

THE VISUAL FIELD

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Definition - a three-dimensional contour representation of differential light sensitivity at each point in the field of vision

- Traquair – “an island of vision in a sea of darkness”
- "Island of vision" - a topographical representation of differential light sensitivity; the peak corresponds to fixation (fovea) and an absolute depression corresponds to the physiologic blind spot (optic nerve) due to an absence of photoreceptors
- Cross-sections through island of vision:
 - Horizontal - maps out isopters, or identical points of retinal sensitivity
 - Vertical - profile plot of differential light sensitivity along a meridian
- Boundaries of the normal visual field:
 - Superior - 60 degrees
 - Inferior - 70-75 degrees
 - Nasal - 60 degrees
 - Temporal – 100 - 110 degrees

Therefore, with both eyes, horizontal visual field encompasses approximately 180 degrees (nasal and temporal fields overlap)
- Goals of perimetry
 - Detection of field defects (screening programs)
 - Quantitation of the size, shape, and depth of all defects

Techniques of Perimetry

- Kinetic - a moving stimulus of fixed intensity and size is moved at a constant rate from non-seeing to seeing regions, identifying points of initial perception. These points map out an isopter, a defined locus of identical retinal sensitivity. An isopter denotes the boundaries of a horizontal cross-section of the hill of vision
- Static - a stationary stimulus at a fixed location is gradually increased in intensity or size until the stimulus is initially perceived. This initial perception determines the retinal threshold

sensitivity at that point and the presence and depth of a scotoma may thereby be determined. Static testing at various points along a meridian defines a profile, or vertical cross-section of the hill of vision

- Automated Perimetry

- Determination of the field of vision via a system whose operation is conducted by a machine programmed to perform explicit mathematical calculations (algorithms)

- Determination primarily by static methods and computer-assisted

- Parameters of background illumination, stimulus size and intensity standardized

- Stimulus size and intensity may be varied

- High reproducibility

- Suprathreshold screening and full threshold programs available

- Minimal technician training and technician-patient interaction

- Objective reliability parameters include fixation losses, false positive responses, false negative responses, and fluctuation

- Standardized and customized programs

- Computerized data storage and analysis

- Advantages of Manual Kinetic versus Automated Static Perimetry:

- Relatively inexpensive

- Relative ease of examination for patient and perimetrist

- Shorter test duration minimizing patient fatigue

- Minimal mechanical servicing ("down - time")

- Perimetrist - patient interaction may facilitate exam

- Advantages of Automated Static versus Manual Kinetic Perimetry:

- Quantification of field defects

- Increased sensitivity and specificity

- Excellent reproducibility

- Standardization of test conditions and programs

- Results less subject to perimetrist experience and technique

- Subjective factors of perimetrist - patient interaction minimized

- Less dependent on patient reaction time

- Comparison with age-matched controls

- Customized programs

- Objective patient reliability indices

Computerized storage of data
 Statistical analysis and comparison of data

Characteristic Glaucomatous Visual Field Defects

Clinically recognizable optic nerve changes precede detectable VF loss in most patients. Normal VFs have greatest sensitivity centrally under photopic conditions; sensitivity declines steadily toward the periphery.

VF defects generally correlate with optic nerve morphology and NFL defects.

1. Ex: Inferior optic nerve cupping commonly associated with superior scotoma

With Goldmann perimetry (Quigley et al, 1982):

- Up to 35% of fibers may be lost prior to detectable VF loss
- > 50% of fibers may be lost by time reproducible early field defects are found
- 2. < 10% of axons may remain by the stage of severe VF loss

With automated perimetry (Quigley et al, 1989):

- A 20% loss of ganglion cells (especially large ganglion cells) in the central 30 degrees of retina correlated with a 5-dB sensitivity loss
- A 40% loss of ganglion cells in the central 30 degrees of retina correlated with a 10-dB sensitivity loss
- Some ganglion cells remained in regions with 0-dB sensitivity

Prior to the development of definitive scotomas, early defects may manifest as:

- Manual perimetry - increased scatter (variable responses)
- Automated perimetry - increased scatter (short- and long- term fluctuation) in affected areas
 and diminished sensitivities in the paracentral region
- Increased scatter may represent warning sign of impending absolute VF defect

VF defect should be at least 3 degrees wide and 6 decibels deep (0.6 log units) to be classified as a scotoma.

In automated perimetry, a cluster of two or more points depressed > 5dB compared to surrounding points or a single point depressed > 10 dB is suspicious. Former more reliable as cluster of points may confirm one another.

Corresponding points above and below horizontal midline should not vary markedly although superior VF may be depressed 1-2 dB compared to inferior VF.

- Typical defects consist of:

Localized defects including arcuate and paracentral scotomas, nasal steps, and temporal wedge defects

Generalized or diffuse reductions in sensitivity

- Location of Characteristic Glaucomatous Defects:

(1) Localized Defects

- Paracentral Scotoma
 - Defect within central 10 degrees though not involving central fixation
 - May represent an initial glaucomatous defect
 - Arcuate (Bjerrum) Scotoma
 - Defect involving arcuate nerve fibers which originate from retina temporal to disc and enter the superior and inferior poles of optic nerve
 - VF loss typically occurs in the area 10 -20 degrees from fixation
 - In complete form, the scotoma extends from the blind spot to the nasal raphe, becoming wider and closer to fixation along nasal extent
 - Examples include:
 - Seidel scotoma - defect in proximal portion of the NFB
Resultant comma-shaped arcuate scotoma connecting with the blind spot
 - Nasal step- defect in distal portion of arcuate NFB involving the nasal VF
Defect respects the horizontal meridian as superior and inferior arcuate bundles do not cross the horizontal raphe of the temporal retina
- Goldmann perimetry - defect should be 10 degrees wide in a single isopter or 5 degrees wide in two or more isopters
- Isolated scotoma - defect of intermediate portion of the arcuate NFB
Not contiguous with the blind spot or nasal step area
 - Temporal Step, Sector or Wedge-Shaped Defect
 - Secondary to involvement of the retinal nerve fibers nasal to the optic nerve head which enter the nasal portion of disc in straight (nonarcuate) pattern
 - Extends from the temporal periphery to the central visual field (usually blind spot) and may not respect the temporal horizontal raphe
 - Rarely initial glaucomatous defect

- Peripheral Defect

- Controversy as to the prevalence of initial field defects that may be missed if only the central VF is examined
- Reported 3 -11% of patients may manifest peripheral defects (usually nasal steps) as the only abnormality detected on automated perimetry (Shields, 1992)
- Peripheral nasal step examination may be performed in patients with full central fields if a high index of suspicion exists

(2) Generalized Defects

3. Baring of Blind Spot (Blind Spot Enlargement)

- Verify by plotting more isopters on Goldmann perimetry
- 4. Rule out peripapillary atrophy and papilledema

- Generalized Constriction of Isopters (Goldmann)

- Generalized Sensitivity Reduction and Peripheral Concentric Constriction (Automated)

May represent early glaucomatous changes though these defects are not specific and may also be caused by pupil miosis, uncorrected refractive errors, media opacification, retinal disease, aging, and malpositioning of the perimetric corrective lens.

May be of greater significance if defect occurs asymmetrically, represents progression from a previous baseline exam, or correlates with clinical findings.

(3) Advanced Glaucomatous VF Defects

- Central Island
- Split Fixation - defect cuts across fixation in either (or both) the horizontal or vertical meridia
 - Greater risk to loss of fixation than central island
 - Not a contraindication to glaucoma filtering surgery as fixation may be lost if glaucoma is uncontrolled on medical therapy
- Temporal Island - often most resistant and may persist after central VF is lost

(4) Progression of Glaucomatous Field Defects

- Manifests most commonly as increased density of pre-existing scotomas
- Scotomas may also increase in extent or new scotomas may develop

PRIMARY ANGLE-CLOSURE GLAUCOMA

I. Mechanisms

A. Anatomic features

1. Biometry- hyperopia common
2. Anterior chamber depth
3. Lens position and thickness
4. Anterior lens curvature

B. Physiology

1. Pupillary block
2. Role of the lens and zonules
3. Role of the iris

C. Methods of examination

1. Anterior chamber depth
2. Gonioscopy
3. Role of Provocative testing

5. Epidemiology

The disease is generally bilateral, although it may be asymmetric. Exceptions include anisometropia, traumatic lens subluxation (conditions in which the AC depth is disparate)

II. Clinical types

A. Intermittent and subacute angle-closure

B. Acute angle-closure

1. More common in Caucasians

C. Chronic angle-closure

1. Diagnosis often missed
2. More common in Blacks and Orientals

D. Plateau iris

E. Secondary angle-closure glaucomas

1. Mechanisms
2. Miotic-induced angle-closure
3. Combined mechanism glaucoma
4. Lens-related angle-closure
5. Malignant (ciliary block) glaucoma
6. Nanophthalmia

III. Therapeutic overview of angle-closure

A. Acute angle-closure

1. medical therapy

Decrease aqueous production -- Beta-blockers, carbonic anhydrase inhibitors, alpha-agonists

Hyperosmotic agents - dehydrate the vitreous

Topical steroids - decrease inflammation

Miotics – should be used sparingly, as excessive therapy

(i.e. Pilo 4% q 5 minutes) may cause contraction of the ciliary ring and anterior lens movement, with a worsening of the angle closure. One drop of Pilo 2% will cause miosis, if this is going to occur.

2. Laser treatment

Iridectomy is the treatment of choice.

Argon laser peripheral iridectomy may be of benefit in recalcitrant case or when the anterior chamber cannot be visualized.

3. Approach to the patient after iridectomy

- B. Chronic angle-closure
- C. Provocative testing

PRIMARY CONGENITAL GLAUCOMA

I Terminology

- A. Congenital - birth to 3 months (or birth to 3 months and infantile 3 months to 3 years)
- B. Late congenital - age 3 to 10
- C. Juvenile - age 10 to 35

II Epidemiology

Incidence - 1 in 10,000 births (1/3 to 1/2 primary, 1/3 secondary, 1/3 associated with systemic anomalies and syndromes)

60% are diagnosed by age 6 mos, 90% within first year.

65% are male.

70% are bilateral.

Inheritance - mostly recessive with incomplete penetrance. Occasional autosomal dominant pattern.

III. Proposed mechanisms - the disease represents a developmental anomaly of the angle

- A. membrane (Barkan)
- B. Faulty cleavage
- C. Sliding of ciliary body-iris root with respect to meshwork
- D. Angle of neural crest origin - same origin as facial bones, teeth, cartilage, meninges - association of congenital glaucoma and defects of these

IV. Diagnostic features

- A. Presenting symptoms - Epiphora, photophobia, blepharospasm
- B. Findings on examination-
 - 1. IOP affected by anesthesia - ketamine increases, inhalation

anesthetics lower.

2. Corneal diameter - >12 mm at birth suspicious
3. Corneal edema and Haab's striae (usually concentric)
4. Gonioscopy - anterior iris insertion, hypoplasia of iris periphery, lack of angle recess.
5. Fundoscopy - early central and deep cupping; may reverse with effective therapy
6. Retinoscopy- usually myopic due to increased axial length
7. Buphthalmos- most of the increased size results from stretching at the limbus.

V. Therapy

1. Medical therapy temporary
 - Acetazolamide dose: 15 mg/kg/day
 - Beta blockers can cause apnea
 - NLD occlusion by the parents will minimize side effects
2. Surgery
 - Trabeculotomy/Goniotomy
 - Long-term prognosis is best for patients who present at 1-24 mos.
 - Trabeculotomy with trabeculectomy
 - Seton or cycloablative procedures
6. Long-term management
 - Amblyopia, myopia are common

VI. Glaucomas of childhood associated with ocular anomalies

1. Secondary glaucomas- trauma, inflammatory, ROP, tumors- RB, JXG, medulloepithelioma
2. Corneal abnormalities- microcornea, sclerocornea
3. Mesodermal Dysgeneses of the Anterior Ocular Segment- Axenfeld's Anomaly, Rieger's Anomaly and Syndrome, Posterior Keratoconus, Peter's Anomalies
4. Aniridia
5. Lens-induced - dislocation and microspherophakia
6. PHPV
7. Nanophthalmos - >10 Diopter hyperope, small anterior segment, high lens/axial length

VII. Glaucoma associated with systemic anomalies:

Congenital rubella, Sturge-Weber, neurofibromatosis, Marfan's syndrome, Homocystinuria, Weill-Marchesani syndrome, Lowe's syndrome, chromosomal abnormalities, Mucopolysaccharidoses (Hurler and Hunter), Pierre-Robin, Oculodentodigital dysplasia.

VIII. Differential diagnosis of Infantile Glaucoma:

Epiphora - NLD obstruction

Corneal enlargement

Megalocornea

High Myopia

Exophthalmos

Corneal Clouding

Birth trauma

Inflammatory corneal disease

CHED

Sclerocornea

Metabolic disorders

Others

OPEN-ANGLE GLAUCOMAS

I. Glaucoma Suspect - any patient at increased risk for developing glaucoma

A. Definition – one of the following must be present

1. Ocular hypertension
2. Glaucomatous visual field loss
3. Optic nerve head damage typical for glaucoma

B. Ocular Hypertension (OHT)

1. IOP > 21 mmHg
2. Distribution curve is shifted to the right (Skewed to higher pressures)
3. 1% of patients with OHT will develop field loss per year

C. Risk factors for ONH damage

1. Increased IOP
2. Race
3. Increased cup/disc ratio
4. family history
5. myopia
6. DM
7. systemic vascular disease
8. other ocular disease (PXS, PG)

- D. Patient factors
 - 1. Expectations of patient
 - 2. Reliability
 - 3. Ability to assess nerve (i.e. cataract)
 - 4. Risk/benefit ratio

- E. Mandatory treatment
 - 1. VF defect
 - 2. Progressive cupping
 - 3. Corneal edema
 - 4. Previous vascular accident
 - 5. Appositional angle-closure (needs LI)

II. Primary Open-Angle Glaucoma (POAG)

- A. Definition
 - 1. IOP >21 mmHg
 - 7. Open, normal appearing angles
 - 8. Typical visual field loss and/or optic nerve head damage
 - 4. No apparent systemic or local disease to account for elevated IOP

 - B. Natural history
 - 9. Damage may take 20 years to develop
 - 10. Once damage has occurred, progressive loss occurs at a faster rate

 - C. Risk factors for development and progression of disease
 - 1. Family history – polygenic or multifactorial inheritance
 - 2. Myopia
 - 11. Diabetes or systemic vascular disease
 - 12. Black race - 8X as likely to develop POAG or progressive damage than whites
 - 13. Age
 - 14. Enlarged physiological/congenital cupping
- D. Adjunctive tests
 - 15. Tonography
 - 2. Other psychophysical tests
 - a. Contrast sensitivity
 - b. Color vision (blue-yellow loss)
- E. Histopathology/Mechanism of Glaucoma
 - 1. Resistance to outflow at level of juxtacanalicular meshwork
 - 2. Collagen of TM - fragmented
 - 3. TM endothelial cell dysfunction cell dysfunction, basement membrane thickening
 - 4. Increased glycosaminoglycans
 - Deratan sulfate
 - Heparan sulfate

chondroitin sulfate

keratan sulfate

Hyaluronic acid - not bound to ECM

-highest concentration of all GAGs

-Hyaluronidase decreases TM resistance

-may act as a gel filtration

(Knepper - binding of water may regulate flow)

5. Narrowed intertrabecular spaces
6. Collapse of Schlemm's canal

F. Steroid Sensitivity

III. Secondary Open-Angle Glaucomas

A. Exfoliation syndrome

1. Histopathology

Deposition of fibrillar material in the anterior segment

- Lens epithelium
- Lens capsule
- Pupillary margin
- Ciliary epithelium
- Iris pigment epithelium
- Iris stroma
- Blood vessels
- Subconjunctival tissue

2. Histochemistry

- Resembles oxytalan
- Microfibrillar component of elastic

3. Epidemiology

- Worldwide distribution
- 40-50% have or will develop OHT/Glaucoma

4. Mechanism of Glaucoma

- No steroid response
- Possibly related to pigment dispersion

5. Clinical deposition

16. Pupil
17. Zonules
18. Ciliary processes
19. Chamber angle
20. Endothelium
21. Anterior hyaloid (aphakia)

22. lens capsule

- a. Central zone
- b. Intermediate clear area
- c. Peripheral granular zone

6. Pre-Clinical Signs

- 23. Pupillary ruff defects
- 24. Iris sphincter transillumination
- 25. Pigment deposits on iris
- 26. Increased TM pigmentat@-cn

7. Compared to POAG, PXE:

- More often monocular (but other eye can become involved)
- IOP higher at time of Dx
- Prognosis worse
- More often develop VF loss, nerve damage
- More often need laser or filtering surgery
- Inc. incidence of ACG
- Lens removal has no effect on glaucoma

B. Pigmentary glaucoma

Pigmentary dispersion syndrome (P)S)

- Krukenberg's spindle
- Heavily pigmented TM
- mid-peripheral slit-like iris transillumination defects
- Iris - zonule rubbing (Campbell theory)
- PDS can occur with or without glaucoma
- Typical patient
 - Male
 - 20 - 50 y.o.
 - Moderate myope
 - If female, tend to be older
- May have symptoms of halo or visual blur
- Often wide IOP fluctuations
- Pigment release after exercise or dilation

C. Lens-induced glaucoma

1. Phacolytic

Denatured lens protein leakage through intact capsule

Hyperature lens

TM blocked by

- Macrophages
- High molecular wt. lens protein

- Pain, A/C reaction
- Require lens removal
- ECCE Vs ICCE

2. Lens particle glaucoma

- Retained cortex after ECCE
- Penetrating injury
- TM blocked by cortex and inflammatory cells
- Medical therapy first
 - Glaucoma meds
 - Mydriatics
 - Steroids
- If unsuccessful - surgery

3. Phacoanaphylaxis

- Rare
- After ECCE or penetrating injury
- Immunologic sensitization to lens protein
- Latent period, then granulomatous reaction (Zonal)
- Hypotony or secondary glaucoma
- Medical first
 - Steroids
 - Glaucoma meds
- If unsuccessful - surgery

D. Corneal endothelial disorders

- a) Fuch's
- b) Posterior polymorphous dystrophy
 - Normal angle
 - Iridocorneal adhesions
 - bilateral

c) Iridocorneal-endothelial (ICE) syndrome

1. Endothelialization of the A/C angle and iris surface
2. Unilateral
3. Females
4. PAS common
 - 1) Iris-Nevus (Cogan-Reese) syndrome
 - Pigmented lesions
 - Small nodular
 - Diffuse
 - 2) Chandler's syndrome

- Minimal iris changes
- Corneal edema, often with normal IOP
- 3) Essential iris atrophy
 - Marked corectopia
 - Stromal and pigment atrophy
 - Hole formation

E. Retinal Disease

- Glaucoma associated with RD
- Unilateral glaucoma look for RD
- Also, increased incidence of RD -in glaucoma (especially PG)
- Glaucoma associated with
 - RP
 - Stickler's syndrome (autosomal dominant, RD, vitreoretinal degeneration, strabismus, skeletal abnormalities)

F. Intraocular tumors

- Mechanism of glaucoma
 - Invasion of angle
 - Intraocular hemorrhage
 - Rubeosis iridis
 - Deposition in TM:
 - Inflammatory cells
 - Debris

- Tumors associated with glaucoma:

- | | |
|--------------------------|----------------------|
| - <u>Adult</u> | - <u>Children</u> |
| - Uveal melanoma | - Retinoblastoma |
| - Metastatic carcinoma | - Xanthogranuloma |
| - Leukemia | - medulloepithelioma |
| - Anterior segment cysts | |

G. Diabetes Mellitus

- Neovascularization
- Vitreous hemorrhage

H. Ocular Inflammation

- a) Acute iridocyclitis
 - Usually IOP down
 - Outflow decreased by
 - WBC's

- Particulate debris
 - Swelling of endothelial cells (TM)
 - Increased viscosity of aqueous
 - DDX from acute angle closure, in iritis:
 - Pupil miotic
 - KP's
 - Rx complicated by steroid response
 - Avoid pilo
- b) Viral inflammations
- Herpes zoster
 - Herpes simplex
 - Rubella
 - Mumps
- a. Glaucomatocyclitic crisis (Posner-Schlossman)
- Recurrent attack
 - Minimal inflammation
 - Corneal edema
 - Usually unilateral

 - Spontaneously subsides
 - Prostaglandins
 - Glaucomatous damage rare, but can occur
- d) Fuch's Heterochromic cyclitis
- "Lighter" colored eye involved
 - Unilateral (usually)
 - Low grade
 - Responds poorly to steroids
 - No PAS or PS
 - Doesn't correlate with glaucoma
 - KP's
 - Stellate and colorless
 - Fine vessels in angle
 - Small vitreous opacities
 - Cataract (good prognosis for removal)
- e) Interstitial keratitis
- Associated with both open and angle closure glaucoma
- I. Elevated episcleral venous
- Retrobulbar tumors
 - Thyroid ophthalmopathy
 - Superior vena cava syndrome

- A-V Fistulas (carotid or dural cavernous)
- Orbital varices (congenital or acquired)
- Sturge-Weber
- Familial cases
- Idiopathic
- Rx with aqueous suppressants

J. Trauma

- Hyphema
- Angle recession
 - Gonioscopically compare two angles
 - Can occur years later
 - Higher incidence of POAG in fellow eye
 - Increased IOP usually noted 6 weeks post trauma
- Siderosis, chalcosis
- Chemical injuries
 - direct damage, prostaglandin, damage to uveal circulation
 - glaucoma can occur years later
- Hemolytic
 - Red cells in AC
 - Hemoglobin-laden macrophages
 - Reddish-brown discoloration of TM
- Ghost cell glaucoma
 - Erythroclasts in A/C and TM
 - Khaki colored cells
 - Pseudohypopyon
 - Hemolytic and ghost cell self limited
 - Rx medically first
 - Washout or vitrectomy

K. Drugs

a) Steroid

- Up IOP correlates with strength and duration
- 95% of POAG are responders
- Up incidence of responders in:
 - Relatives of glaucoma patients
 - Diabetics
 - High myopia
- DDx POAG
- Usually stop steroids, IOP down's (2-3% will have persistent inc.)
- Systemic steroids less frequent but can also up IOP
- May be related to up TM GAG'S.

b) "Surgical" drugs

- 1) Alpha-chymotrypsin
 - TM blocked by zonular debris
- 2) Viscoelastic substances
 - 1 & 2 use cautiously in preexisting glaucoma
 - Beta-blockers, CAI, sometimes hyperosmotic agents needed
 - Usually resolves 1 or 2 days

L. Operative procedures (cat., filter, PK, post. capsulotomy) and lasers (trabeculoplasty, iridectomy, posterior capsulotomy)

- Possible causes
 - Pigment release
 - Inflammatory debris
 - Mechanical deformation of TM

M. Systemic Disease

a) Endocrine disease

- Pituitary tumors
- Cushing's syndrome
- May be steroid responders
- Thyroid disease

b) Renal disease

- Hemodialysis can result in wide swings in IOP (during dialysis)
 - Serum osmolality or pH?
- Renal transplantation
 - Steroid therapy?

IV. Low-Tension Glaucoma ("Normal-pressure glaucoma")

A. Diagnostic Criteria

1. Visual field and disc damage characteristic of glaucoma
2. IOP (mult. readings, diurnal curve) consistently <20

B. Mechanism

1. Poor ONH perfusion
2. Possibly related to ION

C. Diagnostic Findings

1. Non-progressive form
 - possibly related to previous episode of hypotension (GI bleed, auto accident, general anesthesia)
2. Progressive form - chronic vascular insufficiency to ONH
3. Splinter hemorrhages

D. Differential Diagnosis

1. Wide diurnal variation
2. "Burned out" POAG or secondary OAG (i.e. pigmentary glaucoma)
3. Subacute chronic angle-closure glaucoma
4. ONH disease
 - ION
 - post-papillitis
 - optic atrophy
5. Unusual causes of nerve fiber bundle VF defects:
 - ONH pits, drusen, colobomata
 - chorioretinitis, retinal detachment, retinoschisis, tumors

E. Diagnostic Evaluation

1. History: ASHD, carotid disease, D.M., hypotension
2. Physical: murmurs, bruits
3. Lab: ESR (temporal arteritis)
 - CBC (anemia)
 - FTA (syphilis)
 - ?vasculitis
4. Diagnostic Imaging: CT, MRI

F. Management

1. Decide when to treat
 - progression
 - scotoma close to fixation
 - International Low-Tension Glaucoma Protocol
2. Intervention
 - medical (beta-blockers controversial)
 - trabeculoplasty
 - surgery (FTP vs trabeculectomy)

Gonioscopy

- Necessary to view angle because internal reflection at cornea-air interface prevents view of angle.

- Direct Gonioscopy

- Koeppe lens, binocular microscope, Barkan illuminator

- Saline solution as couple

- Advantages:

- Comparison between two eyes (suspected angle recession etc.)

- Can be used in O.R. without need of microscope for EUA or Goniotomy.

- Can use -direct or indirect illumination *via Barkan illuminator. (*Scleral Scatter)

- Smooth-domed Koeppe lens allows view of fundus with direct ophthalmoscope even through miotic pupil.

- Disadvantages:

- Requires supine position

- Very inconvenient

- Indirect Gonioscopy

- uses mirror

- image is inverted (but right-left orientation and up-down orientation are not reversed)

- slightly foreshortened image (foreshortening and erect position of pt. makes angle appear shallower than Koeppe)

- depth of narrow angle often difficult to see (have pt. look toward mirror)

Goldman lens

- requires viscous coupling agent*

- must be rotated to view entire angle

- posterior pressure on lens can falsely narrow angle.

*may make subsequent fundus exam hazy and interfere with VF testing.

Zeiss

- can view entire angle

- no viscous coupling agent

- excellent to distinguish appositional closure from permanent synechiae

Angle Landmarks, (usually best seen inferiorly)

- Schwalbe's line

- at termination of cornea light "wedge"

- scleral spur

- thin pale line between pigmented zone of TM and ciliary face.

Angle Grading System

Width of angle determined by:

- 1) insertion of iris on ciliary face
- 2) convexity of iris
- 3) prominence of peripheral iris roll

Suspect narrow angle at slit lamp if 60 degrees beam just anterior to limbus suggests A/C depth is less than 1/4 of corneal thickness. (not conclusive test)

If angle between iris and TM is >20 degrees, closure unlikely.

Grade IV	45 degrees
Grade III	>20 degrees; <45 degrees
Grade II	≈ 20 degrees, Angle closure possible
Grade I	≈ 10 degrees, Angle closure probable in time
Slit	<10 degrees, Angle closure likely
0	Iris is against TM, Angle closure present

Schlemm's Canal

- usually not visible
- appears as red line in posterior TM when episcleral VP exceeds IOP
 - compression of epi veins by gonio lens (most common cause)
 - hypotony
 - Increased EVP (C-V Fistula, Sturge-Wever)

Blood Vessels

- normal vessels radial in iris, or circumferentially on ciliary face
- rubeosis or Fuch's are fine, branching irregular vessels
- vessels that cross SS to reach TM are usually abnormal

Pigmentation

- increases with age
- usually more in darkly pigmented Irises
- heaviest inferiorly

- heavy pigmentation
 - PXE inflammation
 - PDS hyphema
 - melanoma trauma
 - post surgery

Iris processes (uveal meshwork)

- open and lacv, can see normal angle in spaces between, follow normal angle curvature.
- DDX - Svnchia – more solid, obliterate angle recess.

Angle recession

- abnormally wide CB band
- increased visability of spur
- torn iris processes
- visable sclera
- variation in angle width within same eye (compare with equivalent quadrant of other eye)