud's agar
	Treatment Corneal scrapping and debridement Natamycin (drug of choice), Fluconazole topically Cycloplegics and antiglaucoma drugs
Dry ulcer base	
Satellite lesions Fixed hypopyon Endothelial plaque	

Difference between papilloedema and optic neuritis

<i>Papilloedema</i>	<i>Optic neuritis</i>
Bilateral Marked disc swelling (2-6D) Visual functions intact except in late stages (RAPD) occurs in late stages No pain in ocular movements No Inflammatory cells	Often unilateral Mild disc swelling (<3 D) Visual functions grossly abnormal RAPD is present at the onset Ocular movements painful Vitreous shows inflammatory cells

**Causes of Night Blindness**

- Vitamin A deficiency
- Retinitis pigmentosa
- Advanced glaucoma

Causes of Leucocoria or white pupillary reflex

- Retinoblastoma
- Toxocariasis
- Coat's disease
- Congenital cataract
- PHPV

Blood supply of optic nerve

- Central retinal vessels and its branches
- Scleral vessels (the circle of Zinn-Haller)
- Choroidal vessels
- Pial vessels

Six primary positions of gaze

- Right and out: dextroversion
- Right up and out: dextrosupraversion
- Right down and out: dextroinfraversion
- Left and out: laevoversion
- Left up and out: laevosupraversion
- Left down and out: laevo-infraversion

Treatment modalities of myopia

1. Spectacles
2. Contact lenses
3. Refractive surgery
 - Radial keratotomy (RK)
 - Photorefractive keratectomy (PRK)
 - Laser in situ keratomileusis (LASIK)
 - Intrastromal corneal rings
4. Clear lens extraction
5. Phakic IOL



STYE (Hordeolum Externum)

- Suppurative infection of the glands of Zeiss.
- Caused by staphylococcal or streptococcal infection.
- In cases of recurrent styes, rule out diabetes and refractive errors.
- Epilate the infected lash.
- Express pus if pus point is present.
- Hot compresses and topical antibiotics are recommended.
- Look for preseptal cellulitis.

Steps of Chalazion Surgery

- Anaesthesia: Topical 4% xylocaine drops plus infiltration with 2% xylocaine.
- Clean with povidone iodine and drape the eye.
- Clamp the swelling with chalazion clamp and evert the lid.
- Give a vertical incision (parallel to vessels) over the swelling (conjunctival side).
- Curettage with chalazion curette.
- Use a pad bandage with antibiotic ointment for the day.

Types of Entropion and Ectropion

1. Entropion
 - Involutional or senile
 - Cicatricial
 - Spastic
 - Congenital
2. Ectropion
 - Involutional or senile
 - Cicatricial
 - Spastic
 - Paralytic

WHO Classification of Trachoma

- *TF* : Trachomatous inflammation follicular
Presence of more than five follicles (> 0.5 mm diameter) on upper tarsal conjunctiva.



- *TI: Trachomatous inflammation intense*
Presence of inflammation and papillary hypertrophy obscuring more than half of tarsal vessels.
- *TT: Trachomatous trichiasis*
At least one trichiatic cilia rubbing on the globe or evidence of its recent removal.
- *TS: Trachomatous scarring*
Characterized by obvious trachomatous scarring of upper tarsal conjunctiva.
- *CO: Corneal opacity*
Characterized by trachomatous corneal opacity at least a part of which extends over the pupil.
[Mnemonic – FISTO]

Aetiology and risk factors of keratitis

<i>Aetiology of keratitis</i>	<i>Risk factors for keratitis</i>
Bacterial	Trauma
Viral	Contact lens wear
Fungal	Diminished local resistance, e.g. dry eye, loss of sensations, bullous keratopathy, etc.
Parasitic	
Nutritional	
Degenerative	Altered local microbial flora, e.g. dacryocystitis, blepharitis, etc.
Immunologic	Lid abnormalities like trichiasis.

Lasers In Ophthalmology

1. Argon laser
2. Krypton laser
3. Diode laser
4. Nd-YAG laser
5. Excimer laser



Uses of Argon/Diode Laser

In glaucoma

1. Laser trabeculoplasty for primary open angle glaucoma
2. Laser iridotomy for narrow- angle glaucoma
3. Cyclophotocoagulation for absolute glaucoma

In lesions of retina

1. Diabetic retinopathy
2. Eales' disease
3. Coats' disease
4. Sick cell retinopathy
5. Exudative age-related macular degeneration

Plate - 1



Chalazion



Molluscum contagiosum



Pseudopterygium with
symblepharon



Post burn cicatricial ectropion of
upper lid



Entropion

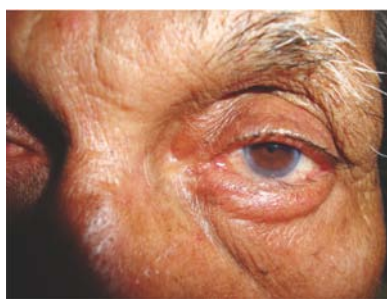


Preseptal cellulitis

Plate - 2



Squamous blepharitis



Trichiasis

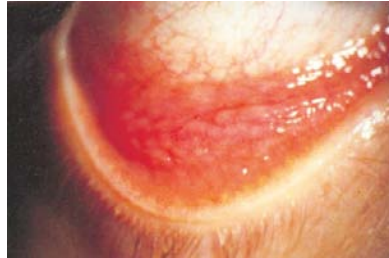


Congenital ptosis

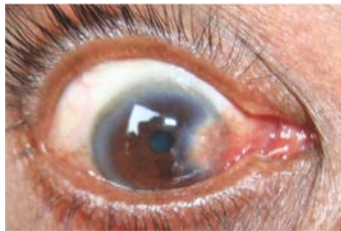
Plate - 3



Acute mucopurulent
conjunctivitis



Follicular conjunctivitis



Atrophic pterygium



Ophthalmia neonatorum



Bitot's spot



Palpebral form of vernal
conjunctivitis

Plate - 4

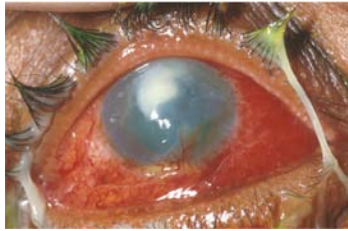


Phlyctenular conjunctivitis

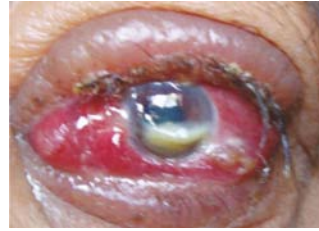


Subconjunctival haemorrhage

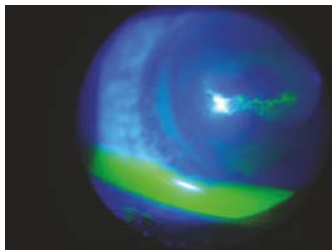
Plate - 5



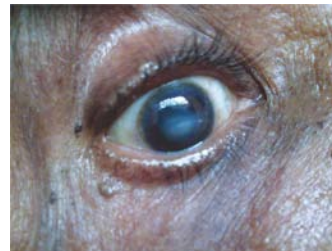
Keratitis with vascularisation of cornea



Exposure keratitis



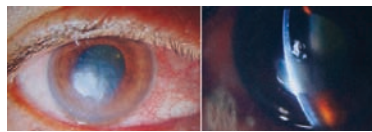
Dendritic keratitis



Corneal leucoma

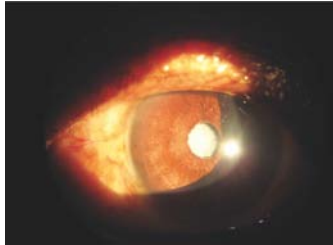


Anterior staphyloma

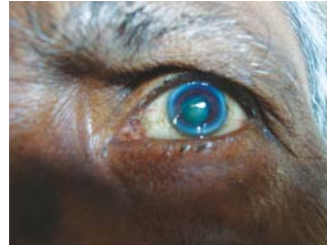


Keratitis with slitlamp view

Plate - 6



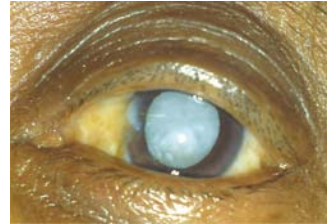
Advance IMSC with
Pseudoexfoliation syndrome



Immature senile cataract



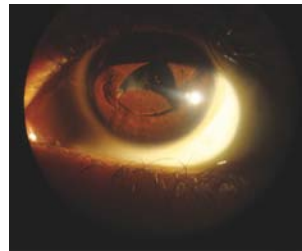
Bilateral subluxated cataractous
lens with aniridia



Mature senile cataract



Bilateral congenital cataract

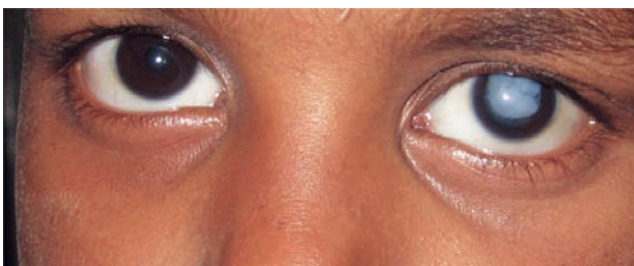


Optic capture, PCO (in a
paediatric pseudophakia)

Plate - 7



Bilateral developmental cataract

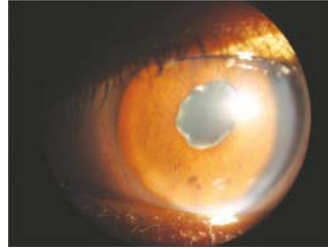


Traumatic cataract

Plate - 8



Ciliary staphyloma



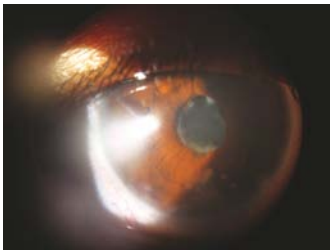
Healed uveitis with posterior
synechiae



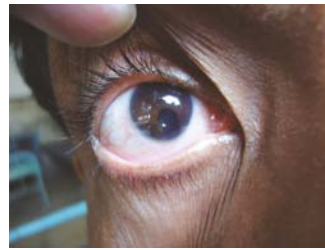
Endophthalmitis with hypopyon



Phthisis bulbi



Granulomatous uveitis with
mutton fat KPs



Typical iris coloboma

Plate - 9



Congenital glaucoma



Normal fundus



Glaucomatous cupping



RE-megalocornea, LE-buphthalmos



Glaucomatous cupping with splinter haemorrhage

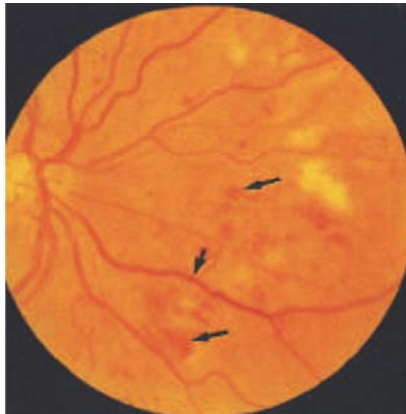
Plate - 10



Bilateral retinoblastoma



CRVO



Nonproliferative diabetic retinopathy

Plate - 11



Anophthalmos



Artificial eye (R)



Socket after evisceration



Proptosis



Dysthyroid ophthalmopathy

Plate - 12



3rd nerve palsy



Left convergent squint

Plate - 13



Retinoscopy mirror



Trial box



Trial frame

Plate - 14



Acute dacryocystitis



Lacrimal sac abscess

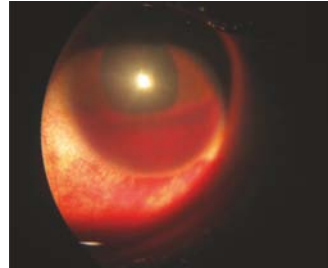


Mucocoele of the lacrimal sac

Plate - 15



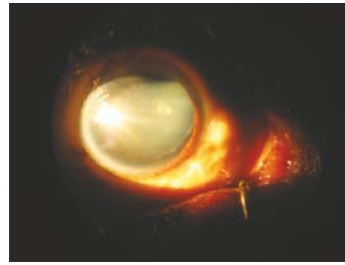
Black eye



Traumatic hyphaema



Fish hook penetrated the right eye near limbus



Traumatic subluxated cataractous lens



Lacerated lid injury



Traumatic cataract

GLOSSARY

ABK	Aphakic bullous keratopathy
AC	Anterior chamber
ACG	Angle closure glaucoma
ACIOL	Anterior chamber intraocular lens
AION	Anterior ischemic optic retinopathy neuropathy
ALT	Argon laser trabeculoplasty
ANA	Anti nuclear antibody
ANF	Anti nuclear factor
ARMD	Age related macular degeneration
BCVA	Best corrected visual acuity
BM	Bowman's membrane
BRVO	Branch retinal vein occlusion
BSK	Band shaped keratopathy
BSS	Balanced Salt Solution
C:D ratio	Cup-disc ratio
CCC	Continuous curvilinear capsulorhexis
CI	Complete iridectomy
CL	Contact Lens
CME	Cystoid macular edema
CRA	Central retinal artery
CRAO	Central retinal artery obstruction
CRVO	Central retinal vein occlusion
CTR	Capsular tension ring
DCR	Dacryocystorhinostomy
DCT	Dacryo-cystectomy
DD	Dextro depression
DE	Dextro-elevation
DM	Descemet's membrane
DV	Dextro-version
ECCE	Extracapsular cataract extraction
EKC	Epidemic keratoconjunctivitis
ERG	Electro retinography



ETO	Ethylene trioxide
FB	Foreign body
FC	Finger counting
FFA	Fundus fluorescein angiography
FL	Fluorescein
HM	Hand movement
HMSC	Hypermaturation senile cataract
HSV	Herpes simplex virus
ICCE	Intracapsular cataract extraction
IMSC	Immature senile cataract
IO	Inferior oblique
IOP	Intra ocular pressure
IR	Inferior rectus
IV	Intravitreal
KP	Keratic precipitates
LASIK	Laser assisted in situ keratomileusis
LD	Levo depression
LGB	Lateral geniculate body
LI	Laser interferometry
LK	Lamellar keratoplasty
LPS	Levator palpebrae superioris
LR	Lateral rectus
LV	Levo-version
MMC	Mitomycin -C
MR	Medial rectus
MSC	Mature senile cataract
NLD	Naso lacrimal duct
NPDR	Non-proliferative diabetic retinopathy
NTG	Normal tension glaucoma
NVI	Neovascularisation of iris
PACG	Primary angle closure glaucoma
PAM	Potential acuity meter
PAS	Peripheral anterior synechiae
PBHI	Peripheral button hole iridectomy
PBK	Pseudophakic bullous keratopathy
PC	Posterior chamber
PCIOL	Posterior chamber intraocular lens



PCO	Posterior capsular opacification
PDR	Proliferative diabetic retinopathy
PHPV	Persistent hyperplastic primary vitreous
PI	Peripheral iridectomy
PL	Perception of light
PMMA	Polymethyl methacrylate
POAG	Primary open angle glaucoma
PR	Projection of rays
PRK	Penetrating keratoplasty
PSC	Posterior subcapsular cataract
RAPD	Relative afferent pupillary defect
RB	Retrobulbar
RD	Retinal detachment
RIOFB	Retained intra ocular foreign body
ROP	Retinopathy of prematurity
RPE	Retinal pigment epithelium
RL	Ringer Lactate
SC	Schlemm's canal
SICS	Small incision cataract surgery
SJS	Stevens-Johnson syndrome
SLE	Systemic lupus erythematosus
SLO	Scanning laser ophthalmoscopy
SO	Superior oblique
SPK	Superficial punctate keratitis
SR	Superior rectus
TB	Tuberculosis
TBM	Trabecular meshwork
USG-B	Ultrasonography –B Scan
VA	Visual acuity
VEGF	Vascular endothelial growth factor
VKH	Vogt- Koyanagi -Harada

INDEX

A

Absolute glaucoma 133
ACG 128
Acquired dacryocystitis 180
Acute congestive glaucoma 245
Acute dacryocystitis 180, 184, 186, 187
Acute iridocyclitis 245, 246, 255, 108, 109, 111
Afferent pupillary defect 156
Age related cataract 246
Age related macular degeneration 145, 246, 260
ALT 127
Altitudinal hemianopia 159
Amaurosis fugax 159, 247
Ametropia 116, 167, 175
Anatomy of the lid 14
Angle Kappa 165
Angle recession 191, 193, 195
Angular conjunctivitis 35, 36
Aniridia 116
Anisocoria 155
Anisokonia 80, 81, 172
Anisometropia 81, 159, 163, 171, 172, 173
Ankyloblepharon 24
Annular tendon of Zinn 161
Ante-flexion of the iris 191
Anterior chamber 121, 128, 129, 130
Antiglaucoma drugs 135, 256
Aphakia 77, 79, 80



Applanation/Schiotz tonometry 124
Aqueous flare 109
Aqueous humor 102
ARMD 247
Arruga's Intracapsular forceps 217, 218
Artery forceps 212
Asthenopia 63, 171, 173
Astigmatism 170, 171
Automated perimetry 6, 124

B

Band keratopathy 112
Bard parker handle 213
Barraquer wire speculum 210
Barraquer's needle holder 211
Bifocal lens 174
Binocular 162, 163
Bitot's spot 248, 40, 41
Blepharitis 24, 25
Blepharophimosis 24
Blindness 197-200
Blood aqueous barrier 120
Blood staining of cornea 191, 195
Blow out fracture 150, 151, 191
Bone punch 233
BRVO 159, 247, 144, 148

C

Calliper (castroveijo's) 237
Canal of Schlemm 130, 137, 138
Capsulorrhexis forceps 221
Capsulotomy 88
Cardinal gazes 7, 8



Cat's paw retractor 232
CCC 92
Central serous Retinopathy 115
Chalazion clamp 229, 258
Chalazion scoop 229
Chalazion 17, 18, 19, 213, 229, 239, 258
Chalcosis bulbi 194
Chemical injury 29, 31, 41, 189, 190
Chemosis 28, 35, 86, 87, 144, 190
Choroid 93, 95, 100, 102, 115, 116, 117, 156, 192
Chronic dacryocystitis 181, 182, 183
Chronic dacryocystitis 180, 181, 182, 183, 231, 60, 65
Ciliary body 119, 120, 121, 129, 130, 133, 135, 172, 191, 193, 196, 56, 61, 83
Ciliary ganglion 133, 152, 153, 154
Ciliary processes 101, 102, 102
Colibri forceps 219
Coloboma 10, 116
Color vision 141, 142, 143
Colour vision test 6, 251
Complicated cataract 94
Concave cylindrical lens 240
Concave spherical lens 239
Concretion 9, 28
Cone cells 141, 142
Congenital dacryocystitis 180, 231, 235
Conjunctival xerosis 40, 41, 49, 248
Conjunctivitis 126
Convex cylindrical lens 241
Convex spherical lens 239
Corneal abrasion 54
Corneal opacity 61, 62, 63
Corneal rupture 191
Corneal spring scissors 215, 216



Corneal transplant 198
Corneal ulcer 54-60, 255-256
Cortical blindness 159
Corticosteroids 83, 135, 203, 204
Cotton wool spots 143, 144
CRAO 5, 144, 147, 159, 159, 247
Crede's method 39
Crescent knife 88, 90, 227
CRVO 5, 144, 147, 159, 247
Cyclocryotherapy 133
Cyclodialysis 191
Cycloplegia 202

D

Dacryocystectomy 183, 185
Dacryocystitis 55, 65, 180, 181, 182, 183, 184, 186, 187, 231, 235, 259
Dacryocystorhinostomy 180, 183
Dalen-Fuchs nodules 196
Dastoor superior rectus forceps 223
Desmarre's upper eye lid retractor 228
De-Wecker's scissors 217
Diabetic retinopathy 117, 246, 247, 260
Digital tonometry 11
Diplopia 162, 163
Direct ophthalmoscopy 12, 75, 115, 149
Direct Simcoe 226
Disposable cystitome 226
Duction 6

E

Eales's disease 148
Ectropion 22, 25, 41, 258
Elschnig's Intracapsular forceps 220



Emmetropia 165
Endophthalmitis 58, 93, 115, 201, 212, 247
Enophthalmos 15, 150
Entropion clamp 236
Entropion 21, 22, 23, 37, 236, 258
Enucleation scissors 234
Enucleation 105, 106, 107, 147, 196, 199, 211, 212, 234, 235
Epidemic Kerato conjunctivitis 36
Epilation Forceps 218
Epiphora 180, 183
Episcleritis 46, 47, 204
Evisceration scoop 234
Evisceration 107, 108, 115, 211, 234
Exophthalmos 150
External hordeolum 70
Eye bank 105, 199, 200

F

Far point 172, 173
Festooned pupil 109, 111
Field of vision 6, 122
Finger counting 3, 68
Fixation forceps 222
Follicle 9, 16, 24, 28, 37
Fovea centralis 142

G

Gland of Krause and Wolfring 16
Gland of Moll 15, 16
Gland of Zeiss 16
Glands of eye lids 15
Glaucoma 120-128 , 132-137
Goblet cells 27, 179
Gonioscopy 124, 129



Granulomatous uveitis 109, 111, 113, 195

Grey line 14, 16, 26

H

Haller's layer 102

Hand movement 4

HCRP 200

Heterophoria 162, 163

Heterotropia 162

High index lens 174

HMSC 74

Hypermetropia 80, 127, 128, 167, 168, 170, 173, 239

Hypertensive uveitis 112, 133

Hypopyon 255, 256, 109, 114

I

IMSC 162

Incomitant squint 163

Indirect ophthalmoscopy 12

Iridocyclitis 108-114, 255

Iris bombe 110, 111, 112

Iris forceps 220

Iris repositor 226

Irrigating vectis 224

Irrigation aspiration cannula 225

Ishihara plates 5, 6

J

Jaeger's chart 4

K

Keratic precipitates 109

Keratome knife 227

Koeppé's nodule 111

**L**

Lacrimal apparatus 3, 11, 177
Lacrimal probe 235
Lacrimal canula 230
Lacrimal dissector with scoop (Lang's) 232
Lacrimal fossa 177, 178, 161
Lacrimal gland 16, 27, 152, 178, 179
Lacrimal passage 177, 178, 183, 230
Lacrimation 27, 28, 129, 180, 255
Landolt C chart 5
LASIK 169, 170, 173
Latent squint 162
Lens 66-68
Lens expressor 91, 224, 238
Lens holding forceps 223

M

Macula lutea 141
Maddox rod test 163
Maddox rod 241
Masquerade syndrome 113
Mc Pherson's Forceps 221
M-K Media 199
Mucocoele 183
Muller's self retaining haemostatic retractor 237
Muscle hook 238
Mydriatic 111, 127, 128, 155, 193, 202
Myopia 167-169 , 239,257

N

Near point 172, 173
Near vision 4
Neovascular glaucoma 134
Nettleship punctum dialator 230



Nodal point 75, 94, 166
Non-granulomatous uveitis 113
NPCB 197, 198, 200
NSAID 20, 83, 204

O

Occlusio pupillae 110, 112
Optic neuritis 142, 156, 157, 247, 256
Optical axis 165
Ora serrata 102, 140, 141
Orthophoria 163

P

Panophthalmitis 28, 58, 105, 107, 114, 234
Papilloedema 143, 159, 204, 247, 256
Paralytic squint 163
Pars plana 101, 115, 116
Pars planitis 113, 115, 116
Pars plicata 101
Perception of light 4, 5
Peritomy 106, 107, 216
Phacoemulsification 91
Phlyctenular conjunctivitis 44-46
PHPV 257
Phthisis bulbi 23, 52, 53, 105, 112, 194
Pin hole 2, 3
Plain smooth forceps 220
Plateau iris 132
Posterior synechiae 109, 110, 111, 112
Presbyopia 170, 172, 173, 239, 246
Projection of rays 4
Pseudoesotropia 165
Pseudoexotropia 165
Pseudomyopia 169



Pseudophakic glaucoma 134
Pterygium 29-33

R

Reduced eye 165
Retinoblastoma 257
Retinoscopy 174, 175
Reverse simcoe 226
Rod cells 42, 102, 141, 241
Rougine 232
Rubeosis iridis 134

S

Sattler's layer 102
Scleral fixation forceps 222
Scleritis 48
Scotoma 123, 124
Seclusio pupillae 110, 112
SICS 88
Sinsky's Hook 227
Snellen's chart 3, 5
Speculum with screw 210
St. Martin's forceps 219
Staphyloma 9, 42, 48, 57, 103, 104, 105
Stenopic slit 243
Sterilisation 243
Steroid induced glaucoma 135
Straight scissors 215
Superficial ocular FB 188
Surgical blade 90, 185, 214
Suture Tier 221, 223, 224
Syringing 60, 75, 181, 182, 183, 185, 230, 231



T

Tear film 179, 9, 16, 48, 49, 51, 138
Tenotomy curved scissors 215
Thermo cautery 212, 213
Trabecular Meshwork 119
Trabeculectomy 126, 137
Trachoma 37

U

Universal eye Speculum 106, 107, 209, 210

V

Vannas' Scissors 217
Vernal conjunctivitis 42-44
Version 7
Vision 2020 198
Visual Acuity 3, 2, 5
Visual angle 165
Visual axis 32, 163, 165
Vitreotomy 115, 116
Vitreous haemorrhage 121
Von graefe's cataract knife 214
Vortex veins 103, 107

W

Wet ARMD 247
Wire Vectis 88, 91, 225

X

Xanthalesma 24
Xerophthalmia 40, 41

Y

Yoke muscles 7